

CARDIAC ANESTHESIA
Practical Aspects

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The Health Sciences Publisher

New Delhi | London | Philadelphia | Panama



Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

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New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
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83 Victoria Street, London
SW1H 0HW (UK)
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Fax: +44 (0)20 3008 6180
Email: info@jpmedpub.com

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City of Knowledge, Bld. 237, Clayton
Panama City, Panama
Phone: +1 507-301-0496
Fax: +1 507-301-0499
Email: cservice@jphmedical.com

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Bhotahity, Kathmandu, Nepal
Phone: +977-9741283608
Email: kathmandu@jaypeebrothers.com

Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

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Cardiac Anesthesia: Practical Aspects

First Edition: 2015

ISBN 978-93-5152-822-7

Printed at

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Foreword

Over the last two decades, there has been a tremendous growth in the field of anesthesia. Anesthesia is not only safer but has reached new heights by accepting the challenge for more complex surgeries. In fact, anesthesia, hitherto a general specialty, is now subspecializing into various branches. Cardiac, pediatric, hepatobiliary as well as intensive care are now established as specialties not only for service purposes but also as educational courses. In the last few years, the Medical Council of India has started DM courses in pediatric and cardiac anesthesia. Intensive care units and hepatobiliary anesthesia are now recognized as fellowship courses by National Board of Examinations and Maharashtra University of Health Sciences. This is great progress. This is not only recognition of the need for specialized care but also an effort to improve outcome in terms of morbidity and mortality.

Seth GS Medical College and KEM Hospital is a prime institute in country and has led the cardiovascular and thoracic (CVT) surgery specialty in the country for the last 60 years. It has produced gems that have been providing excellent clinical and academic services not only across the country but also all over the globe. During this successful journey of CVT department, the department of cardiac anesthesia has not only supported the growth of cardiovascular surgery but has been instrumental in developing cardiac anesthesia as a specialty in the country. It is now one of the pioneering departments in the country that conducts more than 800 complex surgeries per year. This new branch of anesthesia has proved that expertise and high volume centers can make a great difference to patient outcomes. The department has always contributed to the national academic pool in this field.

I am very happy that the department is now publishing a book on cardiac anesthesia for disseminating knowledge and sharing experience gained from years of experience in the field. The book is well planned, and covers all relevant topics that will be of great help to a young budding cardiac anesthesiologist. It is a comprehensive book that covers all topics in a clinically relevant manner. It would also provide a refreshing update for the experienced one. I wish to congratulate the department of cardiac anesthesia for this initiative and hard work that has resulted in this well-edited book. I am sure this will not only benefit cardiac anesthesiologists but also bring hope to all the cardiac patients.

Avinash Supe

MS FICS FCPS DNB PGDME DAY FMAS FIAGES FAIS MHPE
Dean

Seth GS Medical College and KEM Hospital
Mumbai, Maharashtra, India

Foreword

More than a half century ago, cardiac surgery started in India. In these years, there have been changes in the scenarios of cardiac anesthesia.

In earlier years, very few anesthetic agents, muscle relaxants and other drugs were available.

Paucity of different gadgets and monitors resulted in innovation and improvisation, e.g. use of indigenous bubble oxygenator, bubble technique, blood pressure monitoring and others.

While choosing anesthesia agents and techniques, it is imperative to know the pathophysiology of different cardiac ailments, and how this is affected by the use of anesthetic and other agents so as to minimize adverse reactions.

When one is dealing with a patient with cardiac disease who comes for noncardiac surgery, the task is more difficult as we are not improving his original cardiac status. Under these conditions, it is important to understand the pathophysiology of his original condition, what are the factors that affect the type of surgery he/she is to undergo and how to manage during and after surgery. In this book, the authors have described succinctly different types of operations; these patients undergo, and the effects of anesthesia and surgery on them particularly on their heart and peripheral circulation.

It is important to choose anesthesia agents and techniques so as to cause minimal disturbances during and after surgery. This is a herculean task.

This book is a comprehensive write up that gives the basic idea of how to deal with patients with cardiac disease undergoing noncardiac surgery.

I am sure that this will be a good reference book for the anesthetists when they need to deal with patients undergoing noncardiac surgery and will also give guidance to postgraduates.

The anesthetist has multiple roles to play during these surgeries. Whether he/she is physician, physiologist, pharmacologist, as well as the intensivist he/she is also responsible for the wellbeing of the patient during and after surgery.

Meena Bhatta

Formerly Head

Department of Anesthesiology

Seth GS Medical College and KEM Hospital

Mumbai, Maharashtra, India

Foreword

It is always a pleasure to witness growth. The rapid growth of anesthesia and its consequent specialization into various branches has been phenomenal. Our institute, Seth GS Medical College and KEM Hospital, has been at the forefront of this growth. This large department holds the distinction of providing anesthesia care to perhaps all the modern surgical specialties.

We are already affiliated to the Maharashtra University of Health Sciences for the conduct of DM course in cardiac anesthesia. It gives me great pride to present this book, which is a departmental effort. This book attempts to present a complex and extensive subject in a lucid style.

I take this opportunity to congratulate the editors, Dr Manjula Sudeep Sarkar and Dr Sunil Gvalani for this endeavor. The amalgam of knowledge and experience is evident. I am confident that this book shall be of help to the trainees and practitioners alike.

Madhulata Garasia

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Preface

Cardiac anesthesiology became a subspecialty presumably with the publication of the first textbook on cardiac anesthesia by Keown in 1956. It has covered great strides in the last 50 years. It has kept pace with the numerous innovations in cardiac surgery. It is interesting to note that no known surgical treatment for congenital or valvular heart disease was available in 1936. Open cardiac surgery was not possible till the 1950s. Even in 1967, when Dr Barnard performed the first human-to-human heart transplant, the problems of rejection and infection loomed large due to limited comprehension. This is in sharp contrast to contemporary cardiac anesthesia which involves the management of hypothermia, cardiopulmonary bypass, deep hypothermic circulatory arrest (DHCA) and off pump coronary artery bypass. Use of ultrasound and transesophageal echocardiography for advanced monitoring has become routine. Robotic coronary artery bypass grafting and transmyocardial laser revascularization are evolving. The desire to provide a concise and simplified approach to this complex subject has prompted this book.

The Department of Anesthesiology, KEM Hospital, Mumbai, Maharashtra, provides anesthesia care for approximately 900 cardiac surgeries each year. It now conducts DM course in cardiac anesthesia recognized by the Medical Council of India. We feel the book will serve the purpose of guiding examinees faced with the constraint of time.

Manjula Sudeep Sarkar
Sunil Gvalani

Acknowledgments

I take this opportunity to thank:

- ❖ Dr GV Parulekar who epitomizes scientific spirit.
- ❖ Dr Neelima Kshirsagar who combines perfection and patience effortlessly.
- ❖ Dr Pradnya Pai who always supported me.
- ❖ Dr Prasad whose encouragement has been endless.
- ❖ Dr Sanjay Oak whose dynamism has been infectious.
- ❖ Dr Avinash Supe whose achievements are inspiring.

All my senior teachers and colleagues who have moulded me with a lot of affection.

All my students whose unending curiosity has motivated me to self-improvement.

Dr N Agarwal and the Department of Cardiovascular and Thoracic Surgery, Seth GS Medical College and KEM Hospital, for invaluable support.

Last but not least, my husband Dr Sudeep Sarkar and my family, for their love that has made me confident.

Manjula Sudeep Sarkar

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Section 1

General Considerations

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- Introduction and History of Cardiac Anesthesia
- Preoperative Evaluation of Patients with Cardiovascular Disease
- Cardiovascular Monitoring
- Cardiopulmonary Bypass
- Cardiovascular Drugs

Chapter 1

Introduction and History of Cardiac Anesthesia

Manjula Sudeep Sarkar, Madhavi Buddhi

INTRODUCTION

Cardiac anesthesia is a challenging branch. As a specialty, this branch is a remarkably young branch, which has developed since 1950 onwards. The problems are quite different from other branches. Earlier, surgeons used to demand anesthesia as per their assumptions without being well versed with anesthesia. Yet, even though the surgeon's history books are filled with the technical details of the operations and who did what first, the anesthesiologists and their contributions are rarely mentioned.

From what we gather, it appears that the early history of anesthesia for operations on the heart is that of anesthesia itself starting from the late 1840s. Table 1 lists some of the notable developments and contributions in cardiac anesthesia.

Table 1: Notable developments and contributions in cardiac anesthesia

<i>Date</i>	<i>Event</i>	<i>Anesthesiologist</i>
1946	First paper on cardiac anesthesia	MH Harmel, A Lamont
1950	Plastic intravenous cannula	D Massa, JS Lundy, et al.
1951	Anesthesia for mitral commissurotomy	KK Keown
1952	Cardiotachoscope	A Himmelstein
1954	Esophageal stethoscope	C Smith
1956	First textbook on cardiac anesthesia	KK Keown
1957	Anesthesia for cardiopulmonary bypass	EA Gain D Mendelsohn et al. RT Patrick et al.
1955	Electroencephalographic monitoring during cardiopulmonary bypass	RA Theye
1963	Monitoring of left atrial pressure	RA Theye, JW Kirklin
1967	First paper on anesthesia for coronary artery surgery (Vineberg procedure)	JE Wynands et al.
1969	Narcotic (morphine) anesthesia	E Lowenstein et al.

Contd...

Contd...

<i>Date</i>	<i>Event</i>	<i>Anesthesiologist</i>
1972	Pulmonary artery catheter monitoring during cardiac surgery and cannulation of right internal jugular vein	JM Civetta, JC Gabel
1976	Monitoring V5 lead for detecting myocardial ischemia	JA Kaplan
1976	Nitroglycerin infusions to treat myocardial ischemia	JA Kaplan
1978	Founding, Society of Cardiovascular Anesthesiologists	R Marino, first President
1978	High-dose fentanyl anesthesia	TH Stanley et al.
1979	First modern American textbook on cardiac anesthesia	J Kaplan (Editor)
1980	M-mode transesophageal echocardiography monitoring	M Matsumoto, Y Oka
1981	Pulmonary artery catheter to detect myocardial ischemia	JA Kaplan
1982	Alpha-stat blood gas management	AK Ream et al.
1982	Two-dimensional transesophageal echocardiography monitoring	MK Cahalan, P Kremer, M Roizen
1983	Isoflurane steal described	S Reiz
1983	Direct measurement of cerebral blood flow during cardiopulmonary bypass	JG Reves et al. LH Henricksen et al.
1985	Impact of myocardial ischemia on outcome	S Slogoff, A Keats
1986	Cerebral protection with barbituates	NA Nussemeier, S Slogoff
1987	First issue of the Journal of Cardiothoracic Anesthesia	JA Kaplan (Editor)
1987	Color flow transesophageal echocardiography	NP deBruijn et al.
1989	Anesthetic agent does not affect outcome	K Tuman et al. S Slogoff, AS Keats

EARLY DEVELOPMENTS—ANESTHESIA FOR THORACIC SURGERY

The first major challenge to thoracic surgery was how to ventilate the patient when the chest was opened. This was at a time when endotracheal intubation was not yet discovered, monitoring was limited to a blood pressure cuff, stethoscope and a “finger on the pulse”.

Some ingenious approaches were explored, such as placing the patient’s head in a positive pressure box [Brauer (1904)] and Murphy (1905)] or operating within a negative pressure chamber with only the patient’s head outside [Sauerbruch (1904)]; for a while, even into the 1940s, positive pressure ventilation via a tight-fitting mask was used.

It was during this era, when even antibiotics were not available to combat infection that the early cardiothoracic surgeries was performed in 1888, the American surgeon designed metal endotracheal tubes with a conical tip to occlude the larynx and added a bellows device to provide positive ventilation. This was first used and advocated as the best means of preventing pulmonary collapse during thoracic surgery by the surgeon Rudolph Matas in about 1899.

The anesthetic agents in use were mostly ether, chloroform and nitrous oxide when cyclopropane was introduced as the new agent to clinical anesthesia by Ralph Waters in 1933 and was adopted by many cardiac anesthesiologists as an induction if not a maintenance agent. Its major drawback was its inflammability, which led to its ready replacement when halothane became available in the late 1950s. In 1952, Lucien Morris invented the copper kettle, which permitted more precise quantification of anesthetic dosing. Later, in this decade the temperature-compensated agent specific vaporizers (TEC) were introduced by Cyprane Limited of England.

In 1934, John Lundy of Mayo Clinic introduced thiopental into the practice of anesthesia and for many years it was used for both maintenance and induction. Muscle relaxants in the form of curare were first used in 1942 by Griffith and Johnson of Montreal and later in 1947 by Stephens to facilitate thoracic surgery.

Although mechanical ventilators were developed as early as 1907 (the Draeger Pulmotor), and Crafoord introduced his commercially available Spiropulsator in 1940 and Blease the "pulmoflator" after world war II, the use of ventilators in the operating room was not readily accepted until the late 1950s. "Modern" ICU-type ventilators (e.g. Bennett, Bird) did not appear until the 1960s.

Postoperative recovery rooms were established around world war II and although an American Study Commission report published in 1947 suggested that they could improve outcome, they did not become common until the mid-1950s. Open heart surgeries gave way to the development of surgical intensive care units in the early 1960s.

EARLY CARDIAC ANESTHESIA

Documentation of the anesthesia for the earlier cardiac surgical operations was not done, hence will never be known. In 1975, Ellis reviewed the anesthetic management of a 1925 case of mitral commissurotomy operated by Dr Souttar. He states that anesthesia was administered by a surgeon, Eric Lindsay (although in a letter written to Brian Blades 30 years after the event, Dr Souttar gives credit to his anesthetist, Dr John Challis). The patient was premedicated with morphine and atropine; induced with a combination of absolute alcohol, chloroform and ether (ACE); and maintained with ether administered via an endotracheal catheter (? endotracheal tube), and apparently breathed spontaneously throughout the procedure. Blood pressure was monitored about every 5 minutes (presumably noninvasively).

In, 1994, Dobel outlined the anesthetic management of the patient in whom Dr Gross first ligated a PDA in 1938. Anesthesia was provided by a nurse anesthetist (Betty Lank), who used a tight-fitting adult mask that was shrunk (with alcohol) and strapped to the child's face. Monitoring included a finger on the superficial temporal pulse. The anesthetic agent used was cyclopropane. Blood was available but was not needed for Crafoord's first coarctation repairs, accomplished in 1944. Anesthesia consisted of a mixture of cyclopropane and

nitrous oxide administered via an airtight endotracheal tube using controlled ventilation provided by a spiropulsator.

Between 1946 and 1950, the first papers on cardiac anesthesia appeared. These put forth the challenges of providing anesthesia for severely cyanotic and debilitated children undergoing systemic-to-pulmonary artery shunt operations.

The common method of anesthetic management included heavy premedication with morphine and scopolamine, induction with cyclopropane (with or without ether), endotracheal intubation although some tried mask—only ventilation. Most used controlled ventilation (with or without use of curare). The Waters to-and-fro carbon dioxide absorbing canister closed system was usually used. The monitoring methods and fluid management were not discussed in detail except a warning against fluid overload. Usually a cannula was placed in the saphenous vein of an ankle, and plasma was administered.

Between 1952 and 1955, the first papers on providing anesthesia for mitral commissurotomies were published. Moderate premedication including phenobarbital and morphine or meperidine was usually recommended. All groups emphasized the importance of using the lightest possible anesthesia but employed different agents to accomplish this goal. Continuous ECG monitoring was employed by all, some using an oscilloscope. All started large-bore intravenous access and had blood available, including blood pumps and means of giving intra-arterial transfusions, but all emphasized the need to minimize fluid and blood administration to avoid fluid overload. All patients were extubated at the end of the case but given supplemental oxygen postoperatively. Several groups employed paravertebral or intercostal blocks for postoperative analgesia. All authors emphasized the need to move or change the patient's position slowly and gently while the patient was still under anesthesia or emerging, to avoid hemodynamic instability.

Even more challenging was the provision of anesthesia for patients of severe aortic regurgitation for insertion of ball valve prosthesis in the descending aorta. This were first performed by Dr Charles Huffnagel of Georgetown University (without the benefit of left heart or cardiopulmonary bypass) in September 1952. The anesthetic management of these patients was described by John A, O'Donnell and Thomas F McDermott in 1955. All 42 patients were in medically irreversible congestive cardiac failure and 40% had intractable angina pectoris. From their earlier experience, it was realized that these patients do not tolerate a depth of anaesthesia to permit endotracheal intubation. They therefore intubated these patients with topical anesthesia, induced very light general anesthesia with a small dose of pentothal and maintained them on nitrous oxide and oxygen supplemented with intravenous morphine or small amounts of ether. Phenylephrine was the agent used to keep the systolic arterial pressure above 100 mm Hg. Two 15-gauge intravenous needles were placed. The radial artery was surgically exposed, not for monitoring but to use if needed to withdraw blood to treat extreme hypertension during aortic crossclamping or to give intra-arterial transfusion in the case of catastrophic

hemorrhage. Blood transfusion replaced blood loss and an electric defibrillator was always available.

In 1954, SJ Evelyn and I Mackay reviewed their anesthetic techniques and results with cardiac anesthesia in 820 cases operated at the Toronto General Hospital. Their hospital mortality rates were remarkable: tetralogy of Fallot (shunts; 342 patients), 6%; PDA (133 patients), 0%; coarctation (29 patients), 0%; mitral valve surgery (260 patients), 6%.

Not much is known about the anesthetic techniques used for open heart direct-vision surgeries with moderate surface-induced hypothermia. Lewis indicated that anesthesia was induced with pentothal and curare, patients were intubated, and ECG and rectal temperature were monitored. Swan indicated that anesthesia was also induced with pentothal and maintained with cyclopropane and ether, or with ether alone.

Robert W Virtue with Henry Swan in Denver elaborated on the anesthetic management in their 1955 report of their first 100 cases done under hypothermia with brief periods of circulatory arrest. Monitoring included ECG, EEG blood pressure by auscultation. Patients were cooled to less than 35°C sometimes 33°C to 31°C and deliberately hyperventilated to achieve respiratory alkalosis, which they believed reduced the incidence of ventricular fibrillation. They measured right atrial pH (at the patient's temperature) just before arrest and aimed it to be greater than 7.5 (alpha-stat management). After arrest, 100% oxygen was administered, with nitrous oxide (50%) added if required. Patients were extubated when they were breathing adequately. Common complications were ventricular fibrillation and postoperative hemorrhagic shock and mortality rate was high.

ANESTHESIA FOR EARLY CARDIAC SURGERY USING CARDIOPULMONARY BYPASS

In 1953 Dr John Gibbon used the heart-lung machine developed by him to successfully close an ASD in an 18 year old female patient. In his dictated operative notes Dr Gibbon only mentioned that the patient was anesthetized with intravenous pentothal with an endotracheal tube and manual assistance to ventilation. Direct blood pressure readings were made via a needle inserted in the right brachial artery that was connected to a mercury manometer. The veins in both ankles were cannulated for administration of intravenous fluids. The patient was quite light at the end of the operation and was awake and talking one hour after the operation. Many other patients did not survive and according to Anthony Dobell who was a surgery resident under Dr Gross, "A major factor in the deaths of the children was poor ventilation prior to going on cardiopulmonary bypass". There were no physician anesthesiologists at Jefferson at that time and of course mechanical ventilators had not been developed. Some of the children were already moribund by the time they were connected to the heart-lung machine. Thus at Jefferson, lack of expertise in anesthesia, pediatric cardiology and cardiac pathology all played a part in the initial failures.

In 1954, Walton Lillehei and his colleagues at the University of Minnesota initiated a series of direct-vision intracardiac surgery with total CPB using the patient's parent as the "heart-lung machine" (controlled cross-circulation). Anesthesia in these patients was induced with nitrous oxide/cyclopropane and then maintained with pentothal and curare; anesthesia in donors was induced and maintained with pentothal and curare. During CPB, the donor was hyperventilated (two to three times normal values) to induce a respiratory alkalosis and counter balance the metabolic acidosis that regularly occurred during the low-flow (34 to 40 mL/kg/minute) perfusion of the patient. No mention of monitoring, invasive lines, nor fluid management other than blood administration was made. The duration of CPB ranged from 9 to 28 minutes. Of the first eight patients (age 5 months to 5 years) who underwent VSD closure, six survived. According to Arens, Fred van Bergen and Joe Buckley administered anesthesia to the patient while Jim Matthews and Earl Schultz cared for the father donor for the first successful case.

In 1956, the first relevant textbook, '*Anesthesia for Surgery of the Heart*' was published by Kenneth K Keown of Hahnemann Medical College. The book detailed the cardiac anesthesia possible during that decade. It was the time when cardiac surgeons and cardiac anesthesiologists were teaming up, signifying the importance of teamwork and he lists the important teams practising at that time (Table 2).

He also summarized the expected mortality rates and emphasized the role of the anesthesiologist in ensuring that the patient was fit for surgery, the

Table 2: Early cardiac surgery and anesthesia teams

<i>Surgeon</i>	<i>Anesthesiologists</i>	<i>Location</i>
CP Bailey	KK Keown	Philadelphia
A Blalock	MH Harmel, A Lamont	Johns Hopkins Hospital
RC Brock	EH Rink, Lucas	London
DB Effler	DE Hale, JF Vijon	Cleveland Clinic
DE Harken	LD Vandam	Boston
CA Hufnagel	TF McDermott	Georgetown University
EB Kay	D Mendelsohn	Cleveland
JW Kirklin	RT Patrick, RA Theye	Mayo Clinic
CW Lillehei	FH Van Bergen	University of Minnesota
DWG Murray	SJ Evelyn	Toronto
WJ Potts	WO McQuiston	Chicago Children's Hospital
TH Sellors	BA Sellick, Brown	London
H Swan	RW Virtue	Denver
R Varco	JJ Buckley	University of Minnesota

Source: Keown KK, Thomas CC. Anesthesia for surgery of the heart. Springfield, IL: 1956. p. 8.

use of checklists for equipment and supplies and the avoidance of short cuts. Finally, he listed what he hoped would be coming in the near future: a more ideal inhalation anaesthetic (potent, rapid-acting, nonirritating, nonexplosive, mechanical ventilators, arterial lines with partial oxygen pressure (PO_2) and pH measurement, and a practical mechanical heart-lung machine.

Over the next four years, several groups practised varied techniques and reviewed their experiences in delivering anesthesia care for patients undergoing open heart surgery with the use of mechanical heart-lung machines.

These reports reflected major differences of opinion regarding the optimal oxygenator, flow rates, lung management, control of arterial pressure, need to add carbon dioxide, or anesthetic agents to the extracorporeal circuit during surgery and type of monitoring. The range of management varied from a detailed protocol like at the Mayo Clinic to a minimalistic approach like at Baylor/Texas Children's Hospital in Houston.

At Mayo Clinic flows were kept at 2.4 L/minute/ m^2 (60 to 200 mL/kg/minute) and ether was administered via a vaporizer on the oxygenator. Intra-arterial pressure, venous pressure, rectal temperature, EEG, ECG, in-line venous saturation, and arterial blood gases were monitored. Fluid and blood balance was meticulously monitored and written, detailed protocol for step-by-step management was published. Vasopressor drugs and resuscitation drugs were prepared and made ready.

At the other extreme was the practice at Houston (Cooley and Keats). They monitored only one lead of the ECG (and not invasive arterial pressure, venous pressure, EEG, or temperature), ran flows of 35 to 50 mL/kg/minute (adults and infants, respectively), and administered only d-tubocurarine during CPB.

Halothane, though introduced into anesthesia in 1957 was hesitantly used for cardiac surgery because of concern about its myocardial and respiratory effects. However, the desire for a potent, nonexplosive agent led the groups at Mayo Clinic and Indiana University to explore the use of low concentrations (less than 0.8% to 1.0%) in 50% nitrous oxide for adults and children open and closed cardiac surgery. From then halothane remained the favored agent until the landmark article by Edward Lowenstein et al. at the Massachusetts General Hospital in 1969.

High dose morphine (0.5 to 3 mg/kg) was used by Lowenstein and his team as the main anesthetic in 1100 patients because it was devoid of myocardial depression and was considered as the agent of choice in patients with minimal cardiac reserve (e.g. end-stage valvular and coronary artery disease). This approach rapidly became popular and the combination of high-dose narcotics with oxygen and muscle relaxants came to be known as the "cardiac anesthetic". Thereafter for the next 10 years, morphine was the main narcotic until it was replaced by the synthetic narcotics, fentanyl initially in doses of 50 to 100 μ g/kg as popularized in the United States by Theodore H Stanley of the University of Utah. The side effects of morphine like vasodilation and the need for volume expansion were overcome with the use of fentanyl.

ANESTHESIA FOR CORONARY ARTERY SURGERY

The first article on anesthetic management of patients with coronary artery disease was published by Earl Wynands of Montreal in 1967. This was followed by an article by John F Viljoen of the Cleveland Clinic, based on his group's experience with 1500 IMA implant procedures.

Both authors emphasized the desirability of generous premedication, adequate depth of anesthesia, invasive monitoring of arterial and central venous pressure, frequent measurements of arterial blood gases and serum potassium, postoperative endotracheal ventilation with adequate analgesia and sedation for 4 to 24 hours and close cardiovascular surveillance in the intensive care unit. Wynands emphasized the importance of maintaining arterial pressures close to baseline and keeping the hematocrit at about 40 percent. Viljoen advocated intermittent administration of nitroglycerine intramuscularly, and in 1976 Joel Kaplan, then at Emory University, introduced the use of nitroglycerine infusions during coronary artery disease.

Recognizing the importance of integrating cardiology into the practice of anesthesiology, a symposium on this topic, edited by Arthur Keats, was published in the August 1970 issue of *Anesthesiology*.

Allen Ream et al. from Stanford published an article in 1982, which led to a rethinking of the proper management of pCO₂ and pH during hypothermic CPB and a near-complete shift from pH-stat to alpha-stat management during that decade.

A year later, the paper by Sebastian Reiz of Umea, Sweden, raised concern about the risk of coronary steal with isoflurane and led to a flurry of experimental and clinical studies on this topic.

Two years later another influential study by Stephen Slogoff and Arthur Keats of the Texas Heart Institute in Houston documented the frequency of intraoperative myocardial ischemia during coronary artery surgery and showed that it was associated with postoperative myocardial infarction. They also showed that an anesthesiologist can have an impact on the incidence of this problem. This paper helped initiate the intense study for the prevention of perioperative ischemia, that continues to the present day.

In 1986, another study by Nancy Nussmeier and Slogoff demonstrated that use of high-dose (40 mg/kg) thiopental (to produce isoelectric EEG) reduced the rate of postoperative neuropsychiatric dysfunction after open ventricle operations. The general application of this was challenged and generated much interest in the incidence, cause, and prevention of perioperative neurological dysfunction.

In 1989, two similar papers were published. One from Slogoff and Keats at the Texas Heart Institute and the other from Kenneth Tuman and colleagues at Rush-Prebyterian-St Luke's Medical Center in Chicago, documenting in large prospective studies (more than 1,000 patients each) what cardiac anesthesiologists had been saying since 1946: that the choice of anesthetic agents does not significantly affect outcome.

Monitoring

Until the mid-1950s, monitoring was rarely mentioned. The anesthesiologists heavily depended on their five senses, awareness, and clinical acumen.

The first “new” monitor to be introduced was the ECG. Paper recorders were used in adult and paediatric surgery but ECG monitoring was not commonly practiced until the advent of mitral commissurotomy, during which arrhythmias were a major problem and all early reports mentioned use of ECG monitoring.

Soon paper recorders were replaced by oscilloscopic monitors which facilitated continuous ECG monitoring. A major advance in ECG monitoring occurred in 1976, when Joel Kaplan advocated the use of the V5 lead to detect myocardial ischemia in patients with coronary artery disease.

In 1954, Dr Code Smith at Toronto Hospital for Sick Children introduced the simple but effective esophageal stethoscope, which was particularly useful in thoracic surgery.

Sphygmomanometer could no longer provide reliable arterial pressure monitoring when CPB began to be used in cardiac surgery in the mid-1950s. Invasive arterial lines were introduced not only to monitor pressure but also to measure arterial blood gases. Initially these were often placed in the radial artery by cutdown by the surgeons, and it was not until the late 1960s that anesthesiologists began to place these lines percutaneously.

Blood gas analysis was a difficult and time consuming process and was infrequently performed until the late 1950s, when Clark, SEveringhaus, and Astrup introduced their pO_2 , PCO_2 , and pH electrodes. Even then, it took a few years until blood gas analysis was common place.

A common concern after bypass was the estimation of blood volume status and evaluation of ventricular function. Venous pressure monitoring was introduced, initially from catheters placed in arm veins and external jugular veins and then from central lines placed via the groin or arm veins or placed directly into the right atrium at the time of surgery.

However, as early as 1963, the Mayo Clinic group called attention to the frequent discrepancies between central venous (right atrial) and left atrial pressure and described placing catheters directly into the left atrium to monitor function and filling of the left side of the heart. Subsequently, many authors advocated monitoring of left atrial pressure in this manner.

There was a revolutionary change in monitoring in 1970 when the balloon-tipped PAC was introduced by Swan, Ganz, and colleagues. The cardiac surgery/anesthesia team at Massachusetts General Hospital was among the first to advocate use of the PAC in cardiac surgery. Soon, percutaneous introduction of PAC was made available and rapidly became the approach of choice for most anesthesiologists.

Joel Kaplan helped to popularize the use of the PAC during cardiac surgery and emphasized its role in the early diagnosis of myocardial ischemia (although the latter is now largely discounted).

Not only did the PAC allow estimation of the left atrial pressure, but it also provided access to mixed venous blood for oxygen analysis. In 1972, the PAC was modified to permit the easy monitoring of cardiac output by thermodilution.

Although cardiac anesthesiologists were among the first and strongest proponents of the use of the PAC, they were also among the first to point out its limitations and to indicate that not all cardiac surgical patients require a PAC.

From the very beginning in 1955, the Mayo Clinic group used EEG monitoring during CPB and advocated its value as a monitor of anesthetic depth as well as integrity of cerebral perfusion.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

In the past decade, transesophageal echocardiography (TEE) profoundly changed the practice of cardiac anesthesiology. Paul Barash of Yale was perhaps the first to apply echocardiography in anesthesiology, when he evaluated the effects of halothane on ventricular function in children using transthoracic M-mode echocardiography.

Transesophageal Doppler was first described in 1971 and transesophageal M-mode in 1976. In 1980, Masayuki Matsumoto, Yasu Oka, and others at Albert Einstein College of Medicine in the Bronx perceived the future of TEE in cardiac anesthesia when they described the use of M-mode TEE for continuous monitoring of left ventricular function in 21 patients during cardiac surgery. However, M-mode TEE was difficult to apply except by the extremely sophisticated clinician.

Between 1977 and 1980, primitive two-dimensional TEE scanners were described but it was not until Souquet, Schluter, and Hanrath introduced their phased array transducer system mounted on the end of a gastroscope that TEE became a practical reality.

Prototypes were used jointly by cardiology fellow P Kremer and anesthesiologists Micheal Cahalan and Micheal Roizen at University of California in San Francisco (UCSF). In the 1982 meeting of the American Society of Anesthesiology, they created the TEE “revolution” when they presented their results in monitoring cardiac and vascular surgery patients with this new TEE probe. They described its usefulness in assessing filling and function of the left ventricle, myocardial ischemia, and intracardiac air.

RF Cucchiara and colleagues at the Mayo Clinic described its usefulness in detecting air embolism during neurosurgery while the UCSF group described its superiority over ECG in detecting myocardial ischemia.

In 1987 Cahalan et al. and Clement and deBruijn at Duke University wrote review articles on the use of TEE in anesthesiology.

In 1986, Hewlett-Packard introduced color flow Doppler with TEE, and Norbert deBruijn and colleagues at Duke University reviewed their early experience with this new technology. In the same year pulsed wave Doppler was added to TEE, and in 1989, biplane probes first became available.

The Society of Cardiovascular Anesthesiologists in conjunction with the American Society of Anesthesiologists have developed the practice guidelines

for perioperative TEE in 1996. In collaboration with the American Society of Echocardiography they helped initiate the first examination in perioperative TEE, which was administered by the National Board of Echocardiography in 1998. Today TEE is one of the defining characteristics of a cardiac anesthesiologist.

CARDIAC ANESTHESIA AS A SUBSPECIALTY

It's subspecialty status is now accepted by many because of the special knowledge, skill, and activities required of anesthesiologists who attend these patients.

In the beginning, these aspects consisted of intimate knowledge of the pathophysiology of congenital heart disease and how various anesthetic techniques interact with this pathophysiology. Later was added the pathophysiology of valvular heart disease, and finally that of coronary artery disease.

Next was required knowledge of the entirely new techniques that surgeons used to manage these patients: initially hypothermia, then CPB and all of its nuances including deep hypothermic circulatory arrest, coronary perfusion, cardioplegia, and retrograde cerebral perfusion.

In recent years, this has included minimally invasive techniques such as port access system. Advanced monitoring techniques, not usually used in noncardiac anesthesia at the time they were introduced were on the list of new skills required of the cardiac anesthesiologist. These included ECG, EEG, arterial lines, and central venous and left atrial pressure monitoring; next the PAC; and most recently, TEE. Intimate knowledge of blood coagulation and anticoagulation and the administration of blood products (often in large quantities) and of various drugs to modify coagulation was required.

Finally, a thorough understanding of the pathophysiology of abnormal hemodynamic states and manipulation with potent and often novel hemodynamic active agents and mechanical assist devices was required.

Cardiac anesthesiologists have been responsive by developing techniques that facilitate new procedures in cardiac surgery, such as minimal access surgery, transmyocardial laser, the Batista operation, valve repair and use of stentless valves, more aggressive neonatal surgery, and fast-tracking.

ACHIEVEMENTS OF KEM HOSPITAL, MUMBAI

- ❑ The first department of cardiovascular and thoracic anesthesia in India
- ❑ First intensive cardiac care unit in India
- ❑ The first Indian hospital to acquire an ECG machine
- ❑ The first mitral commisurotomy in India (1952)
- ❑ First balloon dilatation of cor-triatriatum in the world
- ❑ First fetal echocardiography-guided interventional therapy in the country
- ❑ First transcatheter closure of ASD in Western India
- ❑ Highest annual collection of blood unit in India (about 30,000/year in 1998, 1999)
- ❑ First intravenous anesthesia with Thiopentone sodium (1940s)

- ❑ First hypothermia technique for ASD (1953)
- ❑ First cardiac catheterization in India 1959–1960
- ❑ Pioneering work on recreation of reptilian heart vascular pattern in mammalian heart (1965)
- ❑ Pioneering work on the association of tuberculosis with nonspecific aortoarteritis (1963)
- ❑ First interventional radiological procedures in India 1975
- ❑ First department of interventional electrophysiology in western India.

SUGGESTED READING

1. Eugene A, Hessel II. Foundation Of Cardiac Anesthesia: History of cardiac surgery and anesthesia; Cardiac Anesthesia: Principles and Practice. In: Estafanous FG, Barash PG, Reves JG (Eds), 2nd edition.
2. Lake CL. History of pediatric cardiac anesthesia, Pediatric cardiac anesthesia. In: Lake CL, Brooker PD (Eds), 4th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 1-6.

Chapter 2

Preoperative Evaluation of Patients with Cardiovascular Disease

Manali Nadkarni

INTRODUCTION

A detailed and thorough preoperative evaluation is necessary for patients with cardiovascular disease posted for surgery as it helps in identification, stratification and if required modification of risk factors. It also helps in optimizing the patient's general condition prior to surgery. It guides in deciding the best suitable anesthesia management for that patient. This in turn decreases the perioperative morbidity and mortality.

The aims of preoperative evaluation are:

- ❑ Identify the patients at risk.
- ❑ Evaluate the severity of underlying cardiovascular disease.
- ❑ Determine the extent of risk and the need for preoperative interventions to decrease the risk of perioperative complications.

The preoperative evaluation includes:

- ❑ History—onset, duration, progress of disease, treatment and complications developed if any. It should include details of medications.
- ❑ Physical examination of the patient.
- ❑ Investigations.
- ❑ Diagnostic tests or procedures.

HISTORY

Depends on the initial diagnosis made:

- ❑ For coronary artery disease
 - Risk factors such as hypertension, diabetes mellitus, smoking
 - Angina patterns—stable, unstable, variant
 - Previous myocardial infarction
 - Dysrhythmias
 - Associated cardiovascular diseases—cerebral, carotid, aortic, peripheral vascular disease if present.

- ❑ For valvular heart disease
 - Dyspnea, orthopnea, paroxysmal nocturnal dyspnea
 - Hemoptysis
 - Embolic events
 - Heart failure
 - Arrhythmias
 - History of valvular surgery, presence of a prosthetic valve
 - Details of anticoagulant therapy
- ❑ For congenital heart disease
 - Cyanotic spells if any
 - History of prematurity, failure to thrive
 - Associated other congenital anomalies
- ❑ Cardiomyopathy
 - Hypertrophic cardiomyopathy—angina pectoris, fatigue or syncope, tachydysrhythmias, heart failure.
 - Dilated cardiomyopathy—chest pain on exertion, dysrhythmias, systemic embolization
- ❑ Patient with pacemaker/implantable cardioverter defibrillator (cardiovascular implantable electronic devices): Cardiovascular implantable electronic devices (CIEDs) are used in patients of arrhythmias (sinus node dysfunction, atrioventricular block, atrial flutter, ventricular tachycardia), structural heart disease (ischemic or nonischemic cardiomyopathy), chronic heart failure or inherited arrhythmia syndromes. The anesthesiologist should be aware not only of patient's specific CIED hardware and programming but also of the underlying cardiac condition for which the device was implanted. In particular, cardiac rhythm and history of ventricular arrhythmia should be reviewed in patients with CIED.
- ❑ Heart failure:
 - Dyspnea, orthopnea, paroxysmal nocturnal dyspnea
 - Fatigue, weakness at rest or with minimal exertion
 - Anorexia, nausea, confusion, anxiety

PHYSICAL EXAMINATION

In addition to general examination, systemic examination, from cardiac point of view will include:

- ❑ Pulse—regularity, volume, radial, carotid, femoral, radiofemoral delay, absent peripheral pulse
- ❑ Blood pressure—upper limbs, lower limbs
- ❑ Pulse pressure
- ❑ Respiration
- ❑ Jugular venous pressure
- ❑ Cyanosis
- ❑ Clubbing
- ❑ Peripheral edema
- ❑ Displaced apical impulse

- ❑ S3 gallop
- ❑ Pulmonary edema
- ❑ Murmurs

INVESTIGATIONS

A. *Blood investigations*

- Hemoglobin, total leukocyte count, differential leukocyte count, platelet count, erythrocyte sedimentation rate
- Renal function tests
- Liver function tests
- Lipid profile
- Coagulation tests—prothrombin time, activated partial thromboplastin time, international normalized ratio (INR)
- Blood sugar estimation
- Arterial blood gas sampling.

B. *Chest radiograph*

- Cardiomegaly
- Signs of ventricular dysfunction
 - Increased pulmonary vascular markings
 - Pulmonary edema
 - Effusions

C. *Electrocardiograph*

- Myocardial ischemia or infarction
 - ST segment changes
 - T wave changes
 - Q wave (significance, location)
- Chamber enlargement
 - Left ventricular hypertrophy
 - Right ventricular hypertrophy
- Arrhythmias

DIAGNOSTIC TESTS OR PROCEDURES

A. *Measurement of biomarkers*

- Serum Troponin T, Troponin I levels
- Serum creatine-kinase levels especially isoenzyme CK MB
- Lactate dehydrogenase
- The above-mentioned biomarkers are useful in detection of acute myocardial infarction.
- B-type natriuretic peptides: Measurement of natriuretic peptides is helpful in assessing patients with heart failure and to diagnose heart failure as a postoperative complication in patients at high risk.
- C-reactive protein levels in patients with history of rheumatic fever to rule out active endocarditis.

- B. *2 D echocardiography*: It is a noninvasive study of heart which gives idea about ejection fraction, chamber dysfunction, regional wall motion abnormalities, chamber sizes and pressures, valve assessment, and pericardial effusion.
- C. *Stress testing*
- Exercise stress testing (treadmill): It is a noninvasive test useful in ambulatory patients with chest pain of unknown cause. It is also done preoperatively in patients with known coronary artery disease for assessing the severity of the disease.
 - Pharmacological stress testing: It is done in patients with coronary artery disease who are not ambulatory or who cannot exercise.
 - Radionuclide myocardial perfusion imaging: Coronary artery vasodilatation is done using vasodilators such as dipyridamole, adenosine or regadenoson. This is followed by thallium imaging to detect myocardial ischemia. Technetium-99m or rubidium-82 can also be used for imaging. Moderate to large perfusion defects which reflect myocardial ischemia carry the greatest risk of perioperative cardiac death or myocardial infarction. An abnormal myocardial perfusion imaging test is associated with very high sensitivity for detecting patients at high risk for perioperative cardiac events. A reversible myocardial perfusion defect predicts perioperative events whereas a fixed perfusion defect predicts long-term cardiac events.
 - Dobutamine stress echocardiography: The coronary flow reserve is tested by using dobutamine infusion, which increases heart rate and contractility. Echocardiography is performed at discrete points to detect new or progressive regional wall motion abnormality.
- D. *Ambulatory ECG monitoring*: Preoperative 24 hours to 48 hours of ambulatory continuous electrocardiographic monitoring is done to detect silent myocardial ischemia or arrhythmias.
- E. *Cardiac catheterization*:
- Coronary angiography provides information about the coronary arteries and its branches in patients with coronary artery disease. Cardiac catheterization provides detailed anatomical information in complex congenital heart disease.
 - For patients undergoing non-cardiac surgery, cardiac catheterization has a limited role (done only in indicated cases) as alternative less invasive techniques are available to assess cardiac function.

ASSESSMENT OF CARDIAC RISK

Various clinical risk factors, surgery specific risk factors and cardiac risk indices are used to assess and stratify the risk involved when a patient with cardiac disease is posted for surgery. They are:

Clinical Risk Factors

Derived from history, physical examination and review of electrocardiograph, they can be grouped into three categories of risk predictors:

1. Major risk clinical predictors—severe valvular disease, decompensated heart failure, significant dysrhythmias, unstable coronary syndrome.
2. Intermediate risk clinical predictors—mild angina pectoris, previous myocardial infarction, compensated or prior heart failure
3. Minor risk clinical predictors—hypertension, left bundle branch block, nonspecific ST-T changes, history of stroke.

Exercise Capacity and Functional Capacity

Functional status is a reliable clinical predictor of perioperative and long-term cardiac events. Patients with decreased functional status preoperatively are at increased risk of perioperative complications. Conversely those with good functional status preoperatively are at lower risk.

Functional status can be estimated from activities of daily living. Functional capacity is expressed in terms of metabolic equivalents (METs) where 1 MET is the basal oxygen consumption of a resting normal adult (3.5 mL/kg/min).

Functional capacity is classified as:

- Excellent—> 10 METs
- Good—7–10 METs
- Moderate—4–6 METs
- Poor—<4 METs

Perioperative cardiac and long-term risk is higher in patients unable to perform 4 METs of work during daily activities.

Functional status can be assessed more formally by activity scales, such as DASI (duke activity status index) or specific activity scale.

Surgery-specific Risk Factors

The cardiovascular risk during surgery depends on the type and site of surgery and the duration and intensity of the perioperative physiological stress associated with the surgery. The surgeries can be grouped as:

- High cardiac risk surgeries—emergency major operations, aortic and other major vascular surgery, prolonged surgical procedures associated with large fluid shifts or anticipated blood loss.
- Intermediate cardiac risk surgeries—head and neck surgery, intraperitoneal and intrathoracic surgery, carotid endarterectomy, orthopedic surgery.
- Low cardiac risk surgeries—endoscopic procedures, superficial surgery, cataract surgery, breast surgery.

Risk Indices

Goldman Cardiac Risk Index

Multifactorial cardiac risk index devised by Goldman et al.

<i>Risk Factors</i>	<i>Points</i>
➤ Age >70 years	5
➤ Myocardial infarction in previous six months	10
➤ S3 gallop or raised jugular venous pressure	11
➤ Important aortic stenosis	3
➤ Rhythm other than sinus or premature atrial contractions on last preoperative ECG	7
➤ >5 VPCs/min any time before surgery	7
➤ PaO ₂ <60 or PaCO ₂ >50 mm Hg; K <3 or HCO ₃ <20 mEq/L3	
➤ BUN >50 or Creatinine >3 mg/dL; abnormal SGOT, signs of chronic liver disease or bed ridden from noncardiac causes	
➤ Intraperitoneal, intrathoracic or aortic operation	3
➤ Emergency surgery	4
Interpretation	
Class I—0–5 points	Low risk
Class II—6–12 points	Intermediate risk
Class III—13–25 points	High risk
Class IV—>26 points	Very high risk

Detsky Modified Multifactorial Index

<i>Risk Factors</i>	<i>Points</i>
➤ Myocardial infarction	
- No history	0
- Within 6 months	10
- Beyond 6 months	5
➤ Canadian Cardiovascular Society Angina	
- Class I - II	0
- Class III	10
- Class IV	20
- Unstable angina within 3 months	10
➤ Pulmonary edema	
- Never	0
- Within 1 week	10
- Ever	5
➤ Valvular disease	
- Possible aortic stenosis of a critical nature	20

- Arrhythmias
 - Abnormal heart rhythm other than sinus with premature atrial beats 5
 - 5 or more premature ventricular contractions per minute 5
- General medical condition
 - $pO_2 < 60$, $pCO_2 > 50$, $K < 3$, $HCO_3 < 20$, $BUN > 50$,
 - Creatinine > 3 , raised SGOT, chronic liver disease,
 - Bedridden 5
- Surgery
 - Emergency 10
- Age > 70 years 5

Risk Index

- 0–5 points—Class I—6% Complications
- 6–12 points—Class II—7% Complications
- 13–25 points—Class III—20% Complications
- 26–100 points—Class IV—100% Complications

Lee's Revised Cardiac Risk Index

Cardiac risk factors in patients undergoing elective major noncardiac surgery

<i>Risk Factors</i>	<i>Inclusion Criteria</i>
➤ High risk surgery	Abdominal aortic aneurysm, thoracotomy, peripheral vascular surgery, major abdominal surgery
➤ Ischemic heart disease	History of myocardial infarction, history of a positive stress test, Q waves on electrocardiogram, use of nitrates, angina
➤ Congestive heart failure	History of congestive heart failure or pulmonary edema, paroxysmal nocturnal dyspnea, rales or S3 gallop, pulmonary vascular redistribution on chest radiograph
➤ Cerebrovascular disease	History of stroke or transient ischemic attack
➤ Insulin dependent diabetes mellitus	
➤ Preoperative serum creatinine concentration > 2 mg/dL	

<i>Risk of major cardiac event</i>	<i>No. of factors</i>
Class 1 (0.4%)	0
Class 2 (0.9%)	1
Class 3 (6.6%)	2
Class 4 ($> 11\%$)	3 or more

New York Heart Association Functional Classification

The parameters considered are:

- ❑ Limitations on physical activity
- ❑ Symptoms (such as undue fatigue, palpitations, dyspnea and/or angina pain) with ordinary physical activity
- ❑ Status at rest

The classification is:

Class I—No limitation on physical activity, no symptoms, comfortable at rest

Class II—Slight limitation on physical activity, symptomatic with ordinary activities, comfortable at rest

Class III—Marked limitations on physical activity, symptomatic at less than ordinary levels of activity, comfortable at rest

Class IV—Unable to perform any activity, discomfort with any activity, symptomatic at rest.

2014 ACC/AHA Guidelines (Table 1)

Table 1: Summary of recommendations for supplemental preoperative evaluation

<i>Recommendations</i>	<i>COR</i>	<i>LOE</i>
<i>The 12-lead ECG</i>		
Preoperative resting 12-lead ECG is reasonable for patients with known coronary heart disease or other significant structural heart disease, except for low-risk surgery	IIa	B
Preoperative resting 12-lead ECG may be considered for asymptomatic patients, except for low-risk surgery	IIb	B
Routine preoperative resting 12-lead ECG is not useful for asymptomatic patients undergoing low-risk surgical procedures	III No Benefit	B
<i>Assessment of LV function</i>		
It is reasonable for patients with dyspnea of unknown origin to undergo preoperative evaluation of LV function	IIa	C
It is reasonable for patients with HF with worsening dyspnea or other change in clinical status to undergo preoperative evaluation of LV function	IIa	C
Reassessment of LV function in clinically stable patients may be considered	IIb	C
Routine preoperative evaluation of LV function is not recommended	IIIa No Banfit	B
<i>Exercise stress testing for myocardial ischemia and functional capacity</i>		
For patients with elevated risk and excellent functional capacity, it is reasonable to forgo further exercise testing and proceed to surgery	IIa	B

Contd...

Contd...

Recommendations	COR	LOE
For patients with elevated risk and unknown functional capacity, it may be reasonable to perform exercise testing to assess for functional capacity if it will change management	IIb	B
For patients with elevated risk and moderate to good functional capacity, it may be reasonable to forgo further exercise testing and proceed to surgery	IIb	B
For patients with elevated risk and poor or unknown functional capacity, it may be reasonable to perform exercise testing with cardiac imaging to assess for myocardial ischemia	IIb	C
Routine screening with noninvasive stress testing is not useful for low-risk noncardiac surgery	III No Benefit	B
Cardiopulmonary exercise testing		
Cardiopulmonary exercise testing may be considered for patients undergoing elevated risk procedures	IIb	B
Noninvasive pharmacological stress testing before noncardiac surgery		
It is reasonable for patients at elevated risk for noncardiac surgery with poor functional capacity to undergo either DSE or MPI if it will change management	IIa	B
Routine screening with noninvasive stress testing is not useful for low-risk noncardiac surgery	III No Benefit	B
Preoperative coronary angiography		
Routine preoperative coronary angiography is not recommended	III No Benefit	C

Abbreviations: COR, class of recommendation; DSE, dobutamine stress echocardiogram; ECG, electrocardiogram; HF, heart failure; LOE, level of evidence; LV, left ventricular; MPI, myocardial perfusion imaging; N/A, not applicable.

SUGGESTED READING

1. 2014 ACC/AHA Guidelines for preoperative evaluation of cardiac patients for noncardiac surgery.
2. Delphin E, Seckin AI, Moore RA. Preoperative evaluation and preparation, Pediatric cardiac anesthesia. In: Lake CL, Brooker PD (Eds), 4th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 95-111.
3. Hines RL, Marschall KE. Preoperative evaluation, Stoelting's Anaesthesia and coexisting disease, 5th edition. Saunders Elsevier.
4. Indian Journal of Anaesthesia vol. 51 no. 4.
5. Tempe D. Clinical Practice of Cardiac Anaesthesia.

Chapter 3

Cardiovascular Monitoring

Manjula Sudeep Sarkar, Pallavi Waghalkar

INTRODUCTION

The vigilant anesthesiologist is the best monitor in the operating room. Monitors do not 'replace' the anesthesiologist; however, they aid in early detection of abnormal parameters. Thus they contribute towards safe management of anesthesia. Current trends in technology favor size reduction, minimally invasive device development, and advance display technology.

ASA MONITORING GUIDELINES

- ❑ **Standard I:** Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthesia, regional anesthesia and monitored anesthesia care.
- ❑ **Standard II:** During all anesthesia, the patient's oxygenation, ventilation, circulation and temperature shall be continually evaluated.

Patients presenting for cardiac surgery require extensive monitoring because of:

- ❑ Severe, unstable cardiovascular disease and hemodynamics
- ❑ The abnormal physiologic conditions associated with CPB
- ❑ Special considerations for minimally invasive cardiac surgery.

Inspection, palpation and auscultation are fundamental elements of perioperative cardiovascular monitoring

- ❑ Electrocardiogram (ECG)
- ❑ Noninvasive blood pressure (NIBP) monitoring
- ❑ Invasive blood pressure (IBP) monitoring
- ❑ Central venous pressure (CVP) monitoring
- ❑ Pulmonary artery (PA) catheter
- ❑ Cardiac output (CO) monitoring
- ❑ Transesophageal echocardiography (TEE)
- ❑ Coagulation parameter
- ❑ Arterial blood gas (ABG) monitoring
- ❑ Temperature

ECG MONITORING

- ❑ ECG facilitates the intraoperative diagnosis of arrhythmia, dysrhythmias, MI, and cardiac electrical silence during cardioplegia.
- ❑ Either three lead ECG or five lead ECG is available, where V5 lead is preferable for cardiac anesthesia which allows simultaneous recording of six frontal limb leads as well as one precordial unipolar lead (Fig. 1).

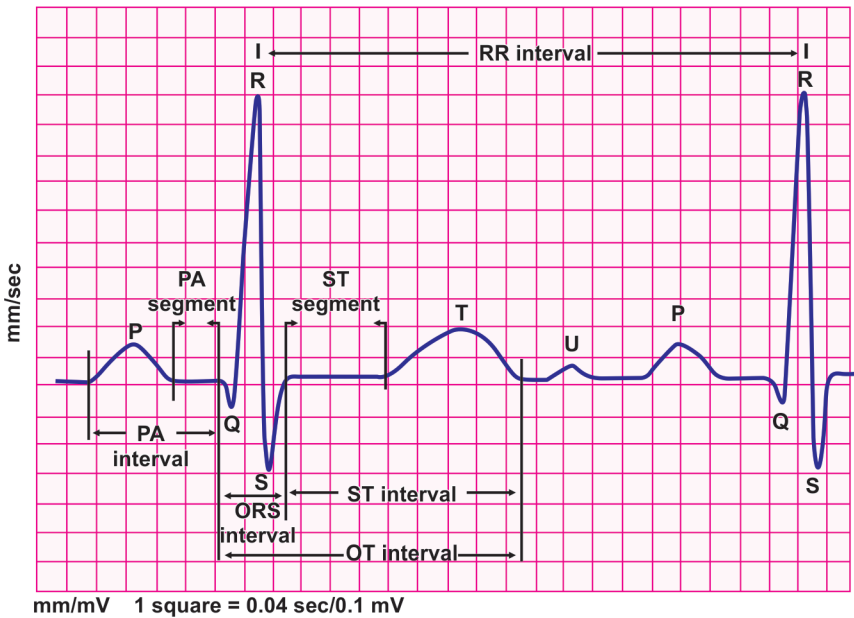


Fig. 1: Normal ECG

Indications

- ❑ For the diagnosis of:
 - Dysrhythmias
 - Arrhythmia
 - Myocardial ischemia, as angina cannot be elicited in an anesthetized patient
 - Conduction defects
 - Electrolyte imbalance
- ❑ Monitor the effect of cardioplegia arrest during cross clamp.

TECHNIQUES

Three Lead ECG System (Fig. 2)

- ❑ It uses electrodes attached to the right arm, left arm and left leg
- ❑ Difference between two electrodes is recorded and third electrode serves as a ground
- ❑ Leads I, II, III, aVR, aVL, and aVF are examined.

- ❑ For monitoring the inferior wall—leads II, III, and aVF are used
- ❑ For lateral wall—leads I, and aVL are useful.

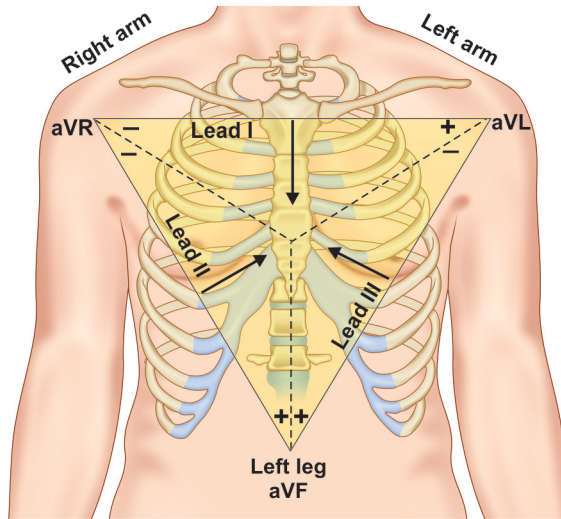


Fig. 2: Three electrode system

Five Lead ECG System (Fig. 3)

- ❑ The limb leads serve as a common ground for precordial unipolar lead which is placed in the V5 position along the anterior axillary line in the 5th intercostal space and monitors the left ventricle
- ❑ The precordial lead can also be placed over the right precordium to monitor the right ventricle.

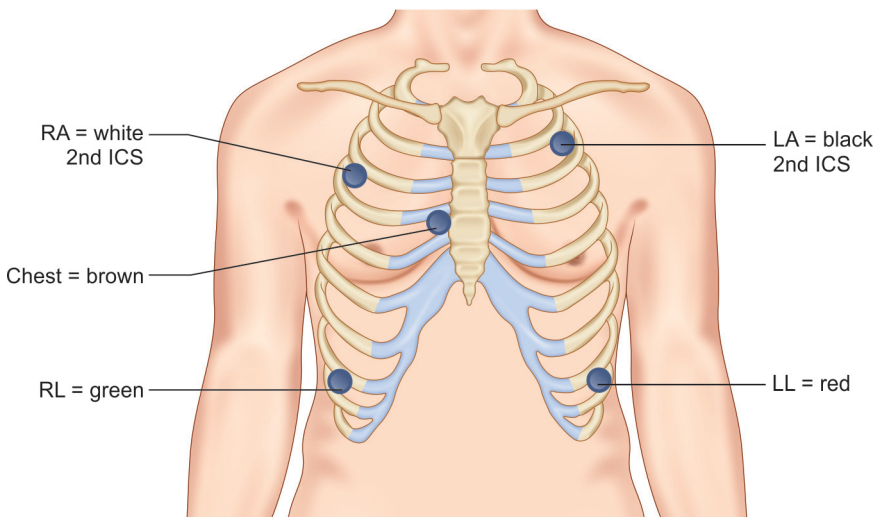


Fig. 3: Five electrode system

Advantages of five lead system

- ❑ All except the posterior wall ischemia can be monitored
- ❑ Up to 95% ischemic events can be detected
- ❑ Useful in differentiating atrial and ventricular dysrhythmias.

Semi-invasive ECG

1. **Esophageal leads:** These help to diagnose atrial dysrhythmias and are also useful in detecting posterior wall ischemia.
2. **Endotracheal leads:** These are useful for detecting atrial dysrhythmias in pediatric patients.
3. **Pulmonary artery catheter electrodes:** These help in recording intracavitary ECG, where atrial, ventricular and atrioventricular dysrhythmias and conduction blocks are diagnosed. They are also helpful in atrial and ventricular pacing.
4. **Epicardial leads:** They are placed intraoperatively before sternotomy closure in the form of pacing wires. They help to record complex conduction problems and dysrhythmias in the postoperative period.

Recommendations

For cardiac anesthesia, five lead ECG should be used with frequency response of 0.05 Hz to 100 Hz. ECG should display at least two leads simultaneously to evaluate ischemia and dysrhythmia for two different areas supplied by two different coronary arteries.

NONINVASIVE BLOOD PRESSURE MONITORING

Methods used depend on the principle that arteries are elastic and can be occluded by inflatable cuff with pressure greater than the systolic blood pressure. The degree of occlusion can then be detected by some means. Hence, correct application of cuff on the skin (snugly fitting) with the cuff lying over the artery is very important.

Techniques

- ❑ Manual intermittent techniques
- ❑ Automated intermittent techniques
- ❑ Automated continuous techniques.

Manual Intermittent Techniques

Palpatory method

- ❑ It relies on sphygmomanometer and mercury manometer to measure cuff pressure
- ❑ Riva-Rocci method: Systolic blood pressure (SBP) is measured by determining pressure at which palpated radial pulse disappears as cuff is inflated.

- ❑ Return to flow technique: Variation of Riva-Rocci method
- ❑ The SBP is recorded during cuff deflation at which pulse reappears and is detected by palpation
- ❑ It can be used with pulse oximeter or indwelling arterial catheter in ipsilateral arm.

Auscultatory method

- ❑ This was originally described by Korotkoff in 1905. It is most commonly used. It uses sphygmomanometer, cuff and stethoscope.
- ❑ It measures both SBP and DBP by auscultating sounds produced by arterial blood flow.
- ❑ Systolic blood pressure coincides with the onset of Korotkoff sounds; diastolic pressure is variably determined as their muffling or disappearance.

Technical aspects

- ❑ The length of the cuff should be 80% and width 40% of the circumference of the arm
- ❑ The cuff should be applied snugly, with bladder centered over the artery
- ❑ The rate of cuff deflation should be 2–3 mm Hg per heart beat.

Automated Intermittent Technique

It provides frequent, regular BP measurement. This is based on the Oscillometry principle. Arterial pulsations cause oscillations in cuff pressure. These oscillations are small if the cuff is inflated above systolic pressure.

Maximum oscillation occurs at mean arterial blood pressure, after which oscillations decrease. A microprocessor derives systolic, mean, and diastolic pressures using an algorithm.

The SBP and DBP are calculated from increasing and decreasing magnitude of oscillations according to empirically derived algorithm.

Advantages

- ❑ Uniform compression of the artery is not necessary
- ❑ No interference with noise
- ❑ Not sensitive to electrosurgical interference
- ❑ Works well with peripheral vasoconstriction.

Limitation

- ❑ It is sensitive to patient's arm movement.

Complications of noninvasive BP measurement

- ❑ Pain
- ❑ Petechiae and ecchymosis (patients on anti-inflammatory drugs, steroids, anticoagulants)
- ❑ Limb edema
- ❑ Venous stasis and thrombophlebitis
- ❑ Peripheral neuropathy (median, ulnar, radial)

- ❑ Compartment syndrome (more common after prolonged periods of frequent cycles of measurement, trauma or impaired limb perfusion).

Automated Continuous Techniques

- ❑ In 1973, Penaz described the volume clamp technique whereby a continuous noninvasive arterial pressure waveform could be obtained from a finger cuff.
- ❑ Device tends to track the MAP in digital arteries underlying the cuff by keeping the volume of the finger constant and thus nulling the transmural pressure.
- ❑ The blood volume of the finger varies in clinical fashion with each cardiac cycle because of the attendant variation in systemic blood pressure. This variation is detectable by a plethysmograph attached to the finger.
- ❑ The waveform is displayed on the screen.

Advantages

- ❑ It is an attempt to improve on the rapidity of determination of NIBP
- ❑ It correlates well with invasive BP in patients not in shock.

Disadvantages

- ❑ Compression of digital veins leads to suffusion of finger, temporary numbness
- ❑ Less reliable when peripheral perfusion is decreased
- ❑ Very sensitive to correct placement on middle phalanx.

Vasotrac System

- ❑ This is the most advanced BP measurement device
- ❑ It accurately measures SBP, DBP, MAP by waveform analysis. It is continuous and noninvasive. The waveform is produced by the variable pressure applied by pressure sensing mechanism directly over artery and the counter pressure in artery.

INVASIVE BLOOD PRESSURE MONITORING

Invasive beat-to-beat arterial blood pressure monitoring is considered the 'gold standard,' as it is both accurate and reliable.

It was described as catheter over needle technique by Barr in 1961.

Whilst not without risk, it has a number of advantages over noninvasive blood pressure measurement (NIBP):

- ❑ It allows continuous beat-to-beat pressure measurement, useful for close monitoring of patients whose condition may change rapidly, or those who require careful blood pressure control; for example, those on vasoactive drugs
- ❑ The waveforms produced may be analyzed, allowing further information about the patient's cardiovascular status to be gained (pulse contour analysis)

- ❑ It may also be useful where NIBP measurement is difficult, e.g. burns or obesity
- ❑ It reduces the risk of tissue injury and neuropraxia in patients who require prolonged blood pressure measurement
- ❑ It allows frequent arterial blood sampling
- ❑ It is more accurate than NIBP, especially in the setting of hypotension or arrhythmia.

Basic Principle

The commonly used IBP measuring systems consist of a column of fluid directly connecting the arterial system to a pressure transducer (hydraulic coupling). The pressure waveform of the arterial pulse is transmitted via the column of fluid, to a pressure transducer where it is converted into an electrical signal. This electrical signal is then processed, amplified and converted into a visual display by a microprocessor.

Components of an IBP Measuring System

- ❑ Intra-arterial cannula
- ❑ Fluid filled tubing
- ❑ Transducer
- ❑ Infusion/flushing system: A bag of either plain 0.9% saline or heparinized 0.9% saline is pressurized to 300 mm Hg and attached to the fluid filled tubing via a flush system. This allows a slow infusion of fluid at a rate of about 2–4 mL/hour to maintain the patency of the cannula. A flush system will also allow a high-pressure flush of fluid through the system in order to check the damping and natural frequency of the system.
- ❑ Signal processor, amplifier and display.

Damping

Anything that reduces energy in an oscillating system will reduce the amplitude of the oscillations. This is termed damping. Some degree of damping is required in all systems (critical damping), but if excessive (overdamping) or insufficient (underdamping) the output will be adversely affected.

These may be a major source of error, causing an under-reading of systolic blood pressure (SBP) and over-reading of diastolic blood pressure (DBP) although the mean blood pressure (MAP) is relatively unaffected.

- ❑ **An overdamped IBP system:** The damping co-efficient is >1 . This system will not oscillate freely and detail such as the dichrotic notch will be lost. It will not overshoot but will tend to under-read SBP and over-read DBP. It will be slow to respond to change due to the frictional drag in the system
- ❑ **An underdamped IBP system:** The damping co-efficient is <0.7 . This system will be quick to respond but will tend to overshoot and oscillate around its resting point, over-reading SBP and under-reading DBP.

Indications

- ❑ Small or rapid changes in arterial perfusion pressure may increase patient risk and require beat-to-beat assessment
- ❑ Wide variations in BP or intravascular volume is anticipated
- ❑ Frequent blood sampling, especially arterial blood gas (ABG) analysis, is required
- ❑ Assessment of BP cannot be done by other methods, e.g. cardiopulmonary bypass (nonpulsatile flow), dysrhythmias, marked obesity.

Sites of Cannulation

- ❑ Radial artery
- ❑ Femoral artery
- ❑ Dorsalis pedis artery
- ❑ Brachial artery
- ❑ Axillary artery
- ❑ Ulnar artery
- ❑ Posterior tibial artery

Technique

- ❑ Blind/traditional method
- ❑ Seldinger's technique
- ❑ USG guided: Especially beneficial when it is difficult to place the catheter via the traditional method
- ❑ Surgical cut down.

Contraindications

- ❑ Inadequate collateral flow to the hand is a relative contraindication
- ❑ Prior vascular surgery
- ❑ Skin infection at the site of cannulation

Femoral artery cannulation: It has advantages over the radial site.

- ❑ Assessment of central arterial pressure
- ❑ Appropriate access for placement of an intra-aortic balloon pump in any patient in whom difficulty in weaning from CPB is expected (e.g. those with markedly depressed ejection fraction, severe wall-motion abnormalities, or significant coronary disease).

Principle of IBP

Arterial Pressure Waveform

It results from ejection of blood from the left ventricle into aorta during systole followed by peripheral arterial runoff of stroke volume during diastole.

The systolic components follow R wave in ECG. It consists of:

- Steep pressure upstroke
- Peak
- Decline

Downslope is interrupted by dicrotic notch, then continues to decline during diastole after T wave of ECG.

Dicrotic notch directly in the central aorta is called incisura (related to closure of aortic valve).

Systolic upstroke of radial artery trace is 120–180 msec after R wave of ECG (time to travel from heart to radial artery to transducer).

MAP is calculated by area under arterial pressure wave divided by beat period.

Interpretation of Arterial Tracings

- a. **Heart rate and rhythm:** This is especially helpful if the patient is being paced or if electrocautery is being used. Presence of atrial or ventricular ectopic beats in the arterial trace can provide useful information on hemodynamic consequences of these dysrhythmias (e. g. if an ectopic beat is perfusing).
- b. **Pulse pressure:** It provides useful information about fluid states and valvular competence. Pericardial tamponade and hypovolemia are accompanied by a narrow pulse pressure on the arterial waveform. An increase in pulse pressure may be sign of worsening aortic valvular insufficiency or hypovolemia.
- c. **Respiratory variations and volume status:** Hypovolemia is suggested by a decrease in arterial systolic pressure with positive pressure ventilation (pulsus paradox).
- d. **Qualitative estimates of hemodynamic indices:** Inference can be made regarding contractility, stroke volume and vascular resistance.

Complications of Arterial Cannulation

- a. Ischemia: The incidence is low with radial artery cannulation.
- b. Thrombosis: Incidence with radial artery cannulation is high but recanalization occurs in majority. Incidence increases with diabetes or severe peripheral vascular disease.
- c. Infection: Risk is minimal with proper sterile technique.
- d. Bleeding: There is a greater tendency to bleed in patients with bleeding diathesis.
- e. False lowering of radial artery pressure immediately after CPB. This is due to forearm vasodilation secondary to rewarming, hypovolemia and vasoconstriction.

Recommendations for BP Monitoring

In high-risk patients, invasive arterial pressure monitoring should commence prior to induction. Under most circumstances, the radial artery pressure

measurement will be sufficient and accurate before and after CPB. In the patient with poor LV function, addition of a femoral arterial catheter before CPB may be warranted. Certain surgical procedures, for example, a thoracoabdominal aortic aneurysm repaired with left heart partial bypass, require both an upper and lower extremity arterial catheters. If an internal mammary artery (IMA) is dissected, retraction of the chest wall and compression of the subclavian artery can dampen or obliterate the radial artery traces. The surgeon should be informed for possible change in retractor position. A dampened radial pressure during IMA harvest may also be associated with brachial plexus injury.

CENTRAL VENOUS PRESSURE MONITORING

The central venous pressure (CVP) measures the filling pressure of the right ventricle (RV).

It gives an estimate of the intravascular volume status and is an interplay of the circulating blood volume, venous tone and right ventricular function.

CVP is the pressure measured at the junction of the superior vena cava and the right atrium.

- ❑ Normal CVP in an awake, spontaneously breathing patient: 1–7 mm Hg or 5–10 cm H₂O.
- ❑ For patient on mechanical ventilation: 3–5 cm H₂O higher.

Methods to Measure CVP

1. **Indirect assessment:** Inspection of jugular venous pulsations in neck.
2. **Direct assessment:**
 - Fluid filled manometer connected to central venous catheter
 - Calibrated transducer.

Indications

- ❑ Major operative procedures involving large fluid shifts and/or blood loss
- ❑ Intravascular volume assessment when urine output is not reliable or unavailable (e.g. renal failure)
- ❑ Major trauma
- ❑ Surgical procedures with a high risk of air embolism, such as sitting position craniotomies. In addition to monitoring, the central venous pressure (CVP) catheter may also be used to aspirate intracardiac air
- ❑ Frequent venous blood sampling
- ❑ Venous access for vasoactive or irritating drugs
- ❑ Chronic drug administration
- ❑ Inadequate peripheral IV access
- ❑ Rapid infusion of IV fluids
- ❑ Special Uses: (i) Insertion of PA catheters; (ii) Insertion of transvenous pacing wires; (iii) Hemodialysis/plasmapheresis.

Contraindications

- ❑ **Absolute**
 - SVC syndrome
 - Infection at the site of insertion
- ❑ **Relative**
 - Coagulopathies
 - Newly inserted pacemaker wires
 - Presence of carotid disease
 - Recent cannulation of the internal jugular vein
 - Contralateral diaphragmatic dysfunction
 - Thyromegaly or prior neck surgery.

Routes of Access of Central Vein

- ❑ **Commonly used veins:**
 - Subclavian vein
 - Internal jugular vein
 - Femoral vein
 - Basilic vein (antecubital fossa).
- ❑ **Less commonly used veins**
 - Axillary (anterior and lateral approach)
 - External jugular
 - Brachial (mid-upper arm approach)
 - Cephalic (antecubital fossa approach)
 - Brachiocephalic (supraclavicular approach).

CVP Waveform Interpretation

The normal CVP waveform consists of three upwards deflections (A, C, & V waves) and two downward deflections (X and Y descents).

These waves are produced as follows:

- ❑ The **'A' wave** is produced by right atrial contraction and occurs just after the P wave on the ECG.
- ❑ The **'C' wave** occurs due to isovolumetric ventricular contraction forcing the tricuspid valve to bulge upward into the right atrium (RA)
- ❑ The pressure within the RA then decreases as the tricuspid valve is pulled away from the atrium during right ventricular ejection, forming the **X descent**.
- ❑ The RA continues to fill during late ventricular systole, forming the **V wave**.
- ❑ The **Y descent** occurs when the tricuspid valve opens and blood from the RA empties rapidly into the RV during early diastole.

Complications of Central Venous Catheterization

- ❑ Arterial puncture with hematoma
- ❑ Arteriovenous fistula
- ❑ Hemothorax
- ❑ Chylothorax
- ❑ Pneumothorax
- ❑ Nerve injury: Brachial plexus, stellate ganglion (Horner's syndrome)
- ❑ Air embolism
- ❑ Catheter or wire shearing.

Complications of Catheter Presence

- ❑ Thrombosis, thromboembolism
- ❑ Infection, sepsis, endocarditis
- ❑ Arrhythmias
- ❑ Hydrothorax.

PULMONARY ARTERY CATHETER MONITORING

Pulmonary artery catheter (PAC) was invented in 1970 by Swan, Ganz and colleagues for hemodynamic assessment of patients with acute myocardial infarction. The standard PAC is 7.0, 7.5 or 8.0 French in circumference and 110 cm in length, divided in 10 cm intervals (Fig. 4).

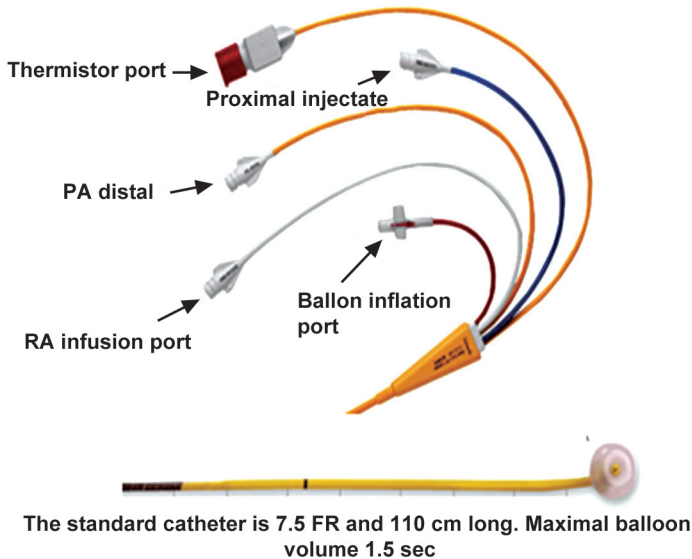


Fig. 4: Pulmonary artery catheter

The PAC has 4–5 lumens:

- ❑ Temperature thermistor located proximal to balloon to measure pulmonary artery blood temperature
- ❑ Proximal port located 30 cm from tip for CVP monitoring, fluid and drug administration
- ❑ Distal port at catheter tip for PAP monitoring
- ❑ Variable infusion port (VIP) for fluid and drug administration
- ❑ Balloon at catheter tip.

Pulmonary Artery Catheterization

A large-bore introducer catheter is used to facilitate PAC insertion. It is inserted through the subclavian or internal jugular vein with the patient in Trendelenberg position. Prior to PAC insertion, distal port is connected to the pressure transducer which is set at the level of the patient's heart and then zeroing is done. Continuous pressure monitoring during PAC insertion is required to determine the location of catheter tip. When the 20 cm mark is reached at the hub of the introducer, the balloon is inflated. The PAC is advanced until the pulmonary capillary wedge pressure (PCWP) waveform is obtained, usually around 45–55 cm at the hub.

PAC Waveform

Waveforms (Fig. 5) are identified by the movement of catheter through right side of the heart into the pulmonary artery and capillary bed:

- ❑ Right atrial (RA)
- ❑ Right ventricular (RV)
- ❑ Pulmonary artery pressure (PAP)
- ❑ Pulmonary capillary wedge pressure (PCWP) *or*
- ❑ Pulmonary artery wedge pressure (PAWP).

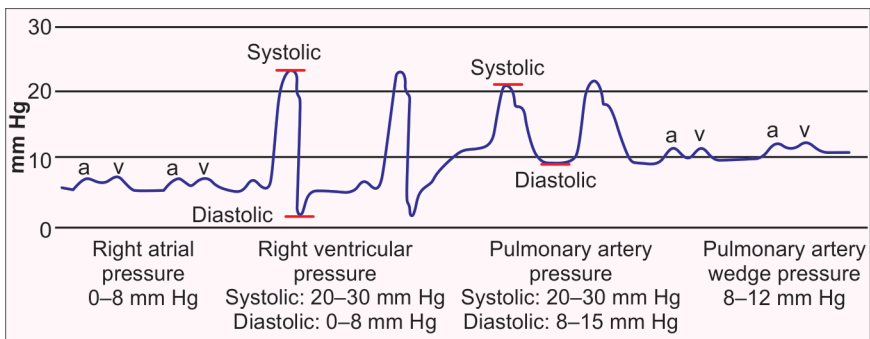


Fig. 5: PAC waveform

Indications**A. Cardiac surgery:**

- Poor LV function (Ejection fraction <0.4; left ventricular end-diastolic pressure (LVEDP) >18 mm Hg)

- Recent MI (<6 months)
- 75% left main coronary disease
- Complications of MI, e.g. MR, VSD, ventricular aneurysm
- Combined lesions, e.g. CAD+MR or CAD+AS
- Complicated lesions, e.g. idiopathic hypertrophic sub-aortic stenosis (IHSS).
- Intra-aortic balloon pump (IABP).

B. Noncardiac situations:

- Shock of any cause
- Severe pulmonary disease
- Bleomycin toxicity
- Massive trauma
- Liver transplantation.

Measured Hemodynamic Parameters

- 1. Central venous pressure (CVP):** It is recorded from proximal port of PAC in the superior vena cava or right atrium
 - CVP = RAP
 - CVP = Right ventricular end diastolic pressure (RVEDP) when no obstruction exists between atrium and ventricle.
- 2. Pulmonary artery pressure (PAP):** It is measured at the tip of the PAC with balloon deflated. It reflects RV function, pulmonary vascular resistance and LA filling pressures.
- 3. Pulmonary capillary wedge pressure (PCWP):** Recorded from the tip of the PAC catheter with the balloon inflated
 - PCWP = LAP = LVEDP (when no obstruction exists between atrium and ventricle).
- 4. Cardiac output (CO):** It is calculated using thermodilution technique. The thermistor at the distal end of PAC records change in temperature of the blood flowing in the pulmonary artery when the blood temperature is reduced by injecting a volume of cold fluid through PAC into RA.

Derived Hemodynamic Parameters

1. Cardiac index (CI) = CO/BSA
2. Stroke volume index (SVI) = CI/HR
3. Systemic vascular resistance (SVR) reflects impedance of the systemic vascular tree

$$SVR = 80 \times (MAP - CVP)/CO$$

4. Pulmonary vascular resistance (PVR) reflects impedance of pulmonary circuit

$$PVR = 80 \times (PAM - PCWP) / CO$$

5. Left ventricular stroke work index (LVSWI) = (MAP - PCWP) × SVI × 0.136
6. Right ventricular stroke work index (RVSWI) = (PAM - CVP) × SVI × 0.136

PAC Complications

Due to establishment of central venous access:

- ❑ Accidental puncture of carotid artery
- ❑ Bleeding
- ❑ Neuropathy
- ❑ Air embolism
- ❑ Pneumothorax

Due to pulmonary artery catheterization:

- ❑ Dysrhythmias:
 - Premature ventricular and atrial contractions
 - Ventricular tachycardia or fibrillation
- ❑ Complete heart block (CHB) in patients with preexisting LBBB
- ❑ Minor increase in tricuspid regurgitation
- ❑ Thromboembolism
- ❑ Mechanical, catheter knots
- ❑ Pulmonary infarction
- ❑ Infection, endocarditis
- ❑ Endocardial damage, cardiac valve injury
- ❑ Pulmonary artery rupture: 0.03–0.2% incidence, 41–70% mortality.

CARDIAC OUTPUT MONITORING

Cardiac output is the volume of blood pumped by the heart, in particular by a left or right ventricle in one minute. An average resting cardiac output (Q) would be 5.6 L/min for a human male and 4.9 L/min for a female.

$$Q = \text{Stroke volume} \times \text{Heart rate}$$

Cardiac output monitoring in the critically ill patient is a standard practice in order to ensure adequate tissue oxygenation and has been traditionally accomplished using pulmonary artery catheter (PAC).

Adolph Fick described the first method of CO estimation in 1870. This method was the reference standard by which all other methods of determining CO were evaluated until the introduction of the PAC in the 1970s.

Methods of CO Monitoring

- ❑ Noninvasive
- ❑ Minimally invasive
- ❑ Invasive

Noninvasive CO Monitoring

1. Esophageal Doppler:

- This technique measures blood flow velocity in the descending thoracic aorta using a flexible ultrasound probe about the size of a nasogastric tube. This measurement can be combined with an estimate of the cross-sectional area of the aorta, which is derived from the patient's age,

height and weight, and allows hemodynamic variables including stroke volume, cardiac output and cardiac index to be calculated.

- The precision of this measurement depends on the following three conditions—the cross-section must be accurate, the ultrasound beam must be directed parallel to the blood flow, and the beam direction should not undergo major alteration between measurements.

2. Transesophageal echocardiography (TEE):

- TEE provides information on cardiac contractility, filling status and output, valvular morphology and function, as well as on the ascending and descending aorta structure in the critically ill patient. With this technique, cardiac output measurement is the result of calculating stroke volume, which can be multiplied by heart rate.
- TEE utilizes Simpson's rule in which the LV is divided into a series of disks to estimate CO without the use of Doppler. End-diastolic and end-systolic dimensions measured by echocardiography are converted to volumes, allowing stroke volume and CO to be determined.

3. Partial CO₂ rebreathing:

A new monitor called NICO is based on the application of the Fick principle to carbon dioxide, in order to estimate cardiac output noninvasively. The monitor consists of a carbon dioxide sensor, a disposable airflow sensor and a pulse oximeter. VCO₂ is calculated from minute ventilation and its carbon dioxide content. The arterial carbon dioxide content (CaCO₂) is estimated from end-tidal carbon dioxide

The Fick equation for carbon dioxide is:

$$CO = VCO_2 / C_vCO_2 - CaCO_2$$

where VCO₂, C_vCO₂, CaCO₂ are CO₂ consumption, venous CO₂ concentration and arterial CO₂ concentration respectively.

Minimally Invasive CO Monitoring

1. **Lithium dilution cardiac output:** It requires a venous line and an arterial catheter. The venous line can be either central or peripheral. A bolus of isotonic (150 mM) lithium chloride (LiCl₂) solution is injected via the venous line. The usual dose for an adult is 0.3 mmol. Arterial plasma concentration is measured by withdrawing blood across the selective lithium electrode at the rate of 4 mL/min. Cardiac output is calculated based on the lithium dose and the area subjected to the concentration-time circulation.
2. **Pulse contour cardiac output:** A long arterial catheter (with a thermistor) placed in the femoral, axillary, or brachial artery, and connected to a pulse contour device. With this catheter, a continuous pulse waveform contour analysis is obtained. The technique uses analysis of the area under the systolic portion of the arterial pressures waveform, from the end-diastole to the end of the ejection phase; this corresponds to the stroke volume. Also, by virtue of a pulse contour analysis device, a beat-to-beat analysis of cardiac output, averaged at 30 seconds, is displayed.

Principle

- The fluctuations of blood pressure around a mean value are caused by the volume of blood forced into the arterial conduit by each systole
- The magnitude of the change in pressure—known, as the pulse pressure, is a function of the magnitude of the change in stroke volume
- One factor, however, that is of particular importance is the compliance of the arterial wall.

Limitations

- Severe arrhythmias may reduce the accuracy of cardiac output measurement
- Use of an intra-aortic balloon pump precludes adequate performance of the device
- It has limited accuracy during periods of hemodynamic instability.

3. Statistical analysis of arterial pressure—FloTrac/Vigileo:

- FloTrac/Vigileo is an uncalibrated pulse contour analysis-based hemodynamic monitor that estimates cardiac output (Q) using a standard arterial catheter with a manometer located in the femoral or radial artery.
- The device utilizes an algorithm that is based on Frank-Starling law of the heart, that pulse pressure (PP) is proportional to stroke volume (SV). The algorithm calculates the product of the standard deviation of the arterial pressure wave (AP) (over a sampled period of time of 20 seconds) and a vascular tone factor (Khi) to generate stroke volume.
- The equation in simplified form is as follows: $SV = \text{Std (AP)} \times \text{Khi}$ or $BP \times k(\text{constant})$.
- Cardiac output (Q) is then derived utilizing the equation $Q = HR \times SV$. Only perfused beats that generate an arterial waveform are counted for HR.

Invasive CO Monitoring

The PAC was the clinical standard for cardiac output monitoring for more than 20 years and the technique has been extensively investigated.

Cardiac output measurement by intermittent pulmonary artery thermodilution, which is based on the Stewart-Hamilton principle, is considered to be the reference cardiac output monitoring standard against which all new cardiac output measuring devices are compared.

- 1. Thermodilution with cold injectate:** This method is the most commonly utilized CO technique because of its ease, and ability to repeat measurements over time. The indicator is an aliquot of saline (typically 10 mL, which is at a lower temperature than the temperature of blood) injected into the RA. The change in temperature produced by injection of this indicator is measured in the PA by a thermistor and is integrated over time to generate a value for RV output, which is equal to systemic CO if no intracardiac shunts are present. This method requires no withdrawal of blood and no arterial line, uses an inexpensive indicator, and is not greatly affected by recirculation.

The thermodilution method underestimates the CO with right-side valvular lesions. Thermodilution remains accurate for forward left ventricular CO for mitral and aortic valve lesions.

$$\text{Cardiac output} = \frac{\text{Quantity of Indicator}}{\int_0^{\infty} \text{Concentration of Indicator} \cdot dt}$$

Limitations

- Volume of injectate: Because the output calculation is based on a specific injectate volume, less volume than that for which the computer algorithm is set, will cause a falsely high value of CO, and vice versa
 - Temperature of injectate: If the temperature of an injectate is incorrect, errors can occur
 - Shunts: Intracardiac shunts will cause erroneous values. This technique should not be used if communication exists between the pulmonary and systemic circulations
 - Timing with the respiratory cycle: As much as a 10% difference in CO will result, depending on when injection occurs during the respiratory cycle due to actual changes in pulmonary blood flow with the respiration
 - Catheter position: The tip of the pulmonary catheter must not be “wedged”; otherwise, erroneous curves are obtained.
- 2. Continuous thermodilution:** A thermal filament in the catheter heats blood, thus generating a temperature change that is measured via a distal thermistor. The input and output signals are correlated to generate CO values. Continuous cardiac output assessment may overcome some of these limitations of intermittent thermodilution method.
- Summary:* The PAC still remains the practical gold standard for evaluating CO. It also provides true mixed venous saturation and pulmonary pressures that cannot be obtained from noninvasive devices. Invasive hemodynamic monitoring remains the standard during surgery. However, postoperatively stable cardiac patients are selected candidates for minimally invasive device monitoring in the ICU.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

The use of echocardiography has advanced greatly in the past few years. It has a great role in diagnosis as well as management of patients perioperatively. The introduction of transesophageal echocardiography (TEE) has provided a new acoustic window to the heart and the mediastinum.

Basic Concepts

Heart and great vessels are probed with ultrasound frequency above 20,000 Hz. The ultrasound is sent in the thoracic cavity and is partially reflected by cardiac structures. From these reflections, distance, velocity and density of objects within the chest are derived.

Imaging Techniques

- ❑ **M-mode:** This is most simple mode. In this mode, the density and position of all tissues in the path of a narrow ultrasound beam are displayed as scroll on video screen. It has limited value because only some part of heart is observed at one time but it is useful for precise timing of events with cardiac cycle. It is called M-mode because it has timed motion display.
- ❑ **Two-dimensional mode:** Rapid repetitive scanning along many different radii within an area in the shape of a sector is done. It thus forms 2D image of section of heart which is displayed in “Real time” on monitor. Image obtained resembles anatomic section of heart and can be easily interpreted.
- ❑ **Doppler technique:** Modern machines combine Doppler capabilities with 2D imaging. First 2D image of heart is obtained then Doppler beam is superimposed on 2D image. Flow velocities are measured by Doppler.

Equipment

All the currently available TEE probes have 3.7–7.5 MHz transducer. The miniature echocardiographic transducer (40 mm long, 13 mm wide and 11 mm thick) is mounted on the tip of a gastroscope housing. The tip of the probe is flexible. Two knobs on the proximal handle are present. One knob controls anterior and posterior movement (anteflexion and retroflexion) while other knob controls side to side movements. Electronic switch is available to scan the heart in various axial views. Heart can be visualized in 0 to 180 degrees.

Basic Views

1. Upper esophageal (20–30cm): It is helpful for evaluating the great vessels including the aortic root and coronary arteries, ascending aorta and the pulmonary artery.
2. Mid-esophageal (30–40 cm): Most of the information is usually obtained in this view.
 - **4 chamber view, 0 degree:** The LV and the right ventricle RV, as well as the LA and RA can be displayed.
 - **5 chamber view, 0 degrees:** The aortic root and left ventricular outflow tract.
 - **2 Chamber view, 90 degrees:** Here LA and LV with LA appendages are seen.
 - **Long axis view, 120–140 degrees:** Best view to look at the LVOT and anteroseptum as well as A2 and P2 of the mitral valve.
 - **Short axis view, 30–60 degrees:** Best view to asses aortic valve.
 - **Bicaval view, 90–110 degrees:** Lays out the intra-atrial septum. This is a great view to look for PFOs, ASDs. This is also an excellent view to evaluate the IVC, SVC, eustachian valve, and right atrial appendage.

3. Transgastric (40–45 cm): It is one of the best views to evaluate left ventricular and right ventricular function and pericardial effusions. Also used to assess ejection fraction and wall motion postoperatively.
 - **Transgastric short axis, 0 degrees:** In this view the anterior, inferior and lateral walls of LV are well visualized as well as the ventricular septum.
 - **Transgastric short axis, 0–30 degrees:** To assess mechanism of tricuspid regurgitation due to restricted leaflet motion (such as pacemaker lead related scarring) vs excess motion in a flail segment.
 - **Transgastric long axis, 90 degrees:** This is an excellent view for evaluating mitral valve vegetations including chordal/papillary muscle involvement and for visualizing ruptured chords.
4. Deep transgastric (45–50 cm): The best views in which one can obtain accurate gradients across the aortic valve to assess the degree of aortic stenosis or regurgitation.

Advantages of TEE over TTE

- ❑ The sound waves emitted from the transducer are passing only through the esophageal wall and pericardium to reach the heart. It does not have to pass through chest wall containing fat and bone. Hence image distortion due to interference with other media is unlikely.
- ❑ As compared to transthoracic transducer, esophageal transducer is in the stable position; hence, continuous recording of cardiac activity for extended period is possible during TEE.
- ❑ It does not interfere with the surgical field.

Disadvantages of TEE

- ❑ It is semi-invasive procedure
- ❑ Chances of injury are present
- ❑ Needs special set up, technique, preparation, instrumentation
- ❑ Needs orientation and skills.

Indications

Indications for perioperative TEE were divided into three categories (I-III) based on the strength of evidence or expert opinion supporting a clinical benefit

Category I

- ❑ Evaluation of acute, persistent, and life-threatening hemodynamic instability in OR or ICU.
 - Intraoperatively during valve repair, congenital heart surgery, repair of hypertrophic obstructive cardiomyopathy (HOCM), to diagnose endocarditis, suspected thoracic aortic aneurysm, dissection or disruption and during repair of aortic dissections.

Category II

- ❑ Perioperative use in patients at increased risk of myocardial ischemia or infarction
- ❑ Perioperative use in patients at increased risk of hemodynamic disturbances
- ❑ Intraoperative assessment of repair of cardiac aneurysm
- ❑ Intraoperative evaluation of removal of cardiac tumors
- ❑ Intraoperative detection of foreign bodies
- ❑ Intraoperative detection of air emboli during cardiac or neurosurgical procedures
- ❑ During intracardiac thrombectomy or pulmonary embolectomy
- ❑ Intraoperative use for suspected cardiac trauma
- ❑ Preoperative assessment of patients with suspected acute thoracic aortic dissections, aneurysms or disruptions
- ❑ Repair of thoracic aortic dissections without aortic valve involvement
- ❑ Intraoperative evaluation of aortic atheromatous disease
- ❑ Intraoperative evaluation of pericardiectomy, pericardial effusions or evaluation of pericardial surgery
- ❑ Intraoperative evaluation of anastomotic sites during heart and/or lung transplantation
- ❑ Monitoring placement and function of assist devices.

Category III

- ❑ Intraoperative evaluation of myocardial perfusion, coronary artery anatomy, graft patency or cardioplegia administration
- ❑ Intraoperative use during cardiomyopathies other than HOCM
- ❑ Intraoperative use for uncomplicated endocarditis during noncardiac surgery
- ❑ Intraoperative monitoring for emboli during orthopedic procedures
- ❑ Intraoperative assessment of repair of thoracic aortic injuries
- ❑ Intraoperative use for uncomplicated pericarditis
- ❑ Intraoperative evaluation of pleura—pulmonary diseases
- ❑ Monitoring placement of intra-aortic balloon pump, automatic implantable cardiac defibrillators or pulmonary artery catheters.

Contraindications

Absolute

- ❑ Previous esophagectomy
- ❑ Severe esophageal obstruction
- ❑ Esophageal perforation
- ❑ Ongoing esophageal hemorrhage

Relative

- ❑ Esophageal diseases—diverticulum, varices, fistula
- ❑ Previous esophageal surgery
- ❑ Previous mediastinal irradiation
- ❑ Unexplained swallowing difficulty.

Clinical Application

- ❑ TEE reveals changes in left ventricular preload and filling pressure. It measures end diastolic volume
- ❑ Real-time TEE images of LV filling and ejection permit qualitative, immediate detection of extreme changes in cardiac output
- ❑ TEE quantify CO, the velocity and the cross-sectional area of blood flow
- ❑ Fractional area change (FAC) during systole is a measure of global LV function
- ❑ Hallmarks of severe RV dysfunction are severe hypokinesia, enlargement of RV, change in shape of RV from crescent to round
- ❑ TEE is an ideal tool for assessment of diastolic function because of its unobstructed view of the mitral valve and pulmonary veins
- ❑ Detection of myocardial ischemia: Acute myocardial ischemia produces abnormal inward motion and thickening of affected myocardium.

COAGULATION MONITORING

Traditionally, coagulation monitoring in the surgical patient has focused on preoperative testing to identify patients at increased risk for perioperative bleeding and intraoperative monitoring of heparin therapy during cardiac and vascular surgery. More recently, availability of increasingly sensitive and specific point-of-care coagulation monitors has provided an opportunity to guide administration of blood components and hemostatic drugs more specifically, without the delays inherent in standard laboratory testing.

Cardiac surgery patients, especially with CPB, are universally anticoagulated with heparin. The vast majority of patients receive protamine to ameliorate the effects of heparin. CPB results in platelet dysfunction, decreased platelet number and dilution of coagulation factors. Monitoring coagulation status is critical.

Commercially available point-of-care coagulation monitors used in the perioperative setting may be divided into four categories:

- 1. Functional measures of coagulation or assays that measure the intrinsic ability of the blood to clot**
 - Activated coagulation time (ACT)
 - Heparin management test (HMT)
 - High-dose thrombin time (HiTT)
 - Prothrombin time (PT)
 - Activated partial thromboplastin time (aPTT)

2. Monitors of heparin concentration:

- Protamine titration
- Ion-selective electrodes

3. Viscoelastic measures of coagulation

- Thromboelastogram (TEG)
- Sonoclot

4. Platelet function analyzers.**Activated Clotting Time (ACT)**

- ❑ ACT testing is probably the most widely used perioperative coagulation monitor because of its simplicity, low-cost, and ability to monitor anticoagulation when large doses of heparin are used as required for cardiac surgical procedures.
- ❑ One of the more widely available ACT monitors employs a glass test tube that contains a small magnet at the bottom, with kaolin or celite as activator. The ACT in normal individuals is approximately 107 ± 13 sec (mean \pm standard deviation).
- ❑ Heparin clearly prolongs the ACT, because this test measures the clotting potential of the intrinsic and common pathways of coagulation. In addition to ACT prolongation by heparin, a prolonged ACT signifies impaired ability of the blood sample to generate clot, for whatever reason. However, the ACT is relatively resistant to platelet dysfunction and is affected only by severe thrombocytopenia and platelet inhibitors such as prostacyclin or monoclonal antibodies directed against GP IIb/IIIa surface receptors.
- ❑ For CPB, prolongation of the ACT to greater than 400s is usually deemed adequate. Off pump CABG procedures sometimes use “partial heparinization” with an ACT target of about 300s.

Limitations

- ❑ ACT is relatively insensitive to low concentrations of heparin, and ACT measurements are not particularly reproducible. These deficiencies are especially important when ACT monitoring is used to verify heparin neutralization with protamine.
- ❑ ACT monitoring also may be influenced by phospholipid-bound microparticles extruded from the platelet surface during activation. These platelet microparticles artifactually shorten the ACT even in the presence of residual heparin.
- ❑ In addition to artifactually low ACT values, high ACT values greater than 600 sec do not represent a linear dose-response relationship to high-dose heparin administration.
- ❑ Both hypothermia and hemodilution prolong ACT.

Thromboelastography (TEG)

The unique aspect of viscoelastic measures lies in their ability to measure the entire spectrum of clot formation from early fibrin strand generation through clot retraction and eventual fibrinolysis.

TEG provide unique advantages over ACT and traditional coagulation parameters because it provides functional information on platelets, clotting factors and fibrinolytic processes.

Various parameters that describe characteristics of clot formation and lysis are inscribed by the TEG recorder.

- ❑ **The R value (reaction time)** measures time to initial clot formation (normal: 7.5–15 min). It is considered comparable to the whole blood clotting time and may be accelerated by adding celite to the TEG sample cuvette. The R value is prolonged by a deficiency of one or more plasma coagulation factors.
- ❑ **Maximum amplitude (MA)** provides a measure of clot strength and may be decreased by either qualitative or quantitative platelet dysfunction or decreased fibrinogen concentration. Normal MA is 50 to 60 mm.
- ❑ **The alpha angle and K (coagulation constant)** values measure the rate of clot formation and may be prolonged by any factor slowing clot generation, such as plasma coagulation factor deficiency or heparin anticoagulation.

One of the more common applications of the TEG analyzer is the real-time detection of excessive fibrinolysis during liver transplantation. The TEG also may be used to differentiate surgical bleeding from coagulopathy following cardiac surgery.

Limitation: Lack of specificity associated with abnormal findings and the qualitative nature of assay interpretation.

ARTERIAL BLOOD GAS (ABG) MONITORING

Monitoring of ABGs is an essential part in the anesthetic management of the high-risk patients as well as in the care of critically ill patients and especially in cardiac surgery on CPB. The pH, PCO₂ and PO₂ measurements are normally accomplished by blood gas analyzers using an arterial blood sample fed into the analyzer as and when the acid base status is required.

In ABG, 3-5 values are actually measured (pH, PaO₂, PaCO₂, Hb & O₂ saturation) and all other values are calculated like ABC (actual bicarbonate), TCO₂, SBC (standard bicarbonate), BE (base excess), SBE (standard base excess).

Principles of Gas Analysis

- ❑ Chemical (Haldane, Orsat-Henderson and Van Slyke)
- ❑ Physical (Magnetic, infrared, gas chromatography)
- ❑ Specific electrodes.

Indications

- ❑ To evaluate the adequacy of ventilation
- ❑ To quantitate patient's response to therapeutic intervention
- ❑ To monitor severity and progression of documented disease process
- ❑ To assess end organ perfusion, tissue oxygenation and acid-base status especially during cardiac surgery on CPB for optimum myocardial protection.

Acid-Base Disorder

In acidosis and alkalosis—acid-base disturbance is at cellular level, there is a change in PaCO_2 or in HCO_3^- but there is no change in pH.

In acidemia there is change in pH below normal (< 7.36) similarly in alkalemia pH goes above normal (> 7.44).

Primary is the cause of acid-base derangement where as secondary is the compensatory change. The end point is constant if compensation is in range. $\text{PaCO}_2/\text{HCO}_3^-$ is constant.

In analyzing the data follow a sequence of order

1. **History and physical examination:** It usually gives an idea of what acid-base disorder might be present even before collecting the ABG sample.
2. **Check pH.** It indicates acidemia or alkalemia
 $\text{pH} < 7.35$ acidemia—the process causing acidemia is acidosis
 $\text{pH} > 7.45$ alkalemia—the process causing alkalemia is alkalosis
 - If pH is within normal range, then acid-base disorder not likely to be present.
 - pH may be normal in the presence of a mixed acid-base disorder, particularly if other parameters of the ABG are abnormal.
3. **Check PO_2** —which indicates oxygenation.
4. **Check PCO_2 and HCO_3^-** —which indicates ventilation and renal function. In simple acid-base disorders, both values are abnormal and direction of the abnormal change is the same for both parameters.

- One abnormal value will be the initial change and the other will be the compensatory response.

Distinguish the initial change from the compensatory response:

The initial change will be the abnormal value that correlates with the abnormal pH.

- If Alkalosis, then PCO_2 low or HCO_3^- high
- If Acidosis, then PCO_2 high or HCO_3^- low.
- Once the initial change is identified, then the other abnormal parameter is the compensatory response if the direction of the change is the same. If not, suspect a mixed disorder

Once the initial chemical change and the compensatory response is distinguished, then identify the specific disorder (Table 1).

- If PCO_2 is the initial chemical change, then process is respiratory.
 - If HCO_3^- is the initial chemical change, then process is metabolic.
5. **If respiratory process, is it acute or chronic?**
 An acute respiratory process will produce a compensatory response that is primarily due to rapid intracellular buffering.
 - A chronic respiratory process will produce a more significant compensatory response that is primarily due to renal adaptation, which takes a longer time to develop.
 - To assess if acute or chronic, determine the extent of compensation.

Table 1: Different types of acid-base disorders

Primary disorder	Initial chemical change	Compensatory response	Compensatory mechanism	Expected level of compensation
Metabolic acidosis	↓HCO ₃ ⁻	↓PCO ₂	Hyperventilation	PCO ₂ = (1.5 × [HCO ₃ ⁻]) + 8 ± 2
				↓PCO ₂ = 1.2 × Δ [HCO ₃ ⁻]
				PCO ₂ = last 2 digits of pH
Metabolic alkalosis	↑HCO ₃ ⁻	↑PCO ₂	Hypoventilation	PCO ₂ = (0.9 × [HCO ₃ ⁻]) + 16 ± 2
				↑PCO ₂ = 0.7 × Δ [HCO ₃ ⁻]
Respiratory acidosis	↑PCO ₂	↑HCO ₃ ⁻		
Acute			Intracellular buffering (hemoglobin, intracellular proteins)	↑[HCO ₃ ⁻] = 1 mEq/L for every 10 mm Hg ΔPCO ₂
Chronic			Generation of new HCO ₃ ⁻ due to the increased excretion of ammonium	↑[HCO ₃ ⁻] = 3.5 mEq/L for every 10 mm Hg ΔPCO ₂
Respiratory alkalosis	↓PCO ₂	↓HCO ₃ ⁻		
Acute			Intracellular buffering	↓[HCO ₃ ⁻] = 2 mEq/L for every 10 mm Hg ΔPCO ₂
Chronic			Decreased re-absorption of HCO ₃ ⁻ , decreased excretion of ammonium	↓[HCO ₃ ⁻] = 4 mEq/L for every 10 mm Hg ΔPCO ₂

6. **If metabolic acidosis, then look at the anion gap.**

$$\text{Anion gap} = [\text{Na}] - ([\text{Cl}] + [\text{HCO}_3^-])$$

- If elevated (> 16), then acidosis due to KULT (Ketoacidosis, Uremia, Lactic acidosis, Toxins).
- If anion gap is normal, then acidosis likely due to diarrhea, RTA.

7. **If metabolic process, is degree of compensation adequate?**

Calculate the estimated PCO₂, this will help to determine if a separate respiratory disorder is present.

8. **If anion gap is elevated, then calculate the delta-ratio (Δ/Δ) to assess for other simultaneous disorders.**

Δ/Δ compares the change in the anion gap to the change in bicarbonate.

$$\text{Delta ratio} = \frac{(\text{AG} - 12)}{24 - \text{HCO}_3^-}$$

- If ratio between 1 and 2, then pure elevated anion gap acidosis
 - If < 1, then there is a simultaneous normal anion gap acidosis present
 - If >2, then there is a simultaneous metabolic alkalosis present or a compensated chronic respiratory acidosis.
- 9. If normal anion gap and cause is unknown, then calculate the urine anion gap (UAG)**
- Urine anion gap** = $[\text{Na}^+] + [\text{K}^+] - [\text{Cl}^-]$
- In normal subjects, the urine anion gap is usually near zero or is positive.
- In RTA, UAG is positive.
 - In diarrhea and other causes of metabolic acidosis, the UAG is negative.

Effect of Temperature

When blood is cooled, CO_2 becomes more soluble reducing its PCO_2 by about 4.5% per degree celsius fall in temperature and the pH rises by about 0.015 per degree celsius fall in temperature.

Available evidence suggests that homeostatic mechanisms center around protein buffers and enzymes, specifically the alpha imidazole group on histidine residues, the so called **alpha stat** regulation. This mechanism requires that, with hypothermia the pH rises and PCO_2 falls, whereas the HCO_3^- concentration remains unchanged. **The alpha stat** concept refers to the use of 37°C temperature-uncorrected pH and PCO_2 values, whereas the **pH stat** concept refers to the use of pH and PCO_2 values corrected to the patient's core temperature.

Combination of pH stat and alpha stat is adopted in complex congenital cardiac lesions along with DHCA.

TEMPERATURE MONITORING

Cardiac anesthesia is unique in that therapeutic hypothermia is utilized frequently and aggressively in many cases. Distribution of thermal energy can be manipulated, via CPB, more rapidly and extensively than any other anesthetic situation. Unique to cardiac anesthesia are circulatory cardiac arrest procedures with cooling up to 15 to 18 degree celsius.

Temperature monitoring for cardiac anesthesia has unique considerations.

Indications

1. Assessment of cooling and rewarming.
2. Diagnosing hazardous hypothermia or hyperthermia. Below 32°C, the myocardium is irritable and subject to complex arrhythmias, especially ventricular tachycardia and fibrillation. The risk of dysrhythmia is particularly high in pediatric patients. Hypothermia inhibits coagulation thus increasing bleeding/transfusion risk. Likewise, significant enzyme desaturation and cell damage can occur with temperatures greater than or equal to 41°C.

Sites of Measurement

Numerous possible sites exist to measure temperature. These sites can be grouped into the core or the shell.

Core Temperature

- a. **General considerations:** The core temperature represents the temperature of the vital organs. The term core temperature used here is perhaps a misnomer because gradients exist even within this vessel-rich group during rapid changes in blood temperature.
- b. **PAC thermistor:** This is the best estimate of the core temperature when pulmonary blood flow is present (i.e. before and after CPB).
- c. **Nasopharyngeal temperature:** Nasopharyngeal temperature provides an accurate reflection of brain temperature during CPB. The probe should be inserted into the nasopharynx to a distance equivalent to the distance between nares to the tip of the earlobe. Nasopharyngeal temperature should be monitored in all hypothermic circulatory cardiac arrest procedures and CPB cases with hypothermia requiring rewarming.
- d. **Tympanic membrane temperature:** Temperature at this site reflects brain temperature and may provide an alternative to nasopharyngeal temperature.
- e. **Bladder temperature:** This modality has been used to measure core temperature, although it may be inaccurate in instances when renal blood flow and urine production is decreased.
- f. **Esophageal temperature:** Because the esophagus is a mediastinal structure, it will be greatly affected by the temperature of the blood returning from the extracorporeal pump and should not be used routinely for cases involving CPB.
- g. **CPB arterial line temperature:** This is the temperature of the heat exchanger (i.e. the lowest temperature during the active cooling and the highest temperature during active rewarming). During either of these phases, a gradient always exists between the arterial line temperature and any other temperature.
- h. **CPB venous line temperature:** This is the “return” temperature to the oxygenator and probably best reflects core temperature during CPB when no active warming or cooling is occurring.

Shell

- a. **General:** The shell compartment represents the majority of the body (muscle, fat and bone), which receives a smaller proportion of the blood flow, thus acting as an energy sink that can significantly affect temperature fluxes. Shell temperature lags behind core temperature during cooling and rewarming. At the point of bypass separation, the core temperature will be significantly higher than the shell temperature. The final equilibrium temperature with thermal redistribution will probably be closer to the shell temperature than the core temperature measured initially.

- b. **Rectal temperature:** Although traditionally thought of as a core temperature, during CPB procedures rectal temperature most accurately reflects muscle mass temperature. If the tip of the probe rests in stool, a significant lag will exist with changing temperatures.
- c. **Skin temperature:** Skin temperature is affected by local factors (warming blanket) and is rarely utilized in cardiac surgery.

Risks of Temperature Monitoring

Epistaxis with nasopharyngeal temperature monitoring: Most cardiac anesthesiologists consider the benefits of neurologic protection (measured by brain temperature) to outweigh the bleeding risk. If the nasal mucosa is traumatized during probe placement, epistaxis can result, especially when patient has been given heparin.

Recommendations for Temperature Monitoring

Monitoring temperature at two sites is recommended, a core site and a shell site. Arterial and venous line temperatures are available directly from the CPB apparatus. Nasal temperature monitoring is recommended for core temperature because it will most rapidly reflect the changes in the arterial blood temperature. Nasal temperature monitoring is recommended for circulatory arrest cases to document brain temperature. Bladder or rectal temperature monitoring is simple to establish and is recommended for monitoring shell temperature.

SUGGESTED READING

1. Gerhardt MA, Walosik-Arnall KM. Monitoring the cardiac surgical patient: A practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravelee GP (Eds), 5th edition. Wolter Kluwer Lippincott Williams and Wilkins, Philadelphia.
2. Reich DL, Mittnacht AJ, London MJ, Kaplan JA. Monitoring of the Heart and Vascular System, Kaplan Essentials of cardiac anesthesia, 1st edition. Saunders Elsevier.

Chapter 4

Cardiopulmonary Bypass

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INTRODUCTION

The heart-lung machine (also called as cardiopulmonary bypass [CPB] machine) is an important surgical development of 20th century, and John Gibbon deserves the major share of credit for its success. The first successful open heart surgery using cardiopulmonary bypass was done by John Gibbon on May 6, 1953. The surgery was done for the closure of an atrial septal defect. The patient, Cecelia Bavolek, was healthy on the 50th anniversary of that operation in May 2003.

Nowadays specialized personnel, the perfusionists manage the CP bypass machine. But still, the anesthesiologist has to ensure the smooth, safe pump function, provide anesthesia and coordinate with the perfusionist during weaning off bypass.

DEFINITION

CPB machine is an artificial heart and lung; in which the blood is drained from the heart into a venous reservoir (containing a priming solution), is oxygenated by an oxygenator at a desired temperature by heat exchanger and pumped back to the aorta. Cardioplegia chamber is incorporated into the machine to deliver cardioplegia and arrest the heart.

COMPONENTS OF CARDIOPULMONARY BYPASS (FIGS 1 TO 3)

The Pump

An ideal pump should:

- ❑ Move large amount of blood against lower pressure
- ❑ Prevent lysis of blood cells
- ❑ Elicit minimal inflammatory response
- ❑ Occupy less dead space
- ❑ Be equipped with safety alarms.

The pump should be able to generate flow by overcoming the resistance according to an equation—Blood flow rate = pressure/resistance.

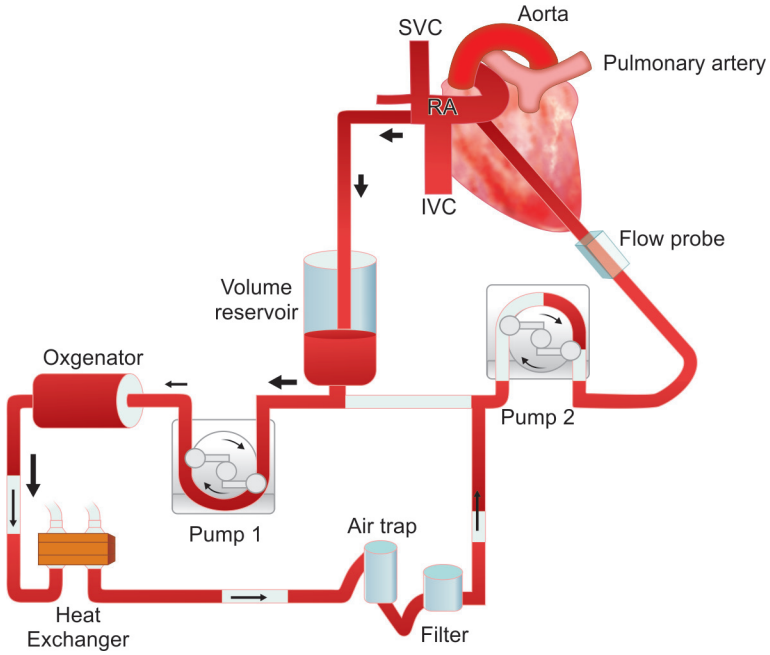


Fig. 1: Schematic diagram showing CPB components

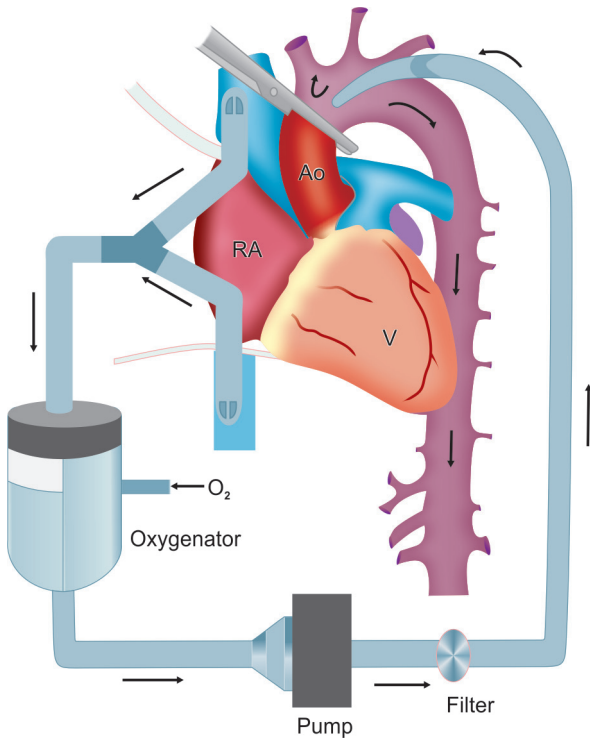


Fig. 2: Simplified diagram showing CPB components

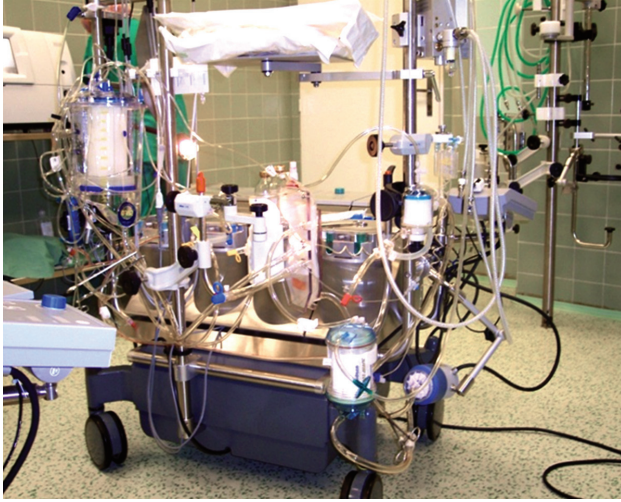


Fig. 3: CPB machine

Types of Pump

Roller pump

It consists of a tubing compressed by roller placed at 180° apart on rotating arms on a horse-shoe shaped metal backing plate (raceway). The direction of compression of the tubing by the rotating arm determines the direction of the blood flow and rollers propel the blood further (Fig. 4).

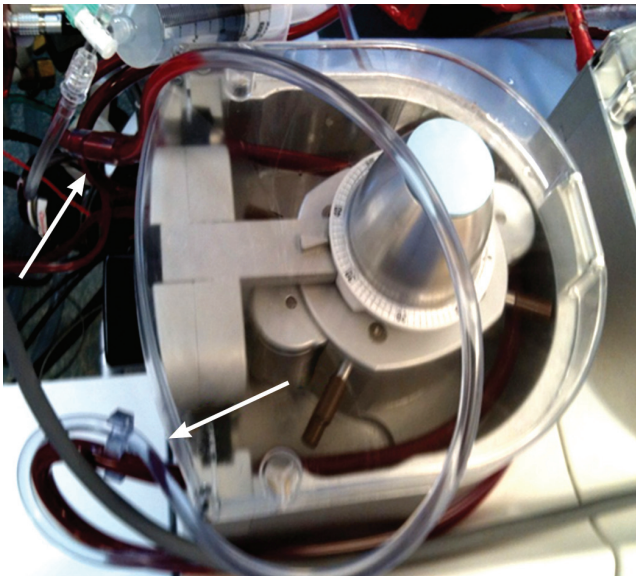


Fig. 4: Roller pump

Centrifugal pump

It consist of a nest of smooth plastic cones or a vaned impeller located inside a plastic housing. These pumps generate a pressure differential that causes the movement of fluid when rotated rapidly. Table 1 shows the comparison of roller pump and centrifugal pump.

Table 1: Comparison of roller pump and centrifugal pump

Roller pump	Centrifugal Pump
Adjustment of occlusion is required	Nonocclusive
Easy to use	Needs vigilance
Not dependent on afterload of patient	Depends on afterload of patient
Flow depends on number of revolutions, length of tube and occlusion	Flow depends on pressure and resistance
Tubing may burst due to overpressure	Maximum pressure is limited here
More risk of embolization	Risk of embolization is less
More hemolysis and platelet destruction	Less hemolysis, platelet structure and function retained, less inflammation

Venous Reservoir

- The deoxygenated blood is drained into a collecting chamber called as the venous reservoir with a capacity of 1–3 lit. It is a low pressure system in which the blood can flow by virtue of gravity (siphon effect); however, kinetic assisted venous drainage and vaccum assisted venous drainage are also practised to augment the venous return (Fig. 5).

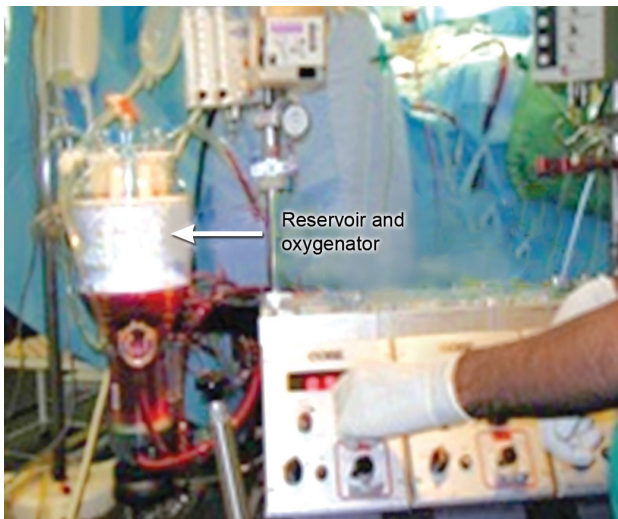


Fig. 5: Venous reservoir with oxygenator

- A reaction time of 10 seconds in case of sudden fall in venous return allows the perfusionist to avoid dry pumping and prevent massive air embolism. Defoaming agent is added to retard the formation of air bubbles.

- ❑ Hard shell plastic open types of reservoirs are easy to operate, can store good amount of volume but the risk of air embolism is high. There is less incidence of blood cell activation, blood loss and blood administration with the closed type. Soft disposable bag adds to the cost, but sterility is better maintained and the risk of embolism is less.

Oxygenators

- ❑ It is also known as artificial lung. Its main function is to oxygenate venous blood and remove carbon dioxide. The physical process of convection, diffusion and chemical reactions that occur during gas exchange in the natural lung also apply to the artificial lung. However, gas exchange is less efficient (Fig. 6).

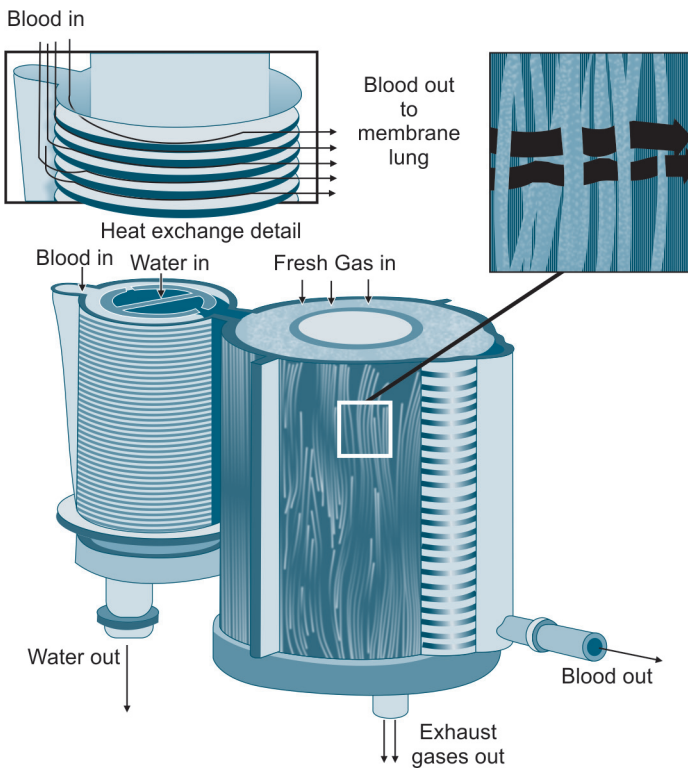


Fig. 6: Oxygenator and heat exchanger assembly

Types of Oxygenators

Bubble oxygenator

It uses a direct blood gas interface, i.e. gases are passed over the blood, time is allowed for the gas exchange and then defoaming of the oxygenated blood takes place before it is returned to the patient. The smaller the bubble size greater is the surface area available for the exchange of gases. Air embolism is a significant risk with the bubble oxygenator. Several studies have proven the efficacy of

membrane oxygenator over bubble oxygenator in terms of lung and brain protection. Hemolysis, and a rise in haptoglobin is also seen in certain cases.

Membrane oxygenator

The diffusion of gases takes place across the membrane, which separates the blood and the gas phases. Microporous polypropylene membranes are commonly used nowadays as they are biocompatible with good exchange capacity. These consist of microhollow fibers. Diffusion distance is approximately 200 μm in comparison with 10 μm in the human alveolus, surface area for gas exchange is 1.7–3.5 m^2 compared with 70–100 m^2 in the human lung. The gas transfer in membrane oxygenator depends upon partial pressure of venous oxygen and carbondioxide, blood flow, ventilation flow (sweep rate), and the gas composition. pO_2 is a function of FiO_2 while pCO_2 depends on sweep rate.

$$\text{Volume of gas transferred } (\alpha) = \frac{\text{Area} \times \text{diffusion constant} \times \text{partial pressure difference}}{\text{Membrane thickness}}$$

Heat Exchanger

- The temperature range on CPB is 4°C to 42°C. Its basic structure consists of two parallel tubings where the perfusate passes in one tube and the hot or cool water in the other. Temperature regulation is done by conduction method. Heat exchanger is commonly placed proximal to the oxygenator.
- Suction ports, filter, ports to inject drugs, and supply gases are also present.

Venous Cannulae

- It drains the deoxygenated blood from right atrium to venous reservoir.
- Single stage cannula: It is inserted in the right atrium, has multiple holes that drain blood from SVC, right atrium, IVC and the coronary sinus. It is performed during on pump CABG, where right atrium is not opened (Fig. 7).

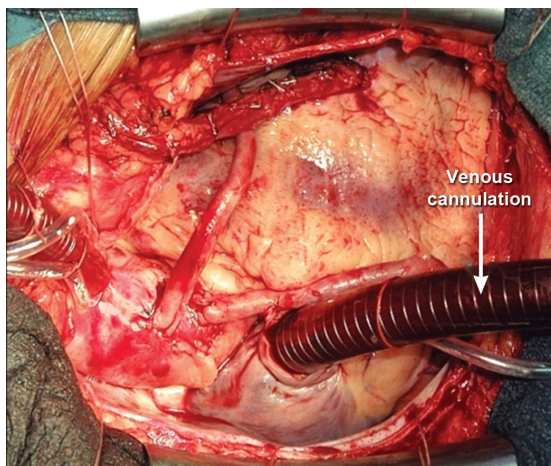


Fig. 7: Single stage venous cannulation in situ

- ❑ Two stage cannula: The superior vena cava and the inferior vena cava are cannulated separately. Both vena cavae are looped and snugged to divert the blood into the venous reservoir. It is mandatory in procedures where the right atrial access is essential, e.g. ASD repair. The coronary sinus is not drained; so an extra vent is needed to drain it.
- ❑ Femoral vein cannulation is done when patient needs to be put on CPB prior to sternotomy, e.g. redo surgery, or aortic aneurysm due to fear of sudden catastrophic hemorrhage. The cannula is long and has multiple holes, however, the venous drainage may not be complete. TEE helps in the assessment of proper placement.

It is important to check with the perfusionist if the venous return is appropriate because inadequate drainage needs readjustment of the cannulae position.

Arterial Cannulation

- ❑ Arterial cannulation is the first thing the cardiac surgeon does after heparinization when target ACT (more than 400 seconds) is achieved.
- ❑ The aortic cannulation is done first to provide volume resuscitation if hypotension occurs due to major bleeding.
- ❑ The systolic blood pressure is generally maintained between 90–100 mm Hg during cannulation to avoid aortic dissection and this is achieved by additional doses of anesthetic agent or a vasodilator.
- ❑ Very low blood pressure can cause injury to the posterior wall of an aorta during cannulation due to relatively collapsed aorta. Potential complications associated with the aortic cannulation include embolization of atheromatous debris or air, inadvertent cannulation of aortic arch vessels and aortic dissection. TEE easily diagnoses the calcification and dissection of the ascending aorta. After aortic cannulation the aortic line is deaired prior to unclamping and starting the pump.
- ❑ Peripheral cannulation: Femoral arterial cannulation is done along with femoral vein in peripheral bypass. Even axillary artery can be cannulated.

Connecting Tubings

Disposable polyvinyl chloride tubings of various diameters are used to set up the pump connection with the patient.

Diameter of tubing	Volume of blood
3/6 inch	7 mL
1/4 inch	13 mL
3/8 inch	27 mL
1/2 inch	54 mL

CONDUCT OF CARDIOPULMONARY BYPASS

Setting up of the CPB Machine

Priming

The deairing of the CPB circuit is done by priming volume. The priming solution is selected according to the body surface area, hematocrit, and the volume required.

- ❑ Cardiac index of 70 kg man with normal metabolism at 37°C is 2.4 L/m²/min; 2.1 L/m²/min at 32°C, 1.8 L/m²/min at 28°C, 1.2 L/m²/min at 25°C, 0.6 L/m²/min at 15°C.
- ❑ Blood flow rate = Body surface area × cardiac index
- ❑ Target hematocrit = 21-24% in adults and 28-30% in children is accepted
- ❑ Target hematocrit on CPB =
$$\frac{\text{Patients blood volume} \times \text{Hct}}{\text{Total circulating volume of pump}}$$
- ❑ Total circulating volume (TCV) = Patient blood volume + priming volume + cardioplegia solution volume
- ❑ Blood required on prime =
$$\frac{(\text{Target Hct} \times \text{TCV}) - (\text{Hct} \times \text{PBV})}{\text{Hct of donor blood}}$$

Priming solution

- ❑ An ideal priming solution should be iso-osmolar to the plasma with similar oncotic pressure, should be easily excreted, should result in less hemolysis and not produce any cellular edema in the post-bypass period.
- ❑ Priming results in hemodilution which improves the microcirculation, and improves oxygen delivery and perfusion even in the setting of hypothermia. Very high pressures would be needed to propel the viscous blood because hypothermia increases the viscosity of blood. But the perfusion with low and nonpulsatile flow is possible due to priming induced hemodilution. However, excessive hemodilution causes edema due to extravasation of fluids into the third compartment.
- ❑ Crystalloids, colloids, albumin, blood and plasma are popular additives used for priming.
- ❑ Balanced salt solution is a fluid of choice for priming. Crystalloid solutions are also considerably cheaper than colloids and are free of the risk of anaphylactoid reactions, but their main drawback is the inability to maintain the oncotic pressure leading to formation of interstitial and intracellular edema. Improved postoperative pulmonary and renal function using crystalloid priming solution alone has been observed in several studies.
- ❑ Colloid: Colloids like gelatins, hydroxy ethyl starch (pentastarch, hetastarch), voluven (130/0.4), and albumin are used. The advantage of colloid is maintenance of oncotic pressure; but additional adverse effects on blood coagulation and the occasional occurrence of allergic reactions have raised questions about their suitability as priming fluids.
- ❑ Blood and Blood products: Homologous blood use continues to be the gold standard for priming in infants and neonates despite exposure of the patient to potential cellular and humoral antigens.

Low hematocrit (Hct < 20) during cardiopulmonary bypass (CPB) is associated with higher mortality and other adverse outcomes. Low Hct is

encountered in patients with small body size and in females. Hence, an aggressive approach in the care of these patients is needed. It is prevented by the use of a low-prime CPB circuit (LP) for all adult patients with a body surface area (BSA) $<1.7 \text{ m}^2$ and use of autologous circuit priming (AP), in addition to the low-circuit volume in some patients.

Setting the Occlusion of the Pump

To ensure accurate delivery of systemic blood flow, an outflow tube is held at 30–40 cm height and occluded to hold the fluid in place and then a gradual drop is allowed at the rate of one inch/minute.

During induction of anesthesia the CPB machine should be ready in case of any emergency situation. Along with resuscitation, the patient can be heparinized and immediately put on bypass.

- ❑ Post-induction, adequate depth of anesthesia is maintained by mounting inhalational agents on bypass. Infusion of anesthetic agents can be used.
- ❑ During sternotomy, patient is disconnected from the ventilator or switched over to low tidal volume ventilation to prevent lung injury.
- ❑ After 3 minutes of heparinization ACT is done. During this period surgeon checks the lines for air bubbles.
- ❑ After adequate anticoagulation the internal suckers are used. Before the aorta is cannulated the systemic pressure is lowered to 100 systolic mm Hg. The line pressure is checked by the perfusionist to confirm lack of resistance. Any resistance raises the suspicion of aortic dissection or cannula malposition. Aortic cannula can be malpositioned in such a way that the jet is directed primarily into the innominate artery, the left common carotid artery (rare) or the left subclavian artery (rare). The latter two can occur with the use of long arch-type cannulae. In the first two circumstances, unilateral cerebral *hyperperfusion*, usually with systemic hypoperfusion, occurs, whereas flow directed to the subclavian artery results in global cerebral *hypoperfusion*. However, transcranial Doppler, and more commonly available cerebral oximetry, is the monitor of choice to detect malperfusion secondary to cannula.
- ❑ As per the requirement, a single caval or bicaval cannulation is done. Adequate venous return is checked by the perfusionist, which should generate blood flow rate of 2.4 L/min/m^2 at a line pressure of 150–200 mm Hg. Poor venous return can occur due to air lock, which is corrected by removal of air. Raising the height of the table aids venous drainage by gravity.
- ❑ Gradually the venous blood is completely drained. Ventilation is stopped once the patient is on complete bypass (perfusionist informs full flows). Normothermic or hypothermic perfusion is maintained.
- ❑ Cardioplegia cannula with a vent is inserted in between aortic root and the cross clamp. Regardless of the cannulation technique selected, some blood

returns to the left ventricle via Thebasian veins, bronchial veins, shunts, aortic insufficiency etc. An LV vent is required in order to prevent warming and distension of LV. Additionally, the bicaval cannulation technique requires the use of a second vent to capture coronary sinus blood that bypasses the IVC and SVC cannulae.

- ❑ Aorta is cross clamped before giving cardioplegia. The duration of cross clamp is noted.
- ❑ All standard monitoring is continued, ET tube is disconnected, and IV fluids and infusions are stopped.

Besides standard monitoring, urine output and temperature at two different sites are meticulously noted.

CONCERNS DURING BYPASS

Temperature

Hypothermic CPB

The temperature is maintained below 34°C (34–28°C). The temperature is gradually reduced. The advantages of hypothermia are:

- ❑ It reduces the oxygen requirement by decreasing the metabolic rate; cerebral metabolic rate is decreased 6-7% per degree decline in the temperature.
- ❑ Flow rate of pump can be lowered in hypothermia due to decreased requirement.
- ❑ A low flow rate protects the cells from trauma.
- ❑ Better organ protection is seen with hypothermia, especially brain and kidney.

Ability of the hypothermic kidney to handle glucose is impaired, and glucose often appears in the urine. Hemodilution in combination with hypothermic CPB improves renal blood flow and protects the integrity of the renal tubules postoperatively.

Hypothermia causes adverse effects on enzyme cascade, delays repair, increases intracellular swelling due to sodium absorption, causes arrhythmia, impairs coronary circulation, inhibits sarcoplasmic reticulum and calcium accumulation.

Normothermic CPB

The temperature is maintained above 34°C. It ensures adequate perfusion, as demonstrated by normal renal function, normal lactates, normal pH, and the high mixed venous saturation during bypass. Normothermic bypass does not precipitate volume overload and high doses of adrenergic drugs are not required during and after CPB. But, continuous cardioplegia delivery is required that keeps the surgical site flooded and causes technical difficulty.

Normothermic bypass narrows the gap between oxygen demand (VO_2) and oxygen delivery (DO_2).

Current evidence suggests that maintaining normothermia during cardiopulmonary bypass in adult cardiac surgery is as safe as hypothermic surgery, and associated with a reduced risk of allogeneic blood transfusion.

Glucose

Hyperglycemia increases the risk of morbidity and mortality in cardiac surgery patients. A tight glucose control is known to reduce multiorgan complications. It lowers the incidence of wound infections, reduces hospital stay and enhances long-term survival.

Glycemic Control in Patients without Diabetes During CPB

Intraoperative glycemic control using intravenous insulin infusions is not necessary in patients without diabetes provided glucose values remain < 180 mg/dL. All patients should be given continuous infusion rather than bolus doses (Level A evidence). Blood glucose level should be maintained below 180 mg/dL for at least 24 hour postoperatively (Level B evidence). The intermittent rise in blood glucose can be treated with bolus dose of insulin, if high dose infusion is needed.

Patient with DM

Patients taking medium to long-acting insulin should stop insulin after dinner the night prior to surgery (level B). Insulin therapy should be initiated prior to elective surgery (level C) and blood glucose should be maintained below 180 mg/dL (level B) while the glycosylated Hb level should be $\leq 7\%$.

Intraoperative elevated glucose levels must be corrected by insulin infusion and the infusion should be continued in the postoperative period. Glucose monitoring should be done every 30–60 minutes during tight control regimen.

pH

The pH-stat and alpha-stat refer to the acid-base management strategies at different body temperatures during CPB. The solubility of CO_2 in the blood increases as the body temperature decreases.

pH Stat

pH is kept constant. Alkalosis occurs as the temperature decreases and it is corrected to desired pH by adding CO_2 to the pump. CO_2 causes cerebral vasodilation and increases blood supply to the brain. Faster cooling can be achieved with this technique, especially in DHCA. The incidence of air embolism also increases. Blood flow becomes pressure dependant as the

cerebral autoregulation is lost. Hoover et al. suggested that in adults with high risk of impaired cerebral blood flow (i.e. aged >70 years old, diabetic, prior stroke, uncontrolled hypertension) pH-stat maintains higher oxygen tension and saturation during CPB.

The α Stat

The amino acid histidine (containing an alpha-imidazole ring with many negatively charged moieties) is the most important buffer. The “alpha” in the term alpha-stat, which denotes uncorrected blood gas measurement, refers to the α -imidazole ring of histidine. Alpha-stat strategy indicates a pH management strategy in which the pH is not corrected and the blood carbon dioxide is allowed to follow its thermodynamically mediated dissociation changes with hypothermia, which results in a decreased hydrogen ion concentration (decreased dissociation) and increased blood pH (alkaline shift). This means in clinical practice no carbon dioxide is added to the oxygenator gas to compensate for these changes in blood pH during cooling. Alpha-stat blood gas management maintains autoregulation, and limits cerebral microemboli load by control of cerebral blood flow and demand. The cellular transmembrane pH gradient and protein function is also maintained. Disadvantages of alpha-stat include less efficiency in homogeneous cooling.

Perfusion Pressure

The mean arterial pressure maintained during bypass is 60–80 mm Hg. The most important function of cardiopulmonary bypass is maintaining perfusion of the vital organs. Autoregulation of most organs is preserved above 50–60 mm Hg. Low-risk patients tolerate mean arterial blood pressures of 50–60 mm Hg without apparent complications, although limited data suggest that higher-risk patients (CVA, chronic hypertension, severe atherosclerotic disease) may benefit from mean arterial blood pressures >70 mm Hg. Advantages of high MAP include better perfusion, opening up of collaterals, and high pump flow rate. Excessive arterial pressure increases noncoronary collateral flow to the heart during aortic cross-clamping (hence “washing out” myocardial protection with cardioplegia) and bronchial flow to the lungs (increases blood return to left heart) and places strain on arterial clamps and suture lines. With a lower MAP there is less trauma to cell, lower pump flow less blood in surgical field, enhanced cardioprotection and less embolic load to CNS.

Adequate MAP is achieved by maintaining flow rate on the pump which is equal to cardiac index in normothermic anesthetized person at normal hematocrit. Proposed advantages of reduced flow rates include less hypertension during hypothermic bypass (due to increased blood viscosity and temperature-induced increases in systemic vascular resistance), improved intracardiac exposure due to less bronchial blood flow returning to the left heart, and reduced warming of the myocardium via noncoronary collateral vessel. During CPB, pump flow and pressure are related through overall

arterial impedance, a product of hemodilution, temperature, and arterial cross-sectional area. This is important because the first two factors, hemodilution and temperature, are critical determinants of pump flow requirements. Pump flows of 1.2 L/min/m² perfuse most of the microcirculation when the hematocrit is near 22% and hypothermic CPB is being used.

Maintenance of Anesthesia

The oxygen and air attached to the bypass machine are started. The flow of gases is adjusted to meet the oxygenation and removal of carbon dioxide. The PO₂ depends on the FIO₂, while the CO₂ wash out is aided by increasing the sweep rate.

The incidence of awareness in patients undergoing cardiac surgery has been reported to be 1.5–23%. Possible reasons for this are, the widely used high opioid based techniques, the almost unpredictable pharmacodynamics of anesthetics under the extracorporeal circulation especially in the rewarming period and at the time of cessation of bypass, interpersonal and interracial differences in drug reactions, hemodilution, and binding on foreign surface areas.

Anesthetic depth should be sufficient (a) to prevent awareness (as assessed ideally by periodic tests of responsiveness, e.g. requesting patient to open eyes), (b) to prevent spontaneous movement including breathing, and (c) to suppress hypertensive or tachycardic responses to surgical stimuli.

Nowadays vaporizers are available which can be fixed to oxygenator gas inlet. Sevoflurane and isoflurane are commonly used, and are known for their preconditioning effect. Nitrous oxide is generally avoided to prevent enlargement of air in case of an embolism.

The IV anesthetic agents are given in the form of bolus doses or by infusion in venous reservoir. Hypothermia itself reduces minimal alveolar concentration and neuronal activity, additional anesthetic drugs are most commonly required during warm periods at the beginning and end of CPB.

METHODS OF MYOCARDIAL PRESERVATION

- A. Cardioplegia
- B. Ischemic preconditioning
- C. Preventing left ventricular distension.

Cardioplegia

- ❑ Cardioplegia solution which perfuses the myocardium after clamping of aorta is used to stop the heart and enable surgery.
- ❑ The metabolic oxygen requirement is reduced by hypothermia and hyperkalemic arrest (soft heart). It is a myocardial preservation technique. Oxygen consumption of beating empty heart at 37°C is 5.5 mL/kg/min. while with K⁺ cardioplegia at 22°C is 0.3 mL/kg/min.
- ❑ Cardioplegia is delivered by a separate pump head at 80 to 100 mm Hg, about 20 mL/kg.

- ❑ The resulting increase in extracellular $[K^+]$ decreases the transmembrane potential. This progressively interferes with the normal Na^+ current during depolarization, decreasing the rate of rise, amplitude and conduction velocity of subsequent action potentials. Eventually the Na^+ channels are completely inactivated, AP's are abolished and the heart is arrested in diastole.
- ❑ Fibrillation and hypercalcemia (systolic arrest) are the other techniques of stopping heart.
- ❑ Components of cardioplegia solution: Blood—provides oxygen. KCL (k—20-30 mEq)—diastolic arrest, $NaHCO_3$ (Na—109 mmol/L, bicarbonate—27 mmol/L)—provides buffer, mannitol (54 mmol/L)—prevents edema, procaine—abolishes vasoconstriction. Aspartate and glutarate are amino acids, Glucose—28 mmol/L, Chloride—114 mmol/L.
- ❑ Blood and cardioplegia solution are mixed in ratio of 4:1, at temperature of 8–10°C and is repeated every 20 minutes, or if there is return of activity. Hotshot the normothermic cardioplegia is given prior to coming off bypass at temperature of 37°C. Normothermia maximally enhances myocardial aerobic metabolism and recovery after an ischemic period.
- ❑ The acceptable perfusion pressure to limit perivascular edema and hemorrhage needs to be less than 50-60 mm Hg. Antegrade delivery is by coronary route, Problem is in severe aortic insufficiency or during aortic root or aortic valve or severe coronary artery disease. Retrograde delivery is through coronary sinus. With retrograde cardioplegia, the delivery is good to left ventricle (by the thebesian and artriovenous communication) but poor perfusion to right ventricle (the perfusion pressure is 40 mm Hg to prevent edema).
- ❑ Delivery is done by cardioplegia cannula inserted proximal to the aortic cannula, while in surgery at aortic route two hockey shaped cannulae are inserted in the right and left coronary.
- ❑ Del nido—This cardioplegia is generally given as a single 20 mL/kg dose antegrade at 8–12°C through a recirculating delivery system. The unique formulation reduces energy consumption, blocks calcium entry into the intracellular environment, scavenges hydrogenions, preserves high-energy phosphates, and promotes anaerobic glycolysis during myocardial arrest. Mannitol 20%, 16 mL, magnesium sulfate 50%, 4 mL, sodium bicarbonate 8.4%, 13 mL. Potassium chloride (2 mEq/mL), 13 mL, Lidocaine 1%, 13 mL.
- ❑ Complications—Injury can occur to coronary vessels and especially the thinned wall coronary sinus. Ischemic changes due to unequal distribution can occur.

Ischemic Preconditioning

Ischemic preconditioning (IPC) is endogenous myocardial protection triggered by exposure to brief (5 to 15 minutes) periods of ischemia. IPC is a natural defense mechanism that permits the heart to better tolerate myocardial ischemia. Mechanism of pre-conditioning is very complex, and

involves activation/translocation of PKC, tyrosine kinases and mitogen—activated kinases. Isoflurane and sevoflurane aid in ischemic preconditioning. Preconditioning helps by reduced infarct size—less generation of lactate—decreased rate of fall in ATP while arrhythmias and contractile dysfunction are decreased.

Venting of Heart

The purpose is to prevent heart distension, reduce myocardial rewarming, prevent cardiac ejection, and provide a clear field to operate. Blood comes to left ventricle from thebesian veins, and bronchial veins as well as blood that gets away from venous cannula. Left ventricle distension can be noted visually, and also by use of TEE. Venting can be done by aortic root cardioplegia cannula, right superior pulmonary vein is usually used. Suction is applied to vents to empty the heart. Complications like bleeding, trauma to intima of the chamber, and air trapping can occur.

POST BYPASS

Weaning from CPB entails the progressive transition of the patient from full mechanical circulatory support to spontaneous heart activity of the patient with an aim to provide sufficient blood flow and pressure through the pulmonary and systemic circulation.

Before initiating the weaning procedure, several prerequisites should be routinely met:

- ❑ Normothermia is achieved by active rewarming by using CPB heat exchanger, by convective air circulation, and by circulating water blanket.
- ❑ Arterial blood analysis to ensure that oxygen content of the blood (hematocrit > 25%, PaO₂ > 100 mm Hg), electrolytes (K⁺, Ca⁺⁺, Mg⁺⁺), blood sugar, and pH are within normal limits; while full anticoagulation is maintained (activated clotting time > 400 s).
- ❑ Lungs are manually re-inflated (FIO₂ > 0.8). Mechanical ventilation is reset, and the alarms of the cardiopulmonary monitoring are reactivated.
- ❑ After aortic unclamping and electrical ventricular defibrillation (if required), a heart rate (HR) between 70 and 100 beats/min should be targeted. Bradycardia and atrioventricular blockade are treated with atropine, beta-adrenergic receptor agonists, or cardiac pacing.
- ❑ The insertion of an intra-aortic balloon pump (IABP) should be considered in patients with ongoing myocardial ischemia or unstable hemodynamic condition. Indeed, by reducing LV afterload and improving diastolic coronary blood flow particularly in subendocardial area, IABP exerts anti-ischemic myocardial effects and increases systemic oxygen delivery.

Temperature

As nasopharyngeal temperature reaches 36.5 to 37° (C), rectal temperature is usually two to three degrees lower. More invasive surrogates of brain

temperature have been obtained using a jugular bulb thermistor. A larger than four degrees gradient between the nasopharyngeal and rectal temperatures is indicative of inadequate rewarming or increased vasoconstriction. In these situations there may occur a two to three degrees decrease in nasopharyngeal temperature during sternal closure and transfer to intensive care unit, which may predispose the patients to unstable cardiac rhythm, shivering, and hypertension. A slow infusion of nitroprusside or other vasodilators may provide a more homogenous rewarming and reduce the occurrence of significant temperature gradients. The rewarming should be gradual as studies have shown that embolism and reperfusion injury is less by gradually bringing up the temperature, hence temperature gradient between the oxygenator and the patient should not be more than 10°C.

Rate and Rhythm

A rate of 80–100 /min with sinus rhythm is ideal to wean off bypass, sometimes atrial fibrillation can be seen which needs cardioversion. Ventricular tachycardia and ventricular fibrillation will require internal cardioversion 20–40 joules. In case of sinus tachycardia rule out common causes like anemia, hyperthermia, hypercarbia, hypovolemia, light plane of anesthesia. Bradycardia is treated with atropine or isoprenaline infusion, common practice is to do epicardial pacing.

Cardiac Output

Simple visual observation of heart contraction and relaxation may provide valuable information about myocardial performance. The cardiac rhythm and the adequacy of ventricular rate are evaluated by the electrocardiogram monitor tracing. CPB exposes the myocardium to some amount of dysfunction and injury during aortic cross clamp, improper perfusion and even reperfusion injury. TEE is a boon to diagnose right and left ventricular dysfunction as well as to identify the ischemic segment. Myocardium is accordingly supported with inotropes.

Coagulation

Activation of coagulation system due to contact with artificial surface, surgical trauma, high doses of heparin, and hypothermia are triggers of a coagulopathy leading to excessive bleeding. Excessive heparin, redo surgery, prolonged duration, platelet activation and dysfunction and inadequate protamine administration are contributory factors. The ACT is checked hourly and prior to coming off bypass. After the pump blood is over a test dose of protamine is given, the ratio of heparin protamine reversal is 1:1.3. Protamine is allergenic and even anaphylaxis can occur. Commonly a drop in blood pressure is noted. Protamine is given gradually over 10–15 minutes, and then ACT is checked. If ACT is less than 180, aortic cannula is removed.

Hematocrit

Hematocrit values from 20 to 25% are usual with most perfusion protocols. By the end of rewarming, depending on renal function and the use of diuretics hematocrit may reach 24 to 30%. Ultrafiltration also helps to decrease the inflammation and build up hemoglobin.

Blood Gas

Rule out acidosis as it impairs myocardial function as well as the action of inotropes.

Resumption of Ventilation

To start ventilation before the patient is off, bypass is an important step. Normal rhythm and pulsatile flow through the bronchial artery is the time when ventilation can be started. Generally, it is started just before some load is given to the heart.

Potassium

Hyperkalemia causes rhythm disturbances and is seen if the cardioplegia is given before coming off bypass, or in case of renal hypoperfusion. It can be treated with glucose insulin drip. Hypokalemia leaves the heart irritable and is treated if less than 3.5 mEq/dL.

Calcium

Is important for contractility and conduction of myocardium. Children with congenital heart disease with failure to thrive are generally hypocalcemic. Monitoring of ionised calcium is very important. However excess calcium is known to cause coronary spasm, reperfusion injury, and inhibit the effect of inotropes at receptor level. Monitors and alarms are restarted.

SEQUENCE OF EVENTS FOR BYPASS

1. Warm cardioplegia is given and patient is warmed gradually, return of electrical activity is noted. The myocardium is given a rest time to resource and compensate for the myocardial injury which is sustained during the bypass period.
2. Deairing—The venous line is partially occluded and some load is given to the heart, venous blood passes from right side of heart to the lungs. The air trapped in pulmonary veins and RA and RV reaches the LA and LV. Surgeons with gentle massage get the air out through the cardioplegia cannula. Trendelberg position helps the bubbles to rise up and exit through the cannula.
3. Unclamping of aorta—After adequacy of deairing aortic cross clamp is removed. Ventilation is resumed.

4. Heart starts filling and starts contracting. Gentle massage can be given to aid contractility, and inotropes are added as per need.
5. Venous occlusion is increased further and blood flow into the heart increases. The flow of the pump is gradually lowered titrating the blood pressure judging the contractility and the resistance of the peripheral vasculature.
6. If there is LV distension that means the heart is impaired to take the load. Surgical trauma, embolization in coronary vessels, stunned myocardium, reperfusion injury and inflammation are the leading causes of ventricular dysfunction. It may be needed to go back on bypass, rest the myocardium, give cardioplegia to the vessels in case of ST-T changes, and institute inotropic support.
7. When the heart resumes the function of contractility patient is taken off bypass. Ventilation and maintenance of anesthesia is continued. The blood is returned to patient slowly.
8. Protamine reversal—After the patient is off bypass, heparin is reversed with protamine. Heparin: protamine ration 1:1.3. Aortic cannula is removed.
9. Maintenance of anesthesia—Rewarming is the stage where the incidence of awareness is high. Extra doses of relaxant, analgesic, and hypnotic agents are given.
10. Sternum is closed, transducers are fixed, invasive lines are taped, and the patient is shifted to ICU on appropriate inotropic support.

COMPLICATIONS OF CPB

1. Venous cannulation—bleeding due to trauma to the vessel. Inadequate venous return is a problem due to improper placement, inadequate height of patient and air lock.
2. Arterial cannulation—Trauma to vessel, bleeding, inability to cannulate, unilateral perfusion due to cannula misplacement, dislodgment of plaque. Aortic dissection is catastrophic. The treatment is to immediately cool the patient, institute circulatory arrest, and cannulate below the dissection.
3. Massive air embolism—The treatment is to stop the pump, start retrograde cerebral perfusion, and arrest the heart. Hyperbaric oxygen is the treatment.
4. Bleeding disorders—Platelets are damaged due to sheer, turbulence and stress of flow through CPB circuit. Structural changes, alpha granule depletion, decrease in ADP and thromboxane activity, hypothermia and inflammatory process leading to destruction of platelets all contribute to bleeding post-bypass.

The concentration of coagulants decreases due to hemodilution. The levels of factor 8, ristocin, vWF are suboptimal due to decreased production. Hemodilution, inflammation and complement activation stimulate the coagulation pathways leading to fibrin formation.

Bleeding is greater in prolonged bypass time, protamine excess, redo surgery, use of pre-operative coagulants, and valve surgery.

5. Renal dysfunction—Kidney is the commonest organ to suffer insult especially if there is pre-existing renal disease. AKI is a major consequence of systemic inflammatory response syndrome. Sepsis is a strong predictor of postoperative AKI, likely mediated through the effects of renal inflammation. Inadequate perfusion in low output state, prolonged bypass time, sepsis, uncontrolled diabetes with high glycosylated Hb are risk factors for kidney disease. Treatment includes pre-operative measures to prevent kidney damage, maintenance of high perfusion pressure, use of N-acetyl cysteine, steroids and calcium antagonist. Renal replacement therapy is required in certain patients.
6. Cerebral dysfunction postbypass includes a spectrum from neurocognitive disorders to stroke. Artheromatous arteries, old age, prolonged bypass time, DHCA, prior history of CVA, arrhythmias, uncontrolled hypertension, sepsis, emboli from artheroma, air embolism and SIRS are all factors responsible for CVA. Strategy to reduce cerebral dysfunction includes maintenance of higher perfusion pressure above the autoregulation threshold, adequate hematocrit, and alpha stat management.
7. Inflammation—Contact of the blood components with the artificial surface of the bypass circuit, ischemia-reperfusion injury, endotoxemia and operative trauma are all possible causes of SIRS. Acute phase reaction is initiated by stimulation and release of:
 - i. Complement activation—Classical and alternative pathway get stimulated. - C3a and C5a anaphylotaxin are secreted. This is normally taken care of by Cofactor which is secreted by endothelium. It is lacking on bypass.
 - ii. Cytokines—They are secreted by tissue in response to injury and include interleukins, tumor necrosis factor, etc. They are proved to cause myocardial depression (TNF alpha, Il1beta). TNF alpha is also known to cause fibrin deposits in kidney. and 'pulmonary edema, by increasing systemic vascular permeability along with nitric oxide.
 - iii. Endotoxins released by bacteria especially in gut due to hypoperfusion and ischemia, along with proinflammatory substances cause toxemia.
 - iv. NO—Cytokines and endotoxins can induce the release of NO by endothelium and smooth muscle cells through the inducible form of the enzyme NOS. It is known to increase vascular permeability (wet lung).
 - v. Ischemiareperfusion injury—occurs at aortic cross clamp. Rewarming can cause stress response and release of inflammatory products.

Steroids, are known to decrease inflammation to some extent. Aprotinine a serine protease inhibitor is known to decrease mediators of contact pathway of coagulation. However, it is less in practise today because of allergic reactions.

Tranexamic acid is proved to decrease bleeding. Antioxidants are also used.

8. Cardiac dysfunction and rhythm disturbances—Rhythm disturbances are very common postbypass, AF, SVT, VT, VF can occur. Optimization of electrolytes, temperature and pH helps. Subclinical myocardial injury with inadequate substrate to the myocardium can occur due to cross clamping of aorta, calcified vessels, and decreased distal supply in coronary bypass patients. Stunning of the myocardium is responsible for immediate dysfunction. Factors that contribute to stunning include not only the metabolic consequences of oxygen deprivation but also the premonitory condition of the myocardium, reperfusion injury, acute alterations in signal transduction systems, and the effects of circulating inflammatory mediators.
9. Electrolyte disturbances
10. Lungs—With techniques of fast tracking minor pulmonary dysfunction can affect the outcome of patients. ARDS and wet lung due to inflammatory process (complement activation and neutrophil sequestration) is seen to some extent in many patients. Anesthesia related issues of atelectasis, and reduced mucociliary clearance, all contribute and require lung protection strategy.

Atelectasis and pleural effusions are the most common pulmonary abnormalities seen after cardiac surgery, presenting in more than 60% of patients.

PEDIATRIC CARDIOPULMONARY BYPASS

Salient features that differentiate pediatric CPB from adult CPB are:

1. It is technically more complex and requires greater skill.
2. The arterial and venous cannulae are smaller in size. The chances of tear in the vessel (thin walled and small calibre vessels) are higher during cannulation.
3. Priming solution causes hemodilution up to 200% in neonates as compared to 25–33% in adults. Even with low volume circuits, the dilution up to 150% is evident. The priming fluid usually chosen is blood, packed cells, fresh frozen plasma or albumin to maintain the hematocrit of 20–25%. In complex congenital heart disease hematocrit of 35–40% is acceptable during CPB. Excessive hemodilution is known to cause wet lung. Steroids and mannitol are added to priming solution to decrease the inflammatory response and cause diuresis respectively. Miniature circuitry is commonly practiced now in which pump prime is just enough to fill the dead space i.e. (i) decreased size of venous reservoir (dead space 150–180 mL), (ii) small size oxygenator, (iii) narrow and short tubings, (iv) Ultrafiltration
4. Temperature—Complex congenital heart surgeries require a still heart and the vessels may need to be resected and resutured (e.g. TGA) in which a circulatory arrest is needed.

Deep hypothermic circulatory arrest (DHCA)—Temperature is dropped below 20°C for 45-60 minutes. Hypothermia leads to increase in the viscosity of the blood hence hemodilution is done. Anerobic metabolism and accumulation of waste products is a hazard of reduced perfusion to vital organs due to circulatory arrest. Cerebral oxygen delivery is an especially important consideration, since cerebral autoregulation is impaired during DHCA. Various pharmacological agents like methyl prednisolone 10 mg/kg, thiopentone sodium (15-50 mg /kg which results in isoelectric line in EEG), and mannitol to prevent edema are used. Cooling of brain by ice on forehead and around both carotids is done to reduce the cerebral metabolism. The benefits of retrograde cerebral perfusion are- uniform cerebral cooling, wash out of air bubbles, delivery of oxygen and nutrients to the brain, and washout of metabolic wastes. A higher Q^{10} in infants suggests a greater metabolic suppression related to hypothermia, which might enable them to tolerate longer periods of “imperfect” perfusion or ischemia. During CPB, as the temperature of the patient decreases, O_2 consumption becomes independent of the flow rate. This is the basis for predicting the minimal pump flow rate (MPFR)—the minimal flow necessary to meet metabolic demands.

5. Glucose—The liver is immature in neonates and the function of gluconeogenesis is immature, so hypoglycemia is a major risk.
6. pH—pH-stat management enhances the distribution of extracorporeal perfusate to the brain and may help in uniform and rapid cooling of the brain, but this strategy increases the risk of embolism. So it is advisable to utilize pH stat during cooling and then shift to alpha-stat strategy.
7. Flow rate—The usual perfusion pressure is 20-50 mm Hg as compared to 50-80 mm Hg because of low temperature and greater hemodilution. Flow rate depends upon body surface area and determinants of organ perfusion like blood gases, urine output, mixed venous oxygen saturation and serum lactate levels.
8. Bleeding—Factors likely to increase bleeding are (i) Hemodilution (ii) activation of the extrinsic coagulation system—consumption coagulopathy, (iii) antithrombin III (AT III) deficiency, heparin-induced thrombocytopenia, and inadequate heparin neutralization (iv) qualitative and quantitative platelet defects. The immune system of the neonate is immature; complement generation is low and neonatal mononuclear cells are dysfunctional.
9. The AT III concentration in healthy newborns is only half that of adults. Using a standard dose of heparin may result in inadequate anticoagulation and intravascular coagulation. Administration of fresh-frozen plasma or recombinant AT III easily corrects this problem.
10. Calcium—Calcium levels are low in neonates due to incomplete development of the sarcoplasmic reticulum and low levels of the enzyme Ca-ATPase required for Ca reuptake.
11. Hemofiltration—The renal system up to 2 years is immature functionally; so hemoconcentration and ultrafiltration is commonly done to remove

excess fluid, increase the hematocrit and attenuate inflammatory mediators. The rate of filtration depends upon membrane permeability, blood flow, transmembrane pressure and hematocrit.

Modified ultrafiltration (MUFF) (i) It is performed at the end of the surgery when the patient is off CPB. It removes excess water, inflammatory products and increases hematocrit. It also improves myocardial function but carries the risk of hypotension and air embolism.

A major advantage of ultrafiltration in pediatric patients is the reduced postoperative blood loss.

SUGGESTED READING

1. Gravelee GP, Davis RF, Stammers AH, Ungerleider RM. Cardiopulmonary bypass: Principles and Practice, 3rd edition. Wolter Kluwer/ Lippincott Williams and Wilkins, Philadelphia.
2. Greeley WJ, Berkowitz DH, Nathan AT. Anesthesia for Pediatric Cardiac Surgery. Miller's Anesthesia. In: Miller RD (Ed). Churchill Livingstone Elsevier, Philadelphia.
3. Hessel EA, Murphy GS, Groom RC, Ghansah JN. Cardiopulmonary bypass: Equipment, circuits and pathophysiology; A practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravlee GP (Eds).
4. Lazar HL, McDonnell M, Chipkin SR, Furnary AP, Engelman RM, Sadhu AR, et al. The Society of Thoracic Surgeons Practice Guideline Series: Blood Glucose Management during Adult Cardiac Surgery. *Ann Thorac Surg.* 2009;87(2):663-9.
5. Licker M, Diaper J, Cartier V, Ellenberger C, Cikirikcioglu M, Kalangos A. Clinical Review: Management of weaning from cardiopulmonary bypass after cardiac surgery. *Ann Card Anaesth.* 2012;15:206-23.
6. Nardo JD. Physiology and Technique of Extracorporeal circulation in Pediatric patient, Pediatric cardiac anesthesia. In: Lake CL, Brooker PD (Eds), 4th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 228-52.

Chapter 5

Cardiovascular Drugs

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INOTROPES AND VASOPRESSORS

DOPAMINE

Dopamine naturally occurs in the body. It works by improving the pumping strength of the heart and improves blood flow to the kidneys.

Dopamine hydrochloride injection is a clear, practically colorless, aqueous, additive solution for intravenous infusion after dilution.

Indication

- ❑ For the correction of hemodynamic imbalances present in the shock syndrome due to myocardial infarctions, trauma, endotoxic septicemia, open heart surgery, renal failure, and chronic cardiac decomposition as in congestive cardiac failure.
- ❑ Hypotension due to inadequate cardiac output can be managed by administration of low to moderate doses of dopamine, which have little effect on systemic vascular resistance (SVR).
- ❑ At high therapeutic doses, the alpha adrenergic activity of dopamine becomes more prominent and thus may correct hypotension due to diminished SVR.

Mechanism of Action

- ❑ Dopamine (dopamine hydrochloride) produces positive chronotropic and inotropic effects on the myocardium, resulting in increased heart rate and cardiac contractility.
- ❑ This is accomplished directly by exerting an agonist action on beta-adrenoceptors and indirectly by causing release of norepinephrine from storage sites like sympathetic nerve endings.

Clinical Pharmacology

Onset of action occurs within five minutes of intravenous administration, and *plasma half-life of about two minutes*, the duration of action is less than ten minutes.

Dopamine is metabolized in the liver, kidney, and plasma by monoamine oxidase (MAO) and catechol-O-methyltransferase to the inactive compounds homovanillic acid (HVA) and 3,4-dihydroxyphenylacetic acid.

Action and Doses

It is used to support BP, cardiac output (CO) and renal perfusion in shock. The predominant effects of dopamine (dopamine hydrochloride) are dose-related.

At low rates of infusion (0.5–2 µg/kg/min) dopamine causes vasodilation that is presumed to be due to a specific agonist action on dopamine receptors (distinct from alpha and beta adrenoceptors) in the renal, mesenteric, coronary, and intracerebral vascular beds.

At intermediate rates of infusion (2–10 µg/kg/min) dopamine acts to stimulate the beta₁-adrenoceptors, resulting in improved myocardial contractility, increased SA rate and enhanced impulse conduction in the heart.

At higher rates of infusion (10–20 µg/kg/min) there is some effect on alpha-adrenoceptors, with consequent vasoconstrictor effects and a rise in blood pressure.

Dosing (Adult)

- ❑ **Refractory CHF:** Initial dose: 0.5 to 2 µg/kg/min.
- ❑ **Renal:** 1 to 5 µg/kg/min.
- ❑ **Severely ill patient:** Initially 5 µg/kg/min, increase by 5 to 10 µg/kg/min (q10 to 30 min) up to max of 50 µg/kg/min.
- ❑ **Cardiac life support (initial):** 2 to 5 µg/kg/min—titrated to effect. Infusion may be increased by 1–4 µg/kg/minute at 10 to 30 minute intervals until optimal response is obtained.

Side Effects

- ❑ **Cardiovascular system:** Ventricular arrhythmia (at very high doses), ectopic beats, tachycardia, anginal pain, palpitation, cardiac conduction abnormality, widened QRS complexes
- ❑ **Respiratory system:** Dyspnea
- ❑ **Gastrointestinal:** Vomiting
- ❑ **Metabolic/nutritional system:** Azotemia
- ❑ **Central nervous system:** Headache, anxiety
- ❑ **Dermatological system:** Piloerection

DOBUTAMINE

Dobutamine is 4-[2-[[3-(*p*-Hydroxyphenyl)-1-methylpropyl]amino]ethyl]-pyrocatechol hydrochloride. It is a synthetic catecholamine.

Mechanism of Action

Dobutamine is a direct-acting agent, whose primary activity results from stimulation of the β_1 -adrenoceptors of the heart, increasing contractility and cardiac output. Increases contractility and to a lesser extent heart rate but little direct effect on BP. Since it does not act on dopamine receptors to induce the release of norepinephrine (another α_1 agonist), dobutamine is less prone to induce hypertension than is dopamine.

Onset of action: 1–10 minutes. Peak effect: 10–20 minutes. Half-life: 2 minutes. The principal routes of metabolism are methylation of the catechol and conjugation. In human urine, the major excretion products are the conjugates of dobutamine and 3-O-methyl dobutamine. The 3-O-methyl derivative of dobutamine is inactive.

Indication

- ❑ Refractory CHF or hypotensive patients in whom vasodilators cannot be used because of effects on BP.
- ❑ Dobutamine is used to treat acute but potentially reversible heart failure, such as which occurs during cardiac surgery or in cases of septic or cardiogenic shock, on the basis of its positive inotropic action.
- ❑ Dobutamine can be used in cases of congestive heart failure to increase cardiac output. It is indicated when parenteral therapy is necessary for inotropic support in the short-term treatment of patients with cardiac decompensation due to depressed contractility, which could be the result of either organic heart disease or cardiac surgical procedures.

Dose

- ❑ Infusion of dobutamine should be started at a low rate (0.5–1.0 $\mu\text{g}/\text{kg}/\text{min}$) and titrated at intervals of a few minutes, guided by the patient's response, including systemic blood pressure, urine flow, frequency of ectopic activity, heart rate and (whenever possible) measurements of cardiac output, central venous pressure, and/or pulmonary capillary wedge pressure. The optimal infusion rates varied from patient to patient, usually 2 to 20 $\mu\text{g}/\text{kg}/\text{min}$.
- ❑ **Cardiac decompensation:** 0.5–1 $\mu\text{g}/\text{kg}/\text{min}$ IV continuous infusion initially, then 2–20 $\mu\text{g}/\text{kg}/\text{min}$; not to exceed 40 $\mu\text{g}/\text{kg}/\text{min}$.
- ❑ **Low cardiac output:** 2–20 $\mu\text{g}/\text{kg}/\text{min}$ IV or IO; titrate to desired effect; not to exceed 40 $\mu\text{g}/\text{kg}/\text{min}$.

Side Effects

Increased heart rate, blood pressure, and ventricular ectopic activity. Approximately 5% of patients have had increased premature ventricular beats during infusions.

EPINEPHRINE

Epinephrine, more commonly known as adrenaline, is a hormone secreted by the medulla of the adrenal glands. In medicine epinephrine is used chiefly as a stimulant in cardiac arrest, as a vasoconstrictor in shock, and as a bronchodilator and antispasmodic in bronchial asthma.

Epinephrine injection, is a clear, colorless, sterile solution containing 1 mg/mL (1:1000), packaged as 1 mL of solution in a single-use clear glass vial. In the 1 mL vial, each 1 mL of adrenaline solution contains 1 mg epinephrine, 9.0 mg sodium chloride, 1.0 mg sodium metabisulfite, hydrochloric acid to adjust pH, and water for injection

Mechanism of Action

- ❑ Epinephrine acts on both alpha and beta-adrenergic receptors
- ❑ Through its action on alpha-adrenergic receptors, epinephrine lessens the vasodilation and increased vascular permeability that occurs during anaphylaxis, which can lead to loss of intravascular fluid volume and hypotension
- ❑ Through its action on beta-adrenergic receptors, epinephrine causes bronchial smooth muscle relaxation and helps alleviate bronchospasm, wheezing and dyspnea that may occur during anaphylaxis
- ❑ Epinephrine also alleviates pruritus, urticaria, and angioedema and may relieve gastrointestinal and genitourinary symptoms associated with anaphylaxis because of its relaxer effects on the smooth muscle of the stomach, intestine, uterus and urinary bladder
- ❑ Epinephrine increases glycogenolysis, reduces glucose up-take by tissues, and inhibits insulin release in the pancreas, resulting in hyperglycemia and increased blood lactic acid
- ❑ Epinephrine causes mydriasis when administered intraocularly or parenterally.

Indications and Usage

- ❑ Relieves respiratory distress due to bronchospasm
- ❑ Provide rapid relief of hypersensitivity reactions to drugs and other allergens (e.g. anaphylactic reactions to drugs, animal serums, insect stings)
- ❑ Prolongs action of local and regional anesthetics
- ❑ Restore cardiac rhythm in cardiac arrest due to various causes, for resuscitation in cardiac arrest following anesthetic accidents, in cardiopulmonary resuscitation
- ❑ Treatment of mucosal congestion of hay fever, rhinitis, and acute sinusitis; relieve bronchial asthmatic paroxysms
- ❑ Symptomatic relief of serum sickness, urticaria, angioneurotic edema
- ❑ For relaxation of uterine musculature and to inhibit uterine contractions
- ❑ Epinephrine injection can be used as a hemostatic agent

- ❑ In syncope due to complete heart block or carotid sinus hypersensitivity
- ❑ Used in open-angle glaucoma
- ❑ Used as a vasopressor in the treatment of anaphylactic shock and under certain conditions in insulin shock.

Dosing (Adult)

- ❑ **Refractory hypotension** (refractory to dopamine/dobutamine): Continuous IV infusion: 1 µg/min (range: 1–10 µg/minute)—titrate dosage to desired effect. Usual rate: 1 to 4 µg/min. Severe cardiac dysfunction may require doses >10 µg/minute (up to max of 20 µg/min in a 70 kg patient).
- ❑ **Endotracheal:** Doses (2–2.5 × IV dose) should be diluted to 10 mL with NS or distilled water prior to administration.
- ❑ **Anaphylaxis (adult):** 0.3 mg IM (0.3 mL of a 1:1000 solution). May be repeated if severe anaphylaxis persists—repeat q10 to 15 minutes PRN or give 0.1 to 0.25 mg IV (1:10,000) over 5–10 min repeat q5 to 15 minutes as needed or start **continuous infusion:** 1 to 4 µg/min.
- ❑ **Asthma:** Inhalational form—start with 1 inhalation, then wait at least 1 min. If not relieved, use once more. Do not use again for at least 3 h. Subcutaneous (SC) form: 0.2–0.5 mg (0.2–0.5 mL of a 1:1000 solution) SC every 2 hours as required. In severe attacks, may repeat dose every 20 minutes for a maximum of 3 doses.
- ❑ **Cardiac arrest**
 - Adults: IV 1:10,000 (0.1 mg/mL) solution: 0.1 to 1 mg (1–10 mL), repeated every 5 min, if necessary.
 - Children: IV 1:10,000 (0.1 mg/mL): 0.005 to 0.01 mg/kg.

Adverse Reactions

- ❑ Cardiovascular: Angina; cardiac arrhythmias including fatal ventricular fibrillation; excessive and/or rapid rise in BP; palpitations.
- ❑ CNS: Anxiety; apprehension; cerebral hemorrhage; dizziness; fear; headache; hemiplegia; restlessness; subarachnoid hemorrhage; tremor; weakness.
- ❑ Dermatologic: Sweating.
- ❑ GI: Nausea; vomiting.
- ❑ Local: Necrosis.
- ❑ Respiratory: Respiratory difficulty.
- ❑ Miscellaneous: Pallor.

NOREPINEPHRINE

- ❑ Norepinephrine (sometimes referred to as *L-arterenol/Levarterenol* or *L-norepinephrine*) is a sympathomimetic amine which differs from epinephrine by the absence of a methyl group on the nitrogen atom.
- ❑ It is a chemical released from the **sympathetic nervous system** in response to stress. It is classified as a **neurotransmitter**, a chemical that is released

from neurons. Because the release of norepinephrine affects other organs of the body, it is also referred to as a **stress hormone**.

- ❑ Norepinephrine is synthesized from dopamine by dopamine β -hydroxylase.

Mechanism of Action

The actions of norepinephrine are carried out via the binding to adrenergic receptors. It functions as a peripheral vasoconstrictor (alpha-adrenergic action) and as an inotropic stimulator of the heart and dilator of coronary arteries (beta-adrenergic action).

Noradrenaline normally produces effects such as increased blood pressure, widening of pupils, widening of air passages in the lungs and narrowing of blood vessels in non-essential organs. This enables the body to perform well in stressful situations.

Indication

- ❑ For blood pressure control in certain acute hypotensive states (e.g. pheochromocytectomy, sympathectomy, poliomyelitis, spinal anesthesia, myocardial infarction, septicemia, blood transfusion, and drug reactions).
- ❑ As an adjunct in the treatment of cardiac arrest and profound hypotension.

Usual Adult Dose

- ❑ **For hypotension**
 - Initial dose: 2 to 4 $\mu\text{g}/\text{min}$
 - Maintenance dose: Adjust the rate for a low normal blood pressure (usually 80 to 100 mm Hg systolic). The average maintenance dose ranges from 1 to 12 $\mu\text{g}/\text{min}$.
- ❑ **For shock**
 - Initial dose: 2 to 4 mcg/min
 - Maintenance dose: Adjust the rate for a low normal blood pressure (usually 80 to 100 mm Hg systolic). The average maintenance dose ranges from 1 to 12 $\mu\text{g}/\text{min}$.

Side Effects

Headache, weakness, dizziness, tremor, pallor, respiratory difficulty or apnea, precordial pain.

ISOPRENALINE

Isoprenaline (INN) or **isoproterenol** is a medication used for the treatment of bradycardia, heart block, and rarely for asthma. It is a nonselective beta-adrenergic agonist and structurally similar to adrenaline.

Mechanism of Action

Isoprenaline's effects on the cardiovascular system (non-selective) relate to its actions on cardiac β_1 receptors and β_2 receptors on smooth muscle within the tunica media of arterioles. By activating β_1 -receptors on the heart, it induces positive chronotropic, dromotropic, and inotropic effects. β_2 -adrenoceptor stimulation in arteriolar smooth muscle induces vasodilation. Its inotropic and chronotropic effects elevate systolic blood pressure, while its vasodilatory effects tend to lower diastolic blood pressure. The overall effect is to decrease mean arterial pressure due to the β_2 receptor's vasodilation.

Pharmacokinetics

Isoprenaline is readily absorbed when given parenterally. The half-life of isoprenaline hydrochloride is brief lasting only a few minutes following intravenous administration and up to 2 hours after subcutaneous administration. Isoprenaline is metabolized by catechol-o-methyl transferase primarily in the liver. The metabolites are excreted through the kidney.

Indications

- Its primary use is for bradycardia or heart block.
- Mild or transient episodes of heart block that do not require electric shock or pacemaker therapy.
- Serious episodes of heart block and Adams-Stokes attacks (except when caused by ventricular tachycardia or fibrillation).
- Uses in cardiac arrest until electric shock or pacemaker therapy, the treatments of choice, are available.
- Bronchospasm occurring during anesthesia.
- As an adjunct to fluid and electrolyte replacement therapy and the use of other medicines and procedures in the treatment of hypovolemic and septic shock, low cardiac output (hypoperfusion) states, congestive heart failure, and cardiogenic shock.
- Used with caution, it can also be used to treat torsades de pointes by acquired defect, in conjunction with overdrive pacing and magnesium sulfate.

Dosage and Administration

It can be administered by the intravenous, intramuscular, subcutaneous or intracardiac routes. It should generally be started at the lowest recommended dose and the rate of administration gradually increased if necessary while carefully monitoring the patient. The usual route of administration is by intravenous infusion or bolus intravenous injection.

Usual dosage is 0.5 $\mu\text{g}/\text{min}$ to 5 $\mu\text{g}/\text{min}$ although doses of 20 $\mu\text{g}/\text{min}$ or greater have been used. For bolus dosing, can dilute 200 μg in 20 mL and administer 1 mL bolus.

Dosage in pediatrics:

- ❑ IV infusion
- ❑ 300 µg/kg in 50 mL of compatible IV fluid.
- ❑ Commence infusion at 0.1 µg/kg/min (1 mL/h) and titrate to effect.

Adverse Reactions

- ❑ CNS: Nervousness, headache, dizziness
- ❑ Cardiovascular: Tachycardia, palpitations, angina, Adams-Stokes attacks, pulmonary edema, hypertension, hypotension, ventricular arrhythmias, tachyarrhythmias
- ❑ Other: Flushing of the skin, sweating, mild tremors, weakness.

PHENYLEPHRINE

Phenylephrine is a selective α 1-adrenergic receptor agonist used primarily as a decongestant, as an agent to dilate the pupil, and to increase blood pressure.

Mechanism of Action

Phenylephrine causes powerful vasoconstriction by stimulating the post-synaptic alpha receptors. Phenylephrine causes increased systemic vascular resistance, decreased cardiac output, increased stroke volume and bradycardia.

Pharmacokinetics

- ❑ Onset of effect: Immediate
- ❑ Peak effect: 2–5 minutes.

Indications and Dosage**Adult**

- ❑ **For hypotension:**
 - IM or subcutaneous: 2 to 5 mg every 1 to 2 hours as needed.
 - **IV bolus:** 0.2 mg/dose (range: 0.1–0.5 mg/dose) every 10 to 15 minutes as needed (initial dose should not exceed 0.5 mg)
 - **IV infusion:** 100 to 180 µg/min initially. The usual maintenance dose is 40 to 60 µg/min.

Alternatively, 0.5 µg/kg/minute; titrate to desired response. Dosing ranges between 0.4 to 9.1 µg/kg/minute have been reported.

- ❑ **For shock:**
 - **IM or subcutaneous:** 2 to 5 mg every 1 to 2 hours as needed.
 - **IV bolus:** 0.2 mg/dose (range: 0.1–0.5 mg/dose) every 10 to 15 minutes as needed (initial dose should not exceed 0.5 mg)
 - **IV infusion:** 100 to 180 µg/min initially. The usual maintenance dose is 40 to 60 µg/min.

Alternatively, 0.5 µg/kg/minute; titrate to desired response. Dosing ranges between 0.4 to 9.1 µg/kg/minute have been reported.

- **For PSVT:** 0.5 mg over 20–30 seconds. If cardiac rhythm fails to revert after 60–90 seconds and blood pressure allows, dose may be repeated with 0.6–0.7 mg given over 20–30 seconds. Duration of effect: 15–20 minutes.
- **For nasal congestion:** Tablets or oral liquid: 10 to 20 mg orally every 4 hours as needed.

Pediatric

- ❑ **For hypotension**
 - **IM or subcutaneous:** 0.1 mg/kg every 1 to 2 hours as needed. Maximum dose: 5 mg.
 - **IV bolus:** 5 to 20 µg/kg/dose every 10 to 15 minutes as needed.
 - **IV infusion:** 0.1 to 0.5 µg/kg/min titrated to effect.
- ❑ **For shock**
 - **IM or subcutaneous:** 0.1 mg/kg every 1 to 2 hours as needed. Maximum dose: 5 mg
 - **IV bolus:** 5 to 20 µg/kg/dose every 10 to 15 minutes as needed.
 - **IV infusion:** 0.1 to 0.5 µg/kg/min titrated to effect.
- ❑ For supraventricular tachycardia: 5 to 10 µg/kg IV over 30 seconds.

Side Effects

Headache, reflex bradycardia (slow heart beat), excitability, anxiety, restlessness, high blood pressure, and rarely abnormal heart beat.

MILRINONE

Milrinone lactate is a member of a new class of bipyridine inotropic/vasodilator agents with phosphodiesterase inhibitor activity, distinct from digitalis glycosides or catecholamines. Milrinone lactate is designated chemically as 1,6-Dihydro-2-methyl-6-oxo[3,4'-bipyridine]-5-carbonitrile lactate.

It works by increasing the force with which the heart pumps blood through the body and widens blood vessels, which allows blood to flow through the body more easily.

Mechanism of Action

Milrinone is phosphodiesterase inhibitor; positive inotrope with little chronotropic effect; direct vasodilator (decreases both preload and afterload).

Milrinone is a phosphodiesterase-3 inhibitor. This drug inhibits the action of phosphodiesterase-3 and thus prevents degradation of cAMP. With increase cAMP levels there is an increase activation of PKA. This PKA will phosphorylate many components of the cardiomyocyte such as calcium channels and components of the myofilaments. Phosphorylation of calcium channels permits an increase in calcium influx into the cell. This increase in calcium influx permits increased contractility.

PKA also phosphorylates potassium channels promoting their action. Potassium channels are responsible for repolarization of the cardiomyocytes therefore increasing the rate at which cells can depolarize and generate contraction. PKA also phosphorylates components on myofilaments allowing actin and myosin to interact more easily and thus increasing contractility and the inotropic state of the heart. Milrinone allows stimulation of cardiac function independently of β -adrenergic receptors which appear to be down-regulated in those with heart failure.

Pharmacology

Absorption

- ❑ Onset: 5–15 min (IV)
- ❑ Duration: 3–5 h
- ❑ Peak plasma time: 2 min
- ❑ Therapeutic range: 100–300 ng/mL (hemodynamic effect)
- ❑ Half-life: 2.5 h
- ❑ Clearance: 2.3 mL/kg/min

Distribution: Protein Bound (70%)

Metabolism

It is metabolized in liver (12%), mainly via glucuronidation. The major metabolite is milrinone O-glucuronide (activity unknown).

Indications and Doses

Used in patients with refractory pulmonary hypertension, low cardiac output (especially after cardiac surgery) and in septic shock.

There is evidence for its use in preventing low cardiac output in patients undergoing cardiac surgery. It is for short-term treatment only and should generally not be used for longer than 72 hours.

Adult

- ❑ **Congestive heart failure**
 - IV 50 μ g/kg loading dose over 10 minutes followed by continuous infusion of 0.375–0.75 μ g/kg/min
 - Maintenance: 1.13 mg/kg/day.

Children

- ❑ **Low cardiac output, septic shock:** 50 μ g/kg loading dose over 10–60 minutes followed by continuous infusion of 0.25–0.75 μ g/kg/min IV.
The infusion rate should be adjusted according to hemodynamic and clinical response.

- ❑ **Congenital heart disease with secondary pulmonary hypertension:** Milrinone is usually started perioperatively. It reduces the pulmonary vascular resistance and improves surgical outcomes. A loading dose of 50 µg/kg during rewarming on pump followed by continuous infusion of 0.25–0.75 µg/kg/min IV.

Adverse Effects

- ❑ Ventricular arrhythmias
- ❑ Supraventricular arrhythmia (4%)
- ❑ Headache (3%)
- ❑ Hypotension (3%)
- ❑ Angina/chest pain (1%)
- ❑ Abnormal liver function test results
- ❑ Anaphylaxis
- ❑ Atrial fibrillation
- ❑ Bronchospasm
- ❑ Hypokalemia
- ❑ Injection-site reaction
- ❑ Rash
- ❑ Thrombocytopenia
- ❑ Torsades de pointes
- ❑ Tremor.

ANTIARRHYTHMIC DRUGS

1. **Class I:** Membrane stabilizing drugs; sodium channel blockers
 - **Class I a: Quinidine, disopyramide**
 - **Class I b: Lidocaine, phenytoin**
 - **Class I c: Propafenone**
2. **Class II:** Beta blockers
 - **Acebutolol, atenolol, esmolol, metoprolol, propranolol**
3. **Class III:** Potassium channel blockers
 - **Amiodarone, sotalol**
4. **CLASS IV:** Calcium channel blockers
 - **Verapamil, diltiazem.**

CLINICAL CLASSIFICATION OF ANTIARRHYTHMIC DRUGS

- ❑ Group 1: Decrease AV conductance, for treatment of SV tachyarrhythmia—verapamil
- ❑ Group 2: For treatment of ventricular tachyarrhythmia—lidocaine (IV), Propafenone, phenytoin
- ❑ Group 3: For treating SV and V tachyarrhythmia—amiodarone, beta blockers
- ❑ Group 4: Drugs for AV block—atropine (M-cholinolytic), ephedrine (indirect adrenomimetics)
- ❑ Group 5: Inhibitors of AV conduction—adenosine (potassium channel opener), digoxin (cardiac glycoside).

AMIODARONE

Amiodarone hydrochloride is a member of a new class of antiarrhythmic drugs with predominantly Class III (Vaughan Williams' classification) effects. It is an antiarrhythmic medication that affects the rhythm of heartbeats. It is used for various types of cardiac dysrhythmias, both ventricular and atrial.

Mechanism of Action

Amiodarone is categorized as a class III antiarrhythmic agent, and prolongs phase 3 of the cardiac action potential, the repolarization phase where there is normally decreased calcium permeability and increased potassium permeability. Amiodarone possesses beta blocker-like and potassium channel blocker-like actions on the SA and AV nodes, increases the refractory period via sodium- and potassium-channel effects, and slows intracardiac conduction of the cardiac action potential, via sodium-channel effects. Amiodarone chemically resembles thyroxine (thyroid hormone), and its binding to the nuclear thyroid receptor might contribute to some of its pharmacologic and toxic actions.

The antiarrhythmic effect of amiodarone may be due to at least two major properties:

1. Prolongation of the myocardial cell-action potential duration and refractory period and
2. Noncompetitive α - and β -adrenergic inhibition.

Action on Heart

Amiodarone is considered a “broad spectrum universal” antiarrhythmic drug:

- ❑ A delay in the rate at which the heart's electrical system “recharges” after the heart contracts (repolarization)
- ❑ Prolongation in the electrical phase during which the heart's muscle cells are electrically stimulated (action potential)
- ❑ Slowing of the speed of electrical conduction (how fast each individual impulse is conducted through the heart's electrical system)
- ❑ Reduction in the rapidity of firing of the normal generator of electrical impulses in the heart (the heart's pacemaker)
- ❑ Slowing of conduction through various specialized electrical pathways (called accessory pathways) which can be responsible for arrhythmias.

Adult Dose for Arrhythmias

Initial dose (IV): 1000 mg over the first 24 hours of therapy, delivered by the following infusion regimen—Bolus dose of 150 mg over the first 10 minutes (15 mg/min), followed by 360 mg over the next 6 hours (1 mg/min) and 540 mg over the remaining 18 hours (0.5 mg/min).

Oral Dose for Arrhythmia

It is usual to take 200 mg three times a day for the first week, then 200 mg twice a day for the second week and then be reduced to 200 mg each day from the third week.

Pediatric Dose for Supraventricular Tachycardia

- ❑ **Less than 1 year**
 - **Oral dose:** 10 to 20 mg/kg/day orally in 2 divided doses for 7 to 10 days; dosage should then be reduced to 5 to 10 mg/kg/day once daily and continued for 2 to 7 months
 - **Intravenous loading dose:** 5 mg/kg given over 60 minutes; may repeat initial loading dose to a maximum total initial load: 10 mg/kg; do not exceed total daily bolus of 15 mg/kg/day.
- ❑ **Greater than 1 year**
 - Initial dose: 10 to 15 mg/kg/day orally for 4 to 14 days given in 1 to 2 divided doses/day.
 - Maintenance dose: 5 to 10 mg/kg/day orally given once a day.

Contraindications

- ❑ Patients with cardiogenic shock
- ❑ Severe sinus-node dysfunction, causing marked sinus bradycardia
- ❑ Second- or third-degree atrioventricular block
- ❑ When episodes of bradycardia have caused syncope (except when used in conjunction with a pacemaker).
- ❑ Patients with a known hypersensitivity to the drug or to any of its components, including iodine.

Side Effects

- ❑ Gastrointestinal: Nausea, vomiting, constipation, anorexia
- ❑ Dermatologic: Solar dermatitis/photosensitivity, blue skin discoloration, rash, spontaneous ecchymosis, alopecia
- ❑ Neurologic: Malaise and fatigue, tremor/abnormal involuntary movements, lack of coordination, abnormal gait/ataxia, dizziness, paresthesias, decreased libido, insomnia, headache, sleep disturbances
- ❑ Ophthalmologic: Visual disturbances
- ❑ Hepatic: Abnormal liver-function tests
- ❑ Respiratory: Pulmonary inflammation or fibrosis
- ❑ Thyroid: Hypothyroidism, hyperthyroidism
- ❑ Other: Flushing, abnormal taste and smell, edema, abnormal salivation, coagulation abnormalities, hypotension, and cardiac conduction abnormalities.

DILTIAZEM

Diltiazem is a nondihydropyridine (non-DHP) member of the class of drugs known as calcium ion cellular influx inhibitor (slow channel blocker), used in the treatment of hypertension, angina pectoris, and some types of arrhythmia. It is a class 3 antianginal drug, and a class IV antiarrhythmic.

Mechanism of Action

Diltiazem is a potent vasodilator, increasing blood flow and variably decreasing the heart rate via strong depression of AV node conduction. Its pharmacological activity is somewhat similar to verapamil.

It is a potent vasodilator of coronary and peripheral vessels, which reduces peripheral resistance and afterload.

Because of its negative inotropic effect, diltiazem causes a modest decrease in heart muscle contractility and reduces myocardium oxygen consumption. Its negative chronotropic effect results in a modest lowering of heart rate, due to slowing of the sinoatrial node. It results in reduced myocardium oxygen consumption.

Because of its negative dromotropic effect, conduction through the AV (atrioventricular) node is slowed, which increases the time needed for each beat. This results in reduced myocardium oxygen consumption.

Indications and Doses

Adult

Hypertension

- ❑ Oral
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day.
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
 - Sustained release (SR): Initial dose: 60 to 120 mg orally twice a day.
 - SR maintenance dose: 240 to 360 mg orally/day.
- ❑ Intravenous
 - Initial bolus doses: 0.25 mg/kg as a bolus administered over 2 minutes. A second bolus of 0.35 mg/kg may be used if necessary.
 - Initial infusion dose: 5 mg/h.
 - Maintenance infusion dose: The infusion rate may be increased in 5 mg/h increments up to 15 mg/h.

Atrial fibrillation

- ❑ Oral
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
 - SR initial dose: 60 to 120 mg orally twice a day
 - SR maintenance dose: 240 to 360 mg orally/day.

- ❑ Intravenous
 - Initial dose: 0.25 mg/kg actual body weight bolus over 2 minutes. If necessary, a second bolus of 0.35 mg/kg may be given. In some cases, an infusion of diltiazem 5 mg/hour may be started, and advanced in 5 mg/hour increments to 15 mg/hour for up to 24 hours.

Atrial flutter

- ❑ Oral
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
 - SR initial dose: 60 to 120 mg orally twice a day
 - SR maintenance dose: 240 to 360 mg orally/day.
- ❑ Intravenous
 - Initial dose (IV): 0.25 mg/kg actual body weight bolus over 2 minutes. If necessary, a second bolus of 0.35 mg/kg may be given. In some cases, an infusion of diltiazem 5 mg/hour may be started, and advanced in 5 mg/hour increments to 15 mg/hour for up to 24 hours.

Supraventricular tachycardia

- ❑ Oral
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
- ❑ Intravenous
 - Initial dose (IV): 0.25 mg/kg actual body weight bolus over 2 minutes. If necessary, a second bolus of 0.35 mg/kg may be given. In some cases, an infusion of diltiazem 5 mg/hour may be started, and advanced in 5 mg/hour increments to 15 mg/hour for up to 24 hours.
 - **Angina pectoris prophylaxis**
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
 - SR initial dose: 60 to 120 mg orally twice a day
 - SR maintenance dose: 240 to 360 mg orally/day.
 - **Congestive heart failure**
 - Initial dose: 30 to 60 mg orally 3 to 4 times a day
 - Maintenance dose: 180 to 360 mg orally/day in divided doses.
 - SR initial dose: 60 to 120 mg orally twice a day
 - SR maintenance dose: 240 to 360 mg orally/day.

Adverse Effects

- ❑ Edema, headache, dizziness, AV block, peripheral edema
- ❑ Bradyarrhythmia, hypotension
- ❑ Nausea, vomiting, diarrhea, constipation
- ❑ Vasodilation, extrasystoles

- ❑ Flushing, drug-induced gingival hyperplasia
- ❑ Myalgia
- ❑ Bronchitis
- ❑ Sinus congestion
- ❑ Dyspnea
- ❑ Congestion.

LIGNOCAINE

Lidocaine is a common local anesthetic and class-1b antiarrhythmic drug used intravenously for the treatment of ventricular arrhythmias (for acute myocardial infarction, digoxin poisoning, cardioversion, or cardiac catheterization) if amiodarone is not available or contraindicated. Lidocaine should be given for this indication after defibrillation, CPR, and vasopressors have been initiated.

Mechanism of Action

Lignocaine hydrochloride acts by decreasing the sensitivity of heart muscle to electrical impulses. This slows the conduction of electrical signals in the heart muscle, which in turn, helps to restore a regular heart rhythm. This enables the heart to pump blood effectively around the body.

In the heart, lignocaine reduces automaticity by decreasing the rate of diastolic (phase 4) depolarization. Lignocaine is considered as a class 1 (membrane stabilizing) antiarrhythmic agent.

The duration of the action potential is decreased due to blockade of the sodium channel and the refractory period is shortened.

Doses

Adult

- ❑ Usual dose of lignocaine is 50 to 100 mg administered intravenously under ECG monitoring. The dose may be injected at a rate of approximately 25 to 50 mg (2.5 to 5.0 mL of the lignocaine 1% solution or 1.25 to 2.5 mL of the 2% solution) per minute.
- ❑ If the initial dose of 50 to 100 mg does not produce the desired response, a second dose may be given after five minutes. No more than 200 to 300 mg of lignocaine should be administered during a one hour period.
- ❑ Intravenous infusions of lignocaine may be administered at a rate of 1 to 4 mg/minute (20–50 µg/kg/minute). Intravenous infusions must be given under ECG monitoring to avoid potential overdosage and toxicity. The infusion should be terminated as soon as the patient's cardiac rhythm appears to be stable or at the earliest signs of toxicity.

Pediatric

Loading dose of lignocaine is 0.5 to 1 mg/kg repeated if necessary up to 3 to 5 mg/kg, followed by continuous infusions of 10 to 50 µg/kg/minute.

Contraindications

Absolute contraindications for the use of lidocaine include:

- ❑ Heart block, second or third degree (without pacemaker)
- ❑ Severe sinoatrial block (without pacemaker)
- ❑ Serious adverse drug reaction to lidocaine or amide local anesthetics
- ❑ Hypersensitivity to corn and corn-related products (corn-derived dextrose is used in the mixed injections)
- ❑ Concurrent treatment with quinidine, flecainide, disopyramide, procainamide (class I antiarrhythmic agents)
- ❑ Prior use of amiodarone hydrochloride
- ❑ Adams-Stokes syndrome
- ❑ Wolff-Parkinson-White syndrome.

Adverse Drug Reactions

- ❑ **CNS excitation:** Nervousness, agitation, anxiety, apprehension, tingling around the mouth (circumoral paresthesia), headache, hyperesthesia, tremor, dizziness, pupillary changes, psychosis, euphoria, hallucinations, and seizures
- ❑ **CNS depression** with increasingly heavier exposure: Drowsiness, lethargy, slurred speech, hypoesthesia, confusion, disorientation
- ❑ **Cardiovascular:** Hypotension, bradycardia, arrhythmias, flushing
- ❑ **Respiratory:** Bronchospasm, dyspnea, respiratory depression or arrest
- ❑ **Gastrointestinal:** Metallic taste, nausea, vomiting
- ❑ **Ears:** Tinnitus
- ❑ **Eyes:** Local burning, conjunctival hyperemia
- ❑ **Skin:** Itching, depigmentation, rash, urticaria, edema, angioedema
- ❑ **Blood:** Methemoglobinemia
- ❑ Allergy

ESMOLOL

Esmolol is a cardioselective beta 1-receptor blocker with rapid onset, a very short duration of action, and no significant intrinsic sympathomimetic or membrane stabilizing activity at therapeutic dosages. It is a class II antiarrhythmic.

Mechanism of Action

Class II antiarrhythmic; selective beta1-blocker with little or no effect on beta 2-receptors except at high doses. It does not have intrinsic sympathomimetic activity.

Pharmacokinetics

Esmolol is rapidly metabolized by hydrolysis of the ester linkage, chiefly by the esterases in the cytosol of red blood cells and not by plasma cholinesterases or red cell membrane acetylcholinesterase. Total body clearance in man was found to be about 20 L/kg/h, which is greater than cardiac output; thus the metabolism of esmolol is not limited by the rate of blood flow to metabolizing tissues such as the liver or affected by hepatic or renal blood flow. Esmolol has a rapid distribution half-life of about 2 minutes and an elimination half-life of about 9 minutes. Onset of action: 2–10 min (IV), duration: 10–30 min, protein bound: 55%.

Indications and Dose

It is commonly used in patients during surgery to prevent or treat tachycardia, and is also used in treatment of acute supraventricular tachycardia.

Adult

Intraoperative tachycardia/hypertension

- ❑ Immediate control: Initial bolus of 80 mg (~1 mg/kg) IV over 30 sec, then 0.15–0.3 mg/kg/min IV infusion
- ❑ Postoperative/gradual control: Loading with 0.5 mg/kg IV over 1 min, then 0.05 mg/kg/min IV for 4 min
- ❑ If inadequate response in 5 min 2nd loading dose of 0.5 mg/kg/min for 1 min, then 0.1 mg/kg/min IV.

Supraventricular tachycardia

- ❑ Loading: 0.5 mg/kg IV over 1 min
- ❑ Maintenance: Start 0.05 mg/kg/min IV for 4 min, may increase by 0.05 mg/kg up to 0.2 mg/kg/min
- ❑ If HR/BP not controlled after 5 min, repeat bolus (i.e. 500 µg/kg/min for 1 min), then initiate infusion of 0.1 mg/kg/min IV.

Hypertensive emergency

- ❑ Loading: 0.25–0.5 mg/kg IV over 1 min, then 0.05–0.1 mg/kg/min IV for 4 minute
- ❑ May repeat loading dose or increase infusion up to 0.3 mg/kg/min if necessary.

Pediatric

Supraventricular tachycardia

- ❑ Loading with 500–600 µg/kg IV over 2 min, then 200 µg/kg/min IV infusion (range 50–250 µg/kg/min).

Postoperative hypertension

Loading with 500–600 µg/kg IV over 2 min, then 200 µg/kg/min IV infusion (range 50–250 µg/kg/min)

Adverse Effects

- ❑ Hypotension
- ❑ Injection site pain
- ❑ Nausea
- ❑ Dizziness
- ❑ Somnolence
- ❑ Agitation
- ❑ Confusion.

NITROGLYCERIN

Nitroglycerin, an organic nitrate, is a vasodilating agent. The chemical name for nitroglycerin is 1, 2, 3 propanetriol trinitrate.

It is used for treating high blood pressure during surgery, controlling congestive heart failure associated with heart attack, treating chest pain in certain patients, and lowering blood pressure during surgery.

Mechanism of Action

Nitroglycerin forms free radical nitric oxide (NO) which activates guanylate cyclase, resulting in an increase of guanosine 3'5' monophosphate (cyclic GMP) in smooth muscle and other tissues. These events lead to dephosphorylation of myosin light chains, which regulate the contractile state in smooth muscle, and result in vasodilatation.

Pharmacological Actions

- ❑ Relaxation of vascular smooth muscle. Although venous effects predominate, nitroglycerin produces, in a dose-related manner, dilation of both arterial and venous beds.
- ❑ Dilation of post capillary vessels, including large veins, promotes peripheral pooling of blood, decreases venous return to the heart, and reduces left ventricular end-diastolic pressure (preload).
- ❑ Nitroglycerin also produces arteriolar relaxation, thereby reducing peripheral vascular resistance and arterial pressure (afterload), and dilates large epicardial coronary arteries.
- ❑ Therapeutic doses of nitroglycerin may reduce systolic, diastolic, and mean arterial blood pressure.
- ❑ Effective coronary perfusion pressure is usually maintained, but can be compromised if blood pressure falls excessively, or increased heart rate decreases diastolic filling time.

- ❑ Elevated central venous and pulmonary capillary wedge pressures, and pulmonary and systemic vascular resistance are also reduced by nitroglycerin therapy.
- ❑ Heart rate is usually slightly increased, presumably due to a compensatory response to the fall in blood pressure.
- ❑ Cardiac index may be increased, decreased, or unchanged. Myocardial oxygen consumption or demand (as measured by the pressure-rate product, tension-time index, and stroke-work index) is decreased and a more favorable supply-demand ratio can be achieved.

Metabolism

A liver reductase enzyme is of primary importance in the metabolism of nitroglycerin to glycerol dinitrate and mononitrate metabolites and ultimately to glycerol and organic nitrate. Known sites of extrahepatic metabolism include red blood cells and vascular walls.

Nitroglycerin plasma concentrations decrease rapidly, with a mean elimination half-life of 2 to 3 minutes. Half-life values range from 1.5 to 7.5 minutes. Clearance (13.6 L/min) greatly exceeds hepatic blood flow. Metabolism is the primary route of drug elimination.

Indication and Doses

Adult

- ❑ **For angina pectoris: Relief of acute anginal attack:**
 - **Lingual spray:** 1 to 2 sprays (0.4–0.8 mg) onto or under the tongue every 3 to 5 minutes as needed, up to 3 sprays in 15 minutes.
 - **Sublingual tablet:** 0.3 to 0.6 mg dissolved under the tongue or in the buccal pouch every 5 minutes as needed, up to 3 doses in 15 minutes.
 - **IV continuous infusion:** 5 µg/min initially, increased by 5 µg/min every 3 to 5 minutes as needed up to 20 µg/min, then gradually by 10 and then 20 µg/min if needed, up to a usual maximum of 200 µg/min and generally no more than 400 µg/min
- ❑ **For congestive heart failure:**
 - **Transdermal patch:** 0.1 to 0.4 mg/h patch applied to a dry and hairless area of the upper arm or body for 12 to 14 hours per day; titrate as needed and tolerated up to 0.8 mg/h.
 - **Transmucosal (buccal) tablet:** 1 mg dissolved between the lip and gum above the upper incisors or between the cheek and gum every 3 to 5 hours during waking hours (approximately 3 times a day); titrate as needed and tolerated. Usual maintenance dosage is 2 mg three times a day.
 - **Oral:** 2.5 every 8 to 12 hours; titrate as needed and tolerated up to 9 mg every 8 to 12 hour.

- ❑ **For myocardial infarction: Initial 24 to 48 hours after an acute myocardial infarction:**
 - **IV continuous infusion:** 5 µg/min initially, increased by 5 µg/min every 3 to 5 minutes as needed up to 20 µg/min, then gradually by 10 and then 20 µg/min if needed up to a usual maximum of 200 µg/min.
- ❑ **For hypertension**
 - **IV continuous infusion:** 5 µg/min initially, increased by 5 µg/min every 3 to 5 minutes as needed up to 20 µg/min, then gradually by 10 and then 20 µg/min if needed up to a usual maximum of 100 µg/min
 - **Congenital heart disease with pulmonary hypertension:** IV continuous infusion of 0.05 to 0.5 µg/kg/min started after the surgery
 - **For coronary perfusion in IHD patients after coronary bypass surgery:** IV continuous infusion of 0.2 to 0.5 µg/kg/min.

Pediatric

Perioperative hypertension or induction of intraoperative hypotension:

- ❑ **IV continuous infusion:** 0.25 to 0.5 µg/kg/min initially, increase by 0.5 to 1 µg/kg/min every 3 to 5 minutes as needed up to 5 µg/kg/min. Usual dose is 1 to 3 µg/kg/min, but doses as high as 20 µg/kg/min have been used.

Side Effects

Headache, vertigo, dizziness, weakness, palpitation, and other manifestations of postural hypotension particularly in erect and immobile patients.

Marked sensitivity to the hypotensive effects of nitrates (manifested by nausea, vomiting, weakness, diaphoresis, pallor, and collapse) may occur at therapeutic doses.

Syncope due to vasodilatation. Flushing, drug rash, and exfoliative dermatitis.

HEPARIN

Heparin also known as **unfractionated heparin**, a highly sulfated glycosaminoglycan, is widely used as an injectable anticoagulant, and has the highest negative charge density of any known biological molecule.

Heparin sodium injection, is a sterile, nonpyrogenic solution of heparin sodium (derived from porcine intestinal mucosa) in water for injection. It contains sodium hydroxide and/or hydrochloric acid for pH adjustment. PH 6.0 (5.0 to 7.5).

Mechanism of Action

Heparin inhibits reactions that lead to the clotting of blood and the formation of fibrin clots both in vitro and in vivo. Heparin acts at multiple sites in the normal coagulation system. Small amounts of heparin in combination with

antithrombin III (heparin cofactor) can inhibit thrombosis by inactivating activated factor X and inhibiting the conversion of prothrombin to thrombin. Once active thrombosis has developed, larger amounts of heparin can inhibit further coagulation by inactivating thrombin and preventing the conversion of fibrinogen to fibrin. Heparin also prevents the formation of a stable fibrin clot in inhibiting the activation of the fibrin stabilizing factor.

Bleeding time is usually unaffected by heparin. Clotting time is prolonged by full therapeutic doses of heparin; in most cases, it is not measurably affected by low doses of heparin.

Pharmacokinetics

- ❑ **Absorption:** Heparin is not absorbed from GI; must be given IV or subcutaneously. T_{max} is 2 to 4 hour (subcutaneous).
 - ❑ **Metabolism:** The liver and the reticuloendothelial system are the sites of biotransformation.
 - ❑ **Elimination:** The average half-life is 1.5 h (range, 1 to 6 h) and is dose dependent and nonlinear. Excreted in the urine, primarily as metabolites.
- Onset: Onset is immediate (IV) and 20 to 60 min (subcutaneous).

Indications

Heparin is generally used for anticoagulation for the following conditions:

- ❑ Acute coronary syndrome, e.g. NSTEMI
- ❑ Atrial fibrillation with embolization
- ❑ Deep-vein thrombosis and pulmonary embolism
- ❑ Cardiopulmonary bypass for heart surgery
- ❑ ECMO circuit for extracorporeal life support
- ❑ Hemofiltration
- ❑ Indwelling central or peripheral venous catheters
- ❑ Treatment of acute and chronic consumption coagulopathies (disseminated intravascular coagulation).

Heparin Dosing Information

- ❑ **Treatment of deep vein thrombosis:**
Continuous IV infusion: 5000 units IV one time as a bolus dose followed by 1300 units/hour by continuous IV infusion. Alternatively, a bolus dose of 80 units/kg IV one time followed by 18 units/kg/hour by continuous IV infusion may be used. *Intermittent subcutaneous injection:* 17,500 units subcutaneously every 12 hours. The dosage should be adjusted to maintain the aPTT at 1.5 to 2.5 times control.
- ❑ **Deep vein thrombosis**
Prophylaxis: 5000 units subcutaneously every 8 to 12 hours. This dosage may be adjusted to maintain the aPTT at the upper end of the normal range.

- ❑ **Pulmonary embolism:**
 - 5000 units IV one time as a bolus dose followed by 1300 units/hour by continuous IV infusion. Alternatively, a bolus dose of 80 units/kg IV one time followed by 18 units/kg/hour by continuous IV infusion may be used.
 - If it is suspected that the patient has experienced a massive pulmonary embolism, a more appropriate initial dosage may be an IV bolus of 10,000 units followed by 1500 units/hour.
 - Intermittent subcutaneous injection: 17,500 units subcutaneously every 12 hours. The dosage should be adjusted to maintain the aPTT at 1.5 to 2.5 times control.
- ❑ **Myocardial infarction:**
5000 units IV one time as a bolus dose followed by 1000 units/hour by continuous IV infusion.
- ❑ **Angina pectoris:**
5000 units IV one time as a bolus dose followed by 1000 units/hour by continuous IV infusion.
- ❑ **Anticoagulation during pregnancy:**
5000 units subcutaneously every 12 hours. This dosage may be adjusted to maintain the 6-hour aPTT at 1.5 times control or greater.
- ❑ **Thrombotic/thromboembolic disorder:**
 - 100 units/mL every 6 to 8 hours for PVC catheters and peripheral heparin locks. Additional flushes should be given when stagnant blood is observed in catheter, after catheter is used for drug or blood administration, and after blood withdrawal from catheter.
 - Addition of 0.5 to 1 unit/mL to peripheral and central TPN has been shown to increase duration of line patency. Arterial lines are heparinized with a final concentration of 1 unit/mL.

Cardiopulmonary bypass for heart surgery

- ❑ For arteriovenous fistula/LIMA harvesting/BT shunt/bidirectional Glenn shunt: Intravenous 100 IU/kg
- ❑ Off pump coronary bypass grafting: Intravenous 200 IU/kg
- ❑ On pump coronary bypass grafting: Intravenous 300 IU/kg
- ❑ On pump valvular/acyanotic congenital heart surgery: Intravenous 300 IU/kg
- ❑ On pump cyanotic heart surgery: Intravenous 400 IU/kg

Side Effects

The most common side effects are hemorrhage (bleeding), thrombocytopenia (decrease platelet count), heparin induced thrombocytopenia (HIT), heparin induced thrombocytopenia and thrombosis (HITT), injection site discomfort/irritation, allergy or hypersensitivity type reactions, increase in liver enzymes and hyperkalemia.

PROTAMINE

Protamines are small, arginine-rich, nuclear proteins that replace histones late in the haploid phase of spermatogenesis and are believed essential for sperm head condensation and DNA stabilization. Protamine sulfate injection, USP is a sterile, non-pyrogenic, isotonic solution of protamine sulfate in water for injection. It acts as a heparin antagonist. It is also a weak anticoagulant.

Mechanism of Action

Protamine that is strongly basic combines with acidic heparin forming a stable complex and neutralizes the anticoagulant activity of both drugs. It is a highly cationic peptide that binds to either heparin or low molecular weight heparin (LMWH) to form a stable ion pair, which does not have anticoagulant activity. The ionic complex is then removed and broken down by the reticuloendothelial system. In large doses, protamine sulfate may also have an independent—however weak—anticoagulant effect.

Pharmacokinetics

- ❑ Half-life elimination: 7 min
- ❑ Onset: 5 min
- ❑ Duration: 2 hour.

Indications

It neutralizes the anticoagulant action of heparin: Before surgery; after renal dialysis; after open-heart surgery, if excessive bleeding occurs and when an overdose has inadvertently been given.

Dosing Forms and Strengths

- ❑ **Heparin neutralization after cardiac surgery:**
 - 1–1.5 mg per 100 IU of heparin
 - Monitor APTT 5–15 min after dose then in 2–8 h or monitor ACT.
- ❑ **Accidental overdoses of heparin:** Consider $t_{1/2}$ heparin 60–90 min
 - In setting without bleeding complications, consider observation, rather than reversal of anticoagulation with protamine (avoids ADR's)
- ❑ **Dalteparin or tinzaparin overdose**
 - 1 mg protamine for 100 units' dalteparin or tinzaparin
 - If PTT prolonged 4 h after overdose, administer protamine 0.5 mg per 100 units of dalteparin or tinzaparin.
- ❑ **Enoxaparin overdose**
 - 1 mg per mg enoxaparin (if enoxaparin overdose given within 8 h)
 - If >8 h of overdose or bleeding continues after 4 h after first dose, give 0.5 mg protamine per mg enoxaparin.

Time Elapsed Since Heparin Dose

Dose of protamine (mg) to neutralize 100 units of heparin:

- ❑ <1/2 h: 1–1.5 mg/100 units of heparin
- ❑ 30–120 min: 0.5–0.75 mg/100 units of heparin
- ❑ >2 h: 0.25–0.375 mg/100 units of heparin.

Adverse Effects

Allergic reactions may manifest as urticaria, hypotension, pulmonary hypertension, bronchoconstriction, facial numbness, and cardiovascular collapse.

Non-IgE-mediated anaphylactoid reactions to protamine sulfate are more common than IgE-mediated anaphylaxis.

Avoiding rapid infusion of protamine sulfate and pre-treating at-risk patients with histamine receptor antagonists (H_1 and H_2) and steroids may minimize these reactions. A 5–10 mg test dose is recommended following pretreatment before administering the full dose.

SUGGESTED READING

1. Butterworth J. Cardiovascular drugs: practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravelee GP (Eds), 5th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia. 23–88.

Section 2

Cardiac Anesthesia

Section Outline

- Congenital Heart Disease
- Valvular Heart Disease
- Anesthesia for Coronary Artery Bypass Grafting
- Anesthesia for Vascular Surgery
- Anesthesia for Pericardiectomy
- Anesthesia for Cardiac Catheterization: Laboratory Procedures
- Pregnancy and Cardiac Surgery

Chapter 6

Congenital Heart Disease

Manjula Sudeep Sarkar, Neeraj Barnwal, Shraddha Mathkar, Sachin Vaishnav

ACYANOTIC CONGENITAL HEART DISEASE

Manjula Sudeep Sarkar, Neeraj Barnwal

Congenital heart disease (CHD) has an incidence of 6 in every 1,000 live births. Acyanotic congenital heart disease accounts for 70% of all congenital heart disease.

Acyanotic congenital heart defects may be due to obstructive lesions (stenosis) or left-to-right shunts. Lesions with left-to-right shunts include atrial septal defect, ventricular septal defect, and patent ductus arteriosus. Obstructive lesions include pulmonary stenosis, aortic stenosis, and coarctation of the aorta.

These defects represent abnormal communication between the high-pressure left side of the heart and the low-pressure right side of the heart. The pressure differential results in left-to-right shunting of blood through the defect consequently leading to turbulence of blood flow producing a heart murmur in systole and sometimes in diastole. Excessive blood flow into the lungs causes pulmonary vascular congestion leading to shortness of breath, increased volume overload of the myocardium resulting in hypertrophy and chamber dilation, and eventual CHF.

These babies are “pink” and present with symptoms of congestive heart failure, which may include poor weight gain, feeding or exercise intolerance (baby sweats when it eats), or prolonged recovery from simple respiratory infections. Patients retain normal levels of oxyhemoglobin saturation in systemic circulation.

The management of acyanotic heart defects depends upon the degree of the defect, the symptoms the defect causes and the age of the person with the defect.

Etiology

Most cases (70–80%) are multifactorial and 6–12% of cases are associated with gross chromosomal anomalies. Associated chromosomal anomalies are:

- ❑ Trisomy 21 (40% have CHD): AV canal defect
- ❑ Trisomy 18 (100% have CHD): VSD, PS
- ❑ Trisomy 13–15: VSD, ASD, TGV
- ❑ XO (Turner syndrome): Coarctation of aorta, AS, VSD
- ❑ XXY (Klinefelter syndrome): Ebstein anomaly, tetralogy of Fallot

GENERAL ANESTHETIC CONSIDERATIONS IN PEDIATRIC OPEN HEART SURGERY

Preoperative Assessment

It includes detailed information about cardiac lesion, altered physiology and its implications. These includes:

- a. Complete understanding of the anatomical changes due to cardiac defect or palliative procedure.
- b. Direction and amount of shunting.
- c. Presence and severity of pulmonary hypertension.
- d. Extent of reduced or increased pulmonary flow.
- e. Degree of hypoxemia, polycythemia: Fatigue, headache, visual disturbances, depressed mentation and paresthesia of toes and fingers are presenting symptoms of polycythemia. It leads to thrombosis and infarction in cerebral, renal and pulmonary region.
- f. Coagulation abnormalities: Due to hypofibrinogenemia and factor deficiencies.
- g. Functional status of the patient: Fatigue and dyspnea on feeding and irritability indicate poor functional status.
- h. Respiratory reserve: Presence of increased respiratory rate, diaphoresis, intercostal muscles retraction, nasal flaring, and use of accessory respiratory muscles indicate poor respiratory reserve.
- i. Compromised heart may show signs of failure, i.e. tachycardia with low volume pulse, a gallop rhythm, tachypnea, difficulty in feeding, excessive perspiration, jugular venous distention, pulmonary congestion or hepatomegaly.
- j. Associated anomalies or syndromes: These include musculoskeletal abnormality (8.8%), neurological defects (6.9%), and genitourinary irregularities (5.3%), the most common is the Down's syndrome (9%). Atlanto-occipital subluxation is common in Down's syndrome which can lead to quadriplegia during laryngoscopy and tracheal intubation.

Investigations

- ❑ Polycythemia is very common which increases blood viscosity. Consider phlebotomy in patients with symptomatic hyperviscosity and hematocrit >65%.
- ❑ Coagulation abnormalities: Platelet count, PT and PTT should be done in all patients.
- ❑ Electrolytes: Done in patients who receive diuretics, digitalis and parental nutrition.

- ❑ Chest X-ray: Heart position (Dextrocardia) and cardiomegaly, atelectasis, acute respiratory infection, vascular markings and elevated hemidiaphragm. High pulmonary flow will lead to increased pulmonary marking while reduced flow causes oligemic lung fields.
- ❑ ECG: It may show ventricular strain or hypertrophy.
- ❑ Echocardiography: For Doppler and color flow mapping.
- ❑ Catheterization: Used for information about pressures in different chambers, magnitude of shunt and coronary anatomy.
- ❑ Blood gas analysis: In patients with cyanotic defects.

Monitoring

Standard ASA monitoring for the children with CHD undergoing cath lab procedure includes: ECG, NIBP, SpO₂, EtCO₂, FiO₂, temperature and airway pressure.

Additional monitoring, IBP, CVP, TEE and urinary catheter may be warranted on an individual basis when either the child's presentation or proposed intervention or anesthetic management predicts the likelihood of circulatory instability.

Caution: In the presence of R to L shunt, the end-expiratory CO₂ consistently underestimates the true arterial CO₂ level.

A fully equipped anesthesia workstation with airway and intubation equipment and emergency cardiorespiratory resuscitative drugs should be available. Pacemaker with external pacing pads and a defibrillator with the appropriate sized pediatric paddles should be confirmed.

Make the chart that will enumerate the calculated dosage, volume and the infusion rate of the anesthetic, resuscitative and vasoactive agents as well as the maintenance fluid in a closed system using programmable syringe pumps.

All intravenous tubing's and syringes should be free of air bubble to prevent paradoxical embolism.

ATRIAL SEPTAL DEFECT

Atrial septal defect (ASD) is a defect in the septum dividing the right and left atria. These account for 10% of all CHD and are more common in females. In our institute, ASD comprises ~18–19% among all congenital heart surgeries in last 5 years.

Introduction

Embryology

The embryonic common atrium undergoes partitioning by the formation of two parallel, overlapping septa, starting in the 4th week of gestation.

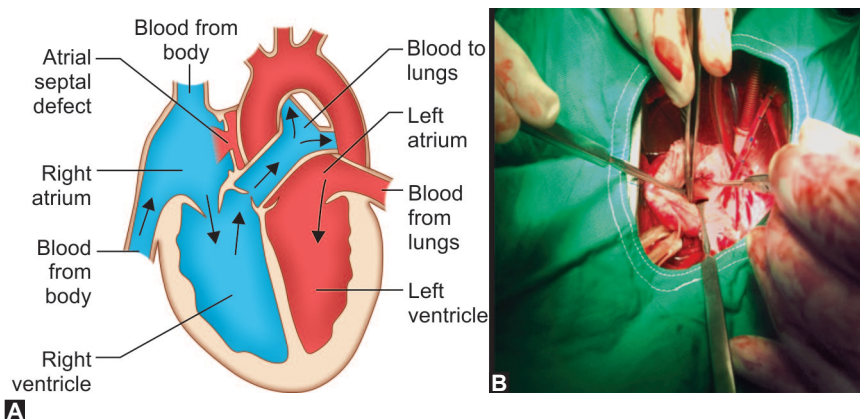
The crescentic septum primum, emerging at the superior and posterior aspect of the left atrial heart field, begins to septate the atria as the endocardial cushion. By the end of 6th week of gestation, the septum secundum begins to form parallel to and immediately rightward of the septum primum, obliterating the remaining ostium primum and circumscribing the fossa ovalis.

Final configuration: The atrial septum consists of two layers, fused all over except for the overlapping offset openings of the fossa ovalis and the ostium secundum. The free edge of the ostium secundum forms a flap valve covering the left side of fossa ovalis, providing free right to left flow through foramen ovale until postnatal physiology closes the valve.

Types of ASD

Classification based on location relative to fossa ovalis and developmental anatomy.

- 1. Secundum atrial septal defect:** The secundum ASD is contained within the area bordered by the limbus of the fossa ovalis. It results from an abnormal reabsorption of the septum primum or defective formation or shortening of the septum secundum. Combinations of these abnormalities may contribute to large defects (Figs 1A and B).
- 2. Primum atrial septal defect:** The primum ASD results from abnormalities in formation of the septum primum. It is frequently associated with atrioventricular canal (AVC) defects, especially the partial atrioventricular canal (PAVC) that includes a cleft in the anterior leaflet of the left atrioventricular valve. These AVC defects are due to defects in fusion of the endocardial cushions.
- 3. Sinus venosus atrial septal defect:** Sinus venosus defects result from abnormal development of the septum secundum or the sinus venosus, the primitive venous collecting chamber. The most common type is located near the superior vena cava (SVC) orifice and is associated with partial anomalous pulmonary venous return (PAPVR) involving the right upper and middle pulmonary veins. Defects near the orifice of the inferior vena cava also exist and may involve PAPVR of the right lower pulmonary vein.
- 4. Patent foramen ovale:** Patent foramen ovale results from failure of fusion of the septum primum to the limbus of the septum secundum. Patent foramen ovale is normal during fetal life as blood passes from right to left bypassing the lungs in fetal circulation. Following birth, as the PVR drops and SVR increases, the foramen ovale closes, but may not fuse.



Figs 1A and B: (A) ASD; (B) Surgeon has put forceps into ASD

Pathophysiology and Natural History

The amount of left-to-right shunting at the atrial level is dependent upon two factors, the size of the defect and the relative compliance of the right and left ventricles. Shunting occurs primarily during diastole, when the atria contract and atrioventricular valves open, and produces a volume burden on the cardiovascular system that is proportionate to the amount of shunting.

Isolated ASDs are usually asymptomatic in infants and during childhood despite the increased volume load on the right ventricle. Congestive heart failure usually occurs after the second or third decade of life due to chronic right ventricular volume overload.

Pulmonary hypertension can occur in up to 13% of unoperated patients younger than 10 years of age; however, progression to Eisenmenger's syndrome is unusual.

The increase in right atrial size may predispose to atrial arrhythmias, and patients with a Qp: Qs of 2: 1 or less have an 11% incidence, whereas those with a Qp: Qs of 3: 1 or greater have a 38% incidence of atrial arrhythmias.

Preoperative

Clinical Features

- 1. Signs and symptoms:** Dyspnea and fatigue are early symptoms of OS-ASD. Orthopnea occurs because the supine position increases the work of breathing in patients with reduced lung compliance. Recurrent lower respiratory tract infections are common especially in children.
- 2. Physical appearance:** Children with an ASD may have a delicate gracile habitus, with weight more affected than height. They may have a left precordial bulge with Harrison's grooves. The distinctive physical appearance of the Holt-Oram syndrome heightens suspicion of a coexisting OS-ASD.
- 3. Jugular venous pulse:** Left atrialization of the jugular venous wave form is seen. The crest of A and V waves tend to be equal as they are in the left atrium.
- 4. Precordial movement and palpation:** Impulse is hyperdynamic but not sustained because the volume overloaded right ventricle empties rapidly into a low resistance pulmonary vascular bed. Impulse is especially prominent at left sternal border during held exhalation and in the subxiphoid area during held inspiration.
- 5. Auscultation:**
 - First heart sound is split at the lower left sternal edge and apex, and the tricuspid component is loud.
 - The pulmonary midsystolic flow murmur begins immediately after S₁ is of Grade 2/6 or 3/6, maximal in the second left intercostal space and is crescendo-decrescendo type, radiating to apex because the right ventricle occupies the apex.
 - The pulmonary component of second heart sound is prominent because of proximity of the dilated pulmonary trunk to the chest wall. Wide fixed

splitting of second heart sound is the auscultatory hallmark of ASD. The aortic and pulmonary components are widely split during expiration, and the degree of splitting does not change during inspiration or during the Valsalva's maneuver.

6. Electrocardiogram:

- Sinus node dysfunction identified as early as age 2 to 3 years. The incidence of atrial fibrillation, atrial flutter and supraventricular tachycardia increases in the fourth decade. Atrioventricular conduction defects are intrinsic component of ASD and are usually age related.
- Abnormal right atrial P waves are peaked rather than tall, but P wave configuration is usually normal. An electrocardiogram hallmark of ASD is an rSr prime or an rsR prime in right precordial leads.

7. Chest X-ray (Fig. 2):

- Increased pulmonary arterial vascularity extends to the periphery of the lung fields.
- The pulmonary trunk and its proximal branches are dilated. Infants with large left to right shunt exhibit both pulmonary arterial and venous vascularity with enlargement of all four cardiac chambers.
- Right atrial enlargement is characteristic, but the left atrium seldom enlarges.
- An enlarged right ventricle occupies the apex and forms an acute angle with the left hemidiaphragm.

8. Echocardiography: The subcostal view provides the best visualization of ASD size and location (Fig. 3). Echo with color flow establishes the location and size of an ASD as also its physiologic consequences.

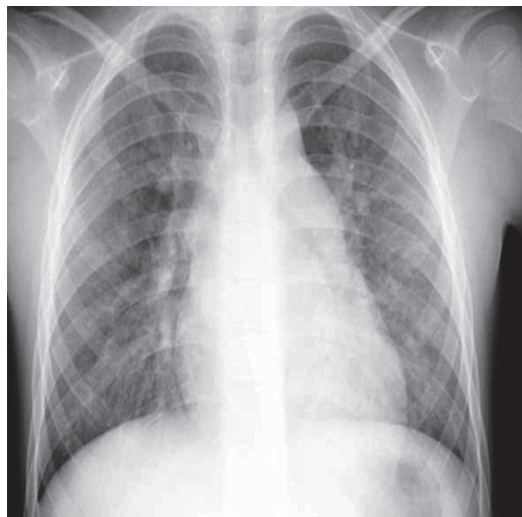


Fig. 2: Chest X-ray in ASD

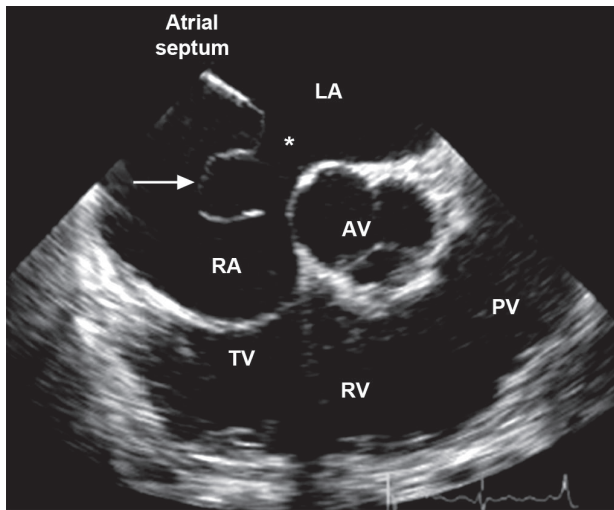


Fig. 3: TEE image showing ASD

Anesthetic Considerations for Intracardiac Repair

Key points

1. Avoid air bubbles in intravenous lines due to risk of paradoxical emboli.
2. The length of IJV line should be precisely guided to avoid inadvertent entry of air into left atrium via ASD. This may predispose to arrhythmia and air embolism.
3. Tailor anesthetic technique to allow early extubation.

Premedication

Oral, nasal or rectal drugs can be used, depending on patient's condition and preference. If intravenous access is present—IV midazolam 0.03–0.05 mg/kg is quite effective in reducing anxiety and improving patient cooperation.

Monitoring

Standard ASA monitoring plus invasive blood pressure via arterial catheter and central venous pressure via central venous catheter usually through right internal jugular cannulation. During surgery, TEE can be helpful to assess de-airing of the left heart and adequacy of the repair.

Induction

- ❑ Patients with ASD are generally asymptomatic, and do not have pulmonary hypertension. The induction of anesthesia can be easily tailored to either inhalation or intravenous technique.
- ❑ Theoretically, inhalation induction is more rapid in patients with a left to right shunt because the alveolar concentration of anesthetic more rapidly approaches the inhaled concentration.

- ❑ Sevoflurane (up to 4–8 %) can be used for induction. Once IV access is obtained, the primary anesthetic is combined technique including volatile agent (Sevoflurane), opioids (IV fentanyl 10 µg/kg) and neuromuscular blocking drugs (IV rocuronium 1 mg/kg). Intravenous induction is preferred in sick patients having severe pulmonary hypertension and drugs need to be titrated cautiously during induction.
- ❑ Maintenance of anesthesia may consist of inhaled agents (Isoflurane), intravenous agents (IV midazolam 0.05–0.1 mg/kg/hr and IV fentanyl 2–3 µg/kg/hr) and muscle relaxant (IV vecuronium 0.08–0.1 mg/kg/hr).
- ❑ The majority of patients have good myocardial function and do not require inotropic support perioperatively. Tracheal extubation in the operating room has been shown to decrease patient charges, without compromising patient care when compared to extubation in the intensive care unit.
- ❑ Whatever technique is chosen, the primary goal for the uncomplicated ASD patient should include preparation for an early extubation either in the operating room or early postoperatively.

Surgical Approaches

Surgical repair of an ASD is usually recommended between the ages of 3 and 5 years. Spontaneous closure of small secundum type ASD can occur in up to 87% of infants in the first year of life, and controversy exists regarding the closure of small ASDs that are asymptomatic.

Conventional surgical treatment involves median sternotomy with the use of CPB to perform a primary repair or patch closure (Fig. 5), with surgical mortality approaching 0%.

Sinus venosus defects are usually repaired using a patch to close the ASD and baffle the anomalous pulmonary veins to the left atrium. Many centers now favor minimally invasive surgery via a partial sternotomy, because of the advantage of improved cosmetic result with morbidity and mortality being similar to complete sternotomy.

Transcatheter Closure Techniques

- ❑ Transcatheter ASD closure (Fig. 4) in the cardiac catheterization laboratory has dramatically reduced the number of operative repairs. The CardioSEAL septal occluder and the Amplatzer septal occluder are the most common devices used. These procedures are usually performed under general anesthesia with the use of TEE to guide placement.
- ❑ Transcatheter closure is safe, associated with decreased hospital stay, lack of a surgical scar, avoidance of CPB, and a reduced need for general anesthesia.

Limitations: Transcatheter closure of ASD is based on patient size (large introducer sheaths), type of ASD (usually only PFO or secundum), and requires the presence of an adequate tissue rim for the device to attach.

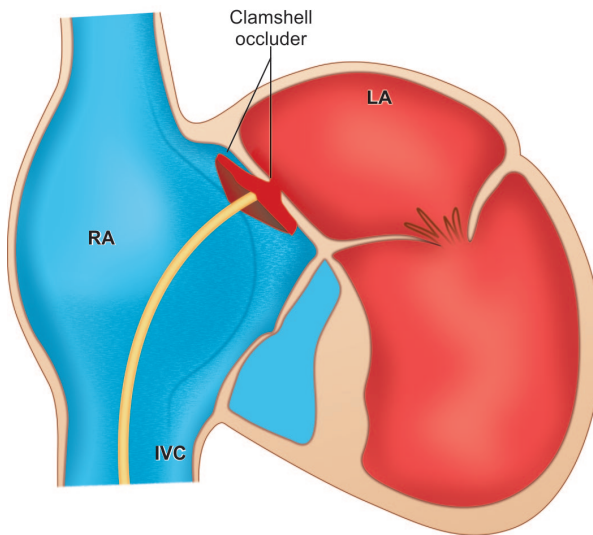


Fig. 4: Transcatheter closure of ASD

Postoperative

Postoperative dysrhythmias are reported in 23% of patients, and as many as 2% of patients may need a pacemaker following surgery. The incidence of dysrhythmia is higher after surgical repair in adults. Atrial fibrillation is the main cause of morbidity in adults with ASD because of the possibility of thromboembolism.

One needs to remember that postoperative fluid management in these patients is tricky because CVP guided fluid administration often leads to pulmonary edema because of the highly compliant right atrium which underestimates CVP.

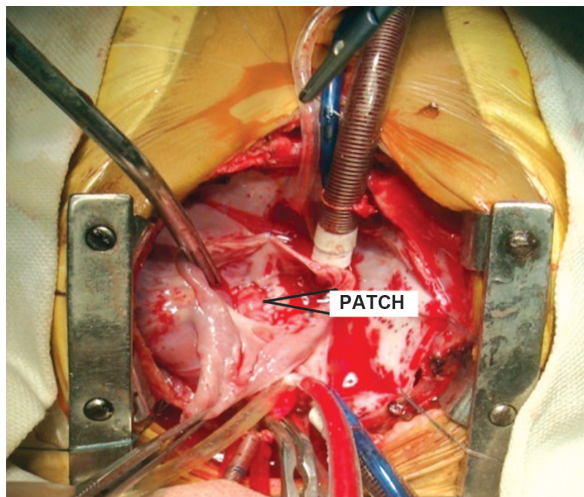


Fig. 5: Pericardial patch closure of ASD (postrepair appearance)

VENTRICULAR SEPTAL DEFECT

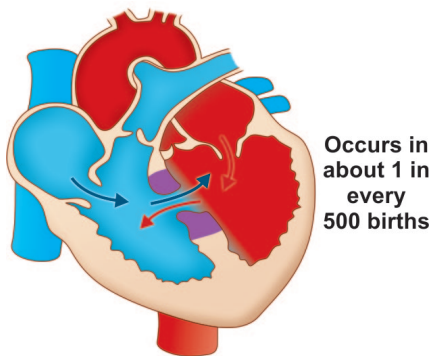
Introduction

Ventricular septal defect is the most common congenital heart defect, comprising approximately 20% of all congenital heart defects, with an incidence between 2.6 and 5.7 in 1,000 live births. In our institute, VSD constitutes 30–32% among all congenital heart surgeries in last five years.

VSD is associated with a variety of inherited conditions, including trisomy 13, 18, and 21 as well as the VACTERL (vertebral, vascular, anal, cardiac, tracheoesophageal, renal, and limb anomalies) association and CHARGE (coloboma, heart anomaly, choanal atresia, retardation, and genital and ear anomalies) syndrome.

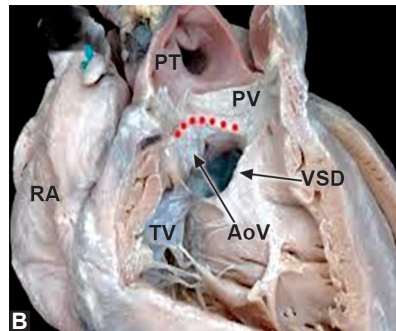
Embryology

The primitive left ventricle is formed from the ventricular portion of the bulbus cordis and the primitive right ventricle is formed from the proximal portion at approximately 23–25 days gestation. A communication between the right and left ventricles defines a VSD (Figs 6A and B).



Ventricular septal defect: The superior part of the inter-ventricular septum fails to form; thus, blood mixes between the two ventricles, but because the left ventricle is stronger, more blood is shunted from left to right

A



B

Figs 6A and B: (A) Embryology of VSD; (B) Dissected heart showing VSD

Anatomical Classification

- i. Perimembranous (80%); (ii) Subarterial/subpulmonary/outlet VSD (5–7%); (iii) Muscular (5–20%); (iv) Swiss cheese/multiple VSD's; and (v) AV canal/inlet VSD (5–8%).
- 1. Perimembranous ventricular septal defect:** The perimembranous VSD is a communication adjacent to a portion of the membranous septum and the

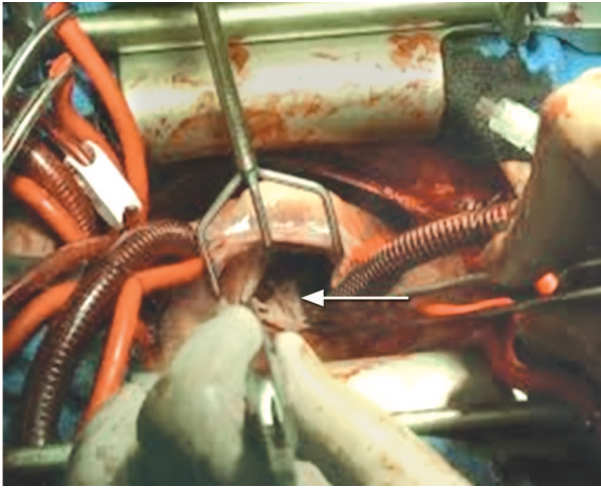


Fig. 7: Intraoperative photograph of VSD (arrow)

fibrous trigone of the heart, where the aortic, mitral, and tricuspid valves are in fibrous continuity. These infracristal defects are the most common VSD subtype, occurring in approximately 80% (Fig. 7).

- 2. Subpulmonary ventricular septal defect:** The subpulmonary (subarterial, supracristal, outlet, infundibular) VSD is located within the outlet septum, above the crista supraventricularis and border of the semilunar valves; and comprises approximately 5–7% of all VSDs. As a result of the location of this defect, a venturi effect may be produced by the jet of blood flowing through the VSD causing the right or noncoronary aortic cusp of the aortic valve to prolapse toward the defect producing aortic insufficiency. This type of lesion is more common in the Asian population.
- 3. Muscular ventricular septal defect:** Muscular VSDs are located within the muscular portion of the interventricular septum. These defects can be multiple and represent approximately 2–7% of VSD. Multiple (>3) defects are also called Swiss-Cheese defects.
- 4. Malaligned ventricular septal defect:** Malaligned VSDs occur from malalignment of the infundibular septum and the trabecular muscular septum. These defects usually occur as a component of a more complex cardiac defect, most commonly tetralogy of Fallot.
- 5. Canal type ventricular septal defect:** Canal type (Inlet) VSD is located in the posterior region of the septum beneath the septal leaflet of the tricuspid valve. These inlet defects accounts for approximately 10% of VSDs.

Pathophysiology and Natural History

Isolated VSDs produce left-to-right shunting at the ventricular level, predominantly during systole. The pathophysiologic consequences of a VSD are shunting, pulmonary hypertension and CHF, and depend essentially on the size of the defect and the pulmonary vascular resistance.

Restrictive VSD refers to a limitation in the amount of flow across the defect based on size; and in this case a pressure gradient exists between the left and right ventricles. It causes little or no functional derangement because the shunt is small, and the pressure and resistance in the pulmonary circulation are normal.

Moderately restrictive VSD is characterized by elevated right ventricular pressure above normal but less than systemic and by low but variable pulmonary vascular resistance.

Nonrestrictive/unrestrictive VSD, the right and left ventricles behave physiologically as a common chamber. It has flow limited only from the relative pulmonary vascular resistance to systemic vascular resistance; and therefore, no pressure gradient exists between the left and right ventricles.

Fifteen percent of patients with large VSDs develop pulmonary hypertension which will progress to the development of pulmonary vascular obstructive disease and Eisenmenger syndrome (PA pressures greater than systemic pressures, reversal of shunt, and cyanosis) by the age of 20 years.

Spontaneous closure of small perimembranous and muscular VSDs occurs in as many as 50% of patients, and such patients are typically asymptomatic. Defects up to 5 mm rarely require surgery, whereas defects 6.5 mm or larger almost always require surgery.

Preoperative

Clinical Features

- 1. Signs and symptoms:** Symptoms range from asymptomatic to signs and symptoms of CHF. The rate and degree of progression of symptomatology depends on the patient age, size of the defect, and the degree of left-to-right shunting. Infants who have unrestrictive VSDs develop symptoms of CHF in the first 3 months of life due to increased pulmonary blood flow. Pulmonary congestion leads to repeated respiratory tract infections.
- 2. Physical appearance:** Failure to thrive due to catabolic effects of congestive heart failure. Harrison's grooves are caused by the thoracic retractions of chronic dyspnea. The physical appearance of trisomy 18 Down syndrome coincides with the presence of an inlet VSD.
- 3. Arterial pulse:**
 - Moderately restrictive VSD with relatively low PVR: Brisk arterial pulse.
 - Nonrestrictive VSD with large left to right shunt: Diminished arterial pulse with pulsus alternans.
- 4. Jugular venous pulse:**
 - Elevated mean jugular pressure and an increase in A and V waves in moderately restrictive and nonrestrictive VSD with congestive heart failure.
 - However, the jugular venous pulse is normal in Eisenmenger's syndrome.

- 5. Precordial movement and palpation:** Restrictive VSD has palpable harsh thrill maximal in the 3rd or 4th left intercostal space at the left sternal border. Nonrestrictive VSD is associated with a dynamic volume overloaded left ventricle, a pressure overloaded right ventricle and a palpable pulmonary closure sound.
- 6. Auscultation:**
- **Restrictive VSD:** Soft localized, high frequency, decrescendo, early systolic murmur.
 - **Moderately restrictive/nonrestrictive VSD** with PVR below systemic vascular resistance: Loud harsh holosystolic murmur, high frequency, grade 4/6, and maximal in the 3rd and 4th intercostal space at the left sternal border.
 - **Nonrestrictive VSD with PVR approaching SVR:** The holosystolic murmur shortens and softens, and its shape becomes decrescendo. The murmur is early systolic before disappearing altogether as the shunt is reversed.
 - **Second heart sound:** Increase in intensity of the pulmonary component. As PVR increases, the degree of splitting decreases, so the 2nd heart sound is single in Eisenmenger's syndrome.
- 7. Electrocardiogram:**
- **Restrictive VSD** with normal pulmonary artery pressure: Normal ECG and occasional rsr prime pattern in lead V1.
 - **Moderately restrictive VSD** with large left to right shunt: Broad notched left atrial P waves in lead 1 and 2 and with a broad deep P terminal force in lead V1. The QRS axis is normal.
 - **Nonrestrictive VSD** with large left to right shunt: Right atrial or combined right and left atrial P waves abnormalities, especially in lead 2 and in lead V1-V2. The QRS axis shifts to right. Biventricular hypertrophy (increased R wave amplitude in lead V1, the deep Q waves, tall R waves, and tall peaked T waves in leads V5-V6).
- 8. Chest X-ray (Fig. 8)**
- In patients with small VSDs, the chest radiographs are usually normal.
 - **Moderately restrictive VSD** with low PVR, increased pulmonary arterial vascularity and pulmonary venous congestion coexist. Large shunts in infant are accompanied by hyperinflated lungs with flat hemidiaphragms. Right atrial dilation accompanies CHF. Enlargement of left atrium and an increase in size of the pulmonary trunk and its branches reflect the magnitude and chronicity of pulmonary arterial blood flow and the level of pulmonary arterial pressure.
 - **Nonrestrictive VSD** with elevated but variable PVR present in infancy with CHF, pulmonary venous congestion, and enlargement of all four chambers.



Fig. 8: X-ray chest showing prominent pulmonary plethora in VSD

- 9. Echocardiography:** Multiple views are required. It is used to diagnose and determine the size and location of VSD. Estimates of right ventricular and PA pressures are obtained and presence or absence of a gradient is noted. The size is expressed relative to aortic root size (Fig. 9).

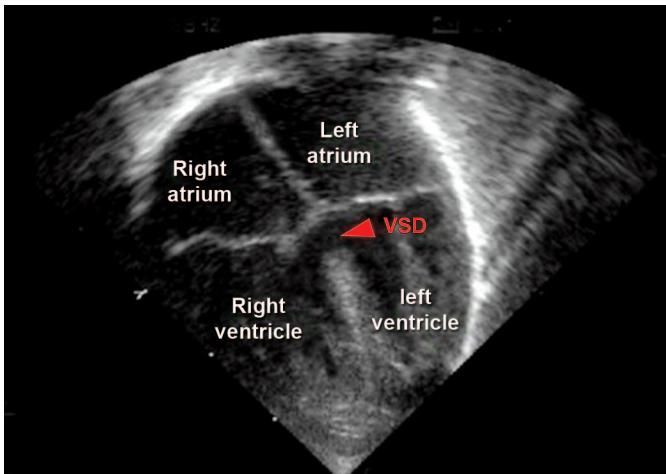


Fig. 9: TTE image showing VSD

- 10. Cardiac catheterization:** It is sometimes indicated to calculate PVR and Qp/Qs ratio in patients who present late especially in our country. This helps to assess feasibility of surgical repair.

Intraoperative

Key points

1. Maintain pulmonary vascular tone prior to repair in patients with pulmonary overcirculation.
2. Diagnose and treat possible dysrhythmias, especially heart block.
3. Patients with uncomplicated VSDs should be considered for early extubation.

Anesthetic management for the patient with VSD is similar to that of ASD. Additional considerations for patients with CHF are to minimize maneuvers that excessively lower PVR (hyperventilation, anemia) and to avoid myocardial depression. **Premedication** is needed in older children in order to avoid exacerbation of pulmonary hypertension. Children less than 6 months usually do not require premedication.

These patients usually receive anti-failure treatment in the form of digoxin and diuretics. So preoperatively potassium level should be optimized.

Pulmonary hypertension may develop early, especially in patients with trisomy 21, and preoperative chest radiograph revealing decreased pulmonary vascular markings is indicative of pulmonary hypertension. Such patients may respond to the use of inhaled NO and/or IV milrinone (50 µg/kg bolus followed by 0.375 to 0.75 µg/kg/min) prior to termination of CPB and/or in the postoperative period. Right heart failure with decreased CO may result if pulmonary hypertension is not controlled, and responds to the use of dopamine, milrinone, dobutamine, or isoproterenol.

Conduction disturbances, particularly atrial-ventricular heart block may be transient or permanent and is reported to occur in up to 10% of patients post-VSD repair. If heart block develops, treatment with atrioventricular synchronous pacing using temporary pacing wires is indicated.

Junctional ectopic tachycardia is sometimes observed in patients less than 1 year of age after repair for lesions that involve VSD, most commonly after tetralogy of Fallot repair. Treatment includes cooling to 35°C, increased anesthetic depth, paralysis, procainamide, esmolol, or amiodarone.

Intraoperative use of TEE will help recognize residual VSDs, intracardiac air, and assess ventricular volume and function. Small muscular VSDs will become apparent after closure of larger VSDs. Frequently these smaller defects, especially if near the apex, may not be amenable to surgical repair or worth the risk of returning to CPB.

Weaning off CPB: Use of pulmonary vasodilator needs to be considered after repair because muscular VSD's are prone to develop pulmonary hypertensive crisis postoperatively within 48 hours.

Antiarrhythmic drugs may be required in case of conduction disturbances which may resolve once edema near conduction tissue subsides. Steroids might be useful in this regard.

Postoperative

Patients with increased PVR may require sedation, continued use of pulmonary vasodilator (IV milrinone 0.375–0.78 $\mu\text{g}/\text{kg}/\text{min}$), elective mechanical ventilation, and aggressive diuresis for 48 to 72 hours.

Patients with uncomplicated VSDs are good candidates for extubation in the operating room or early after arrival in the intensive care unit.

Aortic regurgitation may occur after repair of perimembranous VSD. So postoperative risk of LV dysfunction should be kept in mind.

Junctional ectopic tachycardia is sometimes observed in patients less than 1 year of age after repair for lesions that involve VSD, most commonly after tetralogy of Fallot repair. Treatment includes cooling to 35°C, increased anesthetic depth, paralysis, procainamide, esmolol, or amiodarone.

Analgesia: Various modes of analgesia like opioids, tramadol, paracetamol, Dexmedetomidine infusion are practised worldwide. Use of NSAID's is avoided due to risk of renal injury.

Surgical Approaches

Indications for surgery

1. Timing for surgical repair depends on age and clinical findings.
2. Patients less than 6 months of age are repaired if they manifest uncontrollable CHF and failure to thrive.
3. Patients between 6 and 24 months of age undergo repair to treat CHF symptoms or pulmonary hypertension.
4. Patients older than 24 months undergo repair for Qp: Qs greater than 2: 1.
5. Among patients with subpulmonary VSD, the presence of aortic insufficiency is an indication for surgical repair to prevent further progression of the valvular insufficiency.
6. A defect greater than 5 mm is repaired to avoid progression to aortic cusp prolapse and aortic insufficiency, while defects less than 5 mm can be managed conservatively.

Surgical repair of VSD usually involves patch closure or occasionally primary closure using CPB via median sternotomy. Perimembranous and canal type VSDs are most commonly repaired via a right atriotomy, which may require detachment of the septal leaflet of the tricuspid valve for exposure. Subpulmonary VSDs are most commonly repaired via the transpulmonary approach. Midmuscular VSDs (Fig. 9) are most commonly repaired via right atriotomy, and anterior or apical muscular VSD may be approached using right ventriculotomy. However, the use of right ventriculotomy carries the risks of conduction disturbances and ventricular dysfunction later in life.

At many institutions, symptomatic patients with lesions that are not approachable via right atriotomy are usually treated with pulmonary artery banding until the patient is larger to allow transatrial repair. Pulmonary artery banding is also utilized for multiple muscular VSDs and in patients that are high-risk candidates for CPB.

Transcatheter Closure Techniques

- ❑ Transcatheter closure of muscular VSDs has been performed successfully. The use of the CardioSEAL septal occluder is approved.
- ❑ Indications for the use of devices include all types of muscular VSDs, including apical and multiple.
- ❑ Transcatheter techniques may serve as an adjunct to surgery or an alternative to surgery in selected patients. The major limitation in the application of this technique is related to the size of the sheaths necessary for device delivery, precluding use in infancy.
- ❑ Complications of device closure include need for blood transfusion, tricuspid valve regurgitation, and device embolization.

Long-term Outcome

Mortality for uncomplicated VSD in older patients is less than 1–2%. Mortality for VSD repair in infants during the first year of life is less than 5%. Patients with associated cardiac defects and multiple muscular VSD have poorer outcome. Incidence of residual shunt is 0.5 to 2 %.

PATENT DUCTUS ARTERIOSUS

Introduction

The ductus arteriosus is an essential component in normal fetal circulation; it becomes functionally closed within 10–15 hours after birth, and permanently closes by thrombosis, intimal proliferation, and fibrosis in the first 2–3 weeks after birth.

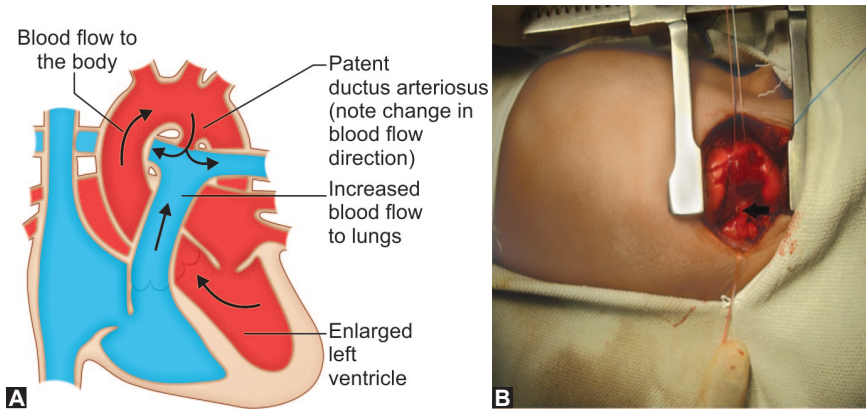
Functional closure is initiated by several mechanisms including aeration of the lungs, removal of prostaglandins produced in the placenta, increased arterial PO_2 , and release of vasoactive substances (bradykinin, thromboxanes, and endogenous catecholamines).

Isolated persistent patent ductus arteriosus (PDA) occurs in approximately 1: 2500 to 1: 5000 live births, the incidence is higher for premature births and this defect is two to three times more common in females than in males. In our institute, PDA ligation constitutes 6–7% among all congenital heart surgeries in last five years.

PDA is also found as part of other complex congenital heart defects. Rubella, prematurity and hypoxemia are predisposing risk factors for PDA.

Embryology

The ductus arteriosus arises from the distal portion of one of the sixth paired aortic arches. The PDA is a vascular communication between the descending aorta and pulmonary artery. The PDA most commonly arises from the aorta, just distal to the left subclavian artery and attaches to the left pulmonary artery (Figs 10A and B).



Figs 10A and B: (A) PDA; (B) Large PDA (arrow)

Pathophysiology

The degree of left-to-right shunting depends on, the duct size as well as the relative ratio of PVR and SVR. The shunt dimensions of importance include the diameter and length of the PDA; shorter connections with larger diameters produce less resistance, i.e. allow greater flow. The pathophysiology of a PDA is similar to a VSD or other lesions with left to right shunt. Both shunt flow and pulmonary edema increase the right heart workload.

In patients with large PDAs, the diastolic runoff into the pulmonary artery results in lowered aortic diastolic pressure, compromising peripheral perfusion which may increase the risk of myocardial ischemia, especially in the presence of anemia or lowered SVR.

A small PDA may be hemodynamically insignificant and unrecognized. The larger the PDA or left-to-right shunt, the more likely the progression to CHF, pulmonary hypertension, and in extreme cases, reversal of the shunt.

In premature infants, PDA is associated with increased morbidity from associated respiratory distress syndrome, necrotizing enterocolitis, and intracranial hemorrhage.

Preoperative

Clinical Features

1. **Signs and symptoms:** Children with PDA are usually asymptomatic. Babies may present with recurrent respiratory tract infections. Signs of isolated PDA in the child can include tachypnea, diaphoresis, decreased exercise tolerance, failure to thrive and other signs of CHF.
2. **Physical appearance:**
 - Maternal rubella is associated with low birth weight and failure to thrive, irrespective of ductal patency, ductal size, or shunt volume. An underdeveloped child with PDA should, therefore, be examined for cataract, deafness, and mental retardation.

- Differential cyanosis and clubbing are important physical signs of PDA with reversed shunt.
3. **Arterial pulse:** Wide pulse pressure is present in PDA with large left-to-right shunt. The typical pulse is characterized by a brisk rise, a single or bisferiens peak, and a rapid collapse. The carotid, brachial, femoral, and even dorsalis pedis pulses can be bounding.
 4. **Auscultation:** Typical murmur is described as a continuous machine murmur, which gets louder throughout systole peaking at the second heart sound and then getting softer during diastole. The murmur is loudest in the first or second intercostal space at the left sternal border.
 5. **Electrocardiogram:** The ECG is usually normal. A nonrestrictive PDA with low PVR is associated with biatrial P waves and combined ventricular hypertrophy. Right ventricular hypertrophy is present if PDA has progressed to pulmonary vascular occlusive disease.
 6. **Chest X-ray:**
 - In a small PDA with limited left to right shunt, the chest X-ray is normal.
 - As the flow increases, the main PA and aortic knob become more prominent.
 - As the shunt flow continues to increase, there will be left heart enlargement and increase in pulmonary vascular markings indicative of failure.
 7. **Echocardiogram:** Echocardiography with color flow imaging and Doppler interrogation establishes the size of the PDA, the flow dynamics through the ductus, and the physiologic consequences of PDA (Fig. 11).

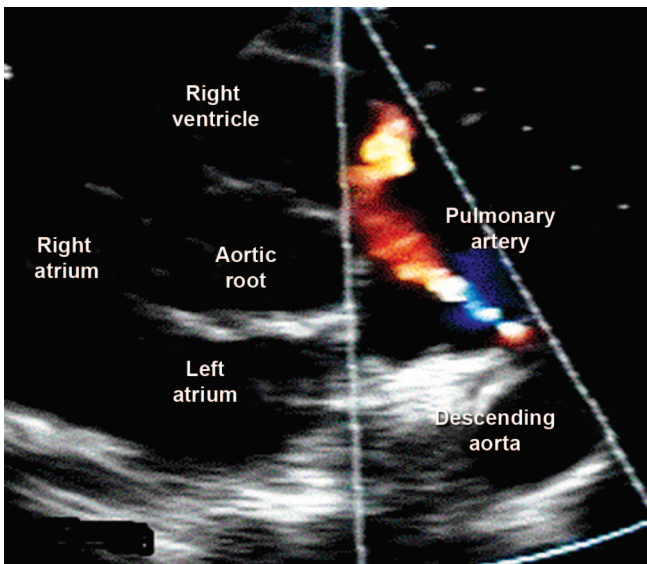


Fig. 11: TTE—color Doppler demonstrating mosaic pattern

Intraoperative

Key points

1. Avoid air bubbles in intravenous lines due to risk of paradoxical emboli.
2. Critically ill neonates may require a high dose narcotic technique to minimize the stress response to surgery.
3. Lung isolation is usually required for video-assisted surgery to allow adequate surgical exposure.

The anesthetic management for PDA ligation depends upon factors such as patient's clinical condition, prematurity, coexisting disease, body weight, and surgical technique.

Standard ASA monitors are used, along with pulse oximetry of both upper and lower extremities which will assist in detecting inadvertent ligation of the descending aorta. In addition, placing a noninvasive blood pressure cuff on both upper and lower extremities will assist in determining if the PDA ligation produced some degree of coarctation of the aorta. Special attention must be paid to temperature monitoring as neonates often land up in hypothermia which predisposes to arrhythmia and delayed recovery from anesthesia. Routine invasive monitoring is usually not required except in very sick neonates.

Large volume venous access (which may be a 22 or 24 gauge IV in a premature infant) is recommended because of the possible blood loss during division and ligation of PDA. Among patients with coexisting disease, intra-arterial pressure monitoring provides a method of assessing arterial blood gases, electrolytes, hematocrit, and acid-base status.

The volume of distribution for many drugs, such as gentamicin, vancomycin, amikacin, and fentanyl, is increased in PDA patients. Antibiotic prophylaxis is indicated whether the closure is done in the catheterization suite or operating suite.

Induction: Neonatal patients commonly develop hemodynamic instability from exposure to inhaled anesthetics and benefit from an intravenous anesthetic technique using opioids such as fentanyl (10–20 $\mu\text{g}/\text{kg}$) and possibly a benzodiazepine (IV midazolam 0.05 to 0.1 mg/kg) along with muscle relaxation (IV rocuronium 1 to 1.5 mg/kg or IV vecuronium 0.1 to 0.2 mg/kg). Fentanyl-based anesthesia reduces the neonatal stress response and improves postoperative outcome. Right lateral position is given in thoracotomy for PDA ligation. So, care should be taken to prevent brachial plexus injury and pressure point necrosis.

Many times, neonate comes to or in already intubated position and it is anesthesiologist's duty not to worsen left-to-right shunt further. Positive pressure ventilation and diuretics often are required in this regard. It is customary to use as low as FiO_2 as possible without compromising oxygenation.

Neonatal PDA ligation is often performed in the newborn intensive care unit to avoid the additional risks of transport, need for ventilator changes, and hypothermic exposure. It is advisable to restrict fluid administration in patients developing CHF prior to surgery.

For VATS, one lung ventilation (OLV) is required for which double lumen endotracheal tube is needed. In neonates, OLV is provided by advancing normal ETT into right bronchus. Relative hypoxia and hypercarbia during VATS in fact prevents pulmonary overcirculation and helps to mitigate left to right shunt further.

Postoperative

A technique providing good postoperative analgesia is important to enhance deep breathing and minimize pulmonary complications. Intravenous narcotics, intercostal nerve block, intrapleural local anesthetics are all effective. Caudal epidural analgesia provides excellent pain relief and helps in early extubation.

Transcatheter Closure Techniques

Many catheter methods have been developed to nonsurgically close a PDA and include the Gianturco coils, the Gianturco–Grifka coil bag and the Amplatzer duct occluder.

These methods are considered safe, efficacious, and cost effective when compared to surgical closure. Risks of transcatheter approaches include arrhythmias, embolization of the device, and incomplete closure. In addition, there are size limitations in small infants.

Surgical Approaches

In newborns, surgical treatment is usually reserved for patients who fail medical treatment with indomethacin, contraindications to indomethacin treatment, and for infants with necrotizing enterocolitis.

The usual surgical options include posterolateral thoracotomy with ligation or division of the PDA or video-assisted thoracoscopic surgery (VATS).

Surgical approaches have mortality rates approaching 0% and minimum morbidity; however, mortality rate in premature neonates is slightly higher.

Complications of surgical treatment include, bleeding, chylothorax, vocal cord paralysis (injury to recurrent laryngeal nerve), pneumothorax, atelectasis, recurrence of patency, and inadvertent ligation of the pulmonary artery or descending aorta.

SUGGESTED READING

1. Davis LK, Husain SA, Weitzel NS. Anesthesia management for patients with congenital heart disease: The pediatric population, a practical approach to cardiac anesthesia, 5th edition. In: Hensley FA, Martin DE, Gravelee GP (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 389-435.
2. Delphin E, Seckin AN, Moore RA. Preoperative evaluation and preparation, Pediatric cardiac anesthesia, 4th edition. In: Lake CL, Brooker PD (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 95-111.
3. Hudson JK, Deshpande JK. Septal and endocardial cushion defects, Pediatric cardiac anesthesia, 4th edition. In: Lake CL, Brooker PD (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 329-43.
4. Park MK. Left to right shunt lesions, Pediatric Cardiology for Practitioners, 5th edition, Mosby Elsevier, Philadelphia; 165-78.
5. Perloff JK, Marelli AJ. Perloff's clinical recognition of congenital heart disease, 6th edition, Saunders Elsevier, Philadelphia; 212-72, 283-315, 368-86, 359-64.
6. Russell IA, Miller HWC, Gregory G, Balea MC, Cassorla L, DeSilva A, et al. The safety and efficacy of sevoflurane anesthesia in infants and children with congenital heart disease. *Anesth Analg.* 2001;92(5):1152-8.

ANESTHESIA FOR CYANOTIC CONGENITAL HEART DISEASES

Manjula Sudeep Sarkar, Shraddha Mathkar, Sachin Vaishnav

TETRALOGY OF FALLOT

Introduction

Anatomy

Described by the French physician Etienne-Louis Arthur Fallot in 1888, tetralogy of Fallot (TOF) consists of four anatomical components (Figs 12A and B):

- ❑ Ventricular septal defect
- ❑ Abnormally positioned aortic valve above (overrides) the ventricular septum/overriding of aorta
- ❑ Right ventricular outflow tract (RVOT) obstruction
- ❑ Right ventricular myocardial hypertrophy.

There are four major variants of TOF:

- ❑ *TOF with pulmonary stenosis*: Subvalvar, valvar or supra-valvar. This is the most common variant.
- ❑ *TOF with pulmonary atresia*: This is a severe variant commonly associated with hypoplastic pulmonary arteries with or without collaterals between the aorta and the pulmonary arteries (major aortopulmonary collateral arteries, MAPCAs).
- ❑ TOF with absent pulmonary valve.
- ❑ *TOF with atrioventricular septal defect*: This is the least common variant.

Abnormal coronary artery anatomy is seen in 10% of cases; most common is right coronary artery crossing the RVOT.

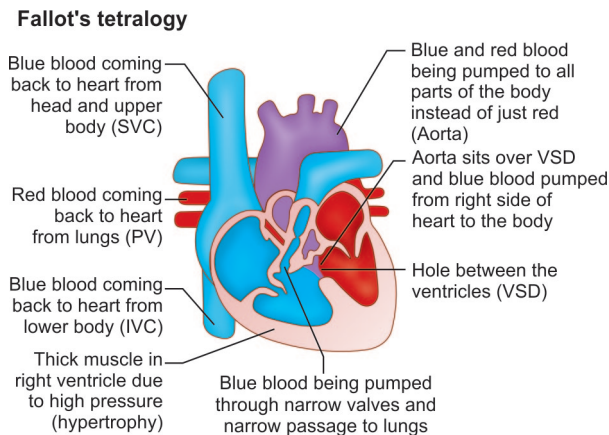


Fig. 12A

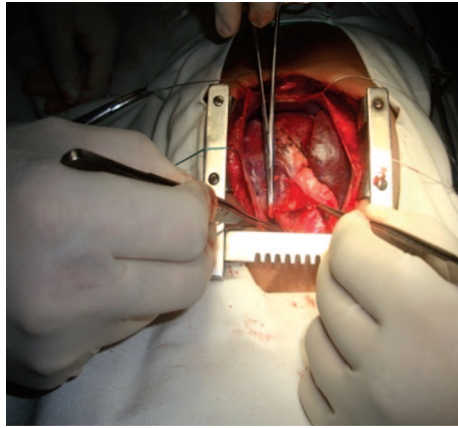


Fig. 12B

Figs 12A and B: (A) Components of TOF; (B) Forceps pointing towards hypoplastic PA in TOF

Incidence of TOF variant in our institute was approximately 26–27% among all the congenital heart surgeries in last five years.

Pathophysiology

Tetralogy of Fallot is the most common form of cyanotic heart disease and accounts for 3.5% of all cases of congenital heart disease. The cause of TOF is unknown, but 25% of patients have a chromosomal abnormality (microdeletion at 11q position on chromosome 22), which may be associated with immune deficiency (Di George syndrome), or velocardiofacial syndrome and submucous cleft palate.

Clinical features of TOF are due to reduced pulmonary blood flow and cyanosis and are variable in severity, depending on the degree of obstruction at the RVOT. Obstruction causes deoxygenated blood to shunt from the right ventricle to left across the usually large (nonrestrictive) VSD. A harsh ejection systolic murmur is present, and is due to turbulent flow across the RVOT.

In most babies, pulmonary blood flow is adequate in the first few weeks of life, but the child becomes increasingly cyanotic with age. Some babies have severely compromised pulmonary blood flow from birth, or in the case of pulmonary atresia without significant pulmonary collateral vessels, the baby will have “*duct dependent*” pulmonary circulation. These babies will require an early palliative shunt, e.g. mBT shunt to supply blood to the pulmonary artery. Some children have minimal right ventricular outflow obstruction (RVOTO), and the history is more typical of a VSD with minimal cyanosis (“*pink tet*”).

Some infants may have dynamic obstruction at the pulmonary infundibulum (subvalvar RVOTO). This leads to “*hypercyanotic spell*”, which is a classical symptom of TOF. The child becomes deeply cyanosed after an episode of crying and may become limp and unresponsive. This is a sign of acute reduction in pulmonary blood flow associated with sudden increase in the dynamic obstruction to the right ventricular outflow tract. It occurs due to muscular

spasm of the infundibulum; secondary to the elevated levels of endogenous catecholamines. During a spell, the cyanosis may be relieved if the baby is placed in the knee-chest position. Such babies respond well to a beta blocker such as propranolol before definitive surgery is carried out.

Older children with uncorrected TOF will develop severe clubbing and may learn to “squat” to increase pulmonary blood flow—this position increases the systemic vascular resistance and reduces the right-to-left shunt across the VSD. They may have polycythemia and suffer complications of long standing polycythemia like:

- ❑ Coagulopathy
- ❑ Intracranial abscess
- ❑ Stroke
- ❑ Hyperuricemia
- ❑ Neurodevelopmental delay

Adolescents and adults will have reduced exercise tolerance and are vulnerable to ventricular arrhythmias.

Without surgical intervention, around 50% of children with TOF will die within the first few years of life and survival beyond 30 years without surgery is uncommon. With corrective surgery in childhood, survival to discharge from hospital is 95–99%, and almost all children can be expected to survive to adulthood.

Management

Medical

- ❑ Before definitive corrective surgery is carried out, the child may be managed medically.
- ❑ Propranolol is prescribed, usually in a dose of 0.5 mg/kg tds. This reduces the spasm of the subpulmonary infundibulum.
- ❑ If the pulmonary blood flow is severely limited or increasing doses of propranolol fails to control the frequency of spells, a palliative shunt or corrective surgery may be required.

Hypercyanotic or Tet spell

It is a paroxysmal episode of acutely worsening cyanosis, usually in response to crying, feeding, defecation, agitation or fright.

It leads to increased right to left shunting due to rise in PVR (Pulmonary vascular resistance) and decrease in SVR (Systemic vascular resistance).

Treatment

- ❑ Squatting position, flexion of leg, abdominal compression, knee chest position
- ❑ High FiO₂ leads to pulmonary vasodilation causing decrease in PVR

- ❑ Hydration (fluid bolus) 20 mL/kg opens RVOT
- ❑ Morphine (0.1 mg/kg/dose) causes sedation and decrease in PVR
- ❑ Ketamine (1 mg/kg) increases SVR and pulmonary blood flow
- ❑ Phenylephrine (1 µg/kg) increases SVR
- ❑ Beta blocker (Esmolol 100–200 µg/kg/min) decreases infundibular spasm
- ❑ Sodium bicarbonate (1 mEq/kg) neutralizes metabolic acidosis.

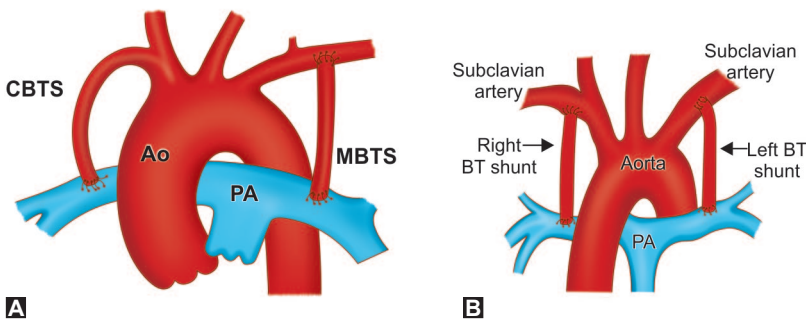
Surgical

Palliative surgery: Modified Blalock Taussig (mBT) Shunt: A palliative shunt is now rarely performed as results of early corrective surgery have improved dramatically with the advent of new technology, improved surgical and anesthetic skills. However, certain patients may benefit from a palliative shunt in the first weeks or months of life:

- ❑ Newborns with duct dependent pulmonary circulation (pulmonary atresia)
- ❑ Marked RVOTO and cyanosis
- ❑ Very small pulmonary arteries
- ❑ Anomalous coronary artery anatomy

The shunt aims to achieve a postoperative target saturation of approximately 80–85% in air to allow child to grow adequately before definitive surgery is undertaken. Shunt also helps to grow pulmonary artery adequate enough for definitive surgery.

The modified Blalock Taussig (mBT) shunt is created by end to side anastomosis of 3–4 mm Gortex tube graft between the subclavian artery and the right or left pulmonary artery (Figs 13A and B). Surgery may be performed via a thoracotomy or sternotomy. Other types of shunts are—the Potts shunt which connects descending aorta to the pulmonary artery, a Waterston shunt which connects the ascending aorta to the main or right pulmonary artery, classic BT shunt connects the right or left subclavian artery directly to the right or left pulmonary artery respectively.



Figs 13A and B: (A) Classical BT shunt; (B) Modified Blalock Taussig shunt

Preoperative Evaluation

History

- ❑ History of frequency of cyanotic spell, duration, triggering factors must be noted which gives an idea regarding severity of the disease.
- ❑ History of prematurity should be sought.
- ❑ In older children signs suggestive of complications of long-standing cyanosis, i.e. stroke, neurodevelopmental delay, intracerebral abscess and arrhythmia are noted.

Associations

The presence of TOF should raise the suspicion of associated anomalies. These include:

- ❑ DiGeorge syndrome (22q11 deletion): It is typically associated with learning difficulties, cleft palate, hypocalcemia, absent thymus (frequent respiratory infections) and characteristic facial appearance
- ❑ Isolated cleft lip/palate
- ❑ Hypospadias
- ❑ Skeletal abnormalities.

Examination

Typical signs in TOF include:

- ❑ Central cyanosis. It is detected by looking at the color of the tongue—blue coloration suggests a saturation of less than 85%. Central cyanosis is not improved by breathing 100% oxygen and its presence should be confirmed by pulse oximeter
- ❑ Clubbing is seen from 3–6 months of age
- ❑ Long harsh ejection systolic murmur heard best over the pulmonary area and the left sternal border. A thrill may be felt along the left sternal border.

Investigations

Oxygen Saturation

Typical SpO₂ is 65–85%—lower SpO₂ denotes increased severity.

Hematology

Full blood count—hemoglobin will be elevated in proportion to the degree of cyanosis. Coagulation studies—severe polycythemia may result in reduced coagulation factors and reduced platelet count.

Serum Electrolytes and Creatinine

Chest X-ray

The classical appearance in TOF is of a ‘boot shaped heart’ due to right ventricular hypertrophy and reduced central pulmonary markings.

ECG

- ❑ Signs of right ventricular hypertrophy and right axis deviation
- ❑ Tall R waves in V1 are diagnostic

Echocardiography

This is usually the only cardiac imaging required before surgery. The intracardiac abnormalities, degree and site of RVOTO and the coronary anatomy can be demonstrated.

Cardiac catheterization

Role is limited but it helps in delineating anatomy of pulmonary artery and assesses MAPCA's.

Anesthesia for mBT Shunt

In our institute, we performed 35 cases of emergency mBT shunt during the period of 2011–2014 with mortality around 65%. (Even in the western literature, mortality of emergency BT shunt is also high.)

Anesthesia for mBT shunt should proceed vigilantly and preparations should be made to manage a hypercyanotic spell during surgery (Fig. 14). If the child has duct dependent pulmonary circulation, ductal patency should be maintained with the prostaglandin infusion intraoperatively.

Challenge: Avoid/treat the Tet spell.

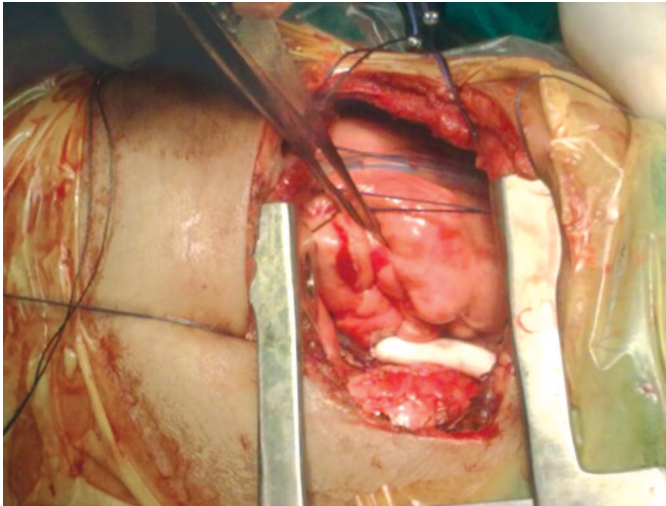


Fig. 14: mBT shunt (intraoperative)

Key points:

- ❑ The surgeon will clamp the subclavian artery during the shunt insertion, so the radial artery cannulation on that side should be avoided.

- ❑ Inotropic support with an adrenaline infusion is occasionally required to maintain cardiac output after the shunt has been opened. Increased infundibular spasm is not a concern in this situation as pulmonary infundibulum has been bypassed by shunt.
- ❑ $ETCO_2$ underestimates PCO_2 , so periodic ABG should be done.

Anesthetic maneuvers:

To decrease PVR—High FiO_2 , hyperventilation, correction of acidosis.

Adequacy of the shunt:

- ❑ The inspired oxygen should be reduced to 30% before the shunt is opened and an arterial blood gas taken to assess the adequacy of the shunt.
- ❑ The ideal situation is SpO_2 80–85% with systolic BP >80 mm Hg.
- ❑ Shunt too small or kinked— SpO_2 <70% despite an adequate BP.
- ❑ Shunt too large— SpO_2 100%, decrease in MAP more than 10, persistent acidosis with low SBP despite inotropes.

Postoperative Care of a Baby with mBT Shunt

Postoperatively, continuous shunt murmur may be heard in the subclavian area, and the shunt flow should be confirmed using ECHO. A low-dose heparin infusion (excess heparin may lead to intracranial bleeding) is started, provided there is no postoperative bleeding, followed by aspirin after a few days. Older child can be extubated early in ICU, sick child may require postoperative mechanical ventilation.

Anesthesia for a Child with mBT Shunt Coming for Noncardiac Surgery

A child with mBT shunt may present for further cardiac or noncardiac surgery. The mBT shunt is prone to obstruction, a complication that may be life-threatening. The pulmonary blood flow is dependent on a patent shunt and adequate systemic perfusion.

- ❑ Check shunt patency preoperatively— SpO_2 >80%, shunt murmur, ECHO
- ❑ Avoid preoperative dehydration, consider intravenous fluids preoperatively
- ❑ Avoid intraoperative hypotension
- ❑ Consider a fluid bolus if the saturation falls.

Corrective Surgery for TOF

Complete repair of TOF consists of closure of the VSD and relief of the RVOTO via median sternotomy on cardiopulmonary bypass. It is ideally performed in the first year of life, usually at 4–6 months of age.

A transatrial approach is taken to repair the VSD using pericardial patch. Relief of the RVOTO depends on the level of obstruction. Approaches to reduce the RVOTO include:

- ❑ Resection of muscle bundles in the pulmonary infundibulum
- ❑ RVOT patch

- Transannular patch
- Pulmonary valvotomy or valvectomy

A pulmonary conduit or homograft from the right ventricle to pulmonary artery may be required in children with pulmonary atresia or coronary abnormalities.

Adequacy of the repair is assessed by intraoperative transoesophageal echocardiography (to check for residual VSD and degree of residual RVOTO) or direct pressure measurements (ideally the right ventricular pressure should be less than 2/3 systemic) $P_{RV}:P_{LV} < 0.7$.

Anesthesia for Repair of TOF

The general approach includes:

- Preoperatively check frequency of hypercyanotic spells and increase hematocrit as it increases chances of spell during induction
- Avoid prolong fasting; it leads to hypovolemia (increases RVOT obstruction), increase viscosity and increase hematocrit (increases potency of spell)
- Premedication: Child should be calm and quiet during induction. In less than 6 month old child, sedation is not required
- Infective endocarditis prophylaxis is needed
- Propranolol should be continued up to the day of surgery, can exacerbate bradycardia during induction
- Goal of induction: Decrease right to left shunt by decreasing PVR, maintaining normal to high SVR, avoiding tachycardia and infundibular spasm
- Induction of anesthesia with halothane (maintains SVR, decreases infundibular spasm) or titrated sevoflurane
- Ketamine is an IV induction agent of choice
- Maintenance anesthesia with isoflurane/sevoflurane/halothane: Oxygen: air. N_2O avoided as it increases PVR and air embolism. Muscle relaxation with bolus dose atracurium/rocuronium/vecuronium
- Intraoperative analgesia—fentanyl/morphine
- Gradient across pulmonary valve less than 40 mm Hg post-repair is acceptable
- Antifibrinolytic drugs (tranexamic acid 10–30 $\mu\text{g}/\text{kg}$). Inotropic support—milrinone 50 $\mu\text{g}/\text{kg}^{-1}$ loading dose, 0.375–0.5 $\mu\text{g}/\text{kg}\cdot\text{min}^{-1}$; low dose adrenaline or noradrenaline as required (0.02–0.1 $\mu\text{g}/\text{kg}\cdot\text{min}^{-1}$), pacing may be needed. Cooling to 32–34°C during bypass, maintaining hematocrit around 35% during bypass.
- Modified ultrafiltration post-bypass to reduce total body water and increase hematocrit for blood conservation. Transfuse blood, FFP and platelet as per requirement.

Management of a Hypercyanotic Spell During Anesthesia

A history suggestive of hypercyanotic spells and use of propranolol should be sought in any child with TOF. These patients may benefit from an oral sedative premedication such as midazolam 0.5 mg/kg^{-1} 30 minutes preoperatively.

Management of a hypercyanotic spell should include measures to reduce infundibular spasm, improve oxygenation and increase cardiac output.

- ❑ Increase FiO_2 to 100%
- ❑ Fluid bolus 10–20 mL/kg
- ❑ Deepen the plane of anesthesia give fentanyl bolus 1 $\mu\text{g}/\text{kg}$
- ❑ Phenylephrine 1 $\mu\text{g}/\text{kg}$ bolus up to 5–10 $\mu\text{g}/\text{kg}$. If repeat boluses are required, start noradrenaline infusion 0.1 $\mu\text{g}/\text{kg}/\text{min}$
- ❑ Esmolol infusion may be considered to reduce infundibular spasm (starting dose 25 $\mu\text{g}/\text{kg}/\text{min}$)
- ❑ Sodium bicarbonate 1 mEq/kg IV
- ❑ Femoral compression increases SVR and decreased right to left shunt.

Weaning off CPB and Post-bypass Care

An infusion of a phosphodiesterase inhibitor such as milrinone is ideal, with or without a low dose of adrenaline to maintain blood pressure.

Adrenaline also helps in maintaining heart rate and prevents increase in the LVEDP.

Serum potassium should be maintained close to 4 mmol/liter.

Normothermia should be maintained and nasopharyngeal temperature should not be targeted to achieve 37°C to avoid the risk of cerebral hyperthermia rather keep it around 36.5°C.

Bleeding should be controlled surgically as well as by adequate use of blood products. Thromboelastography (TEG) is useful to guide the use of blood products.

Postoperative Management

Postoperative ventilation is required until the child is AWaC (alert, warm and comfortable) and there is minimal bleeding. Most children have an uncomplicated course and are discharged home within a week of surgery.

Patients in whom transannular patch is placed intraoperatively to augment RVOT, needs postoperative mechanical ventilation for a little longer because these patients have the higher risk of RV dysfunction in the postoperative period.

Postoperative Restrictive Physiology

Following the repair of TOF, the right ventricle may exhibit signs of “restrictive physiology”. This is due to systolic and diastolic myocardial dysfunction associated with right ventricular hypertrophy. Restrictive physiology is associated with low cardiac output, renal impairment and pleural effusion.

The management includes adequate preload, use of phosphodiesterase inhibitors and inotropes. Diuretic and peritoneal dialysis may be required. Restrictive physiology is associated with increased duration of postoperative ventilation, ICU stay and mortality.

Junctional Ectopic Tachycardia (JET)

Junctional ectopic tachycardia (JET) is frequently seen following TOF repair. It is a notorious condition which is difficult to treat and is exacerbated by the effect of right ventricular dysfunction and low cardiac output.

Treatment includes:

- ❑ Cooling to 34°C
- ❑ Sedation, muscle relaxation to reduce the work of breathing
- ❑ Magnesium sulphate 25 to 50 mg/kg IV
- ❑ Amiodarone/procainamide infusion.

TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION

Manjula Sudeep Sarkar, Shraddha Mathkar

Total anomalous pulmonary venous connection (TAPVC) is an uncommon cyanotic heart disease with an incidence of 1.5 to 2% among congenital heart diseases. In our institute, TAPVC constituted ~ 13–14% among all congenital heart surgeries in last five years.

Anomalous pulmonary venous connection varies from single pulmonary vein to all four veins or a tributary of systemic vein draining into the right atrium.

Embryology

The failure of common pulmonary vein to connect with the pulmonary venous plexus leads to persistence of one or more embryological venous connection to the:

- ❑ Right superior vena cava
- ❑ Left vertical vein/innominate vein
- ❑ Umbilical vitelline/portal vein.

Classification

Three criteria on which the disease is classified are as follows:

- a. Pathway by which the pulmonary venous blood drains into the right atrium.
- b. The presence or absence of obstruction along the course of the pathway.
- c. Nature of an interatrial connection (Table 1).

TAPVC is incompatible with the life unless a communication between the right and left sides of the heart exists; usually via a patent foramen ovale or atrial septal defect (Figs 15 and 16). When interatrial connection is absent; it becomes a life-threatening situation which immediately requires surgical intervention.

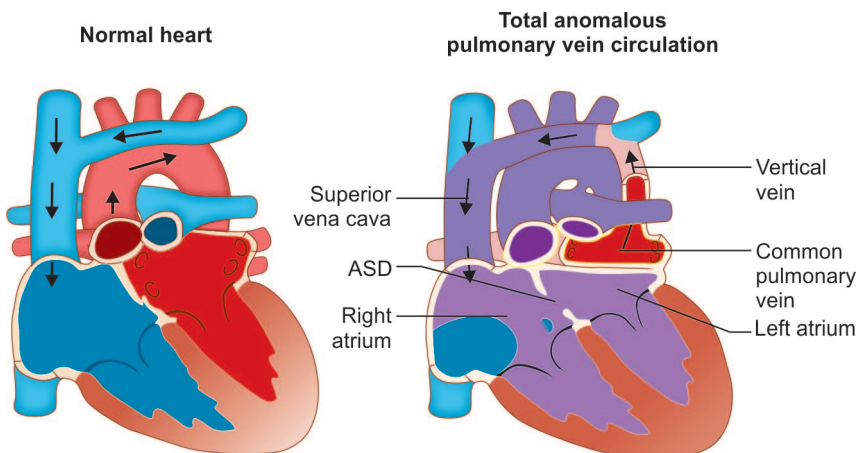


Fig. 15: Total anomalous pulmonary venous connection

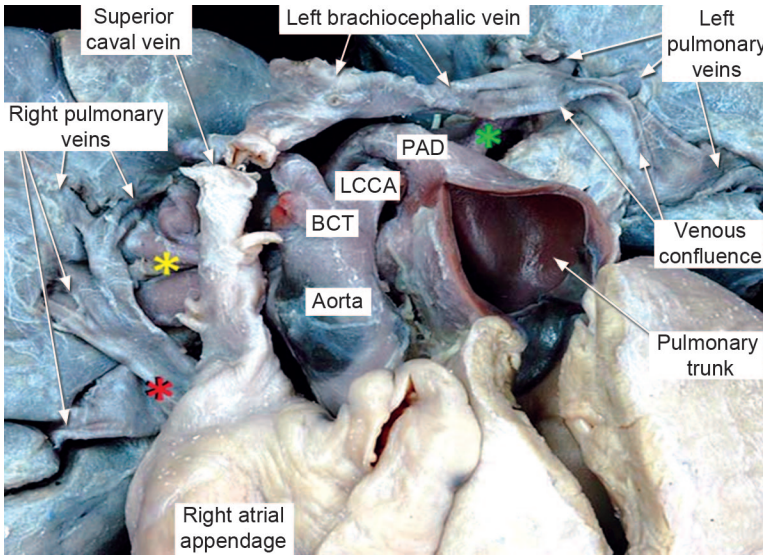


Fig. 16: Supracardiac TAPVC (Note the pulmonary veins draining into SVC through common chamber)

Table 1: Classification of TAPVC and incidence

Type of TAPVC	Incidence	Obstruction
Supracardiac (Right and left pulmonary veins confluence to form a common chamber which opens in right atrium, SVC or IVC via vertical vein)	42–55%	Uncommon (if occurs, then as a result of compression extrinsic or intrinsic narrowing)
Cardiac (Pulmonary vein drains into coronary sinus)	25–30%	Uncommon
Infracardiac (Descending vein joins the portal venous system either at the splenic vein or to the confluence of superior mesenteric vein)	13–30%	Frequent
Mixed	2–5%	Not uncommon

Pathophysiology

See Flow chart 1.

Clinical Features

Depends upon the degree of obstruction

Nonobstructive

- ❑ Asymptomatic at birth
- ❑ Failure to thrive and respiratory infections

- ❑ Cyanosis is mild and clinically not apparent
- ❑ Cardiac failure prior to 6 months of age is commonly seen.

Flow chart 1: Pathophysiology of TAPVC

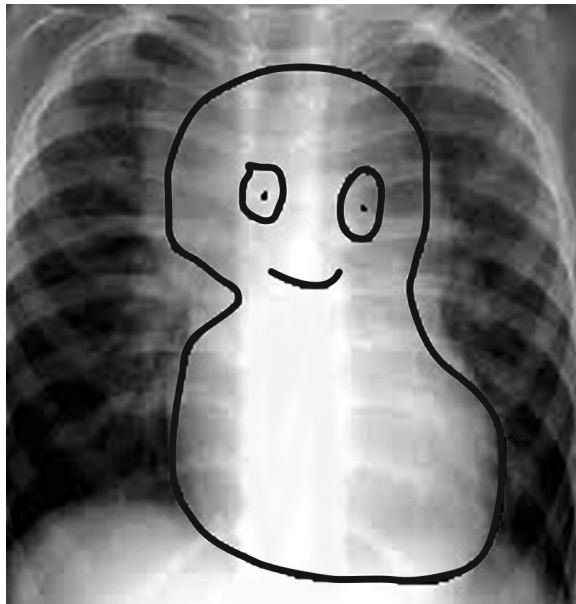
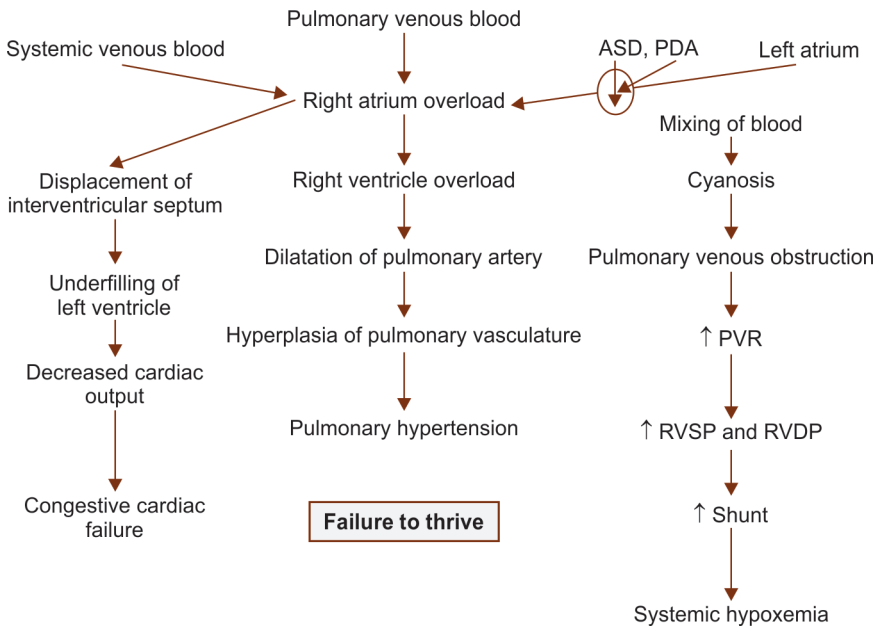


Fig. 17: X-ray chest showing Snowman's heart in TAPVC

Signs:

- ❑ Right parasternal heave
- ❑ S1—loud, S2—widely split
- ❑ P2—loud
- ❑ Pulmonary area—blowing systolic murmur.

Obstructive

- ❑ Symptomatic in the first month of life
- ❑ Cardiorespiratory failure once sets in is stormy and rapid
- ❑ Infradiaphragmatic—symptoms worsen on coughing and crying.

Evaluation (Preoperative)

General physical examination and clinical assessment of cardiac and respiratory status is done. Routine laboratory investigations are done.

2D echo: It helps in the diagnosis of the type of TAPVC and evaluation regarding the presence of an interatrial connection/pulmonary venous obstruction (Fig. 18).

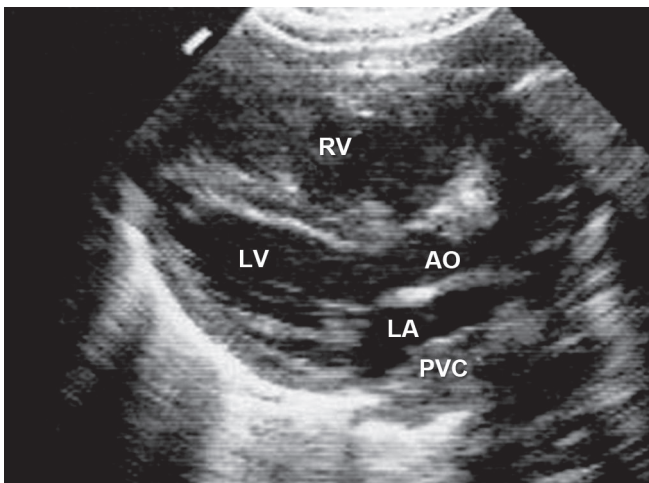


Fig. 18: TTE image showing dilated common chamber (Pulmonary venous confluence—PVC) behind left atrium

Cardiac catheterization guides in oximetry data: There is equalization of oxygen saturation in all the cardiac chambers.

CT angiography is investigation of choice nowadays.

Surgery

Emergency atrial septostomy is done if no communication is present between right and left side of the heart.

Supracardiac TAPVC repair: The principle of surgery is to perform direct anastomosis of common chamber to the left atrium.

Intracardiac TAPVC repair: Right atriotomy is done and the common wall is excised. Patch closure is done in such a way that coronary sinus drains into left atrium. But, postoperative desaturation is expected because some venous return is also directed towards left atrium. Infracardiac TAPVC repair is a complex procedure and the mortality rate is high.

Anesthesia Considerations

Goal: Decrease Pulmonary vascular resistance (PVR) and maintain left ventricular contractility.

Preoperative

Presence of pulmonary venous obstruction is assessed. Child must be kept calm to avoid sympathetic stimulation induced exacerbation of PVR. Neonates do not require any premedication. Prostaglandin PGE1 may help in reducing pulmonary hypertension.

TEE is usually avoided due to the possibility of exacerbating pulmonary venous obstruction.

Induction: *Steal induction* is required in sick child to avoid worsening of pulmonary hypertension (PHTN). In obstructive cases, inhalational induction is slower than intravenous induction. High dose opioid induction is commonly practised. Very sick child fails to tolerate inhalational agents due to already compromised myocardium. Hyperventilation, correction of acidosis, 100% oxygen reduces PA pressures and should be kept in mind during induction.

Maintenance: Balanced anesthesia in the form of oxygen, air, pancuronium and either fentanyl or isoflurane is usually tolerated well. Drugs need to be individualized as per patients status.

Prevention of rise in PVR

- ❑ Avoid nitrous oxide
- ❑ Avoid hypoxia
- ❑ Maintain adequate depth of anesthesia
- ❑ Cautious use of IV fluids
- ❑ Use of nitric oxide 5–80 ppm

On CPB: Inodilator Inj. milrinone 50 µg/kg bolus during rewarming phase helps in decreasing PA pressure during weaning. Also, phenoxybenzamine 1 mg/kg on CPB is practised in our institute in cases of severe PHT.

Weaning off CPB: All the measures to avoid rise in pulmonary artery pressure are carried out. Gentle suctioning needs to be done to prevent traumatic bleeding in already reactive pulmonary vascular bed. Vasodilators reduce afterload and improve LV function. Platelets are administered to control bleeding. Pulmonary congestion is not uncommon during weaning which

requires lung protective ventilatory strategy (low tidal volume and high respiratory rate). Sometimes, PA pressure can be measured by passing a 24–26 G needle with pressure transducer connection at the pulmonary trunk before closure of the chest to know the adequacy of the repair and if PA pressures are systemic/suprasystemic, vertical vein is not ligated which helps to decompress PA system.

Postoperative

Biventricular dysfunction is a common complication encountered in the postoperative period. After TAPVC repair, all the pulmonary venous return drains into the noncompliant left atrium resulting into the rise in the pulmonary system and the RV failure ensues. Left atrial catheter is placed in few patients prior to closure for pressure monitoring and to guide fluid therapy in the postoperative period. Also, left ventricular failure is not uncommon because of the chronically underloaded LV is exposed to increased workload post-repair.

Persistent of pulmonary hypertension and even crisis (40%) occurs frequently; in which mortality is very high. Pulmonary hypertensive crisis is defined as pulmonary artery pressure more than or equal to systemic arterial pressure along with unstable hemodynamic.

SUGGESTED READING

1. Davis LK, Husain SA, Weitzel NS. Anesthesia management for patients with congenital heart disease: The pediatric population. A practical approach to cardiac anesthesia, 5th edition. In: Hensley FA, Martin DE, Gravelee GP (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia, 389-435.
2. Lake CL. Anomalies of systemic and pulmonary venous return, Pediatric cardiac anesthesia, 4th edition. In: Lake CL, Brooker PD (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 480-500.
3. Lell WA, Pearce FB. Tetralogy of Fallot, Pediatric cardiac anesthesia, 4th edition. In: Lake CL, Brooker PD (Eds). Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 344-56.
4. Park MK. Cyanotic congenital heart defects, Pediatric cardiology for practitioners, 5th edition, Mosby Elsevier, Philadelphia, 235-53.
5. Perloff JK, Marelli AJ. DORV, Perloff's clinical recognition of congenital heart disease, 6th edition, Saunders Elsevier, Philadelphia, 359-64.

Chapter 7

Valvular Heart Disease

Manjula Sudeep Sarkar, Pushkar Desai

INTRODUCTION

Valvular heart disease (VHD) forms an important and major portion of cardiac disorders. In developing countries, low socioeconomic status, overcrowding and poor hygiene predispose to streptococcal infections resulting in rheumatic fever and its sequelae. On the other hand, senile degenerative and congenital forms of valvular diseases are prevalent in developed countries. All VHD are characterized by abnormalities of the ventricular loading and the ventricular function is influenced by the progression of volume or pressure overload.

Understanding the pathophysiology of an individual valve disorder is necessary for the implementation of successful anesthesia because hemodynamic conditions differ significantly among valvular lesions. Therefore, detailed history and clinical examination is required to assess the severity of the lesion and plan anesthesia.

In our institute, valvular heart surgery comprises approximately 30% of the cardiac surgery.

MITRAL STENOSIS

Incidence

Developing countries have higher rates of rheumatic fever and consequently higher rates of mitral stenosis (MS) with prevalence of more than 10 cases per 1,000 in India. Incidence is more common in females than males with the ratio 2:1.

Etiology

Causes of mitral stenosis are:

- ❑ Rheumatic heart disease
- ❑ Congenital
- ❑ Carcinoid syndrome
- ❑ Glycogen storage and metabolic diseases like Hunter and Hurler syndrome
- ❑ Systemic lupus erythematosus
- ❑ Amyloidosis

- Infective endocarditis vegetations.

In clinical practice especially in India, it is a dictum that three most common causes of MS are rheumatic, rheumatic and only rheumatic heart disease.

Pathophysiology

MS is characterized by its insidious onset and slow progression over the years. The normal mitral valve area is 4 to 6 cm² (mitral valve index, 4.0 to 4.5 cm²/m²). Symptoms of MS start when the valve area is decreased by 50%. Generally the patient is asymptomatic during the initial 20-30 years. MS can be described in three stages viz. mild, moderate and severe/critical depending upon the extent of the mitral valve apparatus involvement which obstructs left ventricular diastolic filling secondary to a progressive decrease in mitral valve orifice area resulting into an increase in left atrial volume and pressure (Fig. 1).

Stage 1: Mild mitral stenosis (Asymptomatic with physiologic compensation)

With mild mitral stenosis, left ventricular filling and stroke volume are usually maintained at rest by an increase in left atrial pressure. But the tachycardia will limit the stroke volume and the patient complains of dyspnea on exertion and moderate exercise. Cardiac catheterization laboratory, by detecting increased filling pressures with exercise, can identify this stage of the disease. Further progression of mitral stenosis leads to increases in left atrial pressure and volume that are reflected back into the pulmonary circuit.

Stage 2: Moderate mitral stenosis (symptomatic impairment)

As the valve area progresses to 1.0–1.5 cm², increased left atrial pressure is transmitted to pulmonary veins resulting transudation of fluid into the pulmonary interstitial space. Thickening of the capillary basement membrane and increased lymphatic drainage from the lungs occurs when left atrial pressure gradually increases. This leads to decreased pulmonary compliance and increased work of breathing. Pulmonary arterial constriction, intimal hyperplasia and medial hypertrophy eventually results in pulmonary arterial hypertension associated with restrictive lung disease.



Fig. 1: Stenotic mitral valve

Because atrial contraction contributes 30–40% of LV filling in mitral stenosis, the onset of atrial fibrillation can lead to significant impairment in cardiac output.

Stage 3: Critical mitral stenosis (terminal failure)

When the valve area decreases below 1.0 cm², patient is considered to have critical mitral stenosis and symptoms present even at rest. Due to this degree of fixed obstruction, cardiac output also is reduced. With this small valve area, the pulmonary edema is likely to develop when the pulmonary venous pressure exceeds the oncotic pressure of plasma. Episodes of pulmonary edema are precipitated by atrial fibrillation, sepsis, pain, and pregnancy which lead to sudden rise in the left atrial and pulmonary artery pressures. Pulmonary hypertension eventually leads to right ventricular failure. The dilated RV further reduces left ventricular emptying. Functional tricuspid regurgitation ensues in the later stage of the disease due to dilated tricuspid annulus and patients presents with the signs of RV failure like peripheral edema, hepatomegaly, raised JVP etc. Hemoptysis may occur because of pulmonary hypertension. A mitral valve area of 0.3 to 0.4 cm² is the smallest area compatible with life.

PREOPERATIVE

Clinical Features

Symptoms

- ❑ Patients are normally asymptomatic for 20 years or more after an acute episode of rheumatic fever.
- ❑ *Generalized*—Dyspnea, palpitation, fatigue, bronchitis, paroxysmal nocturnal dyspnea.
- ❑ Symptoms suggestive of complications
 - **Hemoptysis**—Due to ruptured bronchopulmonary veins secondary to increased left atrial pressure.
 - **Chest pain**—Associated with IHD or pulmonary HTN. Chest pain may occur in 10 to 20% of patients with mitral stenosis, but it is a poor predictor of the coexistence of coronary disease, which may be present in approximately 25% of patients.
- ❑ Pressure symptoms
 - Hoarseness of voice due to compression of left recurrent laryngeal nerve (Ortner's syndrome)
 - Dysphagia—compression of esophagus
 - Bronchiectasis—pressure on left main bronchus
 - Rarely, erosion of spine may occur resulting into paraplegia
- ❑ *Thromboembolism symptoms*: Stroke, hemiparesis/ monoplegia; due to atrial fibrillation induced thrombus formation in left atrial appendage.

Juvenile Mitral Stenosis

Especially in India, first described in Bengal, characterized by presentation < 20 years age, rapid progression, remarkable pulmonary hypertension but without calcification.

Signs

- ❑ Malar flush—cyanotic face with telangiectasia—called as mitral facies
- ❑ Tapping apical impulse
- ❑ Palpable P2
- ❑ Loud S1
- ❑ Opening snap (indicates pliable valve).

Diastolic murmur over mitral area—low pitched, rumbling, mid diastolic, presystolic accentuation, best heard with bell in lateral position in expiration, accentuated with exercise. Disappearance of murmur indicates progression to severe disease.

Infective endocarditis signs and symptoms like fever, anemia, hemorrhagic spots, splenomegaly are seen more common in pliable valves than in calcific valves.

Atrial fibrillation—Due to enlarged LA, precipitates thrombus formation in LA appendage, irregularly irregular pulse, absent p wave in ECG.

Pulmonary edema—when LAP > 25 mm Hg; presents acutely, uncommon in long standing MS.

Functional TR murmur can be heard over parasternal area in case of pulmonary HTN.

Pregnancy exacerbates MS and often physical signs are found before symptoms develop. Dyspnea increases by one grade in pregnancy.

Twenty percent of patients in whom the diagnosis of symptomatic mitral stenosis is made die within 1 year and 50% die within 10 years after diagnosis, without surgical intervention.

Investigations

Routine

All routine investigations like complete hemogram, renal and hepatic function tests, serum creatinine, serum electrolytes especially potassium due to pretreatment of these patients with digitalis must be done. Many patients receive warfarin or heparin as a treatment/prevention of LA clot; so coagulation parameters like prothrombin time (PT)/INR and aPTT are sought.

Special

ECG

- ❑ P mitrale (notched P wave due to biatrial contribution), features of atrial fibrillation (absent P wave and varying RR interval), features of RVH (in case of pulmonary hypertension).
- ❑ Other common ECG findings include dysrhythmias, conduction abnormalities, evidence of active ischemia, or previous myocardial infarction.

Chest X-ray

- ❑ Double atrial shadow (within right heart border)

- ❑ Splaying of carina
- ❑ Straightening of left heart border (due to prominent PA and LAA)
- ❑ Kerley B lines—towards costophrenic angles when LA pressure reaches 20–30 mm Hg
- ❑ Kerley A line—when LA pressure rises above 30 mm Hg. Seen at apex of the lung fields.
- ❑ Calcification of mitral valve apparatus
- ❑ Vascular markings in the peripheral lung fields are sparse in the presence of significant pulmonary hypertension.

Echocardiography

Wilkin's Score: Echocardiography is used to assess the anatomy of the mitral valve pertaining to calcification, mobility, degree of leaflet thickening and the extent of involvement of the subvalvular apparatus. Each parameter is allotted four points and the total score out of sixteen is calculated. Patients with score of more than 9 are candidates for surgical valve replacement.

The severity of mitral stenosis is assessed by calculation of mitral valve area and measurement of the transvalvular pressure gradient.

Echocardiography also evaluates presence of thrombus in the left atrium. It assesses cardiac chamber dimensions, biventricular function, valve area, transvalvular pressure gradients and the magnitude of valvular regurgitation and helps in measurement of degree of pulmonary hypertension.

Assessment of the Severity of Mitral Stenosis

- ❑ According to the valve area (Fig. 2)
 - Mild 1.6–2 cm²
 - Moderate 1–1.5 cm²
 - Severe < 1 cm²

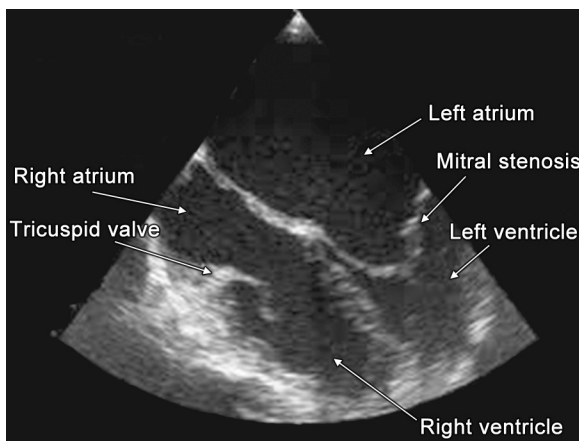


Fig. 2: Transesophageal echocardiography showing mitral stenosis

- According to transvalvular mean gradient (mm Hg) and pressure half time (ms)-

• Mild	< 6 mm Hg	100–200 ms
• Moderate	6–10 mm Hg	200–300 ms
• Severe	>10 mm Hg	> 300 ms

Intraoperative

Management for Mitral Valve Replacement (MVR)

Hemodynamic goals

The goal of anesthesia is to maintain normal cardiac output.

- **Rate:** Optimum heart rate to be maintained between 60–80 beats per minute. Tachycardia will decrease the diastolic filling of the LV and the resultant increase in LA pressure can precipitate pulmonary edema. This is based on Poiseuille's law, which states that the left atrioventricular pressure gradient is proportional to the fourth power of the instantaneous flow across the mitral valve; hence, any increase in instantaneous flow requires a large increase in left atrial pressure. High dose narcotic-based anesthetics help in avoiding intraoperative tachycardia and hence it is preferred. If atrioventricular pacing is initiated in these patients, a long PR interval of 0.15 to 0.20 ms is optimal to allow adequate LV filling across stenotic valve. Decreased in the PR interval reduces diastolic flow and ultimately results into a reduced cardiac output.

At the same time, excessive bradycardia can be dangerous because of the fixed stroke volume which will decrease cardiac output dramatically. ($CO = HR \times SV$).

- **Rhythm:** Atrial contraction contributes approximately 30–40% of the LV stroke volume in MS and so the control of the ventricular rate remains the primary goal in managing patients with atrial fibrillation; although cardioversion should not be withheld from patients with atrial tachyarrhythmias who become hemodynamically unstable. One should aim to maintain sinus rhythm perioperatively.
- **Preload:** Cardiac output in a fixed output states is dependent on adequate preload. But the caution is needed as the too much fluid can result into florid pulmonary edema due to already elevated left atrial pressures.
- **Contractility:** Cardiac output depends upon stroke volume which is generated by the force of the contraction of the ventricle. But in MS contractility of the LV is normal until the end stages of MS. Chronic under filling of the LV, however, leads to cardiomyopathy with depressed ventricular contractility even in the face of restored filling. Also, depression of RV contractility limits left atrial filling and eventually, the cardiac output. Myocardial depressants are better avoided in the perioperative period.
- **Afterload:** Compensatory increased systemic vascular resistance is developed in mitral stenosis to maintain systemic blood pressure in the presence of limited cardiac output. As a result, they cannot compensate for falls in systemic vascular resistance, which result in severe hypertension,

myocardial ischemia, and a downward spiral of reduced contractility causing further falls in blood pressure and coronary perfusion. Spinal anesthesia is also not well tolerated due to sudden vasodilation resulting into hypotension and compensatory tachycardia. It is recommended that the afterload should be maintained in the normal range for these patients. Afterload to the right ventricle is provided by pulmonary vascular resistance and particular attention should be paid to avoid factors like nitrous oxide, acidosis, hypercarbia or hypoxia, hypothermia which increases pulmonary artery pressure.

Premedication

Anxiety-induced tachycardia may be treated with small doses of narcotics or benzodiazepines. But heavy premedication may lead to respiratory depression and resultant hypercarbia will exacerbate pulmonary hypertension. Avoidance of an anticholinergic should be considered to minimize tachycardia. Medications taken by the patient preoperatively to control heart rate, such as digitalis, β -blockers, calcium receptor antagonists, or amiodarone, should be continued.

Monitoring

All routine standard monitors must be applied. Temperature monitoring should be done at two sites (peripheral as well as core temperature). Usually nasopharyngeal and rectal temperature is monitored. Care must be taken not to exceed nasal temperature above 36.5°C in order to avoid cerebral hyperthermia induced neuronal injury.

Placement of radial arterial line sometimes is difficult due to atrial fibrillation and fixed output state in which case femoral arterial line needs to be secured.

Pulmonary artery catheter needs to be inserted farther than usual because of the dilated pulmonary arteries. Special care should be taken when placing the catheters because of the increased risk of pulmonary artery rupture due to pre-existing pulmonary hypertension. It should be remembered that the pulmonary artery diastolic pressure does not accurately reflect left atrial pressure because of significant pulmonary hypertension and even pulmonary capillary wedge pressure overestimates LV filling pressure.

Transesophageal echocardiography is useful to assess the presence of LA clot, helps during weaning off bypass and determines prosthetic valve function.

Induction

High dose narcotic induction is preferred but one should keep in mind the possibility of profound bradycardia and pancuronium negates this vagomimetic effect. Propofol should be used in titrated doses if used at all; taking care not to induce hypotension and tachycardia.

If patient has preexisting left atrial thrombus, patient should be induced in Trendelenberg position to avoid the risk of thromboembolism and LV inflow obstruction.

Anesthetic techniques that avoid increases in PVR prevent additional RV embarrassment. Vasodilator therapy in patients with pulmonary hypertension

generally is ineffective as the venodilation produced further limits LV filling and does not improve cardiac output. The only mitral stenosis patients who may benefit from vasodilator therapy are those with concomitant mitral regurgitation or those with severe pulmonary hypertension and RV dysfunction in whom pulmonary vasodilation can facilitate transpulmonary blood flow and improve LV filling.

Maintenance

Prevention and treatment of tachycardia are central to the perioperative management of these patients. All the strategies to avoid exacerbation of pulmonary hypertension must be followed. Avoidance of hypoxia, hypercarbia, acidosis and excessive PEEP is important. In cases of pre-existing pulmonary hypertension, nitrous oxide is better avoided.

Overzealous use of IV fluids can precipitate pulmonary edema. Appropriate replacement of blood loss and prevention of excessive anesthetic-induced venodilation helps preserve hemodynamic stability intraoperatively. Use of magnesium or amiodarone on CPB reduces risk of postoperative arrhythmia.

Weaning off CPB

After ensuring near normal body temperature, absence of acidosis, acceptable hemoglobin level and good contractility of the heart; patient is weaned off bypass. Inotropic support usually is needed. Sometimes IABP may be required for LV dysfunction because the chronic underfilling masks underlying cardiomyopathy which is exacerbated by ischemic arrest during cardiopulmonary bypass.

Preload augmentation as well as afterload reduction should be undertaken in the immediate postbypass period to improve forward blood flow. Patients previously in chronic atrial fibrillation occasionally can be converted to sinus rhythm by prophylactic treatment with a bolus of procainamide (500 mg to 1 g) or amiodarone (150 mg) during bypass and should be maintained with continuous infusion of amiodarone and/or overdrive atrial pacing at a rate of approximately 110 beats/min. It must be remembered that after prosthetic valve placement, a residual gradient up to 7 mm Hg across the mitral valve is normal.

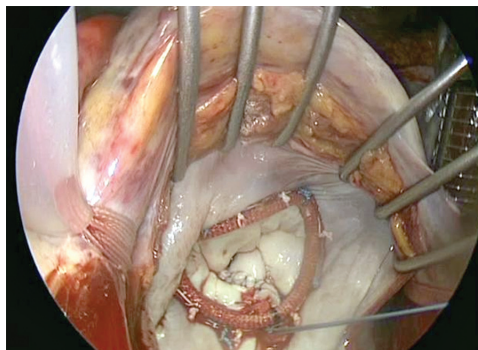


Fig. 3: Mitral ring annuloplasty (technique of mitral valve repair)

Postoperative

One catastrophic complication that can occur within the first few days after valve replacement is atrioventricular disruption. One method suggested to help avoid this complication is **reduction of LVEDP** while maintaining adequate cardiac output. Atrioventricular disruption is a particular risk for the elderly patient with a relatively noncompliant LV who develops increased diastolic tension on the LV wall after surgery and inotropes serve by increasing contractility and reducing LV size and wall tension.

Patients can be extubated at the end of the surgery in case of uneventful perioperative period or can be shifted to ICU and fast tracked within 6 hours. It is better to electively ventilate patients with pre-existing pulmonary hypertension.

Adequate analgesia in the form of IV opioids, paracetamol, opioid skin patches is provided. NSAID's should better be avoided in patients with renal dysfunction.

Most patients with MS are diuretic dependent and they should be maintained on diuretics. RV dysfunction is not uncommon following mitral valve surgery and adequate measures like optimization of preload, use of inodilators, nesiritide and pulmonary vasodilators (inhaled nitric oxide, epoprostenol, iloprost) needs to be applied.

Management for Balloon Mitral Valvuloplasty

Percutaneous balloon mitral valvuloplasty (PBMV) offers a minimal invasive, catheter-based approach to mitral stenosis. First reported by Japanese surgeon Inoue in 1984, the technique of PMC involves directing a balloon-tipped catheter across the stenotic mitral valve. Specifically designed balloons allow sequential inflation of the distal and proximal portions ensuring correct positioning across the mitral valve before the middle portion of the balloon is inflated to split the fused commissures. Patient selection for BMV requires careful echocardiographic evaluation and heavily calcified valves or significant mitral regurgitation are contraindication to the procedure.

BMV is usually carried out under local anesthesia with monitored anesthesia care but sometimes general anesthesia is required in uncooperative patients, pediatric age group and in case of hemodynamic catastrophe following the procedure.

Preoperative

Proper counseling of the patient helps during preoperative period as the procedure is usually performed under local anesthesia and intravenous sedation. Ability of the patient to lie supine comfortably is to be noted.

Presence of pulmonary edema should be ruled out and the need for ventilator support is assessed. Preoperative use of diuretics helps to drain out the excess lung water.

Serum potassium levels should be optimized.

Rule out the presence of moderate to severe MR and LA thrombus in the preoperative assessment.

Ensure functioning defibrillator and the presence of surgical team standby prior to the procedure.

Adequate length of the ventilator tubings, intravenous extension lines, resuscitation equipment, difficult airway cart needs to be available in the catheterization laboratory. One needs to plan for emergency intubation and positive pressure ventilation if catastrophe occurs. Excessive fasting may lead to dehydration and puncture of femoral vein becomes difficult.

Intraoperative

Critical steps during BMV are transeptal puncture and balloon inflation across the stenotic valve. Cardiac tamponade can occur during transeptal puncture and the temporary cessation of blood flow during balloon inflation may result into syncope, thromboembolism and vagal stimulation.

Maintenance of adequate anticoagulation during the procedure is important to avoid the risk of systemic embolization and the stroke. Heparin should be repeated hourly in the dose of 20-30 U/kg after bolus dose of 100 U/kg.

Possibility of local anesthetic toxicity due to an excessive dose and inadvertent intravenous entry during inguinal area infiltration should be kept in mind.

Excessive commisurotomy by the balloon may lead to severe mitral regurgitation and the resultant hemodynamic instability warrants general anesthesia with endotracheal intubation and positive pressure ventilation.

Postoperative

Simple analgesics like paracetamol/NSAID's are usually sufficient. Patients can be mobilized after vascular sheath removal. Medications for heart rate control, diuretics needs to be continued.

AORTIC STENOSIS

Etiology

- Causes: Congenital acquired
 - Bicuspid aortic valve (1-2%)—rheumatic (calcific)
 - Degenerative (atherosclerotic)
 - End-stage renal disease
 - Rheumatoid arthritis

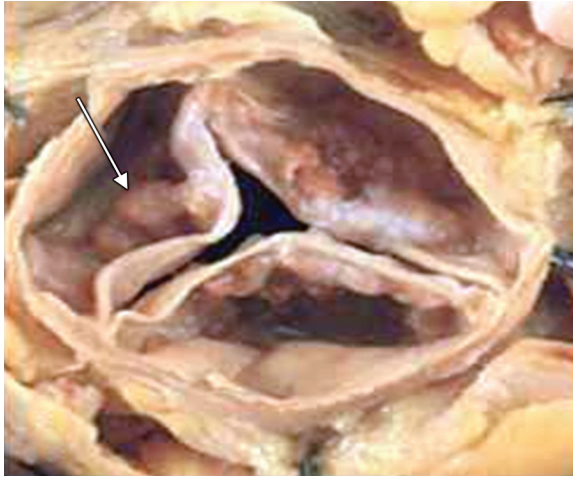


Fig. 4: Calcified aortic stenosis

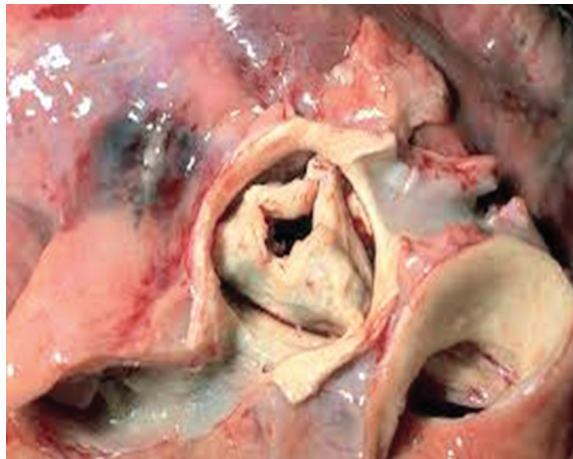


Fig. 5: Stenotic aortic valve

Pathophysiology

The normal aortic valve area is 2.6 to 3.5 cm², with hemodynamically significant obstruction usually occurring at cross-sectional valve area of 1 cm² or less (Figs 4 and 5). Stenosis at the level of the aortic valve results in a pressure gradient from the left ventricle to the aorta. The intracavitary systolic pressure generated to overcome this obstruction directly increases myocardial wall tension (σ) in accordance with Laplace's law. This directly stimulates the multiplication of sarcomeres resulting into the concentrically hypertrophied ventricle. The

consequences of this LV hypertrophy leads to reduced left ventricular (LV) diastolic compliance (i.e. small changes in diastolic volume produce relatively large increases in ventricular filling pressure), potential imbalances in the myocardial oxygen supply and demand relationship and possible deterioration of the intrinsic contractile function of the myocardium.

The rate of progression is on an average a decrease in aortic valve area (AVA) by $0.1\text{cm}^2/\text{yr}$ and the peak instantaneous gradient increases by $10\text{ mm Hg}/\text{yr}$. The rate of progression of aortic stenosis in men older than age 60 years is faster than in women and it is faster in women older than age 75 years than in women 60 to 74 years old. Calcific aortic stenosis (Fig. 4) and coronary artery disease (CAD) both results from active inflammatory process. They are more common in men, older people and patients with hypercholesterolemia. Aortic valve calcification is an inflammatory process promoted by atherosclerotic risk factors. The early lesion of aortic valve sclerosis may be associated with CAD and vascular atherosclerosis.

- 1. Stage 1: Mild aortic stenosis (asymptomatic with physiologic compensation)** Initially, the maintenance of normal stroke volume is associated with an increased systolic pressure gradient between the LV and the aorta but the aortic systolic pressure and stroke volume remain relatively normal. This higher gradient results in a compensatory **concentric LV hypertrophy** without dilation of the chamber.
- 2. Stage 2: Moderate aortic stenosis (symptomatic impairment):** When the aortic valve area reaches 0.7 to 0.9 cm^2 LV hypertrophy as well as dilation occurs, leading to increased LV end-diastolic volume (LVEDV) and LVEDP. This leads to increased myocardial work and O_2 demand. At the same time, the elevated LVEDP decreases coronary perfusion pressure and myocardial O_2 supply is impeded. Finally, the venturi effect of the jet of blood flowing through the aortic valve and past the coronary arteries may lower pressure in the coronary ostia enough to reverse systolic coronary blood flow. These factors cause subendocardial ischemia even in the absence of concurrent atherosclerotic coronary disease.
- 3. Stage 3: Critical aortic stenosis (terminal failure):** Continuation of the disease process with reduction of the aortic valve index to less than $0.6\text{ cm}^2/\text{m}^2$ leads to further reduction in ejection fraction and increased LVEDP. Pressure builds up in the pulmonary venous circuit, leading to pulmonary edema when the left atrial pressure increases to more than 25 to 30 mm Hg. Normally, sudden death will intervene, but if the patient is able to survive, the increasing pulmonary arterial hypertension eventually will produce RV failure.

When the LV dysfunction ensues, there will be reduction in the gradient across aortic valve and this echocardiographic finding should be interpreted carefully. This low-flow, low-gradient aortic stenosis is defined when the mean gradient $< 30\text{ mm Hg}$ and a calculated AVA $< 1.0\text{ cm}^2$ occurs. Dobutamine stress echo helps in this condition.

Aortic Valve Area

Normal	2.6–3.5 cm ²
Mild	1.2–1.8 cm ²
Moderate	0.8–1.2 cm ²
Significant	0.6–0.8 cm ²
Critical	<0.6 cm ²

LV-Aortic Gradient

Mild	12–25 mm Hg
Moderate	25–40 mm Hg
Significant	40–50 mm Hg
Critical	>50 mm Hg

Preoperative***Clinical Features******Symptoms***

- ❑ Pure AS remains asymptomatic for 10–15 years.
- ❑ Classic triad: Angina, dyspnea and syncope—Symptoms do not correlate well to the severity of the stenosis; some patients with small valve areas can remain asymptomatic.

Signs

- ❑ Slow rising pulse with narrow pulse pressure (Pulsus tardus)
- ❑ Ejection systolic murmur maximal at the 2nd intercostal space, right sternal edge radiating to the neck.

Complications

LV failure, arrhythmias, complete heart block, infective endocarditis.

Investigations

- ❑ ECG: Left ventricular hypertrophy and strain (with secondary ST-T wave abnormalities), LBBB, CHB if calcification involves conducting system
- ❑ CXR: Normal until the left ventricle begins to fail, poststenotic dilatation of the aorta, calcified aortic annulus
- ❑ Echocardiogram: Enables calculation of valve gradient and assessment of left ventricular function (Fig. 6)
- ❑ Cardiac catheterization is also used to estimate the gradient across the valve and to rule out any concurrent coronary artery disease
- ❑ Coronary angiogram: Indicated >45 years with severe AS.

Caution

Use diuretics with caution to avoid excess volume depletion.

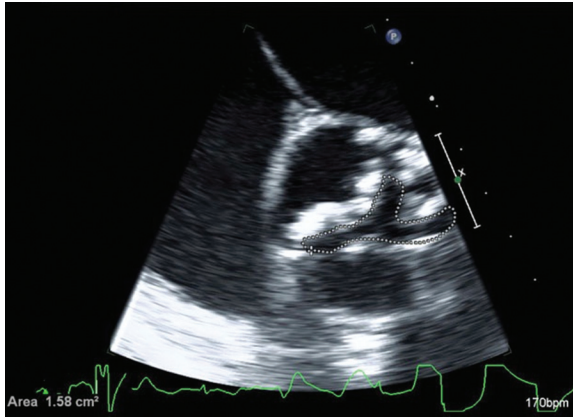


Fig. 6: Transthoracic echocardiogram image of aortic stenosis

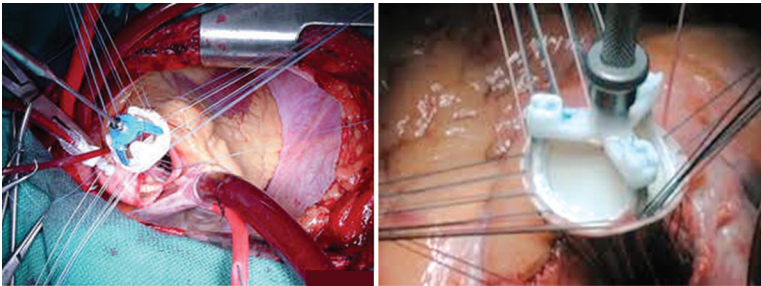


Fig. 7: Prosthetic aortic valve placement during AVR

Intraoperative

□ Hemodynamic goals

- **Rate:** Tachycardia is more hazardous in these patients as compared to that of MS because of an exacerbation of an underlying subendocardial ischemia due to decreased coronary perfusion. Optimum heart rate between 50 to 70 beats/min must be maintained to allow time for systolic ejection across a stenotic aortic valve. At the same time, low heart rate can limit cardiac output in patients with a fixed stroke volume.
- **Rhythm:** The initial appearance of symptoms in patients with aortic stenosis often is associated with the development of atrial fibrillation. Normal patients depend on atrial contraction for approximately 20% of the stroke volume. However, with the reduced ventricular compliance and increased LVEDP that is present in patients with aortic stenosis, passive ventricular filling is reduced, and atrial contraction can supply as much as 40% of ventricular filling during diastole. Therefore, loss of sinus rhythm and atrial contribution to cardiac output can lead to rapid clinical deterioration and it is **essential to maintain a sinus rhythm perioperatively**.

- **Preload:** Preload augmentation is necessary to maintain adequate stroke volume because of an increased LVEDP and LVEDV. At the same time, too much fluid may result into pulmonary edema in critical stenotic stage. Venodilators are poorly tolerated in these patients.
- **Contractility:** Stroke volume depends upon LV contractility and anesthetic agents/factors which depress myocardium should be avoided strictly. Propofol, beta blockers in high doses results in clinical deterioration.
- **Afterload:** Patients with severe aortic stenosis have a fixed cardiac output. They cannot compensate for fall in systemic vascular resistance, which result in severe hypotension, myocardial ischemia, and a downward spiral of reduced contractility causing further falls in blood pressure and coronary perfusion. Use of α -adrenergic agonists like phenylephrine maintains diastolic blood pressure and thereby coronary perfusion.
- ❑ **Premedication:** It helps in preventing stress or anxiety induced tachycardia which is vital in these patients. It can be provided in the form of oral benzodiazepines. Excessive dose of premedication may cause respiratory depression and so heavy premedication is avoided.
- ❑ **Monitoring:** All routine monitors are applied. These patients usually pose problems in the perioperative period especially depressed LV contractility in the postbypass period; so femoral arterial access is preferred which can be used later for IABP insertion.

Intraoperative monitoring should include a standard five-lead ECG system, including a V5 lead, because of the left ventricle's vulnerability to ischemia. These patients usually exhibit strain pattern preoperatively that may be indistinguishable from myocardial ischemia, making the intraoperative interpretation difficult. Simultaneously, lead II should be used for interpreting the P-wave changes.

The central venous pressure (CVP) poorly estimates the LV filling in altered LV compliance states. A normal CVP can significantly underestimate the LVEDP or pulmonary capillary wedge pressure (PCWP).

The principal risks of using a pulmonary artery (PA) catheter in these patients are arrhythmia-induced hypotension and ischemia but it also allows for measurement of cardiac output, derived hemodynamic parameters, mixed venous oxygen saturation and possible transvenous pacing. So, routine use of PA catheter is subject to individual patient characteristics and the severity of the disease.

- ❑ **Induction:** The selected anesthetic technique should maintain afterload and avoid tachycardia to maintain the balance between myocardial oxygen demand and supply. The goal should be to immediately restore the coronary perfusion pressure and then to address the underlying problem (e.g. hypovolemia, arrhythmia). A narcotic-based anesthetic usually is chosen for this reason. Treat hypotension using direct acting α -agonists such as phenylephrine which will increase systemic vascular resistance and

thereby increase coronary perfusion. Arrhythmias must be treated promptly or hemodynamic collapse may ensue. However, central neuraxial blocks must be used with extreme caution because of the danger of hypotension due to afterload reduction. Limb blocks can be used alone or in conjunction with general anesthesia.

- ❑ **Maintenance:** Careful fluid balance is essential, guided by invasive monitoring if required (CVP, esophageal Doppler, transesophageal echocardiography). Intraoperative fluid management should be aimed at maintaining appropriately elevated left-sided filling pressures. Effective analgesia avoids catecholamine-induced tachycardia and hypertension and the risk of myocardial ischemia. Low concentrations of volatile anesthetic are usually safe. The negative inotropy of the inhalation anesthetics is a theoretical disadvantage for a myocardium faced with the challenge of overcoming outflow tract obstruction. The temptation to control intraoperative hypertension with vasodilators should be resisted in most cases.
- ❑ **Weaning off CPB:** Frequently, adequate myocardial preservation with cardioplegic solution during bypass is a challenging task due to myocardial hypertrophy and hence LV dysfunction manifests while coming off bypass. In this scenario, inotropic support is often required. But, in the absence of preoperative ventricular dysfunction and associated coronary disease, inotropic support usually is not required after cardiopulmonary bypass because valve replacement decreases ventricular afterload rather than nitroglycerine infusion needs to be started postoperatively to control hypertension.

Postoperative

Although LV pressure is very high in these patients, significant systolic hypertension is usually not seen immediately in the post bypass period. However, hypertension tends to develop later on in the ICU and should be controlled.

Pre-existing myocardial hypertrophy makes LV preload dependent even in the postoperative period and the hypertrophy regresses over several months.

Patients with adequate intraoperative myocardial protection and smooth weaning off CPB can be fast tracked and usually pose no problem in the postoperative period.

HYPERTROPHIC CARDIOMYOPATHY

Introduction

Incidence: 1 in 500

Pathophysiology: The inciting factor is myocardial hypertrophy which involves septum and LV free wall. This asymmetric septal hypertrophy leads to a

variable pressure gradient between the apical LV chamber and the LV outflow tract (LVOT). The LVOT obstruction leads to increased LV pressure, which creates a vicious cycle of hypertrophy, begetting increased LVOT obstruction. Characteristic features of this obstruction are its dynamic nature (depending on contractile state and loading conditions), its timing (begins early, peaks variably) and its subaortic location. An exaggerated anterior (i.e., toward the septum) motion of the anterior mitral valve leaflet during systole (SAM-Systolic anterior motion of mitral leaflet) accentuates the obstruction.

Factors aggravating the degree of obstruction: Increased contractility, decreased afterload and decreased preload. This occurs due to reduction in ventricular volume which increases the proximity of the anterior mitral valve leaflet to the hypertrophied septum.

Hypertrophy leads to reduced diastolic compliance and the disease is characterized by both systolic and diastolic dysfunction. The poor compliance necessitates a large intravascular volume and the maintenance of sinus rhythm for adequate diastolic filling. The atrial contribution to ventricular filling is even more important in HCM than in valvular aortic stenosis and it may approach 75% of total stroke volume. Also, myocardial oxygen supply demand mismatch occurs and subendocardial ischemia may be precipitated due to increased LVEDP which reduces coronary perfusion pressure.

Treatment

- ❑ Medical— β -Blockers and calcium channel blockers
- ❑ Surgical—Ventricular septal myotomy-myomectomy of Morrow (small amount of muscle from the subaortic septum is resected)
- ❑ Newer treatment—Septal ablation with ethanol
- ❑ Dual—Chamber pacing.

Preoperative

- ❑ Clinical features—Most patients with HCM are asymptomatic.
- ❑ Dyspnea, angina, and syncope. The clinical picture is often similar to that of valvular aortic stenosis.
- ❑ Investigations
- ❑ Similar to that of valvular AS.

Intraoperative

Avoid aggravation of the subaortic obstruction.

Hemodynamic Goals

- ❑ **Rate:** Lower heart rate prevents accentuation of subaortic obstruction and hence optimum rate should be 50–70 bpm. Tachycardia is more hazardous in this condition as compared to aortic stenosis.

- ❑ **Rhythm:** Maintaining sinus rhythm is perhaps more important in this case because atrial contribution to ventricular filling can reach up to 75% and any supraventricular arrhythmia can be detrimental.
- ❑ **Preload:** Aggressive maintenance of intravascular volume keeps heart full and prevents SAM-septal contact. Hypotension is almost always as a result of underlying hypovolemia, which is potentially exacerbated by anesthetic-induced vasodilation and should be corrected by phenylephrine rather than inotropes which exacerbates contractility and worsens subaortic obstruction..
- ❑ **Contractility:** Increased contractility augments SAM and worsens obstruction therefore direct or reflex increases in contractility should be avoided. Myocardial depression is ideal because negative inotropy reduces the degree of SAM-septal contact, which results in LVOT obstruction.
- ❑ **Afterload:** Reduction in afterload is not tolerated at all; rather it increases the degree of obstruction due to compensatory tachycardia and increased contractility. Also, maintaining afterload with phenylephrine during induction improves coronary perfusion pressure.

Preload:	Increased
Afterload:	Increased
Goal:	Myocardial depression
Avoid:	Tachycardia, inotropes, vasodilators

Premedication

Heavy premedication is advisable in order to avoid anxiety-induced tachycardia or to reduce ventricular filling. Chronic β -blockade or calcium channel blockers or both should be continued up to and including the day of surgery.

Monitoring

Particular attention must be paid to arrhythmia monitoring with the help of V_5 and lead II. Abnormal Q waves can be seen in 20% to 50% of patients with HCM. These waves should not raise concern about a previous myocardial infarction; instead, they probably represent accentuation of normal septal depolarization or delay in depolarization of electrophysiologically abnormal cells. Some patients exhibit a short PR interval with initial slurring of the QRS complex, and they may be at increased risk for supraventricular tachyarrhythmias on the basis of pre-excitation.

Due to the reduced diastolic compliance, PCWP overestimates the patient's true volume status and so PCWP should be maintained in the high-normal to elevated range. A PA catheter with pacing capability is ideal because atrial overdrive pacing can effect immediate hemodynamic improvement in the event of episodes of junctional rhythm.

Induction and Maintenance

The inhalation anesthetics are commonly used for patients with HCM. Their dose-dependent myocardial depression is ideal because negative inotropy reduces the degree of SAM-septal contact, which results in LVOT obstruction.

Aggressive replenishment of intravascular volume and concurrent infusion of phenylephrine is beneficial to treat hypotension during induction.

Inotropes, β -adrenergic agonists, and calcium are all contraindicated because they worsen the systolic obstruction and perpetuate the hypotension.

MITRAL REGURGITATION

Etiology

RHD	Connective tissue disorders like ankylosing spondylosis, rheumatoid arthritis, SLE, Marfan's syndrome
Congenital	Mitral annular calcification in DM, HTN, renal diseases, elderly women
Infective endocarditis	Cleft mitral leaflet with ostium primum ASD
Amyloidosis	Dilated cardiomyopathy
Sarcoidosis	IHD

Causes of Acute Mitral Regurgitation

- Infective endocarditis trauma, acute rheumatic fever, MI, LA myxoma.

Pathophysiology

Mitral regurgitation is characterized by the decrease in forward left ventricular stroke volume and cardiac output. A portion of every stroke volume is regurgitated through the incompetent mitral valve back into the left atrium and combines with the normal LA volume and returns to the left ventricle during each diastolic period. This elevated preload leads to increased sarcomere stretch and, in the initial phases of the disease process, augmentation of LV ejection performance by the Frank-Starling mechanism. The fraction of left ventricular stroke volume that regurgitates into the left atrium depends on the size of the mitral valve orifice; heart rate, which determines the duration of ventricular ejection; and pressure gradients across the mitral valve. When mitral regurgitation develops gradually, the volume overload produced by mitral regurgitation transforms the left ventricle into a larger, more compliant chamber that is able to deliver a larger stroke volume. Development of ventricular hypertrophy and increased compliance of the left atrium permit the accommodation of the regurgitant volume without a much increase in left atrial pressure. This allows patients to maintain cardiac output and remain free of pulmonary congestion and be asymptomatic for many years.

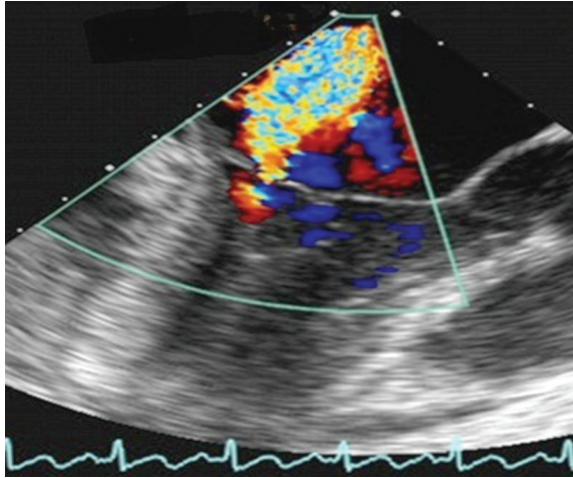


Fig. 8: Transesophageal echocardiogram showing mitral regurgitation

In cases of acute, severe mitral regurgitation (Fig. 8), such as patients with a ruptured papillary muscle after acute myocardial infarction, the sudden increase in preload enhances LV contractility by the Frank-Starling mechanism. Despite the increased preload, LV size is initially normal. Normal LV size combined with the ability to eject into a low-pressure circuit (i.e. the left atrium) results in decreased afterload in the acute setting. However, because the left atrium has not yet dilated in response to the large regurgitant volume, LA pressure rises acutely and may lead to pulmonary vascular congestion, pulmonary edema and dyspnea.

Preoperative

Clinical Features

Symptoms

- ❑ Usually asymptomatic in chronic MR
- ❑ With the progression of the disease, patient presents with decreased functional capacity in the form of dyspnea, palpitation, increased precordial activity, fatigue etc.

Signs

- ❑ Wide pulse pressure
- ❑ Hyperdynamic apical pulse
- ❑ Parasternal lift
- ❑ Soft S_1
- ❑ Pansystolic murmur radiating to axilla
- ❑ Severity—Systolic thrill over apex, S3, large LV
- ❑ Complications—AF, thromboembolism, IE.

Investigations

- ECG—P mitrale, LVH
- Chest X-ray—Cardiomegaly, signs of pulmonary venous hypertension, pulmonary edema, calcification of annulus and leaflets
- Echocardiographic grading of mitral regurgitation (Table 1).

Table 1: Grades of MR

	<i>Area of MR jet (cm²)</i>	<i>MR jet area as percentage of left atrial area</i>	<i>Regurgitant fraction (%)</i>
Mild	<3	20–30	20–30
Moderate	3.0–6.0	30–40	30–50
Severe	>6	>40	>55

Intraoperative

Goal—Decrease the regurgitant fraction and improve forward left ventricular stroke volume.

Hemodynamic Goals

- **Rate:** Higher heart rate favors a smaller mitral annular area and decreases regurgitation fraction into LA and hence rate should be kept around 90–100 bpm. Bradycardia increases LV volume overload potentially resulting in LV distention and mitral annular dilatation. Also, regurgitant volumes may increase at slower heart rates.
- **Rhythm:** Patients with pure mitral regurgitation generally have no impedance to LV filling and atrial fibrillation is usually better tolerated than in patients with stenotic lesions. Maintaining sinus rhythm is less important in these patients compared to patients with stenotic valves.
- **Preload:** Maintaining adequate LV preload is essential. An enlarged left ventricle that operates on a higher portion of the Frank-Starling curve requires adequate filling. At the same time, excessive volume administration is to be avoided because it may cause unwanted dilatation of the mitral annulus and worsening of the mitral regurgitation. Excessive fluid administration may precipitate RV failure in patients with pulmonary vascular congestion and pulmonary hypertension. Optimization of preload is aided by analysis of data obtained from PA catheter measurements and TEE images.
- **Contractility:** Heightened contractility also favors a smaller mitral annular area and decreases regurgitation fraction. Anesthetic agents that lead to decreased contractility should be avoided. High-dose narcotic relaxant anesthetics are used most commonly.
- **Afterload:** Decreased systemic vascular resistance improves left ventricular function and forward flow. Increases in systemic vascular resistance can lead to decompensation of the left ventricle and increased regurgitation fraction may precipitate pulmonary edema.

Premedication should be used **judiciously** because oversedation can lead to hypercapnia and marked increases in pulmonary vascular resistance in frail patients with ventricular dysfunction.

Monitoring

PA catheter—Changes in the amplitude of the V wave in CVP gives an idea about the degree of regurgitation and can help during anesthetic maneuvers. PA catheters allows optimal maintenance of the left-sided filling pressures; avoiding volume overload. Another benefit of PA catheter insertion is the ability to pace the ventricle in case of hemodynamically significant bradycardia.

Intraoperative TEE identifies the mechanism of mitral regurgitation, thereby guiding the surgical approach and it assesses the function of the cardiac chambers. The appearance of SAM of the mitral apparatus immediately after valve repair which is evident on TEE allows the anesthesiologist to intervene with volume infusion and medications such as esmolol or phenylephrine as appropriate.

Induction

Anesthetics induced hypotension favors regurgitant lesions due to improved forward left ventricular stroke volume. Reduction in afterload decreases regurgitant volume into left atrium. Both intravenous and inhalational induction agents can be used but inhalational agents are better avoided in patients with left ventricular dysfunction. Pancuronium produces a modest increase in heart rate, which can contribute to maintenance of forward left ventricular stroke volume.

Maintenance

Adequate fluid volume is very important for maintaining cardiac output in these patients. Anesthetic drugs with minimal effects on systemic vascular resistance and without myocardial depression should be preferred. Inhalational gases desflurane, isoflurane and sevoflurane can be used safely. Alternatively, opioid based anesthesia is preferable in sick patients.

Mechanical ventilation is directed towards prevention of hypercarbia, hypoxia and maintaining proper acid-base status.

Weaning off CPB

An interesting phenomenon called as 'Afterload mismatch' occurs after surgery; in which LV ejects fully into an aorta due to absence of low pressure regurgitant outlet postcorrection. This enhanced afterload is sometimes poorly tolerated by the LV and afterload reduction with a vasodilator drug such as nitroprusside with or without an inotropic drug/ IABP improves left ventricular function.

Patients with increased pulmonary vascular resistance preoperatively often presents with RV dysfunction after CPB and inodilators like milrinone, dobutamine may be needed to improve RV systolic function after adopting proper ventilatory strategy. Inhaled nitric oxide and prostaglandins (PGE₁) also helps in this scenario.

Postoperative

- ❑ Elective mechanical ventilation postoperatively is required in patients with ventricular dysfunction.
- ❑ Inotropic support and IABP frequently is required in the postbypass period in patients with papillary muscle dysfunction secondary to ischemia.
- ❑ Adequate analgesia in the form of IV opioids, paracetamol, opioid skin patches is provided. NSAID's should better be avoided in patients with renal dysfunction.

Aortic Regurgitation

Incidence—Common in males.

Etiology

- ❑ Aortic valve involvement: RHD, IE, congenital bicuspid valve, AV prolapse with VSD.
- ❑ Aortic wall involvement: Syphilis, RA, ankylosing spondylitis, Marfan's syndrome, Takayasu arteritis, osteogenesis imperfect, cystic medial necrosis
- ❑ Acute aortic regurgitation: Infective endocarditis, aortic dissection.

Pathophysiology

- ❑ *Chronic AR*: It is characterized by left ventricular volume overload. The amount of regurgitation depends upon the size of the regurgitant orifice, the aorta-ventricular pressure gradient and the diastolic time. Progressive volume overloading from aortic regurgitation increases end-diastolic wall tension (i.e., ventricular afterload) and produces a pattern of eccentric ventricular hypertrophy. This dilation of the ventricle, in accordance with Laplace's law, also elevates the systolic wall tension, stimulating some concentric hypertrophy. The diastolic pressure-volume curve is shifted to the right.

This permits a tremendous increase in LVEDV with minimal change in filling pressure resulting in a high diastolic compliance. In the terminal stage, progressive volume overload increases ventricular end-diastolic volume to the point that compensatory hypertrophy is no longer sufficient to compensate, and a decline in systolic function occurs. As systolic function declines, end-systolic dimension increases further, LV wall stress increases, and LV function is further compromised by the excessive ventricular afterload and myocardial oxygen supply demand mismatch occurs.

- ❑ *Acute AR*: In acute aortic regurgitation, sudden diastolic volume overload in a non-adapted left ventricle results in a precipitous rise in the end-diastolic pressure which equilibrates with aortic diastolic pressure and exceeds the LA pressure in late diastole. This causes early closure of the mitral valve and protects pulmonary capillaries from elevated LVEDP. Severe LV distention often follows and produces mitral annular enlargement and functional mitral regurgitation. The reduced stroke volume cause reflex tachycardia and peripheral vasoconstriction. Eventually, catastrophic hypotension and high LVEDP combine to cause accentuated ischemia and ventricular dilation.

Preoperative

Clinical Features

Symptoms: Chronic aortic regurgitation: Asymptomatic for many years. Symptoms such as shortness of breath, palpitations, fatigue, and angina usually develop later on.

Acute AR: Sudden onset of dyspnea, hemoptysis, pulmonary edema.

Signs

Patients with **chronic aortic regurgitation** may be asymptomatic for up to 20 years. The 10-year mortality for asymptomatic aortic regurgitation varies between 5% and 15%. However, once symptoms develop, patients progressively deteriorate and have an expected survival rate of 5 to 10 years. Early symptoms include dyspnea, fatigue, and palpitations. Angina pectoris normally is a late symptom and is an ominous sign. Patients with **acute aortic regurgitation**, on the other hand, may deteriorate rapidly, and the prognosis is guarded.

Investigations

- ECG—LVH, q waves in V5-V6
- X ray—Cor bovinum (huge cardiomegaly)
- Calcification of valve in RHD or bicuspid valve
- Echocardiography—AVR indicated if end systolic dimensions > 55 mm or end systolic volume > 55 mL/m² or EF < 55% (Rule of 55) Or end diastolic dimensions > 75 mm (Table 2).

Table 2: Severity of aortic regurgitation graded by echocardiography

Parameter	Mild	Moderate	Severe
Regurgitant jet width as percentage of LVOT width	25–46	47–64	>65
Regurgitant jet area as percentage of LVOT area	4–24	25–59	>60
Aortic diastolic flow reversal	None		Holodiastolic retrograde flow in the descending aorta

Intraoperative

Hemodynamic Goals—Similar to that of MR (maintain the forward left ventricular stroke volume)

- **Rate:** Optimum heart rate should be around 90 bpm bradycardia increases the degree of regurgitation producing acute left ventricular volume overload and the increased diastolic run off decreases coronary perfusion pressure, and the LV function may rapidly deteriorate.
- **Rhythm:** Atrial fibrillation is better tolerated and maintenance of normal sinus rhythm assumes less importance.

- ❑ **Preload:** Preload augmentation is needed to maintain forward stroke volume in already volume overloaded LV. Too much dehydration prior to the surgery and the use of NTG can reduce preload significantly which should be avoided.
- ❑ **Contractility:** LV contractility must be maintained and in fact, slight increase in contractility helps in reducing LVEDV and LVEDP. In patients with impaired LV function, use of pure β -agents can increase stroke volume through a combination of peripheral dilation and increased contractility.
- ❑ **Afterload:** An abrupt increase in systemic vascular resistance can also precipitate left ventricular failure by increasing the amount of regurgitant flow into the LV and so modest decrease in afterload should be applied.

Premedication

Light premedication is recommended to maintain myocardial contractility and heart rate because tachycardia actually can be helpful for these patients. Increases in systemic vascular resistance that may arise from anxiety, however, may be detrimental. Also, avoid drugs that dilate the capacitance vessels and reduces preload.

Monitoring

ECG monitoring is important because ischemia is a potential hazard.

PA catheter allows determination of basal filling pressures and cardiac output. It monitors ventricular preload and cardiac output response to pharmacologic interventions. The other requirement for a PA catheter is to allow for pacing when it is anticipated. Capturing the ventricle with a PA-based transvenous wire can be difficult because of the very large ventricular cavity size.

- ❑ TEE monitors LV function and provide immediate feedback concerning the integrity of valvular function and perivalvular leaks.

Induction

- ❑ Both inhalation as well as intravenous induction drugs can be used. Regional anesthesia is beneficial during noncardiac surgery. The ideal induction drug should not decrease the heart rate or increase systemic vascular resistance.
- ❑ Use of an intra-aortic balloon pump is contraindicated in the presence of aortic regurgitation because augmentation of diastolic pressure will increase the amount of regurgitant flow.

Maintenance

- ❑ Isoflurane, desflurane and sevoflurane are better tolerated due to modest increase in heart rate, decrease in systemic vascular resistance and minimal

myocardial depression. In patients with severe left ventricular dysfunction, high-dose opioid anesthesia may be preferred. Pancuronium is preferred due to increased heart rate. Bradycardia and myocardial depression from concomitant use of nitrous oxide or a benzodiazepine are risks of the high-dose narcotic technique. Mechanical ventilation should be adjusted to maintain normal oxygenation and carbon dioxide elimination and adequate time for venous return. Adequate fluid balance must be maintained.

- ❑ Problems during CPB
- ❑ Cardioplegia delivery results in LV distension due to non-competent aortic valve. This increases LV wall tension and myocardial oxygen requirement. So, LV venting has to be done during this period for myocardial protection.
- ❑ Due to large LV and eccentric hypertrophy, myocardial protection is often a problem which leads to difficulty in weaning from cardiopulmonary bypass and LV dysfunction. Inotropic support may be indicated in order to maintain cardiac output and avoid further LV dilation and dysfunction. Preload augmentation must be maintained to maintain filling of the dilated LV.

Postoperative Care

In the early postoperative period, a decline in LV function may necessitate inotropic or intra-aortic balloon pump support. Adequate preload must be maintained. Patients with LV dysfunction may require prolonged ventilator support.

PULMONARY STENOSIS

Agents that produce some pulmonary vasodilation and those that maintain venous return without myocardial depression should be used. SVR should not be lowered due to fear of reduction in coronary flow.

TRICUSPID STENOSIS

Most tricuspid pathology occurs in the context of significant aortic or mitral disease, and anesthetic management is primarily determined by the left-sided valve lesion.

- ❑ **Rate:** Tachycardia impairs RV filling and therefore heart rate must be maintained on a lower normal range. At the same time, bradycardia can be harmful because it reduces total forward flow.
- ❑ **Rhythm:** Adequate RV filling is hampered in supraventricular tachyarrhythmias and so sinus rhythm should be maintained.
- ❑ **Preload:** Forward flow of blood across the stenotic tricuspid valve depends on maintenance of high preload.
- ❑ **Contractility:** Cardiac output depends upon right ventricular contractility and hence myocardial depression must be avoided.
- ❑ Reduction in the afterload is deleterious in stenotic conditions and at the same time increased pulmonary vascular resistance impedes RV output; so avoid elevation in PVR and reduction in SVR.

TRICUSPID REGURGITATION

- ❑ Paradoxical air embolism is a possibility due to high right atrial pressures—Ensure de-airing of intravenous fluid systems.
- ❑ Maintain high normal RV preload for adequate LV filling and augmented vasodilation may be problematic.
- ❑ Maintain heart rate in higher normal range for adequate emptying of RV.
- ❑ Avoid RV contractility depression. Remember that propofol in high doses exhibit myocardial depressant properties.
- ❑ Maintain low pulmonary vascular resistance by treating factors like hypoxia, hypercarbia, acidosis, hypothermia.
- ❑ Nitrous oxide can be a weak pulmonary artery vasoconstrictor and could increase the degree of tricuspid regurgitation.
- ❑ Ventilator strategy—maintain low tidal volume, high rate and minimum PEEP.
- ❑ Inodilators like milrinone, dobutamine helps in maintaining forward RV stroke volume in the post operative period.
- ❑ Blood prime is usually not required on CPB due to high venous return.

MIXED VALVE LESIONS

General Principles of Management

- ❑ Stenotic lesion takes priority over regurgitant lesion and the formulation of an anesthetic plan should follow hemodynamic goals for stenotic lesion.
- ❑ Aortic stenosis should be given priority over mitral stenosis.

INFECTIVE ENDOCARDITIS (IE)

Definition: It is the colonization of the cardiac valvar apparatus by microbes leading to formation of vegetations rendering valve to be fragile and injury prone.

Types

Acute IE

- ❑ Most commonly caused by *Staphylococcus aureus*
 - Occurs in normal valve

Subacute IE

- ❑ Common organism is *Streptococcus viridians*
 - Occurs in previously normal/injured valve
- Infective endocarditis (IE) induced vegetation is class I indication for valve replacement.

Clinical Features

- ❑ Symptoms: Generalized like fever, weight loss, anorexia, fatigue, etc.
- ❑ Signs:

- Pallor
- Splinter hemorrhages under nailbeds
- Roth spot (retinal hemorrhage)
- Splenomegaly

Due to immune complex deposition- Osler's nodes (tender, over pulp of the fingers)

- ❑ Janeway lesion (nontender macules over palms and soles)
- ❑ Arthralgia
- ❑ Clubbing (Fig. 9)



Fig. 9: Grade III/IV clubbing

Complications

Perforation of valves, embolism, cerebral abscess, nephrotic syndrome, mycotic aneurysm

Diagnosis

DUKE'S criteria

- ❑ *Major*—Positive blood culture
 - Endocardial involvement in the form of abscess, perforation or vegetations
- ❑ *Minor*—Fever $>38^{\circ}\text{C}$
 - Presence of risk factor i.e. IV drug abuse
 - Positive blood culture
 - Positive echo findings
 - Vascular or immune complex lesions
 - Diagnosis: 2 major or 1 major and 2 minor or all 5 minor criteria.

Cardiac conditions associated with the highest risk of adverse outcomes from endocarditis for which prophylaxis for dental procedures are (Table 3)

1. Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
2. Previous infective endocarditis
3. Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits

Table 3: Antibiotic prophylaxis regimens for a dental procedure

Situation	Agent	Single dose 30 to 60 minutes before procedure	
		Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin OR Cefazolin or Ceftriaxone	2 g IM or IV	50 mg/kg IM or IV
		1 g IM or IV	50 mg/kg IM or IV
Allergic to penicillins or ampicillin—oral	Cephalexin OR Clindamycin OR Azithromycin or Clarithromycin	2g	50 mg/kg
		600 mg	20 mg/kg
		500 mg	15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medication	Cefazolin or ceftriaxone OR Clindamycin	1 g IM or IV 600 mg IM or IV	50 mg/kg IM or IV 20 mg/kg IM or IV

Note: Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin

4. Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure
 5. Repaired congenital heart disease with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
 6. Cardiac transplantation recipients who develop cardiac valvulopathy:
- In summary, (a) Antibiotic prophylaxis for endocarditis prophylaxis is recommended only for the conditions mentioned above. It is no longer recommended for any forms of congenital heart disease except above conditions. Antibiotic prophylaxis is recommended for dental procedures that involve manipulation of gingival tissues or the periapical regions of teeth or perforation of the oral mucosa. (b) Antibiotic prophylaxis is recommended for invasive procedures (i.e., those that involve incision or biopsy) on the respiratory tract or infected skin, skin structures or musculoskeletal tissue. (c) Antibiotic prophylaxis is not recommended for GU or GI tract procedures.

SUGGESTED READING

1. Cook DJ, Housmans PR, Rehfeldt KH. Valvular Heart Disease—Replacement and Repair, Kaplan Essentials of cardiac anesthesia. In: Kaplan J (Eds), 1st edition.
2. Herrera A. Valvular Heart Disease, Stoelting's Anaesthesia and coexisting disease. In: Hines RL, Marschall KE, (Eds), 5th edition. Saunders Elsevier; 27-40.
3. Park MK. Infective endocarditis and acute rheumatic fever, Pediatric Cardiology for Practitioners, 5th edition, Mosby Elsevier, Philadelphia; 351-59, 381-7.
4. Townsley MM, Martin DE. Anesthesia management for valvular heart diseases, A Practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravlee GP (Eds), 5th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia.

Chapter 8

Anesthesia for Coronary Artery Bypass Grafting

Manjula Sudeep Sarkar, Pushkar Desai

INTRODUCTION

Anesthesia for myocardial revascularization forms the core of cardiac anesthesia. It is a great challenge for the entire team comprising anesthesiologist, surgeon, perfusionist and the nursing staff. With the changing lifestyle and rise in the number of diabetic patients, coronary artery disease is increasing at an alarming rate in India. But, with the recent technological advances, more patients are being managed with percutaneous coronary intervention (PCI). As a result, coronary artery disease patients with multiple risk factors are undergoing coronary artery bypass graft (CABG) which further makes perioperative management a challenge. The anesthesiologist plays a significant role in the successful outcome of the surgery.

ANESTHESIA FOR ON-PUMP CORONARY ARTERY BYPASS GRAFTING

Incidence: In our institute, CABG comprised ~35–40% of all adult open heart surgeries in the last five years.

Preoperative Consultation

Preoperative visit by the anesthesiologist gives the patient a realistic scenario of events surrounding surgery which helps to alleviate the fear of the patient. At the same time, the anesthesiologist is able to identify the risk factors and perform risk stratification for the surgery. A full preanesthetic history and examination should be performed with particular reference to the following points which may affect anesthetic management.

History

Symptoms: Symptoms of cardiac failure, pulmonary edema and recent myocardial infarction should be noted.

Co-existing diseases: Diabetes, hypertension, cerebrovascular disease, renal insufficiency, asthma (COPD), peripheral vascular disease (presents as claudication), hemostatic disorder and hypercoagulable disorder affects anesthetic as well as surgical management.

Risk of perioperative stroke increases with age over 65, hypertension, diabetes and history of previous stroke.

History of reflux/dysphagia (GERD): Rapid sequence induction in these patients may pose special challenges. The placement of TOE probes may be hazardous in patients with esophageal disease.

Social/cultural: These factors determine the cooperation of the patient required during invasive line placement and also in post-extubation period.

Allergies: History of an allergy especially to heparin, protamine (patients having seafood allergy and those who underwent vasectomy are susceptible to the protamine reaction), iodine and antibiotics should be elicited.

Drugs: Antihypertensive, antianginal, antiarrhythmics, specific regimes for diabetes and asthma should be noted and these medications should be continued till the day of the surgery.

ACE inhibitors should be stopped one day prior to surgery to avoid excess hypotension in the perioperative period.

Heparin: Patients who receive heparin for more than 5 days should be evaluated for the platelet count before proceeding to the surgery.

Newer antiplatelet agents: Enoxaparin (Clexane) should be stopped about 24 hours prior to the surgery. Similarly clopidogrel should be stopped 5–7 days preoperatively. Abciximab—avoid CPB for 5–7 days. Tirofiban—avoid CPB for 24–48 hours, Streptokinase—avoid CPB for 2–3 hours.

Physical Examination

Airway, dentition, head and neck movement examination helps in anticipating a difficult intubation cases.

Carotid bruit suggests heightened risk of atheroma embolization during aortic cannulation and perioperative stroke.

High JVP denotes congestive heart failure which should be optimized using diuretics.

Also, it is important to **check for distal pulses** which give an idea about the feasibility of an arterial access during the surgery. Radial artery access should preferably be considered in the nondominant hand.

Investigations

- ❑ CBC—Hb, platelets
- ❑ Blood sugar, HB A1C
- ❑ Electrolytes—especially potassium and magnesium
- ❑ LFT's
- ❑ Creatinine
- ❑ Coagulation profile: Prolonged APTT in the absence of heparin (suspect lupus antibody)
- ❑ ABG
- ❑ CXR—cardiomegaly, effusions, aortic calcification, lung pathology, hiatus hernia
- ❑ ECG—rate, rhythm, conduction abnormalities, territory of infarcts

- ❑ Carotid Doppler—especially in patients with symptomatic cerebral symptoms
- ❑ Blood group and cross match.

Cardiac catheter and echo report: Note following things.

- ❑ Coronary vasculature—number, site and severity of stenosis especially left main coronary artery disease or equivalent
- ❑ Left ventricular function—LV ejection fraction, LVEDP and pulmonary artery pressures
- ❑ Valvular lesions—area and gradients which denotes the severity.

Risk Stratification

Following factors *greatly increase the risk* associated with cardiac surgery:

- ❑ Age >80 years, decompensated cardiac failure, cardiogenic shock, acute renal failure.

The following factors *moderately increase* the risk:

- ❑ Age >70 years, re-operation, emergency surgery, pulmonary hypertension, chronic renal failure.

Other factors resulting in increased risk:

- ❑ Diabetes, hypertension, obesity, ejection fraction <40%, concomitant valvular surgery, LV aneurysm, female gender.

Categorization: Before anesthetizing any patient with ischemic cardiac disease undergoing CABG; it is useful to categorize them. Easiest classification would be depending on ventricular function.

- ❑ **Class 1:** Those with normal/good left ventricular (LV) function—(EF >50%, CI >2.5 L/min/m², LVEDP < 12 mm Hg, no areas of dyskinesia or aneurysm, no history of CCF)
- ❑ **Class 2:** Poor LV function—(LVEF <40%, CI <2.0 L/min/m², LVEDP >15 mm Hg, areas of dyskinesia, akinesia, hypokinesia or aneurysm, history of CCF, cardiomegaly)

Patients with preserved LV function usually respond to pain and stress by developing tachycardia and hypertension, while those with poor LV function respond with evidence of LV failure such as decreasing cardiac index, increasing LVEDP, bradycardia and hypotension.

Anesthesia goal: Prevention/treatment of intraoperative myocardial ischemia by minimizing myocardial oxygen demand and maximizing oxygen supply.

Premedication

Lorazepam 2 mg/alprazolam 0.25–0.5 mg orally night before and in the morning along with aspiration prophylaxis should be given.

All patients should get their usual antianginals, antiarrhythmics and antihypertensives. Aspirin, clexane, NSAIDs and oral hypoglycemics should be

stopped accordingly. ACE inhibitors should be stopped 24 hours before surgery to prevent excess hypotension during induction. Heparin can be continued but stopped 6 hours prior to surgery.

Diabetics on insulin should be taken first for the surgery if possible.

Monitoring

All standard ASA monitors (ECG, pulse oximetry, capnography, invasive blood pressure, temperature) should be applied.

Cardiac output monitoring using PA catheter (in patients with LV dysfunction) and/or Flotrac helps in determining adequacy of peripheral tissue perfusion to some extent.

Temperature monitoring: Both core (rectal) and surface (nasopharyngeal) temperature should be measured.

Depth of anesthesia monitoring using BIS monitor helps in avoiding excessive anesthetic dose and facilitates fast tracking.

Neuromuscular monitoring prevents excess dose of the muscle relaxants and early extubation becomes possible.

Ischemia monitoring: Monitoring leads II and V5 will pick up the majority of ischemic episodes detectable by ECG. When the blood flow to myocardium is insufficient, regional wall motion abnormality develops within 5 to 10 seconds. At 60 to 90 seconds, changes in the ST-T wave become evident.

Transesophageal echocardiography (TOE) monitoring will detect ischemia earlier than the ECG and abnormal wall motion due to ischemia may occur without ECG changes. The best view for monitoring for ischemia is the short-axis mid-papillary view.

Communication

Cardiac anesthesia involves a series of repetitive procedures which absolutely have to be done correctly. Communication with other anesthesiologist and surgical team is necessary.

Induction and Maintenance

Anesthesia induction for CABG should be done in presence of surgeon and perfusionist because of high chance of patient collapse during induction and need to commence CPB immediately in this situation.

For a standard (non-fast tracked) case, induction consists of fentanyl 5–10 µg/kg, thiopentone 50–200 mg/propofol 0.5–1 mg/kg or midazolam 0.1 mg/kg and 0.1 mg/kg pancuronium. The vagolytic action of the pancuronium counteracts the bradycardia induced by the high narcotic doses. Following intubation, the TOE probe is inserted (unless there is a contraindication).

No form of anesthesia has been demonstrated to be better than another. Isoflurane, sevoflurane, high and low dose narcotic-based anesthesia are

equivalent *provided* hemodynamics are well controlled. Nitrous oxide is avoided because of potential problems with expansion of gaseous emboli in open heart procedures.

Heart rate control is particularly important as intraoperative tachycardia is associated with myocardial ischemia in these patients.

Hypotension during induction should be tackled promptly with the use of vasopressors like phenylephrine or ephedrine taking care not to induce tachycardia. To avoid such a complication, careful titration of anesthetic drugs during induction is necessary.

Sternotomy

A long period of little stimulation occurs following induction. Once surgery starts, there is a relatively short period before the painful stimuli of sternotomy and (even more stimulating) sternal retraction occurs. Hence it is important to ensure that the patient is adequately anesthetized prior to incision. Disconnect the patient from the ventilator during sternotomy to avoid tearing the pleura but never forget to restart the ventilation. During redo sternotomy with an oscillating saw it may not be necessary to cease the ventilation.

Redo Surgery Sternotomy

In redo cases, adhesions may bring the ventricle close to the sternum. The sternal saw may cut through the right ventricle or innominate vein resulting in massive hemorrhage. So it is always advisable to have blood available and a large bore IV (14/16 G) should be taken prior to the surgery. It is also possible to cut through the internal mammary artery (IMA) or a saphenous graft, with instant and severe myocardial ischemia as a result. It is less hazardous if the IMA and grafts are not functional. A functional graft that the patient is dependent on is potentially the grave situation. One way to avoid this is to ventilate patient with high tidal volume so that lungs may cover LIMA/saphenous graft and the injury to the graft may not result.

Internal Mammary Artery (IMA) Dissection

The surgeon may want the table tilted to the left and elevated for LIMA harvesting. The tidal volume should be reduced and the rate increased to help with the dissection and a brief period of hand ventilation during this period may be required.

Heparinization

NO CPB without Heparinization!

The central line is chosen for heparin administration. Blood is aspirated from the line before and after the heparin dose to ensure that the heparin is in the circulation. If blood is not aspirated then a different lumen of the central line

should be used. If an IMA is being harvested, the surgeon will ask for heparin prior to detaching the distal end.

Usually heparin is given during purse string suture on aorta or right atrium. Heparin should be given as a bolus over 10–15 seconds. There might be a slight drop in systemic pressure due to ionized hypocalcemia. The dose of heparin is 400 U/kg. The ACT is checked at least one minute after the dose (usually 3 minutes).

ACT (Activated Clotting Time)

The ACT should be greater than 400 seconds prior to initiating CPB. If aprotinin has been used, it needs to be above 750 seconds. If the patient is on heparin preoperatively, the same dose (Heparin 400 U/kg) should be repeated. If the ACT is still less than 400 seconds after the second dose of the heparin then FFP/antithrombin III complex concentrate is infused until the ACT is above 400 seconds (heparin resistance).

Cannulation

During aortic cannulation the BP should be lowered to 90–100 systolic in order to minimise the risk of aortic dissection. The small bore cannula in the aorta should not have any bubbles in it. If a bubble is seen, the surgeon should be informed immediately.

Checklist for going on bypass

- ❑ Heparin: Always give prior to bypass.
- ❑ ACT: Always check before going on bypass (400 seconds)
- ❑ Drugs: Ensure additional doses of nondepolarizing neuromuscular blocker and narcotic.
- ❑ Infusions: Turn off the inotropes and other infusion.
- ❑ Pull the PA catheter back 5 cm to avoid pulmonary arterial occlusion/rupture.
- ❑ Alarms: Disable alarms tones (ECG, BP, CO₂, etc.)
- ❑ Ventilator: Turn off once patient is safely on bypass (“full flow”)
- ❑ Air: Check the arterial cannula for air bubbles.

On initiating bypass, a perfusionist removes the clamp from the venous drain line and a siphon effect drains blood from the right atrium and inferior vena cava into the venous reservoir. It is important to maintain the siphon effect to keep this flow going. Since there is no or less blood going into the right ventricle, the cardiac output drops. The perfusionist then turns on the pump and returns the blood via the arterial cannula into the patient's aorta. The blood will be heated/cooled and oxygenated by the heater/cooler/oxygenator. The perfusionist will say “Full flow” which means that the pump flow has reached around 2.2 L/min/m². At this point the ventilator should be turned off alongwith IV infusions. Anesthetic drugs need to be given directly into venous reservoir or through peripheral vein.

Anesthesiologist should always ensure that heparin is given prior to commencing CPB; oxygen source is connected into CPB and blood in the reservoir remains above the minimum level to avoid risk of massive air embolism.

Air lock: The venous line drains by siphon effect. Air introduced into the venous system can cause the loss of this siphon effect. If the perfusionist notes bubbles in the venous return line, integrity of the tubings is checked, closure of all stop cocks is ensured, and the surgeon checks the atrial purse string. It is rectified by reducing pump flow so that the venous pressure will rise and the air leak will diminish. The lines can be refilled with saline if complete airlock occurs.

Hemodynamic

Hypotension

Profound hypotension can occur with cardiac manipulation. If the blood pressure suddenly drops or VPC's develop, it is better to rule out surgical manipulation before taking definitive steps.

Prebypass Hemodynamics

In general, the blood pressure is kept around 90–120 mm Hg (systolic) and the heart rate between 50 and 80 depending on the clinical situation prior to commencing CP bypass. During purse string insertion, systolic BP should be brought down to 90–100 mm Hg to prevent aortic dissection during cannulation. Papaverine injection by the surgeon during mammary artery harvest may cause transient hypotension. Tachycardia and hypertension should be treated by increasing the depth of anesthesia first. Esmolol or metoprolol are useful drugs if tachycardia and hypertension coexist. Clonidine 0.5–1 µg/kg is also useful in these cases.

Bypass Hemodynamics

MAP is generally kept between 40–50 mm Hg during the cold period of bypass (cross clamp on) and between 50–60 mm Hg during warm bypass (cross clamp off). There will be exceptions—such as patients with carotid vascular disease or chronic renal insufficiency that may need higher pressures (60–80 mm Hg) on CPB.

Postbypass Hemodynamics

Systolic blood pressure greater than 80 mm Hg is accepted very well. If it is greater than 120 mm Hg then the patient is likely to bleed more in the postoperative period. Cardiac index should be greater than 2.0 L/min/m². PA diastolic pressure should be less than 20 mm Hg and the CVP should be less than 15 mm Hg. If CVP is greater than PA pressure; then the problem of poor calibration or right ventricular failure should be suspected. If hypotension

occurs, always consider surgical manipulation of the heart if the chest is open or tamponade if the chest is closed. Difficult hemodynamic problems can be sorted out with the use of TEE.

Checklist for weaning off bypass

- ❑ *Temperature:* Nasopharyngeal temperature should be between 36.5–37 C.
- ❑ *Rhythm:* Sinus rhythm is ensured.
- ❑ *Monitors (Alarms):* Turn on all the monitors along with alarms limit.
- ❑ *Ventilation:* Commence ventilation before going off CPB.
- ❑ *Perfusion:* Vasoconstrictors may be necessary if the MAP remains below <50 mm Hg despite “full flows”
- ❑ *Non-acidotic* milieu must be achieved.

Weaning from bypass

One weaning plan would be to calculate the systemic vascular resistance (SVR):

$$\text{SVR} = [(\text{MAP} - \text{CVP})/\text{CO}] * 80$$

MAP: Mean arterial pressure, CVP: Central venous pressure, CO: Cardiac output (pump flow rate corresponds to cardiac output).

SVR should be in the 1000 to 1200 unit range. If it is 600–800 then the cardiac output necessary to develop a reasonable pressure post-bypass may well be too high. Vasoconstrictors or a catecholamine with some vasoconstrictive effects (dopamine, adrenaline, noradrenaline) are commonly necessary to raise the MAP.

In this scenario, the problem is simply low resistance and hence a vasoconstrictor rather than an inotrope is needed.

Another simple way to decide about the requirement of an inotrope is to look for the contractility of the ventricle directly and on the TEE. A well contracting heart produces an audible snap. A poorly contracting heart prior to bypass most likely will require an inotrope while coming off bypass. If the inotropic state of the ventricle was good prior to bypass and cross clamp times were reasonable (60 minutes or less) then it is likely that no inotropes will be needed.

The default catecholamine support is either dopamine or adrenaline.

Careful attention is paid to both the right and left ventricles to make sure they are not distending while coming off bypass.

Protamine

Protamine should not be given until the patient is off bypass. The standard dose of the protamine is 1 mg for every 100 units of heparin. The post-reversal ACT should be less than 120% of normal (<150 seconds). It should be given slowly with vigilance for an allergic reaction manifested as hypotension, bronchospasm, rash, or pulmonary hypertension. Protamine should be stopped if these occur. Severe hypotension from protamine is treated with phenylephrine, steroids, H₁ and H₂ blockers, vasoconstrictors, inotropes, and occasionally, it may be necessary to return to bypass again. Perfusionist must be informed when one-third of the protamine dose is completed so that they

can stop the pump suckers and avoid clotting in the pump. This is important because of the need to return to bypass at anytime thereafter. Once all the protamine is finished another ACT is repeated and more protamine is given if ACT does not return to 20% of the baseline. If pump blood is given after this point, extra protamine should be given due to the heparin in the pump-blood.

Postbypass bleeding: First step is to correct ACT keeping in mind that excess protamine can itself result in bleeding. Most nonsurgical bleeding is due to platelet dysfunction and hence platelet transfusion may be necessary. Epsilon aminocaproic acid has been used as an antifibrinolytic. It reduces postbypass bleeding but is not as effective as aprotinin. Aprotinin is an antifibrinolytic protease inhibitor that reduces bleeding and transfusion associated with redo CABG surgery and patients on aspirin. Aprotinin prolongs the celite ACT hence ACT on bypass must be kept over 750 seconds in patients who have received aprotinin. Aprotinin is occasionally allergenic so extra care should be taken in patients who have received it before, especially within 3 months.

Returning to bypass: If there is severe hypotension, bleeding, low cardiac output, or other problems, it may be unavoidable to return to bypass once again. Another dose of heparin (400 U/kg) is repeated and ACT is checked. When returning to bypass it is better to be over heparinized.

Before the aortic cannula is removed, if the blood pressure is low despite inotropes, it is important to communicate to the surgeon who either delays the removal of the aortic cannula or returns to bypass again. It is very detrimental to the heart to be dilated by high filling pressures resulting in low coronary perfusion pressure.

Intra-aortic balloon pump: It may improve ventricular function in a failing heart by increasing the coronary perfusion pressure. Placement may be aided with the use of TOE. The pump is synchronized with the ECG or the arterial pressure trace. LV Assist Device or ECMO are also available for the failing myocardium.

Closing the chest: It may cause hypotension if the patient is hypovolemic. If the lungs seem too large or if the heart is lifting out of the chest then bronchospasm with air trapping should be considered. Bronchodilators, ventilator and ETT adjustment can help in this regard.

Removing the TEE: It should be unlocked before removal.

Planning for Early Extubation (“Fast-tracking”)

Early extubation (within 6 hours postoperatively) lead to reduced ICU stay and lower costs without increasing patient morbidity. Generally only low-risk cases are suitable candidates for early extubation. On-table extubation may be possible in OPCAB patients.

Early extubation should be discussed at the beginning because it requires planning. Attention should be paid to limiting intravenous fluids, limiting the total narcotic and benzodiazepine dose and most importantly, keeping the patient warm and pain free. If the temperature is less than 35.5°C at the end of the surgery extubation is avoided. Careful control of blood pressure with emergence may be necessary.

Transport: The bed transfer often leads to hypotension and arrhythmia and so it is important to monitor the patient during transfer to the intensive care unit.

ANESTHESIA FOR OFF-PUMP CORONARY ARTERY BYPASS

Introduction

Off-pump coronary artery bypass (OPCAB) surgery is one of the few surgeries where anesthesia management is at par with the surgery itself. Anesthesia for OPCAB surgery demands thorough knowledge and anticipation of the steps of the surgery, continuous vigilance and skilled hemodynamic management.

The first OPCAB surgery was performed by Kolesov in 1964. The OPCAB procedure is characterized by the absence of CPB, surgery on a beating heart and moving lungs, use of an epicardial suction stabilizer, temporary interruption of coronary blood flow during anastomosis of distal vessels and early extubation.

Advantages of OPCAB

The main advantage of OPCAB surgery is avoidance of the systemic inflammatory insult inflicted by the cardiopulmonary bypass pump. The key advantage is the reduced incidence of neurologic dysfunction (confusion, delirium, stroke) when compared to surgery on pump. Incidence of neurologic complications has been reported to be less than 1% when compared to 2–3% with CPB. OPCAB also avoids potential risk of myocardial ischemia during CPB due to inadequate myocardial protection. Numerous studies have shown lesser release of troponin T and troponin I after OPCAB as compared to conventional CABG. These have also shown less requirement of inotropes post-surgery and a reduced incidence of arrhythmia and bleeding complications. This is attributable to the lack of hemodilution and an absence of systemic inflammatory response as well as an absence of platelet sequestration which occurs during CPB. Renal function is better preserved with OPCAB, as demonstrated by fewer instances of postoperative renal insufficiency.

There are also other advantages in terms of reduced respiratory complications and facilitation of early extubation.

Preoperative Considerations

Premedication: Use of short acting benzodiazepines and opiates is warranted since early awakening and extubation is desirable after OPCAB surgery. One of the regimes is to use relatively small doses of benzodiazepines (Alprazolam 0.25 mg) preoperatively and supplement with intravenous midazolam 0.005 mg/kg IV and fentanyl 1 µg/kg IV in the operating room during placement of invasive lines.

All cardiac medications needs to be taken till the morning of surgery with the exception of ACE inhibitors which should be stopped 24 hours prior to the surgery in order to prevent excess hypotension during the perioperative period.

Investigations: Apart from routine investigations, special attention should be paid to the ventricular function, coexisting valvular pathology specially mitral regurgitation which warrants careful fluid administration and avoiding vasoconstrictors during verticalization of the heart in case of anastomoses to obtuse marginal/high ramus coronary artery.

Monitoring: All standard ASA monitors must be used. Apart from this, special monitoring includes use of pulmonary artery catheter (PAC) for cardiac output monitoring and measurement of PCWP to guide fluid therapy. Some centers use continuous cardiac output monitors like flotracs during surgery for hemodynamic management. Transesophageal echocardiography has a limited role in OPCAB as compared to the conventional CABG due to the difficulty in obtaining useful information while the heart is retracted for many of the distal anastomoses. Temperature monitoring deserves special mention during OPCAB as maintaining normothermia is the crux during surgery to avoid hypothermia induced ventricular fibrillation and to facilitate early extubation. Accordingly it is better to keep patients warm preoperatively and ensure use of forced air warmers, hotline for warm fluids and blood products and other active warming measures. Sometimes, it is essential to raise the temperature of the OR to keep patient warm. Always ensure presence of sterile internal defibrillator paddles before start of the surgery. It is better to back up invasive arterial monitoring with NIBP in case problems arise with IBP. For ischemia monitoring, ST segment analysis on cardioscope should be turned on. More than 2 mm ST elevation and 1 mm ST depression is considered significant and should be informed to the surgeon who will reposition heart accordingly. The plethysmograph on the pulse oximeter is very helpful in assuring adequate perfusion. Depth of anesthesia monitoring using BIS is helpful to avoid anesthetic overdose and consequently delayed extubation.

Induction and Maintenance

The induction of anesthesia is tailored to the patient's status and the aim is to extubate at the end of the case. Etomidate or propofol are most often used for induction, along with a loading dose of opioid. For patients with ventricular dysfunction, induction with high narcotics or etomidate should be preferred. Sudden fall in blood pressure during induction is countered by using vasopressors like ephedrine (3–6 mg boluses) or phenylephrine (20–100 µg boluses). At the same time stress response due to laryngoscopy must be prevented by opioid, esmolol, lidocaine or topical lignocaine prior to laryngoscopy. For coronary vasodilation, background infusion of nitroglycerine is started prior to induction taking care not to exaggerate hypotension. Anesthesia is maintained using a volatile agent (isoflurane/sevoflurane) and occasionally, a propofol infusion is also used to control high blood pressure. Nitrous oxide is avoided in patients with pulmonary hypertension. Muscle relaxation is provided with intermediate acting neuromuscular blockers which can be reversed easily at the end of surgery.

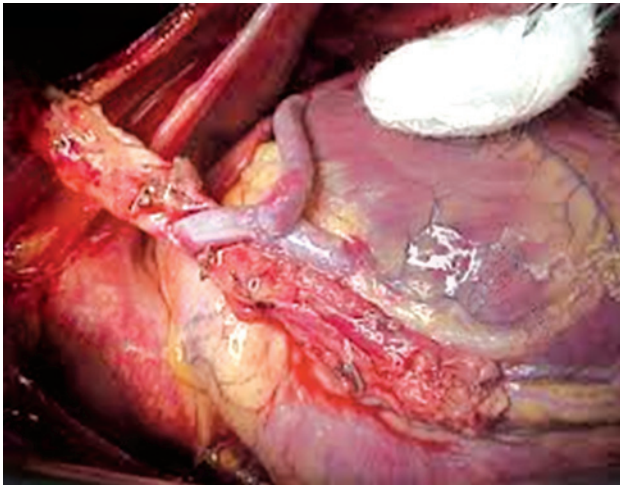


Fig. 1: Photograph showing LIMA to LAD anastomosis

Inotropes and Vasopressors

Sudden hemodynamic perturbations during OPCAB surgery require vasodilator and vasopressor/inotrope for immediate infusion. Dopamine and noradrenaline depending upon the preoperative contractile status of the heart are frequently used. Inodilators like milrinone and levosimendan reduce PA pressure intraoperatively in patients with ventricular dysfunction and coexisting mitral regurgitation. But in these cases noradrenaline is required to counter excessive peripheral vasodilation and low SVR. If the cardiac index continues to fall during the anastomosis (e.g. CI <1.5), bolus doses of epinephrine (10–20 μ g) are given immediately to avoid progressive cardiac failure. Maintenance of cardiac output appears to be more important than maintaining systemic blood pressure. Metabolic acidosis is treated with sodium bicarbonate to keep the corrected pH greater than 7.30 to maintain hemodynamic stability.

Needless to mention that metabolic acidosis should be corrected first before using inotropes.

Heparin

Injection heparin 200 U/kg IV to keep ACT greater than 250 seconds during anastomoses is given. ACT is monitored every hourly and accordingly heparin is repeated in the dose of 20–50 U/kg.

Fluid Management

OPCAB differs from conventional CABG in liberal fluid administration (if ventricular function is normal preoperatively) in order to maintain adequate

preload for better hemodynamics during distal anastomoses which require retraction and verticalization of heart. Pulmonary artery catheter helps in guiding the fluid management in the perioperative and postoperative period.

Some centers administer glucose (D 5%), potassium (20 mEq) and 10 U insulin (GPI) solution perioperatively (depending upon baseline potassium level) which has been shown to have an ischemic preconditioning effect.

Transfusion Criteria

The ultimate goal of anesthesia management is to maintain optimal oxygen supply demand ratio of the myocardium. In order to increase or maintain physiologic oxygen supply, it is desirable to maintain adequate hemoglobin level. Till date no single transfusion trigger is recommended in the literature. The trigger varies according to the clinical situation and patient profile. But still it is a usual practice to maintain hemoglobin at or above 10 gm% in the perioperative period. Numerous methods have been described to alleviate serious side effects of allogenic blood transfusion;. Use of antifibrinolytics (aprotinin, tranexamic acid), autologous blood, acute normovolemic hemodilution and cell saver definitely reduce transfusion requirements.

Antiarrhythmic Agents

It is usual to detect arrhythmias during distal anastomoses particularly of right coronary artery (posterior descending artery); atrial fibrillation being most common. So it is advisable to administer injection lidocaine 1.5 mg/kg IV bolus during this period. Also injection magnesium sulfate 2–4 gm IV prevents arrhythmia intraoperatively because most of the patients have depleted total magnesium level in the body due to chronic treatment with diuretics. Sometimes, it is necessary to start infusion of amiodarone to treat refractory arrhythmia after reperfusion of the occluded coronaries.

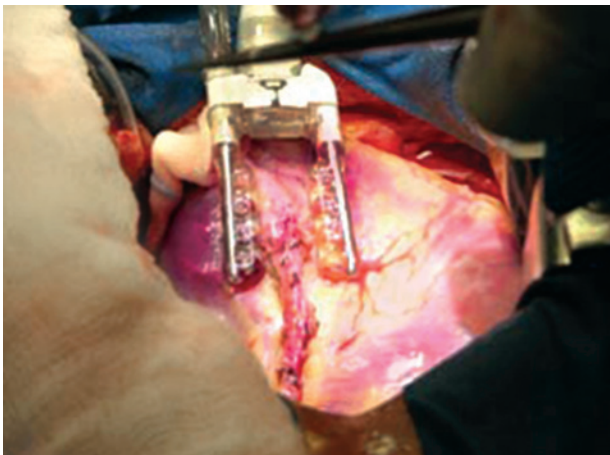


Fig. 2: Photograph showing OCTOPUS—myocardial stabilizer

Surgical Part and Anesthetic Maneuvers

Period of distal anastomoses is the most challenging part of the surgery and demands keen observation and anticipation from the anesthesiologist. Vigilance and timely interventions can prevent catastrophes. Communication with the surgical team is a must during this period.

The key to anesthetic management during a distal anastomosis is to aggressively maintain hemodynamic stability.

To optimize surgical exposure during a distal anastomosis, the anesthesiologist may need to hand ventilate or even stop ventilating for short intervals. This would be most likely, for example, during the anastomosis to a marginal branch of the left circumflex artery. It may be necessary to subsequently reexpand the lungs and hyperventilate to prevent patchy atelectasis and hypercarbia.

Once the anastomosis is complete, and coronary flow is reestablished, both cardiac index and ST segment changes usually improve. Blood pressure and cardiac output should return to near baseline levels before the surgeon attempts the next anastomosis, especially if it involves displacement of the heart.

Anastomosis of the marginal and ramus branches of the circumflex artery deserves special mention here. It requires deep pericardial retractors or a sling may be used to retract the heart into an optimal position for the surgical approach. This displacement of the heart results in verticalization with the apex pointing anteriorly, causing right ventricular compression and dysfunction. This deterioration in circulatory status is due primarily to a severe reduction in stroke volume, as the geometrically distorted right ventricle cannot sufficiently expand during diastole. During this retraction phase, the ECG tracing is altered and ST analysis also becomes unreliable. If a TEE is being used, its images of the retracted heart provide little useful information. Fortunately, cardiac output measurements and plethysmography are often a reassuring guide of peripheral perfusion in addition to visual impression of the contractility of the heart.

In order to combat these hemodynamic perturbations during heart displacement, the patient is placed in approximately 20-degree Trendelenburg position. Steep Trendelenburg position causes decrease in pulmonary compliance and functional residual capacity and may compromise adequate ventilation, especially in obese patients.

After completing distal anastomoses, the surgeon applies partial cross clamp to the aorta for the proximal anastomoses. This step requires rapid lowering of blood pressure to 90–100 mm Hg, usually with volatile agent, nitroglycerin, or nitroprusside to prevent aortic dissection. This period of proximal anastomoses provides some rest to the heart and allows it recover from repeated ischemic insults during distal anastomoses. Once proximal anastomosis is completed, hemostasis is achieved and closure is started. During hemostasis, heparin is neutralized usually by half dose of protamine or may not be reversed at all if there is concern regarding graft thrombosis.

Extubation

After the sternum is approximated, the muscle relaxant is reversed (cell saver blood is returned at this time in some institutes). In order to facilitate extubation in the OR, patient must be awake, normothermic, non-acidotic, adequately breathing and most importantly pain free. If the patient does not meet these criteria, extubation can be done later in the intensive care unit.

Postoperative Analgesia

Adequate analgesia is an integral part rather prerequisite for early extubation after OPCAB. It is a good strategy to infuse 1 gm IV paracetamol to all patients after sternal closure so that its peak effect is achieved at the time of extubation. Later on IV paracetamol 8 hourly in addition to opiate (fentanyl) provides adequate analgesia. Continuous dexmedetomidine infusion following extubation to keep the patient calm and pain free is one of the strategies. It is also better to give noninvasive ventilation (CPAP) postextubation for 1–2 hours for better respiratory mechanics.

Different modalities of pain relief described in the literature are as follows:

- ❑ Intravenous opioids
- ❑ Patient controlled analgesia
- ❑ Thoracic epidural analgesia
- ❑ Paravertebral nerve block
- ❑ Intercostal nerve block
- ❑ Intrapleural analgesia
- ❑ Intrathecal opioid
- ❑ Alpha-2 agonists

Summary

The goals of anesthetic management for OPCAB are, monitoring and prevention of coronary ischemia, maintaining anesthetic depth and hemodynamic stability and appropriate plan for postoperative care such as early extubation and adequate pain relief.

ANESTHESIA FOR MINIMALLY INVASIVE CARDIAC SURGERY: MID-CAB

- i. “MID-CAB” is nothing but a mini-thoracotomy with no CP bypass. This procedure requires a double-lumen tube and one lung ventilation. The standard anastomosis is a single IMA to the LAD. The heart is stabilized by placing latex sutures under the LAD proximal and distal to the site of the anastomosis. A small stabilizer presses on the myocardium. Blood flow is stopped in the target vessel by the stabilizing sutures. External defibrillator pads must be attached to the patient as the heart cannot easily be defibrillated internally via this incision. On-table extubation is usually

possible, so a warming blanket is required to prevent hypothermia which can delay extubation.

- ii. “OPCAB” or “Octopus” or “Platypus”: This is a sternotomy without CP bypass with the heart stabilized by two arms with rows of suckers like octopus legs. Another retractor can also be used. Air and saline blower keeps the field dry while the surgeon operates.

Both these techniques require improved technical skill on the part of the surgeon since the heart is in constant motion. (heart contraction as well as respiratory movements). It also requires increased technical skill on the part of the anesthesiologist because an area of myocardium is necessarily ischemic. Hemodynamic instability and arrhythmias can occur.

Challenges

There are two major challenges in MIDCAB viz ventricular fibrillation and hypothermia. One has to have a backup plan to tackle ventricular fibrillation as there is limited space for internal defibrillator paddles. So it is imperative to apply external defibrillator paddles preoperatively. Another problem with these cases is hypothermia. On-pump cases can be allowed to cool down pre-bypass because they are routinely rewarmed to 37deg prior to weaning from bypass. OPCABs must be kept warm to prevent fibrillatory episodes and to allow early extubation.

Summary

- ❑ An anesthetic that lowers the heart rate (this will minimize the risk of myocardial ischemia occurring during target vessel occlusion) is chosen.
- ❑ Perfusionist should be immediately available if required.
- ❑ Positioning of the heart and placement of stay sutures/footplate may require preload adjustments. Fluids and /or vasoconstrictors are frequently necessary. Beta-agonists may produce pro-arrhythmic effects. The use of the Trendelenberg position is usually used to aid right ventricular filling.
- ❑ Additional full dose of the heparin (in case of need to go on bypass urgently) is needed.
- ❑ The ventilator is adjusted to reduce the motion of the lungs (small tidal volumes with increased rate).
- ❑ Reperfusion arrhythmias occur very often with release of the stay sutures.

Anesthesia for Robotic CABG

Robotic cardiac surgery offers numerous advantages like decreased blood loss and surgical stress as well as better cosmesis. Anesthetic management of robot-assisted coronary artery bypass surgery is challenging which needs to focus on respiratory mechanics, pain management and fast tracking.

Patients who can tolerate one lung ventilation (OLV) for a prolonged period of time are suitable for this surgery. Patients with COPD and its sequelae may not tolerate hypercarbia, hypoxia and barotrauma resulting from OLV

and carbon dioxide insufflation. Pulmonary function tests (PFT) should be performed, with proper optimization using bronchodilators and steroids.

The patient is positioned supine with a slight lateral tilt with the arm on the side of chest ports which are suspended from a support at the level of head. Proper arm positioning is crucial to prevent any inadvertent nerve plexus injury. Once the robotic arms are inside the patient's chest, changes in patient or instrument panel position are not possible.

External defibrillator pads should be applied before induction of anesthesia since internal defibrillation is not possible.

ECG monitoring and analysis becomes more difficult due to the capnothorax-induced changes and the placement of chest ports which preclude the ideal ECG lead placement. PA catheter is highly desirable as it not only helps in monitoring CO, PAP, CVP and PCWP but also gives vital information about the peripheral oxygen delivery through SvO₂ measurement. TEE has a significant role in minimally invasive cardiac surgery to check the placement of various cannulae as well as monitoring for myocardial ischemia and cardiac function. Also, de-airing of the heart can be better monitored with TEE in view of the space constraint.

One lung ventilation (OLV) and a capnothorax are prerequisites to this surgery and can cause significant hypoxemia during the operative period. Insufflation pressures should be continuously monitored and insufflation of CO₂ should be maintained at 2–3 liters per minute to maintain intrapleural pressure less than 10 mm Hg. Positive end-expiratory pressure (PEEP) should be applied to the ventilated lung along with the continuous positive airway pressure (CPAP) to the non-ventilated lung if required.

Capnothorax reduces venous return decreasing cardiac output (CO). It increases central venous pressures (CVP), pulmonary capillary wedge pressure (PCWP) and decreases mixed venous oxygen saturation (SvO₂).

Maintaining the operating room temperature with proper air conditioning and use of ambient air warmers and fluid warmers can help in preventing hypothermia.

Regional analgesia like thoracic paravertebral block (PVB), thoracic epidural analgesia (TEA) and intercostal nerve block have also been used in these patients. The advantage of PVB is minimal hemodynamic perturbations with no risk of epidural hematoma.

ANESTHESIA FOR AWAKE CABG

Awake CABG is the novel strategy which requires high level of expertise from the surgeon, anesthesiologist and immense cooperation from the patient. This type of surgery is beneficial to the patients who are not suitable to undergo general anesthesia due to preexisting pulmonary pathology or cerebrovascular episode.

Key features of the management includes:

- i. High thoracic epidural (T1-T2/T2-T3 interspace) to provide sympathetic, motor and sensory block from C6 to T8 level. Preferably, epidural catheter

- is put one day prior to the surgery and the positioning is confirmed by radiologic method. Care must be taken to stop antiplatelets/heparin prior to the placement of the epidural catheter.
- ii. High level of suspicion and extreme vigilance is required to detect local anesthetic systemic toxicity (LAST) due to high volume and concentration of the local anesthetics. Surgical expertise is warranted to avoid prolonged anesthesia.
 - iii. Inadvertent opening of the pleura may produce intraoperative altered respiratory mechanics which may require conversion to general anesthesia.

ANESTHESIA FOR TRANSMYOCARDIAL LASER REVASCULARIZATION

This procedure is performed usually via left anterior thoracotomy and the management is same as that of the minimal invasive CABG apart from the precautions taken during the laser surgery.

- i. Monitor with radial arterial line and PA catheter
- ii. *TEE is needed to confirm laser "hits"*
- iii. Arms should be kept by the side and, *saline bag under the left chest*
- iv. External defibrillator pads are applied preoperatively.
- v. Groin remains uncovered for possible IABP insertion
- vi. Anesthesia plan for on-table (or soon after) extubation is desirable.

SUGGESTED READING

1. London MJ, Mitanachat A, Kaplan JA. Anesthesia for myocardial revascularization. Kaplan Essentials of cardiac anesthesia, 1st edition. Saunders Elsevier; 2008.
2. Mehta V. Off pump CABG Anesthetic and Surgical Consideration. Austin J Anesth Analg. 2014;2(1):8.
3. Okum G, Horrow JC. Anesthetic management of myocardial revascularization. Practical approach to cardiac anesthesia, 4th edition. In: Henseley FA Jr, Martin DE, Gravlee GP (Eds). Lippincott Williams and Wilkins.
4. Watanabe G, Tomita S, Yamaguchi S, Yashiki N. Awake CABG under thoracic epidural: impact and recovery. Eur J Card Thorac Surg. 2011;40:788-93.

Chapter 9

Anesthesia for Vascular Surgery

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INTRODUCTION

The changes in the practice of vascular surgery from the conventional open reconstructions and repairs to percutaneous minimally invasive endovascular repairs have reduced the morbidity and mortality of the high-risk vascular surgical procedures. Rapidly increasing elderly population is likely to increase the number of vascular procedures performed.

Anesthesia specialization in vascular surgical procedures is associated with improved outcomes. As shown by Walsh et al. in their retrospective cohort study, vascular anesthesia specialization reduced early (within 30 days of surgery) and medium-term (within two years of surgery) mortality rates following both elective and emergency major vascular surgeries (lower limb revascularization, abdominal aortic aneurysm (AAA) repair, endovascular aneurysm repair and carotid endarterectomy).

PREANESTHETIC EVALUATION

Atherosclerosis is a systemic inflammatory disease affecting regional circulations (coronary artery disease, cerebral vascular disease, renal artery disease, peripheral artery disease) of predominantly elderly population. Ischemic heart disease and cerebrovascular disease are two of six independent risk predictors for patients undergoing nonurgent major noncardiac surgery (also including congestive heart failure, insulin dependent diabetes, preoperative renal insufficiency and high-risk surgery).

Most of the open vascular surgeries belong to the category of high-risk procedures.

ACC/AHA 2014 Perioperative Guidelines use algorithmic approach for evaluation and care for noncardiac surgery. The stepwise approach takes into consideration urgency of surgery (emergency cases are taken to the operating room without further cardiac evaluation), patient's preoperative functional status, presence of clinical risk factors and surgical risk stratification.

Active cardiac conditions are unstable coronary syndromes, decompensated heart failure, significant arrhythmias, and severe valvular disease.

Clinical risk factors are history of ischemic heart disease, history of compensated or prior heart failure, history of cerebrovascular disease, diabetes

and renal insufficiency. Four metabolic equivalents (MET) equal moderate intensity physical activity.

Open aortic aneurysm repairs and lower extremity revascularization procedures are considered high-risk surgical procedures with combined incidence of cardiac mortality and morbidity more than 5%. Carotid endarterectomy and endovascular aortic aneurysm repair are intermediate cardiac risk procedures with combined incidence of cardiac mortality and morbidity 1% to 5%.

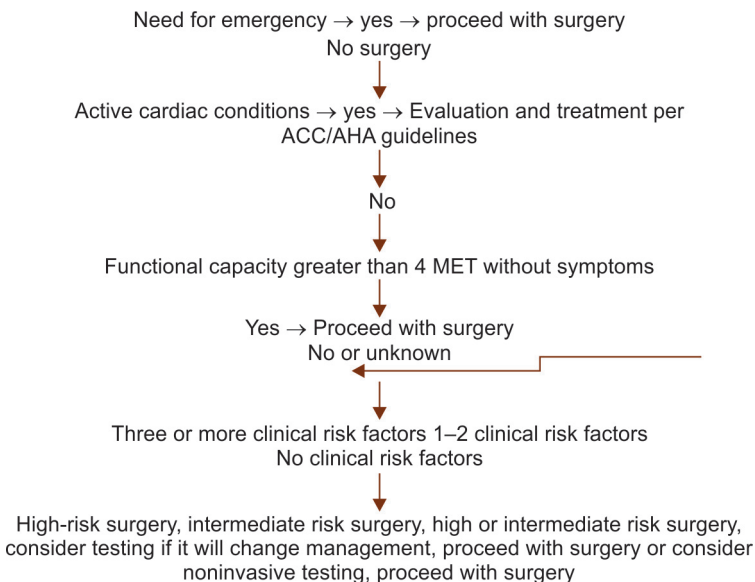
Preoperative Coronary Revascularization

Coronary Artery Revascularization Prophylaxis (CARP) Trial published in 2004 studied impact of prophylactic coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI) on long-term mortality of patients scheduled for vascular surgery. The study showed no benefit of prophylactic revascularization. However, authors excluded the patients with unstable coronary syndrome, severely reduced left ventricular function, left main coronary artery disease, and aortic stenosis. This methodological concern limits generalization of the results to high-risk patients.

Patients undergoing elective noncardiac procedures who are found to have prognostic high-risk coronary anatomy and in whom long-term outcome would likely be improved by coronary bypass grafting should generally undergo coronary revascularization before a noncardiac elective vascular surgical procedure or noncardiac operative procedures of intermediate or high risk (Flow chart 1).

Percutaneous coronary revascularization should not be routinely performed in patients who need noncardiac surgery unless clearly indicated for high-risk coronary anatomy, unstable angina, myocardial infarction, or hemodynamically or rhythmically unstable active coronary artery disease amenable to percutaneous intervention.

Flow chart 1: Cardiac evaluation algorithm for high- and intermediate-risk vascular surgery



PERIOPERATIVE MEDICAL THERAPY

Beta-blockers

2009 American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) Focused Update on Perioperative Beta Blockade revised the existing guidelines published in 2007. Based on this update, beta-blockers should be continued in patients undergoing surgery who are receiving beta-blockers for treatment of conditions with ACCF/AHA indications (Class I). Beta blockers titrated to heart rate and blood pressure are recommended for vascular surgery patients with high cardiac risk due to coronary artery disease or due to presence of more than one clinical risk factor (Class IIa) (Table 1). Although initial studies showed cardiac benefit with the use of perioperative beta-blockers, more recent studies question the benefit of perioperative beta blockade, especially in patients at moderate- to low-risk of cardiac events. Metoprolol after Vascular Surgery study published in 2006 showed no difference in cardiac events in patients receiving perioperative beta-blockers versus those receiving placebo (10.2% vs. 12%, $P = 0.57$). The rate of intraoperative hypotension requiring treatment (46.3% vs. 33.6%, $P < .001$) and bradycardia requiring treatment (21.5% vs. 7.6%, $P < .001$) was significantly higher in metoprolol group.

Table 1: Classification of recommendations

Class I
Benefit >>> Risk: Procedure/treatment should be performed/administered
Class IIa
Benefit >> Risk: It is reasonable to perform procedure/administer treatment
Class IIb
Benefit \geq Risk: Procedure/treatment may be considered
Class III
Risk \geq Benefit: Procedure/treatment should not be performed/administered

In the perioperative ischemic evaluation (POISE) study published in 2008, fewer patients in the metoprolol group than in the placebo group reached the primary endpoint—a composite of cardiovascular death, non-fatal myocardial infarction, and non-fatal cardiac arrest (5.8% vs. 6.9%, hazard ratio (HR) 0.84, 95% CI 0.70–0.99; $p = 0.0399$). However, there were more deaths in metoprolol group than in the placebo group (3.1% vs. 2.3% patients, HR 1.33, 95% CI 1.03–1.74; $p = 0.0317$) and more patients in the metoprolol group than in the placebo group had a stroke (1% vs. 0.5%, HR 2.17, 95% CI 1.26–3.74; $p = 0.0053$).

Based on recently published data, ACCF/AHA guidelines restrict Class I recommendations to those patients already on beta-blockers.

Beta-blockers have potential beneficial effects outside the prevention of cardiac events. In addition to reducing anesthetic and analgesic requirements during the perioperative period, beta-blockers have neuroprotective effects in patients with brain trauma and possible effectiveness in the management of intraoperative awareness-induced post-traumatic stress disorder.

Statins

Chronic therapy with statins should continue through perioperative period. Statins are very effective at reducing the incidence of myocardial infarction, stroke and other manifestations of vascular disease. The adverse event rates are very uncommon and the benefit risk ratio is extremely high. The effects of statins extend far beyond their effects on cholesterol levels: pleiotropic effects include vasoprotective mechanisms, comprising improved endothelial function, increased bioavailability of nitric oxide, immunomodulatory and antiinflammatory properties, stabilization of atherosclerotic plaques, as well as antioxidant and stem cell regulating capacities.

Angiotensin Converting Enzyme Inhibitors (ACEI) and Angiotensin Receptor Blocking (ARB) Agents

Risk of hypotension requiring vasopressors during anesthesia makes it controversial whether to stop or continue ACEI/ARA until the day of surgery. Rosenman et al. published in 2008 random-effects meta-analysis from 5 studies totaling 434 patients which suggested that patients receiving an immediate preoperative ACEI/ARA dose were more likely (RR 1.50, 95% CI 1.15–1.96) to develop hypotension requiring vasopressors at or shortly after induction of anesthesia.

An observational cohort study of 883 consecutive patients undergoing elective open abdominal aortic aneurysm repair showed increased mortality associated with preoperative renin-angiotensin system (RAS) blockade. The estimated odds ratio for 30-day mortality associated with RAS blockade was 5.0 (CI 95% 1.4–27). Perhaps more complex review by ACC/AHA task force and practice guidelines would better assist physicians in clinical decisions regarding ACEI/ARA preoperative administration.

LOWER EXTREMITY REVASCULARIZATION

Vascular surgery patients typically suffer from multiple organ systems comorbidities.

Incidence of severe coronary artery disease reaches 25% and there is an increased risk of myocardial infarction after vascular surgery. Complexity of the preoperative clinical condition combined with high-risk vascular surgery makes vascular surgery anesthesia a challenge even for an experienced anesthesiologist.

One of the unsolved controversies of anesthetic management is related to choice of regional (mostly neuraxial) and general anesthesia.

Almost all anesthetic agents in various combinations have been used successfully for these operations. Successful conduction of general anesthesia depends more on thorough preoperative evaluation and careful intraoperative management with appropriate monitoring than on anesthetic technique per se.

Regional anesthesia is usually more time consuming, has a finite failure rate, and there is a category of patients in whom lying still for several hours is

an unachievable goal (chronic pain, dementia, orthopnea). There is obvious need of general anesthesia when vein harvesting from patient's upper extremity is part of the planned procedure. Employment of regional anesthesia avoids the periods of hemodynamic instability during induction, and emergence from general anesthesia.

General anesthesia does not attenuate the stress response as well as regional anesthesia. Attenuated sympathetic nervous system activation in patients receiving epidural anesthesia/neuraxial opiate analgesia is associated with a reduced frequency of hypertension in the early postoperative period. In addition, increased plasma catecholamine values are associated with postoperative vascular graft failure and suppression of fibrinolytic activity.

American Society of Regional Anesthesia and Pain Medicine Practice guidelines (Third Edition) published in 2010 summarize evidence-based recommendations for neuraxial blocks in patients receiving drugs inhibiting platelet aggregation, blocking coagulation factors or inhibiting fibrinolysis. Neuraxial techniques, particularly epidural anesthesia and continued epidural analgesia attenuate hypercoagulable response and reduce the frequency of thromboembolism after major vascular surgery. However, the effect of neuraxial techniques is insufficient as the sole method of thromboprophylaxis and routine DVT prophylaxis is now standard part of perioperative care. While earlier studies showed a decreased rate of cardiovascular complications in patients receiving neuraxial block, more recent studies revealed no significant difference in cardiac outcomes of patients receiving either neuraxial block or general anesthesia for peripheral vascular surgery.

A systematic review of four studies that compared neuraxial anesthesia with general anesthesia for lower limb revascularization surgery showed no evidence of differences in the postoperative risks of death, myocardial infarction or leg amputation between the two types of anesthetic. The risk of pneumonia was less after neuraxial anesthesia in one of the studies which reported this outcome. Femoral nerve block can also be given for unilateral lower limb vascular surgeries.

Monitoring

Monitoring during anesthesia for peripheral vascular surgery is largely affected by choice of open or endovascular technique. Open vascular technique in addition to standard ASA (American Society of Anesthesiology) monitors frequently requires arterial line which also facilitates blood sampling for blood gas, electrolytes, glucose, and hematocrit in patients with multiple clinical risk factors and significant blood loss. There is a general trend in hemodynamic monitoring towards less invasive techniques. When using general anesthesia with positive pressure ventilation, the fluid responsiveness monitoring methods (PiCCO, LiDCO, FloTrac/Vigileo, ECOM) are available for goal-directed fluid management. Respiratory variations in pulse pressure (PPV) have been considered the gold standard for fluid responsiveness monitoring.

CAROTID ARTERY REVASCULARIZATION

Stroke is the third most common cause of death and the leading neurologic cause of long-term disability. The majority of strokes are ischemic in nature. Up to 20% of ischemic strokes are result of carotid artery atherosclerotic disease and most ischemic events are caused by embolization of material of the atherosclerotic plaque. Carotid endarterectomy, (CEA) remains the gold standard for carotid revascularization, although more recently, carotid angioplasty and stenting (CAS) emerged as a minimally invasive alternative. Based on the recommendations of the American Heart Association, symptomatic (history of TIA or stroke) patients with 50% to 99% stenosis are best treated with CEA if the risk of perioperative stroke or death is less than 6%, with greatest benefit in those with severe stenosis 70% to 99%. For asymptomatic patients with 60% to 99% stenosis, CEA is recommended if the perioperative risk of stroke or death is less than 3% and if the patient has a life expectancy greater than 5 years.

Current data seem to support the use of CAS as an alternative to CEA in patients with anatomical risk factors, with contralateral occlusion, and severe, multiple medical comorbidities. CAS is best avoided in patients older than 80 years of age, those with complex vascular anatomy and specific unfavorable lesion characteristics, and possibly those with symptomatic disease.

Preanesthetic Evaluation

Carotid artery disease is a local manifestation of systemic disease. Coronary artery disease, hypertension, diabetes mellitus and renal insufficiency are frequent comorbidities of patients with carotid vascular disease. Ouriel and colleagues compared outcomes of high-risk patients versus low-risk patients undergoing CEA. They found a significant increase in the perioperative risk of stroke, death, and myocardial infarction in high-risk patients (with severe coronary artery disease, chronic obstructive pulmonary disease, or renal insufficiency).

The ACC/AHA 2007 guidelines consider CEA an intermediate-risk procedure.

Risk of perioperative stroke is most strongly associated with an active neurologic process prior to surgery. Approximately 25% of strokes associated with CEA occur intraoperatively.

Preoperative hypertension should not delay elective surgery unless the blood pressure >180/100 mm Hg. Perioperative glycemic control and elimination glucose-containing intravenous infusions are strongly recommended.

Choice of Anesthesia for CEA

Regional or general anesthesia can be used for carotid endarterectomy. Recently the third option, patient cooperating during general anesthesia, was introduced. During carotid clamping, general anesthesia is reduced so that

patient is able to respond to verbal statements and neurological monitoring can be performed.

General anesthesia usually implies balanced technique. Sevoflurane and desflurane have shortest extubation times. The use of nitrous oxide given its potential to increase the size of air emboli is controversial. Propofol and narcotics provide good hemodynamic stability. Almost all commonly used anesthetics reduce cerebral oxygen requirements. Patients are kept normothermic and normocarbic. Blood pressure is maintained at patient's "normal" to high values in most situations. Besides standard ASA monitoring, invasive arterial pressure is monitored.

The routine use of shunting in patients under general anesthesia largely depends on surgeon's preference. Recent randomized trial compared selective shunting (SS) versus routine shunting (RS). Selective shunting was used only if systolic stump pressure was <40 mm Hg. Study showed no significant difference in combined perioperative transient ischemic attack and stroke rates (2% in RS vs 2.9% in SS, $p>0.99$). CNS monitoring is prudent when surgeons use shunts selectively. The 16-channel EEG is considered a gold standard. Other monitoring options include EEG, somatosensory evoked potentials (SSEP), measurement of stump pressure, cerebral oximetry, near-infrared spectroscopy, and transcranial Doppler.

No difference in stroke rate has been demonstrated with the use of particular monitoring technique.

Regional anesthesia for CEA can be achieved but not very popular. Superficial cervical plexus block, deep cervical plexus block, epidural anesthesia, or straight local anesthesia (frequently performed by surgeon) have been tried for this purpose. Serious potential complications of the uncommonly used epidural anesthesia include dural puncture, epidural venipuncture and respiratory muscle paralysis. Hypotension and bradycardia are the most frequent side-effects of this technique.

Cervical plexus block (deep, superficial, or their combination) is a technique performed by anesthesiologist. The most common complication of cervical plexus block is systemic local anesthetic toxicity, caused by either intravascular injection or vascular absorption in this highly vascularized region.

Awake patient monitoring during local or regional anesthesia seems the most reliable method of predicting the need for a shunt after carotid clamping and can be regarded as the accepted standard for the evaluation of patient's intraoperative neurologic status. Aleksic et al. did not find significantly different outcome between the group of high-risk patients (ASA 4, "hostile neck", recurrent ICA stenosis, contralateral ICA occlusion, and age ≥ 80 years) and the group of low-risk patients who underwent CEA under local anesthesia. It does not appear justified to refer high-risk patients principally to carotid angioplasty and stenting when local anesthesia can be chosen to perform CEA.

Meta-analysis of the randomized studies published in 2009 showed that there was no evidence of a reduction in the odds of operative stroke or death with regional anesthesia (odds ratio (OR) 0.85, 95% confidence interval (CI) 0.63 to 1.16). There was a trend towards lower operative mortality with regional anesthesia (OR 0.62, 95% CI 0.36 to 1.07).

Carotid artery stenting requires minimal to moderate sedation and standard monitors.

Dexmedetomidine seems to be an alternative to more traditional sedation with benzodiazepines and opioids.

Postoperative Considerations

Hemodynamic instability is common after CEA or CAS. Hypertension particularly in patients with preoperative poorly controlled hypertension is more frequent than hypotension. Hypertension may worsen cardiac outcome by myocardial infarction, and neurologic outcome by triggering the hyperperfusion syndrome (may lead to intracerebral hemorrhage). Increased sensitivity of the baroreceptors after atherosclerotic plaque removal may lead to postoperative hypotension and bradycardia, which when properly treated, are usually not associated with serious cardiac adverse outcomes. Some institutions keep post CEA/CAS patients longer in the recovery room so that airway obstruction caused by hematoma can be promptly diagnosed and managed. Stroke during the postoperative period is mostly embolic in origin.

AORTIC RECONSTRUCTION

Thoracoabdominal aortic aneurysms are one of the most challenging surgeries for the anesthetists. They account for 10% of aneurysms of the aorta. A thorough understanding of pathophysiology, anatomy, and surgical interventions including extracorporeal circulation are essential to achieve a good outcome.

Crawford classified them according to their extent and location in four types. Patients with Crawford type II aneurysms are at greatest risk for paraplegia and renal failure from ischemia to the spinal cord and kidneys during cross-clamp. Neurologic and renal complications are significant for the most extensive forms of aneurysms. Mortality has improved over time as a consequence of either increased surgical experience, the adoption of a protocolized strategy for repair, or secular improvements in anesthetic and intensive care treatment. Long-term survival after elective TAAA repair is good (Table 2 and Fig. 1).

A true aortic aneurysm is a dilation of the entire aorta, as measured across from the adventitia to the adventitia, and it is associated with degenerative changes of the aortic wall where the original histological constituents can still be recognized. Only one-half of TAAA are atherosclerotic in origin. The remainder cases are caused either by trauma or by connective tissue diseases involving the aortic wall from conditions such as Marfan syndrome, cystic medial degeneration, Takayasu arteritis, or syphilitic aortitis. Patients with aneurysmal disease have a poor prognosis without surgery. Nutritional blood flow to the aorta is compromised, and the increasing diameter is associated with increased wall tension (LaPlace's law), even when arterial pressure is constant. In view of the often lethal consequence of ruptured aneurysms, for men age 65 to 75 years, an invitation to attend screening reduces aneurysms-

related mortality. Crawford classified TAAA in type I, II, III and IV according to the extent of aneurysm.

Table 2: Crawford's classification for thoracoabdominal aortic aneurysm

Group	Location and extent of aneurysm	Description
Type I	Involves the descending thoracic aorta from the left subclavian artery to the celiac artery, just below the diaphragm	Mostly thoracic aorta
Type II	Includes aneurysms involving most of the descending thoracic and most of the abdominal aorta, from the left subclavian artery to the bifurcation of the aorta	Most of thoracic and abdominal aorta
Type III	Involves the lower portion of the thoracic aorta and most of the abdominal aorta	Lower thoracic and abdominal aorta
Type IV	Most of the abdominal aorta up to and including the celiac artery just above the diaphragm	Mostly abdominal aorta

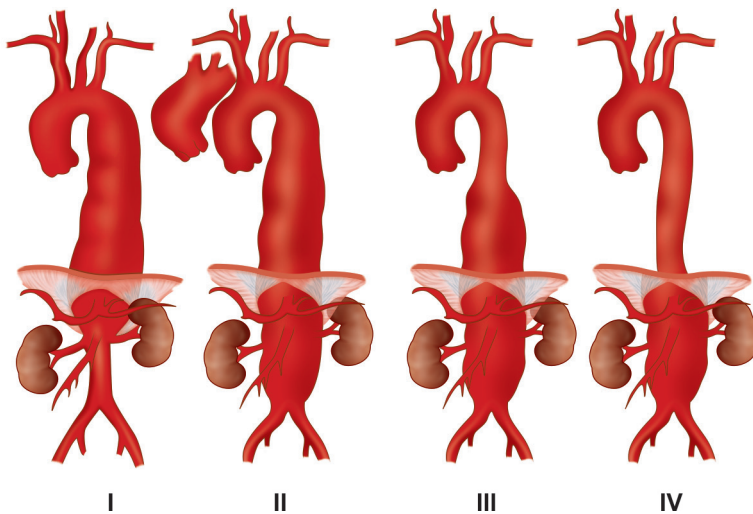


Fig. 1: Crawford's classification for TAAA

Patients with Crawford type II aneurysms are at greatest risk for paraplegia and renal failure from ischemia to the spinal cord and kidneys during cross-clamp. Even with extracorporeal circulatory support, there is an obligatory period of time when blood flow to these organs is interrupted. For this reason, protective measures to prevent ischemic injury are important in reducing morbidity.

Preoperative Comorbidities and Evaluation

Patients with thoracoabdominal aortic aneurysms often have multiple comorbidities which affect not only life expectancy but also perioperative

outcome. Because of the importance of preoperative cardiac, pulmonary and renal status on morbidity and mortality, the importance of a thorough preoperative assessment of these systems should be emphasized. In addition, the preoperative nutritional status is important and needs to be carefully evaluated and, if necessary, improved. Low preoperative serum albumin level has been associated with postoperative negative outcomes in surgical patients. For this reason, it is our practice to evaluate in all patients, albumin and total serum proteins. In those with low albumin or with clinical evidence of malnutrition, our practice is to supplement their diet with a high protein/calorie beverage (e.g. ensure 2 cans per day) during the month before surgery.

Airway and Chest

The chest X-ray should be available in the operating room and should be examined preoperatively for evidence of aneurysm compression of the left mainstem bronchus.

Preoperative pulmonary assessments provide a baseline measurement to aid in risk stratification, and to identify patients whose preoperative respiratory status may be improved. Poor pulmonary function is associated with prolonged respiratory ventilation and increased mortality. All patients should have spirometry, arterial blood gas and analysis study of gas diffusion. The lower limit for FEV1 below which significant activity restriction and carbon dioxide retention occurs is around 0.6 to 0.7 L. In patients with carbon dioxide retention, the five year survival is less than 10%. In patients undergoing abdominal aneurysm surgery, the transient postoperative decrease in FEV1 is at least 0.6 L. Because of the extensive involvement of the abdomen and thorax, the division of the diaphragm and other physiological derangements in TAAA surgery, the decrease in FEV1 will be greater. Svenson suggests an FEV1 of below 1.2 L, a carbon dioxide level of greater than 45 mm Hg, an FEV 25% of less than 0.5 L/sec, and a PaO₂ of less than 55 mm Hg as preoperative indicators of high-risk for postoperative respiratory complications.

Blood Loss and Blood Products Utilization

Repair of TAAA is associated with major blood losses, often exceeding the patient's intravascular volume, and complex intraoperative and postoperative coagulopathies necessitating large-volume transfusion of blood products. Abnormalities sufficient to cause thrombocytopenia or clinically important prolongation of clotting parameters are rarely present before surgery in elective aneurysms but are more common with ruptured aneurysms. The coagulation abnormality identified before surgery is that of higher PTT values, suggesting a disturbance of the extrinsic coagulation pathway.

The finding of intraoperative and postoperative deficiencies of clotting factors, along with thrombin generation and activation of the thrombolytic system, is reflective of massive blood losses, visceral ischemia, and massive transfusions. An aggressive strategy of transfusion of blood products is critical to the prevention of clinically significant coagulopathy during surgery. Adjuncts

to reduce blood losses and blood product use include low-dose aprotinin, tranexamic or epsilon-aminocaproic acid, intraoperative blood salvaging, and acute normovolemic hemodilution. In TAAA repair, an average blood loss of 5000 to 6000 mL and average transfusion of allogeneic blood products of 50 to 60 units are to be anticipated. Blood and clotting factors should be available. We recommend an initial cross-match 12–16 units of packed red blood cells, 10 units of fresh frozen plasma and 10 units platelets.

Renal Function

A preoperative serum creatinine concentration of greater than 200 mmol/L has been associated with a 42% incidence of postoperative renal failure defined by creatinine greater than 265 mmol/L or the need for dialysis, following TAAA repair. Renal failure is also associated with a higher incidence of respiratory failure and postoperative paraplegia.

Acetylcysteine used as an adjunct during TAAA was associated with a trend to improved renal function.

Peripheral Vascular Disease

Manifestations of peripheral vascular disease need to be identified. If there is evidence of carotid artery stenosis, surgical correction may be indicated prior to surgery. In addition attention to femoral and radial pulses is important since multiple arterial lines for upper and lower body systemic blood pressure are often necessary for monitoring during surgery. In addition if an atrio-femoral bypass is used, disease of the aorto-iliac-femoral system may require special surgical considerations that need to be carefully discussed with the surgeon.

Intraoperative Management

In order to reduce complications and mortality, multiple surgical and anesthetic techniques such as different adjuncts for distal aortic perfusion (DAP), cerebrospinal fluid drainage, and regional cooling of the spinal cord and kidneys have been developed. These techniques are controversial and not universally accepted because complications such as renal failure and paraplegia still occur.

A care plan should be drawn for the intraoperative phase.

Monitoring and Infusion Lines

If partial bypass is not used, the primary focus should be on massive blood loss and the ability to rapidly transfuse. It is preferred to place at least three large bore intravenous lines for transfusion.

The right jugular vein is avoided to prevent the possibility of pneumothorax in a patient who will be on the right lateral decubitus and single right lung ventilation. A Swan-Ganz catheter is also routinely inserted. In cases when partial bypass is used, the primary focus is often the coordination of upper

body blood flow and lower body blood flow. The perfusionists usually maintain a mean distal arterial pressure of 60 to 70 mm Hg. Since the left atrium is supplying the left ventricle (for upper body perfusion) and the bypass circuit (for lower body perfusion), it is apparent that if the perfusionists increase the pump flow enough, it will eventually compromise left ventricular filling, which immediately compromises proximal pressure and perfusion (e.g. brain and heart). The use of electromagnetic pump which is volume-controlled (as opposed to a rotary pump) minimizes the risks of this complication.

Maintenance of adequate intravascular volume and communication between anesthetists and the perfusionists are essential. We prefer to use three arterial lines, one in each radial (the left radial may be lost if the clamp is placed proximal to the left subclavian artery), and one in the right femoral artery to monitor proximal and distal pressures. The use of transesophageal echocardiography is helpful. Chronic diastolic dysfunction (DD) is a well-recognized problem in hypertensive patients and in patients with high afterload presenting for TAAA. The relationship between left ventricular diastolic relaxation and systolic function may be uncoupled. As a result, many of the traditional monitors of cardiac systolic function may not reflect the extent of diastolic dysfunction. Transesophageal echocardiography (TEE) is necessary to measure the alterations in intracardiac flows and velocities to assess the extent of DD. Filling of the left ventricle occurs in diastole in two different phases.

The first phase, known as E-wave, starts with mitral valve opening, and blood flow occurs during the relaxation of the LV. The second, known as A-wave, occurs at the end of diastole as a consequence of atrial contraction. The majority of filling of the LV occurs during the E-wave (>85%).

Echocardiographically, with normal diastolic function, the velocity of blood during the E-wave is, therefore, greater than during the A-wave. In conditions of diastolic dysfunction, atrial contraction contributes relatively more to ventricular filling, and the blood velocity during A-wave becomes greater than in the E-wave causing an inversion of the E : A ratio. In TAAA repair, there is a sudden increase in afterload during aortic clamping. The reversal of E : A ratio identified by TEE during aortic clamping in TAAA repair was reported. Other uses of TEE in TAAA repair include visualizing the position of left atrial (LA) cannula when left atrio-femoral bypass (LAFB) is used and to monitor the LV function and volume. Beta-blockers may be protective of intraoperative acute diastolic dysfunction.

Airway Management

Patients operated on an emergency basis will be considered to have a full stomach. In these situations, the benefits of a rapid sequence (airway protection) must be balanced against the patient's hemodynamic status. We often use bronchial blockers for one lung anesthesia. They are easier to place than double lumen tubes. However, it takes a longer time for the lung to deflate and they do not allow for separate lung ventilation. A disadvantage of double lumen tubes is that at the end of the case, they need to be changed to a single

lumen endotracheal tube at a time when airway and facial edema are present, or they must be left in place until the edema is resolved with consequence caused by pressure due to their stiffness and increased airway resistance. The position of the bronchial blocker should be checked (and rechecked in the lateral position) with the fiberoptic bronchoscope.

The Pre-clamp Period

Blood pressure should be well controlled to prevent rupture, to allow easier management of the post-clamping hypertension, and to reduce blood losses. Before the aortic cross-clamp is applied, mannitol at approximately 0.5–1.0 g/kg intravenous is usually administered because of its diuretic and free oxygen radicals scavenger properties, which may be beneficial for renal and spinal cord protection. If partial bypass is used (atrial or pulmonary vein to femoral artery), there is usually partial heparinization (100 IU/kg). If full bypass is used (femorofemoral with an oxygenator), full heparinization is used to maintain an activated clotting time of 400 to 450 seconds. The senior author described the use of acute normovolemic hemodilution with partial exchange transfusion (ANHPET) in surgery of TAAA. During TAAA repair, ANHPET was used to withdraw of up 3 L of blood. This was returned to the patient at the end of the reconstruction. Albumin 5% and stored packed red blood cells (PRBCs) were used for replacement. ANHPET reduced blood product transfusion, improved postoperative hemostatic parameters and simplified the management of cross-clamping hypertension. This appears to be a useful adjunct in the anesthetic management of TAAA patients.

Preparation for Aortic Cross-clamping without Bypass

Nitroprusside, labetalol, esmolol and/or intravenous nitroglycerine should be available for immediate infusion as needed to control blood pressure. However, arterial blood pressure should be maintained at a mean of 70 to 90 mm Hg after the clamp is applied. This is because perfusion distal to the clamp (spinal cord, kidney, etc.) is severely diminished and the use of vasodilators further exacerbates low end-organ perfusion. The needs of the heart (which would prefer a lower afterload) need to be balanced against those of the poorly perfused end-organs. Once the aneurysm is opened, there may be substantial blood loss. A cell-saver and rapid infusion systems should be used with large bore intravascular access for rapid transfusions.

Platelets and fresh frozen should be given to avoid complex forms of dilution and consumption coagulopathies. Calcium chloride should also be given, either guided by ionized calcium levels or one gram for every three units of packed cells.

Preparation for Removal of the Aortic Cross-clamp without Bypass

Additional platelets and fresh frozen, and cryoprecipitate are often given just before aortic unclamping to prevent bleeding secondary to coagulopathy and

to provide increased preload. If a continuous infusion of bicarbonate was not administered during clamping (usually 0.005 mmol/kg/minute), 100 mmol of bicarbonate is slowly administered at this point to balance reperfusion acidosis. One additional gram of calcium may be also given at this point. An infusion of low dose (0.005–0.01 µg/kg/min) norepinephrine is often started approximately 5–10 minutes before the aortic cross-clamp is removed. At this point, it is often advisable to have another anesthetist to help in this critical phase. All blood products should be in the room and checked. Normally with cross-clamp removal, there is reactive hyperemia, vasodilatation, and decreased systemic vascular resistances and preload. The surgeon should remove the cross-clamp slowly to prevent profound hypotension and the clamp may be reapplied, if necessary until the volume status and hemodynamic drugs are optimized.

THORACOABDOMINAL AORTIC ANEURYSM (TAAA)

Table 3: Summary of anesthesia care

Intraoperative Care
<ul style="list-style-type: none"> • One lung anesthesia <ul style="list-style-type: none"> – Double lumen tube or endobronchial blocker – Confirmation of position with patient supine and in lateral decubitus.
<ul style="list-style-type: none"> • Transfusion therapy – ANH <ul style="list-style-type: none"> – Anticipatory treatment of coagulation disorders (FFP, cryoprecipitate, inhibitors of fibrinolysis)
<ul style="list-style-type: none"> • Antimicrobial therapy <ul style="list-style-type: none"> – Cefazolin 2 g before induction of anesthesia and 1 g after loss of 1 blood volume
<ul style="list-style-type: none"> • Prevention of renal dysfunction <ul style="list-style-type: none"> – Cold perfusion (blood or crystalloid) – Target parenchymal temperature <20°C – Low dose dopamine, frusemide, mannitol – N-acetylcysteine.
<ul style="list-style-type: none"> • Prevention of paraplegia <ul style="list-style-type: none"> – CSF drainage – Target pressure <10 cm H₂O – Reattachment of intercostal arteries from T₇–L₁

Preparation for Aortic Cross-clamping with Bypass

Partial bypass is initiated before cross-clamping: minimal effect on blood pressure is seen with cross-clamping. The proximal cross-clamp is then applied slowly, and distal flows are titrated to maintain adequate proximal and distal pressures. If proximal pressure falls, it is usually because there is inadequate volume to support the flow rates that the perfusionists are trying to achieve. Flow rates must temporarily be decreased, and slowly reestablished. Typically, distal pressures can be about 50 to 60 mm Hg, while proximal pressures should be equal or slightly below preclamp pressures. We use a modified circuit, which consists of a primary circuit with a centrifugal pump and heat exchanger to perfuse and warm the systemic circulation and a parallel secondary circuit

with a roller pump and a second heat exchanger to perfuse the viscera with cold blood. A progressive sequential cross-clamping technique is used. This technique offers theoretical hemodynamic and metabolic advantages and may prove to be useful in preventing ischemic and reperfusion injury to the spinal cord and kidneys.

Preparation for Removal of the Aortic Cross-clamp with Bypass

With perfusion to the lower body with partial or full bypass, acidosis and hypovolemia are often less troublesome compared with clamp-and-go technique. The perfusionists gradually reduce the flow rates allowing proximal pressure to rise and the clamp is gradually released. Volume or pressors may be needed initially, so blood should be in the room and checked (usually the hematocrit is kept at 25–30% throughout the case and have at least 2 units of PRBC pressurized and ready to infuse at cross-clamp removal). However, as the surgeons are assessing their repair, it is important to avoid hypertension.

SPECIAL CONSIDERATIONS

Renal Protection

Lasix, mannitol or dopamine may be used in an attempt to preserve renal function. Any of these adjuncts are able to augment flow through the renal tubules, however, there is little scientific basis on which to choose one or a combination of them. We know, however, that hypovolemia or decreased cardiac output increases the likelihood of renal failure. Patients at increased risk include those with pre-existing renal compromise, older patients, longer cross-clamp times (greater than 30 minutes), and failure to use atrial-femoral bypass. Acute renal failure occurs in 3–14% with a mortality rate approaching 40%. Many renal protective agents have been used, including acetylcysteine with some encouraging results.

Spinal Cord Protection

Paraplegia may result from prolonged ischemia (long cross-clamp times, particularly those above 30–45 minutes) or the occlusion of the artery of Adamkiewicz. Aortic occlusion increases cerebrospinal fluid pressure (CSFP), which increases after aortic cross-clamp (likely caused by increased venous congestion of dural veins) and decreases distal aortic systolic pressure, thereby, decreasing perfusion of the spinal cord. Theoretically, decreasing CSFP by cerebrospinal fluid drainage (CSFD) should improve spinal cord blood flow and decrease the risk of spinal cord ischemic injury. One approach is to place a lumbar epidural catheter into the subarachnoid space to drain CSF. This is done before the procedure begins. The hope is that draining CSF will lower the CSF pressure and allow more blood flow, since spinal cord perfusion pressure (SCPP) is equal to mean arterial pressure (MAP) minus cerebral spinal fluid pressure (CSFP). Dropping the CSF pressure to 10 cm H₂O might double

blood flow by doubling the gradient of pressure. If catheters are placed, they should not be removed until it has been documented that coagulation status has returned to normal.

Usually 20–40 cc is drained before cross-clamping, then either they can passively drain, or about 10 cc/hr of CSF can be removed, throughout the case. Evidence from randomized and nonrandomized trials and from cohort studies support the use of CSF drainage as an adjunct to prevent paraplegia when this adjunct is used in centers with large experience in the management of TAAA. Some centers use electrophysiologic monitoring with somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) to monitor for spinal cord ischemia. However, there are some problems associated with SSEP monitoring.

These monitoring techniques may be helpful in identifying the important intercostals arteries that perfuse the spinal cord so they can be implanted into the graft. If ischemia is identified, cross-clamps can often be moved, upper or lower body blood pressure can be increased to provide perfusion through collaterals. Other measures may include CSF drainage, induced hypothermia, or intrathecal papaverine.

Anesthesia Summary of TAAA

- ❑ Higher risk, ASA III-IV elderly patients
- ❑ One lung ventilation (OLV)
- ❑ Profound hemodynamic changes caused by clamping and unclamping of the thoracic aorta
- ❑ Massive blood losses
- ❑ Coagulopathy
- ❑ Severe metabolic derangement
- ❑ Vital organs ischemia or damage, e.g. renal and spinal cord
- ❑ Prolonged postoperative course
- ❑ Expensive operation.

Intraoperative Phase

- ❑ One lung anesthesia
 - Double lumen tube or endobronchial blocker
 - Confirmation of position with patient supine and in lateral decubitus
- ❑ Transfusion therapy—ANH
 - Anticipatory treatment of coagulation disorders (FFP, cryoprecipitate, inhibitors of fibrinolysis)
- ❑ Antimicrobial therapy
 - Cefazolin 2 g before induction of anesthesia and 1 g after loss of 1 blood volume
- ❑ Prevention of renal dysfunction
 - Cold perfusion (blood or crystalloid)
 - Target parenchymal temperature <20°C
 - Low dose dopamine, furosemide, mannitol
 - N-acetylcysteine

- ❑ Prevention of paraplegia
 - CSF drainage
 - Target pressure <10 cm H₂O
 - Reattachment of intercostals arteries from T₇-L₁

Postoperative Phase

- ❑ PEEP of 10 mm Hg in the first 12 hours
- ❑ Maintain normo- or moderate hypervolemia
- ❑ Aggressive treatment of coagulation disorders
- ❑ CSF drainage for 48–72 h
- ❑ Close surveillance of infections
- ❑ Liberal use of broad spectrum or multiple antibiotics
- ❑ Early use of AVF or dialysis
- ❑ Early use of TPN.

Late Operative and Postoperative Considerations

By the end of the procedure, patients could be hypovolemic, edematous, very cold with marginal lung function. So very often patients are ventilated overnight while kept in a 30 degree head up position so that head edema can resolve before extubation or change in the tracheal tube

ENDOVASCULAR SURGERIES

Endovascular surgery encompasses a large number of different cases, from endovascular aneurysm repair (EVAR), to carotid stenting, to coiling of intracerebral aneurysms. For the purposes of this abstract, anesthesia for EVAR will be considered.

Preoperative Preparation

Patients selected for EVAR are likely to be older and have more comorbidity than patients selected for open repair. This is because EVAR is viewed as a “minimally invasive” option, causing less physiological insult, and has been deemed suitable for nonagenarians. Also, vascular surgeons are more likely to offer open repair to younger patients in order to avoid years of annual angiographic follow-up and reduce the need for reintervention. As a result, the patient for EVAR may be a greater anesthetic challenge, even without trespass into the abdomen and aortic cross-clamping.

General vs Regional Anesthesia for EVAR

General anesthesia is frequently more practical than regional anesthesia for the following reasons:

- ❑ Considerations for anticoagulation are reduced. These patients are frequently on antiplatelet agents preoperatively and will certainly require

heparin intraoperatively. This is usually asked for within 15 minutes of anesthetic induction.

- ❑ Blood pressure control is easier and can be achieved by titration of anesthetic agents and vasopressors in the majority of cases. Should aneurysm rupture occur, the patient's airway is already secure and transport to theater is less complicated.
- ❑ Breath-holding on the ventilator is easy, exact and can be prolonged, if necessary.
- ❑ Complex fenestrated grafts may take lengthy periods of time, which may be tolerated poorly by some patients. The possibilities for regional anesthesia extend across the spectrum of infiltrated local anesthetic to neuraxial blockade. The potential benefits of local/regional anesthesia include the reduced need for hemodynamic support, reduced numbers of ICU admissions and reduced length of hospital stay.

Intraoperative Monitoring

Distinct from open surgery, blood loss during EVAR may be occult. Further, bleeding may occur from numerous sites, most commonly from the aneurysm itself, but also from the stent edge at deployment and at the groin access sites.

Therefore, continuous arterial pressure monitoring is vital. Any unexplained decrease in blood pressure should prompt an inspection of the groin and a top to bottom angiographic run to look for a potential site of bleeding.

Other Considerations

Renal Protection

These patients may have significant background renal disease and are exposed to large volumes of intravenous contrast. The mainstay of renal protection is administration of intravenous fluid and maintenance of circulating volume. Consideration should also be given to bicarbonate supplementation and the use of N-acetyl cysteine.

Blood Pressure Control

Unless aortic occlusion balloons are used (usually in ruptured aneurysms), hemodynamic instability is minimal. Persistent hypertension is probably best managed with a beta-blocker such as metoprolol or labetalol. Immediate control of hypertension is easily managed with nitrates and/or short-acting beta-blockers. Hypotension is common after induction of anesthesia, as the magnitude of surgical stimulus is small. Infusion of low-dose vasopressors, such as phenylephrine, is often required.

Emergency Situations

This may occur in two scenarios. Firstly, the patient with a ruptured aneurysm may be emergently stented in the radiology department. This is appropriate

for relatively stable patients with ruptured abdominal aneurysms, and often the modality of choice in patients with thoracic aortic dissection and traumatic transection of the aorta. Secondly, hemorrhage may result from aneurysm rupture, which may occur during wiring or stent deployment, or endo-leak after stent deployment.

Significant hemorrhage may occur from groin vessels. In this setting, the patient needs to be moved rapidly to an operating theater (as most of us are not blessed with hybrid operating theaters), with adequate light and a full range of vascular surgery instruments.

Logistically, plans need to be in place for such an eventuality. Several recommendations can be made:

- ❑ The vascular theater team (surgeons, anesthesiologists and nurses) is used to perform the EVAR with or without radiological assistance.
- ❑ Keep one theater empty—possibly the theater that would usually have been occupied by the vascular surgeons anyway. This, with the above point, ensures that the entire team can rapidly translocate to a theater and deal with the rupture.
- ❑ The patient trolley/bed needs to remain outside the radiology suite with oxygen, a self-inflating bag, appropriate monitoring and emergency drugs.
- ❑ There must be an identified system of rapid transport to theater.

CONCLUSION

The aim of this chapter is to summarize information related to anesthesia for vascular surgery in a simplified manner.

Optimization and standardization of perioperative care may result in improvement of outcomes of the vascular surgery. Therefore, guidelines prepared by multidisciplinary team of professionals and employing “protocolized medicine” may help to achieve this goal.

SUGGESTED READING

1. Aburahma AF, Stone PA, Hass SM, et al. Prospective randomized trial of routine versus selective shunting in carotid endarterectomy based on stump pressure. *J Vasc Surg.* 2010;51(5):1133-8.
2. Aleksic M, Luebke T, Brunkwall J. Outcome of carotid endarterectomy under local anesthesia with respect to the patients' risk profile. *Vasa.* 2009;38(3):225-3.
3. Barbosa FT, Cavalcante JC, Juca MJ, Castro AA. Neuraxial anaesthesia for lower-limb revascularization. *Cochrane Database of Systematic Reviews* 2010, Issue 1. Art. No.: CD007083.
4. Bevilacqua S, Romagnoli S, Ciappi F, et al. Anesthesia for carotid endarterectomy: The third option. Patient cooperating during general anesthesia. *Anesth Analg.* 2009;108(9):1929-36
5. Bode RH, Lewis KP Jr, Zarich SW, et al. Cardiac outcome after peripheral vascular surgery. Comparison of general and regional anesthesia. *Anesthesiology.* 1996; 84:3-13.
6. Bonnet F, Derosier JP, Pluskwa F, et al. Cervical epidural anesthesia for carotid artery surgery. *Can J Anaesth.* 1990; 37(3): 353-8.

7. Brennan JM, et al. Radial artery pulse pressure variation correlates with brachial artery peak velocity variation in ventilated subjects when measured by internal medicine residents using hand-carried ultrasound devices. *Chest*. 2007;131(5):1301-07.
8. Brown WV. Safety of statins. *Curr Opin Lipidol*. 2008;19(6):558-62.
9. Chopra V, Plaisance B, Civosoglu E, Flanders SA, Eagle KA. Perioperative beta-blockers for major noncardiac surgery: Primum non nocere. *Am J Med*. 2009;122,222-9.
10. Cristopherson R, Baettie C, Frank SM, et al. Perioperative morbidity in patients randomized to epidural or general anesthesia for lower extremity vascular surgery. Perioperative ischemia randomized anesthesia trial study group. *Anesthesiology*. 1993;79:422-34.
11. Devereaux PJ, Yang H, Yusuf S, et al. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): A randomized controlled trial. *Lancet*. 2008;371:1839-47
12. Fleischmann KE, Beckman JA, Buller CE, et al. 2009 ACCF/AHA Focused Update on Perioperative Beta Blockade. American College of Cardiology Foundation, American Heart Association Task Force on Practice Guidelines, American Society of Echocardiography, American Society of Nuclear cardiology, Heart Rhythm Society, Society of cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular medicine, Society for Vascular Surgery. *J Am Coll Cardiol*. 2009; 07.004.
13. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *J Am Coll Cardiol*. 2007;50:e159-241.
14. Hashem M, Cina CS. Anaesthesia for surgical repair of thoracoabdominal aortic aneurysms. *Acta Anaesth Belg*. 2007;58:45-54.
15. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in patient receiving antithrombotic or thrombolytic therapy. American society or regional anesthesia and pain medicine evidence-based guidelines (Third edition). *Reg Anesth Pain Med*. 2010;35:64-101.
16. Howell GM, Makaroun MS, Chaer RA. Current management of extracranial carotid occlusive disease. *J Am Coll Surg*. 2008;12:442-53.
17. Kadoi Y, Saito S. Possible indications of beta-blockers in the perioperative period other than prevention of cardiac ischemia. *J Anesth*. 2010;24(1):81-95.
18. Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100:1043-9.
19. McFalls EO, Ward HB, Moritz TE. et al. Coronary artery revascularization before elective major vascular surgery. *N Engl J Med*. 2004;351:2795.
20. Moore WS, Barnett HJ, Beebe HG, et al. Guidelines for carotid endarterectomy. *Circulation* 1995;91:566-79.
21. Moritz S, Kasprzak P, Arlt M, et al. Accuracy of Cerebral Monitoring in Detecting Cerebral Ischemia during Carotid Endarterectomy: A Comparison of Transcranial Doppler Sonography, Near-infrared Spectroscopy, Stump pressure and Somatosensory Evoked Potentials. *Anesthesiology*. 2007;107(4):563-9.
22. Ouriel K, Hertzner NR, Beven EG, et al. Preprocedural risk stratification: identifying an appropriate population for carotid stenting. *J Vasc Surg*. 2001;230:70-6.

23. Parker SD, Breslow MJ, Frank SM, et al. Catecholamine and cortisol responses to lower extremity revascularization: Correlation with outcome variables. Perioperative ischemia randomized anesthesia trial study group. *Crit Care Med.* 1995;23:1954-61.
24. Prinz V, Endres M. The acute (cerebro) vascular effects of statins. *Anesth Analg.* 2009;109(2):572-84.
25. Railton CJ, Wolpin J, Lam-McCulloch J. Renin-angiotensin blockade is associated with increased mortality after vascular surgery: Anesthesia for Vascular Surgery 132. *Can J Anaesth.* 2010;57(8):736-44.
26. Rerkasem K, Rothwell PM. Local versus general anaesthesia for carotid endarterectomy. *Cochrane Database of Systematic Reviews* 2008, Issue 4. Art. No.: CD000126
27. Robertson S. The Canberra Hospital, Australia; Refresher Course: Anaesthesia for endovascular surgery. *South Afr J Anaesth Analg.* 2011;17(1).
28. Rosenman DJ, McDonald FS, Ebbert JO. Clinical consequences of withholding versus administering renin-angiotensin-aldosterone system antagonists in the preoperative period. *J Hosp Med.* 2008;3(4):319-25.
29. Sacco RL, Adams R, Albers G, et al. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack. *Circulation.* 2006;113:e409-49.
30. Tuman KJ, McCarthy RJ, March RJ, et al. Effects of epidural anesthesia and analgesia on coagulation and outcome after major vascular surgery. *Anesth Analg.* 1991;73:696-704.
31. Walsh SR. *Eur J Endovasc Surg.* 2010;39(6):719-25.
32. White CJ, Gray WA. Endovascular therapies for peripheral arterial disease: An evidence-based review. *Circulation.* 2007;116:2203.
33. Yang H, Raymer K, Butler R, et al. The effects of perioperative beta-blockade: Results of the metoprolol after vascular surgery (MaVS) study—a randomized controlled trial. *Am Heart J.* 2006;152:983-90.

Chapter 10

Anesthesia for Pericardiectomy

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Pericarditis is inflammation of the pericardium, often with fluid accumulation. Pericarditis may be caused by many disorders (e.g. infection, MI, trauma, tumors, metabolic disorder) but is often idiopathic. But in India, tuberculous pericarditis is widely prevalent.

Anatomy

The pericardium has two layers:

1. Visceral
2. Parietal

The sac created by these layers contains a small amount of fluid (<25 to 50 mL), composed mostly of an ultrafiltrate of plasma. The thickened pericardium along with the effusion limits distension of the cardiac chambers and decreases the efficiency of the heart.

The pericardium has rich innervations of sympathetic and somatic afferents. Stretch-sensitive mechanoreceptors sense changes in cardiac volume and tension and may be responsible for transmitting pericardial pain. Also, phrenic nerves are embedded in the parietal pericardium and are vulnerable to injury during surgery on the pericardium.

Pathophysiology

Pericardial effusion is accumulation of fluid in the pericardium. The fluid may be serous (sometimes with fibrin strands), serosanguinous, blood, pus or chyle.

Cardiac tamponade occurs when a large pericardial effusion impairs cardiac filling, leading to low cardiac output and sometimes shock and death. If fluid (usually blood) accumulates rapidly, even small amounts (e.g. 150 mL) may cause tamponade because the pericardium cannot stretch quickly enough to accommodate it. Slow accumulation of up to 1,500 mL may not cause tamponade. Loculated effusion may cause localized tamponade on the right or left side of the heart.

Constrictive pericarditis results from marked inflammatory, fibrotic thickening of the pericardium. The visceral and parietal layers adhere to each other or to the myocardium. The fibrotic tissue often contains calcium deposits.

The stiff, thickened pericardium markedly impairs ventricular filling, decreasing stroke volume and cardiac output. Rhythm disturbance is common. The diastolic pressures in the ventricles, atria, and venous beds become virtually the same (diastolic equalization of pressure in all chambers). Systemic venous congestion occurs, causing considerable transudation of fluid from systemic capillaries, with dependent edema and later on, ascites. Occasionally, pleural effusion develops. Chronic elevation of systemic venous pressure and hepatic venous pressure may lead to liver scarring, called cardiac cirrhosis, in which, patients may initially present for evaluation of cirrhosis. Constriction of the left atrium, the left ventricle, or both may elevate pulmonary venous pressure.

Symptoms and Signs

Pericardial Effusion

Pericardial effusion is often painless, but when it occurs with acute pericarditis, pain may be present. Considerable amounts of pericardial fluid may cause muffled heart sounds, increased area of cardiac dullness, and change in the size and shape of the cardiac silhouette. A pericardial rub may be heard. With large effusions, compression of the base of the left lung results in decreased breath sounds and moist adventitious sounds. Arterial pulse, jugular venous pulse, and BP are normal unless intrapericardial pressure increases substantially, causing tamponade.

Cardiac Tamponade

The clinical findings are similar to those of cardiogenic shock:

- ❑ Decreased cardiac output
- ❑ Low systemic arterial pressure
- ❑ Tachycardia
- ❑ Dyspnea
- ❑ Raised JVP
- ❑ Severe cardiac tamponade is nearly always accompanied by a fall of > 10 mm Hg in systolic BP during inspiration (pulsus paradoxus)
- ❑ In advanced cases, pulse may disappear during inspiration
- ❑ Heart sounds are muffled unless the effusion is small. Loculated effusions and eccentric or localized hematoma may cause localized tamponade, in which only selected cardiac chambers are compressed. In these cases, physical, hemodynamic, and some echocardiographic signs may be absent.

Constrictive Pericarditis

Fibrosis or calcification rarely causes symptoms unless constrictive pericarditis develops. The only early abnormality may be elevated ventricular diastolic, atrial, pulmonary, and systemic venous pressures. Symptoms and signs of peripheral venous congestion are:

- ❑ Peripheral edema
- ❑ Hepatomegaly

- ❑ Early diastolic sound (pericardial knock), often best heard during inspiration. This sound is due to abrupt slowing of diastolic ventricular filling by the rigid pericardium. Ventricular systolic function (based on ejection fraction) is usually preserved
- ❑ Prolonged elevation of pulmonary venous pressure results in dyspnea (particularly during exertion) and orthopnea
- ❑ Fatigue may be severe
- ❑ Distension of neck veins with a rise in venous pressure during inspiration (Kussmaul sign) is present; it is absent in tamponade. Pulsus paradoxus is rare and is usually less prominent than in tamponade. Lungs are not congested unless severe left ventricular constriction develops.

ANESTHETIC CONSIDERATIONS

Preoperative Period

Investigations

- ❑ Liver function tests: These are done in view of liver congestion and/or anti-tubercular medications
- ❑ Coagulation profile: These patients usually have abnormal coagulation due to deranged LFT's and also chances of postoperative diffuse bleeding are higher
- ❑ 2D Echo: It helps in assessment of the severity of the disease and to rule out coexisting cardiac pathology
- ❑ Fasting status: Prolonged NPO duration predisposes to dehydration and should be avoided as these patients are preload dependent. Clear liquids may be allowed 2 hours prior to the surgery.

INTRAOPERATIVE

Hemodynamic Goals

- ❑ Rate: Cardiac output in these patients is rate dependent; so bradycardia may be hazardous and the heart rate should be maintained on a higher side (90-100/bpm)
- ❑ Rhythm: Due to already limited ventricular filling, arrhythmias are poorly tolerated and should be aggressively treated.
- ❑ Preload: Avoid decrease in the preload. Maintenance or slight augmentation of preload is desirable.
- ❑ Contractility: Myocardial depression should be avoided.
- ❑ Afterload: Profound reduction in the afterload is not tolerated. Anesthetic induced peripheral vasodilation should be treated promptly.

Monitoring

- ❑ All cases of pericardiectomy warrant invasive monitoring like intra-arterial pressure and central venous catheter apart from routine monitoring.

- ❑ PA catheter monitoring is recommended due to the occurrence of postoperative low CO syndrome.
- ❑ Arterial access: Preserve upper limb arteries in cases of uremic pericarditis for AV fistula creation later on.
- ❑ Temperature: Usually these patients are cachexic and rapidly become hypothermic; so adequate warming measures must be adopted intraoperatively. Hypothermia also increases amount of microvascular bleeding from the surgical raw surface.

Induction

- ❑ Ketamine is generally used for induction as it maintains heart rate and systemic vascular resistance.
- ❑ Drugs causing myocardial depression are avoided. Propofol and volatile agents except isoflurane may not be tolerated well.

Maintenance

- ❑ Usually inhalational agent with less cardiovascular depressant activity like isoflurane is used.
- ❑ Inotropic support may be required to maintain heart rate, afterload and contractility.
- ❑ Fluid administration should be directed towards increasing the effective filling pressure and maintaining adequate cardiac output.
- ❑ Low CO, hypotension, and arrhythmias (atrial and ventricular) are common during surgery.
- ❑ Arrhythmias: Patients are prone to arrhythmias during stripping of the pericardium. If significant hemodynamic changes occur, drugs like magnesium sulfate, lignocaine (preservative free) or amiodarone should be used.
- ❑ Heart rate may have to be maintained with beta agonists or pacing.
- ❑ CP bypass machine should always be kept ready in case of catastrophic hemorrhage from the perforation of a heart chamber during dissection.
- ❑ Ongoing blood losses must be replaced promptly as bleeding is usually significant in these already sick and cachexic patients.

SUGGESTED READING

1. Modak R. Pericardial diseases and trauma, Stoelting's anaesthesia and coexisting disease. In: Hines RL, Marschall K (Eds). 5th edition; 125-30.
2. Oliver WC Jr, Nuttall GA. Uncommon Cardiac Diseases. Kaplan essentials of cardiac anesthesia. Saunders Elsevier, 1st edition.
3. Park MK. Constrictive pericarditis, Pediatric Cardiology for Practitioners, 5th edition, Mosby Elsevier, Philadelphia; 366-7.
4. Townsley M, Shelton ML. Anesthetic considerations for patients with pericardial disease: a practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravelee GP (Eds), 5th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 571-80.

Chapter 11

Anesthesia for Cardiac Catheterization: Laboratory Procedures

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INTRODUCTION

The cardiac catheterization laboratory has expanded its role from mere a diagnostic suite to more advanced center performing complex therapeutic procedures which obviates the need to go under a knife. With the advent of newer technology, the number and the complexity of cardiac catheterization procedures are rapidly progressing. Some of the procedures like placement of ventricular assist device (VAD), percutaneous implantation of valves (TAVI), cardiac resynchronization devices (CRTD), etc. are carried out in extremely sick patients who are at risk of hemodynamic instability which demands the thorough understanding of the procedure and the airway management as well as stabilization of the patients by an anesthesiologist.

WHY IS AN ANESTHESIOLOGIST NEEDED IN THE LABORATORY?

The main role of an anesthesiologist is to provide an airway management, hemodynamic support and resuscitation of the patient in the catheterization laboratory. But nowadays more complex procedures are being performed under transesophageal echocardiographic (TEE) guidance and the skilled anesthesiologist can play a significant role during TEE-guided procedures. Needless to mention that the prerequisite of making the patient cooperative and immobile during intervention is a forte of the anesthesiologist. The anesthesiologist can provide analgesia, amnesia and comfort to the patient while maintaining hemodynamic stability and airway control.

The presence of a cardiac anesthesiologist in the cardiac catheterization/ electrophysiology lab greatly benefits the care of these patients. Also he can escort patient safely to the operation theater if the patient requires an emergency open heart surgery for salvage. Providing safe anesthesia to the patient requires comprehensive preoperative assessment, involvement in multidisciplinary planning of these cases and detailed understanding of the procedure with their potential complications.

ANESTHESIA CHALLENGES

Unfamiliar Environment

The cardiac cath lab is designed to meet the needs of the interventional cardiologist not the anesthesiologist. The monitors, airway equipment, anesthesia work station, drugs, suction, defibrillator, etc. are often unfamiliar. So, an anesthesia cart stocked with usual anesthesia equipments should be readily available within catheterization lab. This cart should include difficult airway equipments, extra length intravenous tubing, anesthesia circuit extensions, extrapressure tubing for invasive monitoring as patient is often at some distance from the anesthesiologist. The access to the patient is often limited by the C-arm.

Radiation Hazard

Ionizing radiation is a hazard to both the patient and staff. Malignancy and genetic injury are unpredictable side-effects of radiation. The traditional dose of the radiation dose absorbed by any material is radiation absorbed dose [rad = gray (Gy) in SI units]. The SI derived unit Sievert (Sv) quantifies the biologic effect of ionizing radiation. Normal annual radiation exposure is about 0.5 mSv/year. In the United States, annual radiation occupation limit is 50 mSv and the lifetime limit is 10 mSv multiplied by age in years. In most of the cardiac catheterization studies, reported dose exposure is well below this limit, usually in the range of 2–4 mSv/year.

Strategies to reduce radiation exposure:

- ❑ Maximize distance from the radiation source: As the intensity of the radiation is inversely proportional to the square of the distance and hence the establishment of the remote anesthetic monitor in the console will limit the exposure to the radiation and one should stand as far as possible from the C-arm.
- ❑ Limit the duration of exposure
- ❑ Use of adequate radiation protective devices like lead aprons, thyroid shield, gonadal guards, goggles, etc.

Also, protective radiation barriers must be used. It is important to understand the mechanism of radiation exposure, i.e. scattering from the body of the patient; so it is advisable to remain on the receiver end of the X-ray arm.

Good communication between anesthesia and cardiology team is essential

Pre-anesthesia checkup is important as most of these patients are on the borderline hemodynamics and fragile. The whole purpose of less invasive intervention in these patients is that they are not suitable candidates to undergo conventional surgical repair.

The anesthesiologist must be familiar with the interventional procedure and should anticipate its requirements like immobility, cooperation and

potential for blood loss and any other major complications. The backup or rescue plan for complications such as cardiac perforation, great vessel rupture, cardiac arrest must be kept ready. Patients with cardiogenic shock require on table IABP insertion, emergency cardiopulmonary bypass or temporary VAD support as rescue therapy. Surgical team standby should always be confirmed prior to the high-risk interventions. The potential for major blood loss must be anticipated and the appropriate blood products should be made available beforehand.

Cardiac catheterization procedures performed in the catheterization laboratory are:

DIAGNOSTIC PROCEDURES

- ❑ Coronary angiography
- ❑ Diagnostic catheterization in congenital heart disease.

INTERVENTIONAL PROCEDURES

- ❑ Balloon atrial septostomy
- ❑ Valvuloplasties: BMV, BPV, BAV, BTV
- ❑ Device closures: PDA, VSD, ASD, RSOV
- ❑ Coil embolization of: BT shunt, MAPCA'S, anomalous coronary arteries, rupture of sinus of Valsalva, vertebral vein coiling
- ❑ Intravascular stent placement in coronary artery, renal arteries, pulmonary arteries, coarctation of aorta
- ❑ Transcatheter implantation of aortic (TAVI), pulmonary valves
- ❑ MitraClip procedure for mitral valve repair
- ❑ Electrophysiologic procedures: Radiofrequency ablation of arrhythmogenic foci
- ❑ Pacemakers and automatic cardioverter defibrillator implantation (AICD) insertion
- ❑ Cardiac resynchronization therapy device (CRTD) implantation.

PREOPERATIVE EVALUATION

In adults, detailed history and examination pertaining to the disease and its complications; functional status of patient is assessed. Investigations include hemogram, ECG, chest X-ray, renal function tests including BUN, serum creatinine and electrolytes, coagulation profile (PT, PTT and platelet count), baseline ABG in adult cyanotic heart disease.

Clinical status of children is assessed by enquiring about child's exercise tolerance or infants feeding habits. Reduced activities with increasing fatigue or feeding associated with irritability, dyspnea, cyanosis indicates poor cardiopulmonary compensation. The patients are examined for signs of heart failure. CHD may be associated with other congenital anomalies like

musculoskeletal abnormalities (8.8%), neurological defect (6.9%), atlanto-occipital subluxation (20% of Down syndrome), macroglossia and micrognathia which may lead to difficulty in intubation. Repeated respiratory tract infections are common in children with left-to-right shunts. In our country, children present for surgery later in life by which time severe pulmonary hypertension had already progressed to severe grade.

Children with cyanotic CHD may have hematocrit more than 65%. In such a situation, preoperative phlebotomy is done to decrease chances of cyanotic spells during the procedure. Also patients with cyanotic heart disease may have thrombocytopenia, hypofibrinogenemia, low levels of vit-K dependent clotting factors. Therefore, coagulation tests like PT, PTT and platelet count should be done. Serum electrolytes are important in patients receiving digoxin or diuretics.

Fasting guidelines: In children who require sedation, fasting guidelines are same as those who require general anesthesia.

2 hours—Clear fluids without fruit pulp

4 hours—Breast milk; fruit pulp

6 hours—Solid foods

Consent: Informed consent is taken for GA even if case is done under sedation. Consent is also taken for blood transfusion, ICU treatment and CP bypass in case of complications which warrants patient to be taken on bypass for surgery.

Premedications: In IHD patients long-term medications like antihypertensives, antianginal drugs are continued till the morning of procedure. Antibiotics are given to prevent infective endocarditis.

Sedation for children is carried out by midazolam—0.02–0.25 µg/kg IV, 0.5 mg/kg oral or intranasal OR, triclofos—75–100 mg/kg oral OR IV, fentanyl—1–2 µg/kg bolus followed by infusion.

In sick or premature neonates premedication may be completely avoided due to the fear of respiratory depression.

Monitoring: Standard ASA monitoring includes ECG, NIBP, IBP, pulse oximetry, end tidal CO₂ (ETCO₂), ABG, temperature, ACT and urine output.

PROCEDURES

Balloon Atrial Septostomy (BAS)

BAS was introduced by Rashkind. It a life-saving and emergency procedure performed in a neonate with TGA presenting with severe hypoxia, cyanosis and acidosis. It is also done in tricuspid and mitral atresia with hypoplastic right and left ventricle and in some cases of total anomalous pulmonary venous connection (TAPVC). It can also be done as a bed side procedure using echocardiographic guidance. This procedure usually requires GA with ET intubation as baby is severely cyanotic, hypoxic and acidotic. The balloon is inflated in LA and then jerked across the septum to increase the interatrial

communication. This helps in increasing the arterial oxygen saturation of a deeply cyanotic baby. Hypothermia, acidosis, hypoglycemia and hypocalcemia are other complications associated with neonates which should be taken care of perioperatively.

Valvuloplasties

BPV: Historically the pulmonary valve was the first valve to be balloon dilated. It is an effective method of relieving RV outflow obstruction and treatment of choice for pulmonary valve stenosis.

Vascular access is performed via femoral vein and the course of the catheter is right atrium to right ventricle into the pulmonary artery. When the balloon is inflated across stenotic valve, cardiac output decreases transiently due to decrease in pulmonary blood flow resulting into hypotension and bradycardia. All this manifestation usually recovers spontaneously once the balloon is deflated. Occasionally, complete heart block/ventricular tachycardia may occur in cases of long standing PS resulting into RV dysfunction.

The procedure is usually done under conscious sedation. Care must be taken to avoid factors increasing pulmonary vascular resistance.

Mitral valvuloplasty (BMV): Mitral valve is accessed via interatrial septal puncture and the balloon is passed into the left ventricle and pulled back across the mitral valve and then inflated till the prescribed volume. The Inoue-balloon is composed of latex and is inflated using radiopaque dye. The balloon is inflated to the prescribed volume noting the presence of central waist which disappears at maximum balloon pressure. Success is considered when the gradient across the valve is reduced by 50% or the increase in the valve area evident on echocardiography.

This procedure is usually done under local anesthesia and sedation. Patient with critical valve stenosis may be in pulmonary edema where it is important to intubate the patient and give positive pressure ventilation with PEEP.

Complications include cardiac tamponade during septal puncture, acute severe MR due to excessive commissural splitting/chordae rupture which may necessitate general anesthesia for better hemodynamic control. Sometimes iatrogenic ASD, which is created during transseptal puncture, fails to close. Another issue is to keep an eye on the adequate heparin dose and its timing during the procedure to avoid shedding of emboli into the cerebral circulation when the catheter is in the left-sided chamber of the heart.

Balloon Aortic Valvotomy (BAV)

In this procedure, balloon is inflated across aortic valve and so only arterial access is required. Transient decrease in cardiac output and vasovagal response occurs during balloon inflation due to interruption of the blood flow which subsides on its own or sometimes atropine is needed. During balloon inflation, transient phase of ventricular fibrillation is created using ventricular pacing which may produce hemodynamic instability.

Device Closure

Patent Ductus Areriosus

Patent ductus areriosus (PDA) closure was the first transcatheter closure procedure performed using percutaneous technique. Gianturco coils are used to occlude small PDAs, but self-expanding occluders such as the AMPLATZER duct occluder (St Jude Medical, Plymouth, MN) are used for larger ducts (Fig. 1).

These procedures require retrograde placement of a fine guidewire into the PDA from the femoral or upper-limb artery under fluoroscopic guidance. Sedation or general anesthesia may be required because the procedures can be of unpredictable duration and because patients must be immobile to allow accurate device/coils deployment across the orifice of the PDA. In patients with secondary pulmonary hypertension and right-heart failure, general anesthesia may be preferable if supine positioning is poorly tolerated. Increases in pulmonary vascular resistance (PVR) caused by hypoxia, hypercarbia or high ventilating pressures must be avoided because this will exacerbate right-heart failure. At the same time increase in the magnitude of the shunt should be avoided in patients without pulmonary hypertension by proper ventilatory support, i.e. using low FiO_2 and PEEP. Pediatric patients can become hypothermic rapidly so adequate warming methods must be followed. Systemic heparinization should be used throughout the procedure to prevent arterial thrombotic embolization. Usual dose of heparin is 100 U/kg IV followed every hour by 20–30 U/kg.

Atrial Septal Defect

Atrial septal defect (ASD) closure usually is performed for the prevention of stroke or right ventricular volume overload and pulmonary hypertension. Current American Heart Association guidelines recommend closure of ASDs

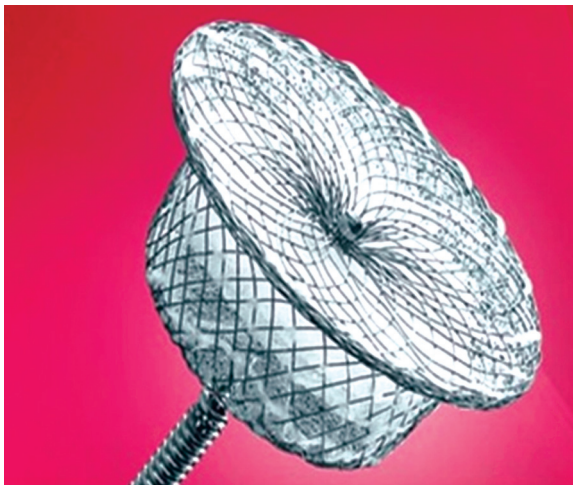


Fig. 1: The AMPLATZER duct occluder



Fig. 2: AMPLATZER PFO Occluder (St Jude Medical, Plymouth, MN)

for right atrial or right ventricular enlargement (with or without symptoms), paradoxical embolism, documented orthodeoxia-platypnea (a rare condition in which dyspnea and deoxygenation are induced by standing), and pulmonary hypertension. Patient selection is important because anatomic features, such as limited ASD rims, large or irregular ASD size, and anomalous pulmonary venous drainage limit the effectiveness of closure devices.

Transcatheter percutaneous device closure is an attractive and novel approach to close ostium secundum atrial septal defect in which umbrella devices (Fig. 2) have been used. As the procedure is done under TEE guidance GA with intubation with one size smaller ETT is used. In cases where transthoracic echo is used for device placement, sedation is sufficient. Again patient must be immobile during device placement.

During device deployment there are chances of bundle branch block and heart block which must be treated with atropine or pacing. Occasionally, device embolization may result in device engagement into RVOT/PA's producing hemodynamic catastrophe. Meticulous deairing of all fluid lines should be ensured to reduce the potential for right-to-left air embolization during device placement.

Heparin is administered during this procedure in a dose of 100 U/kg and repeated every hour in a dose of 20 U/kg to prevent thrombotic complications.

Sternal pain should alert the anesthesiologist to the possibility of myocardial perforation, which complicates 0.1% of ASD closures.

Most perforations occur within 72 hours, but about a third of cases gets delayed for up to 3 years. Acute or delayed aortic root perforation also can occur if the closure device abuts the aortic root; this may present as aortoatrial fistula or cardiac tamponade. Other potential complications include acute or delayed device embolization and stroke although these are extremely rare. Atrial thrombus, predominantly left-sided and attached to the implanted

device, is a more common complication that affects 2% of patients and usually resolves with oral or intravenous anticoagulation. Post-procedure, patients usually receive oral low dose aspirin for six months.

Ventricular Septal Defect

In contrast to ASD and PDA device closures, ventricular septal defect (VSD) device closures are prolonged procedures usually associated with hemodynamic instability and blood loss, which can be significant in infants and children. TEE is used to position the device, evaluate the residual shunt and atrioventricular valve function. VSD device closure is done under GA with endotracheal intubation. Potent and short-acting drugs like fentanyl, midazolam, propofol are used. Muscle relaxants like atracurium and vecuronium are used. Sevoflurane is the agent of choice for maintenance.

Hemodynamic problems are related primarily to the procedure. Contributing factors includes acute aortic or mitral regurgitation, wire and catheter manipulation. Hemodynamic instability may require treatment varying from fluid replacement, aggressive pharmacological support to full cardiopulmonary resuscitation with cardiac massage. There is likelihood of complete heart block during device deployment for which pacing equipment must be kept ready.

In view of potential hemodynamic complications, airway protection with ET intubation is a must. Also TEE is used to assist device placement. Right IJV may be cannulated to approach apical muscular VSD. All these issues make intubation and ventilation of these patients a necessity.

Coil/device can get embolized after it is released from the sheath. In such a situation, GA is preferred as retrieval of coil/device may prolong the procedure. Anesthesiologist must keep an eye on blood loss during the procedure which is an important aspect in pediatric patients.

Analgesia is usually provided by intravenous paracetamol and local anesthetic infiltration at the puncture site.

Left Atrial Appendage (LAA) Occlusion

The LA appendage is a nidus for thrombus formation because of its long, narrow lumen and trabeculations. It is thought to be a source of most atrial thrombi in patients with nonvalvular atrial fibrillation (AF). Atrial appendage occlusion devices such as the WATCHMAN LAA Closure Device (Atritech, Minneapolis, MN, Fig. 3) are similar to PFO/ASD are placed in the orifice of the LAA. In a randomized trial of 707 patients with AF, closure of the LAA was shown to be as effective as warfarin therapy alone in stroke prevention. However, the Watchman device group was associated with a higher rate of adverse events, particularly bleeding complications. The AMPLATZER device also is effective for LAA closure (96%), with a similar reported rate of serious complications to the Watchman closure device (7%, including pericardial effusions in 3.5%). The anesthetic plan includes invasive monitoring and preparation for the management of pericardial tamponade and major bleeding.



Fig 3: WATCHMAN left atrial appendage occlusion device.
(Image courtesy: Atritech © 2011)

Anesthetic Considerations in Left-to-right Shunts

The principle is to avoid the worsening of the shunt. It is beneficial to decrease the afterload in these cases and so they are best induced with inhalational agents as they have increased pulmonary blood flow. Halothane or sevoflurane are used for induction. Intravenous induction can be achieved with Injection thiopentone 4–6 mg/kg or propofol 2 mg/kg and maintained with propofol infusion. Hypoxia, hypercarbia, acidosis, hyperinflation, sympathetic stimulation which worsens pulmonary hypertension should be avoided at all times. Nitrous oxide, ketamine which increase pulmonary hypertension should be avoided. Expansion of air emboli with nitrous oxide is also a consideration in all shunt cases. It is important to keep lowest possible FiO_2 to decrease excessive pulmonary blood flow. Maintenance can be achieved with oxygen, air and inhalational agents like halothane, isoflurane, sevoflurane. Fentanyl and Midazolam are used for sedation and analgesia. But in patients with systemic or suprasystemic pulmonary artery pressures, avoiding all the factors which increase PA pressures takes priority.

Coil Embolization

Systemic arterial to pulmonary artery collaterals results in left-to-right shunt. Certain groups of patient such as those with pulmonary atresia may have large collaterals supplying lung segment or lobes. Before definitive surgery or bypass, thrombogenic coils may be introduced into these collaterals that are not suitable for unifocalization. These coils are coated with thrombogenic material which induces clot formation, once introduced into bloodstream. Similarly coils can be used to close the BT shunt before the child goes for intracardiac repair. Rupture sinus of valsalva, anomalous coronary artery can also be closed by coils. This avoids major surgery. Besides, this procedure can be done under sedation in older child. Coil embolization is usually done under GA in children. Vertical vein closure after TAPVC repair is also done by coil embolization under general anesthesia as the patients are in the pediatric age group.

Anesthetic Considerations in Right-to-left Shunts

In children with right-to-left shunts, a slight heavier premedication is preferable as crying and struggling precipitates a cyanotic spell. Also overstarvation should be avoided. Children who have a difficult IV or are uncooperative for IV can be induced with inhalational agents. If IV is already in place, ketamine is a good choice for induction. The child can be maintained with ketamine and midazolam if the procedure is short. If the child is very sick or the procedure is long, it is preferable to have LMA or ETT inserted. Maintaining fluids, ABG and temperature are important considerations in anesthetic management. Baseline saturation should be noted. A cyanotic spell should be suspected in the event of any intraoperative drop in saturation. Cyanotic spell is treated with β -blocker like metoprolol 0.1 mg/kg or esmolol 0.5 mg/kg bolus followed by infusion 50–300 μ g/kg/min, morphine 0.1 mg/kg, NaHCO_3 1 mL/kg (send for ABG and correct acidosis), Ringer lactate 20 mL/kg. If saturation does not come up in spite of these drugs, phenylephrine 5–10 μ g/kg IV bolus followed by infusion 2–5 μ g/kg/min to increase SVR is given.

Intravascular Stent Placement

PA Stenosis

Stenting of the right ventricular-to-pulmonary artery conduit may be used to prolong conduit lifespan. Endovascular stents also have been used in cases of pulmonary arterial compression caused by tumor or after lung transplantation. Coronary arterial compression has been reported during stenting of a right pulmonary arterial conduit. In a patient with single lung, extracorporeal circulatory support is required during pulmonary artery stenting.

Aortic Coarctation

Coarctation of the aorta represents approximately 5% to 10% of CHD cases and is associated with poor long-term survival if untreated. Rapid ventricular pacing is used to prevent stent movement during deployment, and intravenous adenosine also can be used for this purpose. General anesthesia is used in most cases, with invasive blood pressure monitoring in the right arm. Because of the potential for aortic dissection or rupture, rapid transfusion facilities and cardiac surgical backup should be available immediately. Delayed arterial complications such as stent migration and pseudoaneurysm also have been reported.

In adults, dilatation is usually done under sedation and local anesthesia. In children GA is preferred. Dissection, aneurysms are known complications. Patient may complain severe pain in chest and abdomen in case of dissection which should be managed promptly.

Electrophysiologic Study and Anesthesia Implications

The demand for cardiac EP procedures is increasing, as is the range of procedures performed. Although diagnostic studies may be performed in isolation, they

are almost always combined with therapeutic ablations if treatable aberrant conducting pathways are identified. Ablation creates an endocardial scar, which can be produced by RF energy, cryotherapy, or high-intensity focused ultrasound. Ablation procedures can be classified by the level of complexity or by the location [e.g. supraventricular tracts, AV node, ventricular re-entrant pathways, and pulmonary vein isolation (PVI) procedures].

Noncomplex Catheter Ablations

Noncomplex ablations include procedures for AV nodal reentry tachyarrhythmia, atrial flutter, and tachycardia mediated by accessory pathways, such as Wolff-Parkinson-White (WPW) syndrome. Ablation catheters are deployed under fluoroscopic guidance via large peripheral veins (usually the femoral vein). These procedures commonly are performed under sedation and generally take less than 2 hours to perform.

Complex Catheter Ablations

Complex catheter ablations are performed for AF and for tachyarrhythmia because of ventricular foci, multiple foci, or CHD. Electro-anatomic mapping systems using previously acquired 3D computed tomography scanning or magnetic resonance imaging reconstructions of the heart and thoracic vessels commonly are used to guide these procedures. These systems provide a 3D representation of the cardiac chambers and catheters as well as displaying the cardiac electrical activation timing.

Diligent attention to the volume of fluid administered by the cardiology team is important because this may amount to several liters during the procedure; hence, a urinary catheter should be placed. Hypothermia can occur during these long procedures, particularly in elderly patients, so the use of forced air warming devices also is recommended.

These patients should receive heparin to prevent catheter thrombosis and thromboembolic stroke, particularly when transarterial or transseptal catheters are used. Clear delineation of the responsibility for the management of anticoagulation must occur. The target ACT range for these cases is based on the PCI guidelines.

Complex ablations may produce prolonged periods of induced or persisting tachyarrhythmia. Hemodynamic instability is a particular concern in patients with pre-existing ventricular dysfunction or CHD. Hypotension may be tolerated poorly in patients with coronary artery disease, carotid artery stenoses, or chronic hypertension. Acute elevations in PVR (e.g. because of hypercapnia, hypoxemia, or acidosis) are tolerated poorly in patients with severe pulmonary hypertension or passive pulmonary circulations. Therefore, external pads should be placed for defibrillation or external pacing.

Reactive pericardial effusion can occur in patients undergoing extensive left atrial ablations. Therefore, it is important to remain alert to the risk of cardiac tamponade in these patients. TEE is useful for the early detection of this complication, which is more likely if multiple, extensive ablations are performed. The recognition of tamponade may otherwise be delayed if

hypotension is attributed to poor underlying ventricular function. Facilities for pericardiocentesis or surgical drainage should be readily available. Ablations near the coronary sinus and coronary arteries may be complicated by acute occlusion of these vessels, especially in patients with aberrant coronary arterial anatomy. This rare complication should be considered if unexplained hemodynamic deterioration occurs.

The ideal anesthetic for EP procedures should not affect intrinsic pacemaker function, impulse propagation, refractoriness, or autonomic tone although most anesthetic agents have some effect on cardiac conduction. Despite anecdotal preferences and published opinion, there is little data on the effects of anesthetic agents on cardiac conducting pathways relevant to EP procedures.

Radiofrequency energy is the low power high frequency current which causes controlled heat at the tip of catheter. Radiofrequency ablation is a painless procedure which does not require GA. During this procedure, arrhythmia are induced with drugs like isoprenaline and accessory pathway is detected and ablated. Adenosine is used to block the AV node then atria or ventricle is stimulated to check the aberrant pathway. Inhalation agents may alter the cardiac electrophysiology, thereby making it difficult to locate the accessory pathway. Ectopic atrial tachycardia is seen to be terminated with propofol and cannot be induced by isoprenaline infusion. Propofol should be avoided in such cases.

Automatic Implantable Cardioverter Defibrillator

The indications for ICDs are broad and include patients at risk of sudden cardiac death because of ischemic or nonischemic dilated cardiomyopathy, inherited arrhythmia syndromes, hypertrophic cardiomyopathy, long QT syndrome, and syncope with inducible sustained ventricular tachycardia. The technique for ICD insertion is the same as for pacemakers, but the leads and pulse generators are larger.

The decision to perform these cases under general anesthesia or sedation will depend on patient comorbidities and the expected duration of the procedure. Invasive arterial pressure monitoring is routine in patients with poor ventricular function for hemodynamic monitoring during defibrillation. External defibrillator pads must be placed before ICD testing in the event of device failure. TEE sometimes is used to exclude intracardiac thrombus before testing. Careful attention must be provided to infection prophylaxis because infected devices usually require removal and replacement.

This device is implanted pectorally which delivers the electric shock internally within 10–15 seconds of arrhythmia onset correcting the abnormal rhythm back to normal sinus rhythm thus preventing sudden death. AICD can also deliver pacing (anti bradycardia, antitachycardia) in addition to defibrillation and synchronized cardioversion. These cases are usually done under local anesthesia with conscious sedation. The external defibrillator is kept ready for use at all times. GA is preferred during checking of defibrillation threshold. Propofol is usually used during defibrillator testing. If ejection fraction of the patient is low, careful titration of sedatives is used. After defibrillation, ABG is checked and acidosis if any is corrected.

Cardiac Resynchronization Therapy Devices

Cardiac resynchronization therapy (CRT) improves heart failure symptoms and reduces mortality in patients with broad QRS complexes, impaired ejection fractions, and class III or IV NYHA heart failure symptoms. Patients with NYHA class I and II symptoms may also benefit.

CRT involves the placement of biventricular pacemaker leads that are adjusted to optimize intraventricular conduction. Most CRT devices also function as ICDs (CRTD). CRT device insertion usually is performed under general anesthesia, with invasive arterial pressure monitoring. The leads may be placed *de novo* or as an upgrade to a previously implanted right ventricular pacemaker. The LV pacing lead can be attached to the epicardium during cardiac surgery or percutaneously via the coronary sinus to pace the lateral wall of the LV. Coronary sinus lead placement can be technically challenging, of prolonged duration and can be complicated by cardiac perforation or arrhythmias. Preoperative computed tomography scanning of the coronary sinus may aid planning of these cases. TEE also can be useful in guiding the placement of the coronary sinus lead.

As the procedure is of prolonged duration, and so, urine output must be monitored. Complications include hypothermia, cardiac tamponade and significant arrhythmia.

Conscious sedation using dexmedetomidine is also an attractive mode of anesthesia but one must be vigilant as these patients usually have low ejection fraction. Appropriate analgesia must be provided. Occasionally, general anesthesia is needed in case of hemodynamic instability during the procedure.

Cardioversion

Cardioversion is a procedure where synchronized electric shock is given to restore normal sinus rhythm. It differs from defibrillation in the nature of the shock (synchronized with R wave) and generally of the less magnitude. It is a short but painful procedure which demands amnesia and analgesia. It can be a elective procedure or an emergency in case of hemodynamic instability. Its indications are supraventricular tachycardia and stable VT's.

Conscious sedation under propofol is generally enough. In cases of borderline hemodynamics, etomidate is a safe option. Analgesia is provided using short acting opioid (fentanyl). One should be vigilant in managing airway during the procedure and all resuscitation equipment must be available bedside.

ANESTHETIC IMPLICATIONS OF PACEMAKER

Introduction

The number of elderly patients with electrophysiological disturbances requiring permanent pacemaker therapy has significantly increased in recent times. These patients may need to undergo geriatric surgeries like TURP, joint replacement, etc. Therefore, anesthesiologist must be well-versed with anesthetic implications of a pacemaker.

Cardiac pacing is most reliable treatment for various cardiac arrhythmias. The American College of Cardiology/American Heart Association (ACC/AHA) indications for permanent pacemaker or antitachycardia devices are as follows:

- ❑ Acquired AV block:
 - Third degree AV block: Symptomatic bradycardia
 - Refractory postoperative AV block
 - AV block with escape rhythm <40 bpm or asystole >3 seconds
 - Second degree AV block: Permanent or intermittent block with symptomatic bradycardia
- ❑ After myocardial infarction:
 - Persistent second degree or third degree block
 - Infranodal atrioventricular (AV) block with left bundle branch block (LBBB)
 - Symptomatic second or third degree block
- ❑ Bifascicular or trifascicular block:
 - Intermittent complete heart block with symptoms
 - Type II second degree AV block
 - Alternating bundle branch block
- ❑ Sinus node dysfunction:
 - Sinus node dysfunction with symptoms as a result of long-term drug therapy
 - Symptomatic chronotropic incompetence
- ❑ Hypertensive carotid sinus and neurocardiac syndromes:
 - Recurrent syncope associated with carotid sinus stimulation
 - Asystole of >3 sec duration in absence of any medication

Pacemaker Coding

Modified North American society of pacing and electrophysiology/British pacing and electrophysiology group (NASPE/GPEG) coding consists of a five position system using a letter in each position to describe the programmed function of a pacing system. The first letter indicates the chamber being paced, the second letter designates the chamber being sensed, third position designates response to sensing (I and T indicates inhibited or triggered responses, respectively). The fourth and fifth positions describe programmable and antitachyarrhythmia functions, but these two are rarely used. An R in fourth position indicates that the pacemaker incorporates a sensor to modulate the rate independently of intrinsic cardiac activity such as with activity or respiration.

Generic codes for pacemaker

<i>Pacing</i>	<i>Sensing</i>	<i>Response</i>	<i>Programmability</i>	<i>Tachycardia</i>
O-None	O-None	O-None	O-None	O-None
A-Atrium	A-Atrium	I-Inhibited	C-Communicating	P-Pacing
V-Ventricle	V-Ventricle	T-Triggered	P-simple programmable	S-Shocks
D-Dual (A+V)	D-Dual (A+V)	D-Dual (I+T)	M-multiprogrammable	D-Dual (P+S)
S-Simple (A or V)	S-Simple (A or V)		R-Rate modulation	

Unipolar Lead Pacing

Only single electrode stimulates heart and so current has to traverse a longer distance to complete a circuit sensing extracardiac signals and myopotentials.

Bipolar Leads Pacing

Two different electrodes located close to each other, pace the chamber. As a result, less sensing of extracardiac signals occurs and more accurate pacing is performed.

Pacemaker Battery

Lithium-iodine batteries are being used nowadays which have longer shelf life (5–10 years).

Threshold

It is the minimum energy required for successful pacing. Increased threshold means more energy is needed to initiate pacing and so battery also drains out faster. Factors which increase threshold are hyperkalemia, acidosis/alkalosis, severe hypoxia, hypothermia, hypothyroidism, hypoglycemia, amiodarone and myocardial infarction.

Anesthetic drugs are not likely to change the pacing threshold. Combination of opioids and equipotent doses of halothane, enflurane, or isoflurane after cardiopulmonary bypass did not increase pacing threshold.

Asynchronous Pacing (A00, V00, and D00)

In this, heart is paced at a fixed rate irrespective of the intrinsic heart rate. It may result in tachyarrhythmias and continuous pacing also reduces the battery life.

Single Chamber Pacing (AAI, AAT, VVI, and VVT)

Either atrium or ventricle is sensed and paced. VVI is most commonly used pacing mode in which intrinsic R wave is sensed and pacemaker function is inhibited. Single chamber ventricular pacing is not recommended for patients with sinus node disease, as these patients are more likely to develop the pacemaker syndrome.

Dual Chamber AV Sequential Pacing (DDD, DVI, DDI, and VDD)

Both atrium and ventricle are paced sequentially. The atrium is paced first, then after an adjustable PR interval, ventricle is paced. These pacemakers preserve the normal AV synchrony and are beneficial in situations where atrial kick should be preserved (e.g. MS, AS).

ANESTHESIA CONSIDERATIONS

Preoperative Evaluation

Evaluation of the patient with respect to the severity of cardiovascular disease, functional status, and medications should be done. Routine investigations should be performed as per the need. Measurement of serum potassium needs to be done because it changes pacing threshold and may pose intraoperative problems. 12 lead ECG and X-ray chest (for visualization of continuity of leads) is needed. The most important question to answer is whether the patient is pacemaker-dependent or not.

Evaluation of pacemaker should include the indication and the time of insertion, type of pacemaker, and half-life of the pacemaker battery. This can be easily done by referring to the pacemaker manual kept with the patient and consulting the cardiologist.

Pacemaker reprogramming: ACC/AHA guidelines advise that all antitachycardia therapy should be disabled before anesthesia. If the risk of electromagnetic interference (EMI) is high, alternative temporary cardiac pacing device should be available.

Use of Magnet

Previous generation pacemakers were using magnet operated reed switches. Placement of a magnet over the pulse generator activates the reed switch which shuts down the demand function converting pacemaker into a nonsensing asynchronous mode with a fixed pacing rate.

However, not all pacemakers switch to asynchronous mode on the application of magnet and it is advisable to consult the manufacturer to know the magnet response before use. The new generation pacemakers are relatively immune to magnet application and may not convert to asynchronous mode. Also, it should be kept in mind that switching to asynchronous pacing mode may trigger ventricular asynchrony in patients with myocardial ischemia, hypoxia and electrolyte imbalance.

MONITORING

Continuous ECG monitoring is essential to monitor pacemaker function.

External defibrillator must be checked and in pacemaker-dependent patients, temporary pacing mode should be available in case of an emergency.

Central venous or PA catheter should be inserted only in the indicated cases because guidewire can dislodge pacemaker leads. Besides, these are potentially arrhythmogenic.

Intraoperative Considerations

Both narcotic and inhalational techniques can be used safely as anesthetic agents are not likely to change the pacing threshold. It has been observed that

the combination of opioids and equipotent doses of halothane, enflurane, or isoflurane after cardiopulmonary bypass did not increase pacing threshold. However, fasciculations, myoclonus, shivering and electroconvulsive therapy can affect the pacemaker function.

Etomidate and ketamine should be avoided as these cause myoclonic movements. Scoline induced muscular fasciculations can inappropriately trigger or inhibit pacemaker.

Ventilation: Positive pressure ventilation may cause dislodgement of pacemaker leads.

Nitrous oxide can also diffuse and expand into the pacemaker pocket and may alter pacemaker function.

Rate responsive mode should be deactivated before surgery. If this is not possible, then conditions causing changes in paced heart rate need to be avoided perioperatively. For example, shivering and fasciculations should be avoided in 'activity' rate responsive pacemaker, ventilation (respiratory rate and tidal volume) should be controlled in case of 'minute ventilation' rate responsive pacemakers, and temperature must be kept constant in 'temperature' rate responsive pacemakers.

Electromagnetic interference (EMI)

- ❑ Sources of EMI are:
Surgical electrocautery, MRI, orthopedic saw, mechanical ventilators, lithotripters, cellphones, etc. Diagnostic radiology and computed tomographic (CT) scans do not affect the function of the pacemaker.
- ❑ Following principles should be kept in mind while using electrocautery (More important in pacemaker-dependent patients):
 - Bipolar cautery should be preferred but if unipolar cautery is to be used during operation, the grounding plate should be placed close to the operative site and as far away as possible from the site of pacemaker with good skin contact.
 - Avoid using cautery within 15 cm of pacemaker pulse generator.
 - Use cautery in short bursts with long pauses and the frequency should be limited to 1-second burst every 10 seconds to prevent repeated asystolic periods.
 - Ensure that the programmer is available in the operation theater and pacemaker is programmed to asynchronous mode.
 - During the use of cautery, magnet should not be placed on pulse generator as it may cause pacemaker malfunction.
 - Alternative mode of temporary pacing and isoproterenol (pharmacologic pacemaker) should be available in the OT.
 - Anterior-posterior positioning of defibrillator paddles is preferred and paddles should be positioned as far away as possible from the pacemaker generator.
 - ECG monitoring can also be affected by interference and becomes unreliable.
 - The device should always be rechecked after the procedure.

SPECIFIC PROCEDURES AND EFFECT OF PACEMAKER

Electroconvulsive Therapy

Skeletal myopotentials resulting from seizures may inhibit the pacemaker and hence, it should be changed to asynchronous mode.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) in patients with pacemaker is contraindicated because reed switch activation by high static field of 0.5–1.5 T can result in switching off the pacemaker. It can also interfere with pacemaker output circuits and result in rapid pacing, pacemaker reprogramming and destruction of electronic components. It may also cause thermal injury to endocardium and myocardium.

If MRI is absolutely essential, then it should be done in presence of a cardiologist with temporary pacing equipment readily available. But, patients with pacemakers should not routinely undergo MRI scanning.

Extracorporeal Shock Wave Lithotripsy

High-energy vibrations produced by lithotripsy machine can cause closure of reed switch resulting in asynchronous pacing. Dual chamber pacemaker is especially sensitive to shock waves and should be reprogrammed to a simpler mode (VOO, VVI) preoperatively. 'Activity' rate responsive pacemaker can be affected due to damage caused to the piezoelectric crystals by extracorporeal shock wave lithotripsy (ESWL). The shock waves can produce ventricular extrasystoles, if not synchronized with R wave.

Therefore, rate responsive pacemaker should have their activity mode deactivated prior to the procedure. Focal point of the lithotripter should be kept at least six inches (15 cm) away from the pacemaker. Patients with abdominally placed pacemaker generators should not be treated with ESWL. Low shock waves (<16 kilovolts) should be used initially followed by a gradual increase in the level of energy.

Transurethral Resection of Prostate (TURP) and Uterine Hysteroscopy

The cutting current at high frequencies can suppress the output of a pacemaker and preoperative reprogramming of pacemaker to the asynchronous mode is advisable.

Radiation

Complementary metal oxide semiconductor (CMOS) inside pacemaker can be damaged by radiation. If pacemaker cannot be shielded from the field of radiation, consideration should be given to reimplanting the pacemaker at a

distant site. Temporary damage to pacemaker may recover after reprogramming but there may be permanent damage to the pacemaker as well.

Transcutaneous Electronic Nerve Stimulator Unit

Transcutaneous electronic nerve stimulator unit (TENS) creates repeated frequency similar to the normal range of heart rates, so it can create a far field potential that may inhibit pacemaker function. Pacemaker mediated tachycardia has been induced by intraoperative somatosensory evoked potential stimulation. So, these patients should be monitored during initial application of TENS.

Summary

- ❑ Cardiologist should be consulted for device evaluation regarding its proper function and life of the batteries
- ❑ While using electrocautery, precaution for minimal EMI should be taken
- ❑ Magnet should not be placed over pacemaker in presence of electrocautery
- ❑ Rate responsive pacemakers should have rate responsive mode disabled before surgery
- ❑ Provision of temporary pacing should be available in the OT to deal with emergency situation of pacemaker malfunction
- ❑ Pacemaker should be rechecked after the procedure.

DIAGNOSTIC CATHETERIZATION

The number of adult survivors of pediatric congenital heart surgery (GUCH-grownup adults with CHD) are increasing. Although the leading cause of death in patients with congenital cyanotic heart disease is arrhythmia followed by heart failure, in noncyanotic patients, it is now ischemic heart disease. Right-heart catheter studies frequently are indicated in these patients to define the anatomy of previous corrective procedures, to determine the direction and size of shunts and to assess the reversibility of pulmonary hypertension. These patients also may present for coronary angiography or EP procedures for the management of arrhythmias.

These patients present a number of anesthetic challenges. Careful preoperative assessment is essential to determine the presence and severity of heart failure and cyanosis. Consultation with the attending cardiologist regarding the procedural aims may aid in anesthetic planning in patients with complex cardiac anatomy. Preoperative evaluation of noncardiac comorbidities is important (such as cervical spine instability in trisomy 21) together with correct timing of antibiotic prophylaxis.

In patients with right-to-left shunts, it is important to avoid increasing PVR and decreasing system vascular resistance (SVR) because this will increase shunt flow and worsen cyanosis. Systemic vasodilatation, hypercarbia, and high ventilating pressures must be avoided. Volatile anesthetics should be

titrated carefully and SVR maintained with vasopressors if required. In patients with systemic-to-pulmonary shunts, decreased SVR and increased PVR should be avoided because this will decrease pulmonary blood flow.

Right-to-left shunting also affects end-tidal CO_2 measurement; the arterial-to-alveolar pCO_2 gradient increases as shunting worsens. The direct measurement of arterial CO_2 will be more accurate than end-tidal CO_2 monitoring in patients who are at risk of elevated PVR because of hypercarbia.

Acute elevations in PVR caused by high positive airway pressures may be tolerated poorly in patients with passive venoatrial pulmonary circulations. In these patients, the driving pressure gradient providing pulmonary blood flow is the central venous pressure (rather than mean pulmonary artery pressure) minus left atrial pressure. If positive-pressure ventilation is used, the inspiratory time and pressure should be titrated to the lowest level that will maintain normocarbia at the lowest mean airway pressures. Spontaneous ventilation, with inspiratory pressure support if required, may be used as an alternative to positive-pressure ventilation to minimize ventilating pressure, provided that normocarbia is strictly maintained.

The venous anatomy should be considered when placing central catheters. In a Fontan circulation, caution should be exercised when administering drugs that may be thrombogenic, such as calcium, because the SVC is connected directly to the pulmonary artery. Arterial pressure monitoring in the presence of arterial shunts (such as the Blalock-Taussig shunts or variants) should be performed on the contralateral side to avoid under-reading systemic pressures. Venous access can be difficult in these patients because they have often had multiple and prolonged hospitalizations. Finally, thromboprophylaxis should be considered in patients with high hematocrits because of cyanotic heart disease.

General complications in catheterization laboratory are:

- ❑ Hypercarbia: Due to upper airway obstruction and deep sedation.
- ❑ Acid-base imbalance, hypothermia, hypocalcaemia, hypoglycemia, especially in children
- ❑ Cardiovascular instability related to catheter manipulation, arrhythmia, perforation leading to tamponade, regurgitation following BMV, cardiac failure
- ❑ Vascular thrombosis, hematoma at the site of puncture
- ❑ Embolization of devices requiring prolonged anesthesia for retrieval
- ❑ Neurological complication due to embolization of clot, thrombus, infarction due to decreased blood supply to brain during temporary cessation of circulation in some of the procedures
- ❑ Contrast-related complications: Anaphylaxis; renal failure (contrast nephropathy CIN) which can be prevented by the use of nonionic dye; maintaining well hydration and limiting the amount of the dye upto 2.7 mL/kg body weight. Preprocedure administration of N-acetyl cystein (NAC) is followed at some centers but the results of renoprotective effects of NAC are inconclusive.

SUMMARY

Procedures in the catheterization laboratory allow poor access to the patient, are carried out in a not so illuminated environment and may lead to devastating complications like cardiac tamponade. Monitoring ABG, fluids, electrolyte and temperature, especially in children and keeping the patient immobile at the time of placement of coils, devices or ablation procedure is the mainstay of anesthesia management. All resuscitation equipment, emergency drugs, blood grouping and crossmatching and cardiac surgery standby should be confirmed before carrying out the cardiac catheterization procedures.

SUGGESTED READING

1. Albers DD, Lybrand FE 3rd, Axton JC, Wendelken JR. Shockwave lithotripsy and pacemakers: Experience with 20 cases. *J Endourol.* 1995;9:301-3.
2. Asroff SW, Kingston TE, Stein BS. Extracorporeal shock wave lithotripsy in patients with cardiac pacemaker in an abdominal location: Case report and review of the literature. *J Endourol.* 1993;7:189-92.
3. Atlee JL, Bernstein AD. Cardiac rhythm management devices (Part II). Perioperative management. *Anesthesiology.* 2001;95:1492-506.
4. Chien WW, Foster E, Phillips B, Schiller N, Griffin JC. Pacemaker syndrome in a patient with DDD pacemaker for long QT syndrome. *Pacing Clin Electrophysiol.* 1991;14:1209-12.
5. Dresner DL, Lebowitz PW. Atrioventricular sequential pacemaker inhibition by transurethral electrosurgery. *Anesthesiology.* 1988;68:599-601.
6. Erlebacher JA, Cahill PT, Pannizzo F, Knowles RJ. Effect of magnetic resonance imaging on DDD pacemakers. *Am J Cardiol.* 1986;57:437-440.
7. Hollinger I, Mittinacht A. Cardiac catheterization. Pediatric cardiac anesthesia, In: Lake CL, Brooker PD. 4th edition, Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 112-36.
8. Mitrani RD, Myerburg RJ, Castellanos A. Cardiac pacemakers. In: Fuster V, Alexander RW, O'Rourke RA (Eds), 10th edition. *Hurst's The Heart*, New York, McGraw-Hill. 2001;963-92.
9. Muller-Runkel R, Orsolini G, Kalokhe UP. Monitoring the radiation dose to a multiprogrammable pacemaker during radical radiation therapy: A case report. *Pacing Clin Electrophysiol.* 1990;13:1466-70.
10. Philbin DM, Marieb MA, Aithal KH, Schoenfeld MH. Inappropriate shocks delivered by an ICD as a result of sensed potentials from a transcutaneous electronic nerve stimulation unit. *Pacing Clin Electrophysiol.* 1998;21:2010-11.
11. Rastogi S, Goel S, Tempe DK, Virmani S. Patients with Pacemakers and Defibrillators *Annals of Cardiac Anaesthesia.* 2005;8:21-2.
12. Simon AB. Perioperative management of the pacemaker patient. *Anesthesiology.* 1977;46:127-31.
13. Souliman SK, Christie J. Pacemaker failure induced by radiotherapy. *Pacing Clin Electrophysiol.* 1994;17:270-73.

Chapter 12

Pregnancy and Cardiac Surgery

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GENERAL CONSIDERATIONS IN PREGNANT PATIENTS

- ❑ **Aspiration prophylaxis:** The aim is to achieve a gastric pH >2.5 and reduce the volume of gastric contents to < 25 mL. Appropriate antacids and prokinetic drugs should be given preoperatively.
- ❑ Pregnant patients should be considered full stomach. Hence rapid sequence induction with **Sellick's manœuvre** should be done.
- ❑ **Supine hypotension syndrome:** Pregnant patients (>20 weeks gestation) should be given left lateral tilt to prevent aortocaval compression due to gravid uterus.
- ❑ **Anticipated difficult airway:** Changes in pregnancy especially in the presence of preeclampsia can lead to facial and laryngeal edema, making the airway difficult.
- ❑ **Placental drug transfer:** Anesthetic agents readily cross placenta and can affect the fetus adversely. Feto-maternal ratio (F/M) indicates the proportion of drug that crosses the placenta. Drugs with a lower ratio are safer for the fetus. Table 1 depicts common drugs with their F/M ratio.

Table 1: Common drugs with their F/M ratio

Drug	F/M Ratio
Scoline	0
Atracurium	0.07
Vecuronium	0.06–0.1
Rocuronium	0.06
Thiopentone	0.4–1.1
Propofol	0.65–0.85 (bolus 2–2.5 mg/kg) 0.5–0.54 (@6–9 mg/kg/h)
Ketamine	1.26 (in 1.25 min)
Etomidate	0.5
Morphine	0.6
Fentanyl	0.37–0.57
Remifentanyl	0.88

Contd...

Contd...

Drug	F/M Ratio
Diazepam	1
Midazolam	0.76 (within 20 min level rapidly decreases)
Pethidine	0.6–1
Bupivacaine	0.3
Lignocaine	0.55
Ropivacaine	0.2
Halothane	0.71–0.87
Isoflurane	0.7
N ₂ O	0.83
Clonidine	0.89
Dexmedetomidine	0.12
Heparin	0
Atropine	0.93
Glycopyrrolate	0.22
Metoprolol	1
Esmolol	0.2
Labetalol	0.3
Ephedrine	0.7

BMV IN PREGNANCY

BMV is the procedure of choice in pregnancy in cases of severe to critical MS due to increased risk of surgery. It is usually carried out under local anesthesia with monitored anesthesia care (MAC) but sometimes general anesthesia may be required in uncooperative patients, and in case of hemodynamic catastrophe following the procedure.

Preoperative

Proper counseling of the patient helps during the perioperative period as the procedure is usually performed under local anesthesia and intravenous sedation. Ability of the patient to lie supine comfortably is to be noted.

Presence of pulmonary edema should be ruled out and the need for ventilatory support is assessed. Preoperative use of diuretics helps to drain out excess lung water.

Serum potassium levels should be optimized.

Rule out the presence of moderate to severe MR and LA thrombus in the preoperative assessment.

Ensure functioning defibrillator and the presence of surgical team standby prior to the procedure.

Adequate length of the ventilator tubings, intravenous extension lines, resuscitation equipment, and difficult airway cart needs to be available in the

catheterization laboratory. One needs to plan for emergency intubation and positive pressure ventilation in case of a catastrophe. Excessive fasting may lead to dehydration and puncture of femoral vein becomes difficult.

Intraoperative

Slight left lateral tilt by keeping wedge under right hip is advisable.

Placement of radioprotective shield both over and under the abdomen must be ensured.

Radiation exposure level in the C-arm machine must be switched to the *low mode*.

Critical steps during BMV are trans-septal puncture and balloon inflation across the stenotic valve. Cardiac tamponade can occur during trans-septal puncture. Temporary cessation of blood flow during balloon inflation can result in syncope, thromboembolism and vagal stimulation.

As pregnancy is a prothrombotic state, maintenance of adequate anticoagulation is important to avoid the risk of systemic embolization and stroke. Heparin should be repeated hourly in the dose of 20–30 U/kg after a bolus dose of 100 U/kg.

Possibility of local anesthetic toxicity due to a large dose and inadvertent intravenous entry during inguinal area infiltration should be kept in mind.

Excessive commisurotomy by the balloon may lead to severe mitral regurgitation and the resultant hemodynamic instability warrants general anesthesia with endotracheal intubation and positive pressure ventilation.

Postoperative

Simple analgesics like paracetamol/NSAIDs are usually sufficient. Patients can be mobilized after vascular sheath removal. Medications for heart rate control, and diuretics need to be continued.

OPEN HEART SURGERY IN PREGNANCY

Pregnant women undergoing cardiac surgery present a unique challenge because normal physiological changes during pregnancy pose an additional stress on the already compromised cardiovascular system due to preexisting cardiac disease. That is why pregnancy creates a high-risk situation in women with significant cardiac pathology. With proper counselling and improved management, however, increasing number of obstetric patients are being posted for cardiac surgery. Hence, apart from usual anesthetic implications of pregnancy, anesthesiologist should be aware of interactions between cardiopulmonary bypass and maternal-fetal circulation.

The incidence of heart disease in pregnant women varies from 1% to 4%, with rheumatic mitral valve disease being the most common. Also, recent data suggest similar maternal mortality rate (1.47%) to that associated with cardiopulmonary bypass in nonpregnant women, unless the surgery is emergent. However, fetal mortality remains at 16–33%.

NORMAL FINDINGS IN PREGNANT PATIENTS

Pregnant ECG: Right axis deviation and ST changes are normal due to hyperdynamic circulation. So ECG should be interpreted carefully.

Chest X-ray: Cardiomegaly is common due to enlargement of atria and great vessels. However, it should be interpreted same like nonpregnant women because the magnitude of radiographic changes is too small.

Echocardiography: LVH and increased chamber dimensions is a common finding along with mild regurgitative changes.

So, diagnosis of cardiac pathology needs to be done carefully in pregnant patients.

Radiation hazard: Cardiac catheterization may cause exposure up to 0.01–0.1 Gy depending upon fluoroscopy time. Thallium 210 myocardial perfusion scan results in exposure of less than 0.01 Gy. Termination of pregnancy is generally not recommended for less than 0.05 Gy radiation exposure.

TIMING OF CARDIAC SURGERY IN PREGNANCY

The best time to perform cardiac surgery during pregnancy is late second trimester/early third trimester (before 30 weeks) because fetus has already passed beyond the period of organogenesis and it can be salvaged if immediate delivery is required. Also, late third trimester and postpartum period put maximum stress on the heart which may cause cardiac decompensation. Data suggest maternal mortality of 6% as compared to 14% when surgery is performed in second trimester rather than in postpartum period. Also, cardiac surgery performed immediately after vaginal delivery is associated with 12% maternal mortality.

If cesarian section needs to be done simultaneously during cardiac surgery, then it should be done after heparinization but before cannulation and abdomen be kept open during open heart surgery to assess uterine bleeding.

CARDIOPULMONARY BYPASS AND PREGNANCY

Cardiopulmonary bypass (CPB) in a pregnant woman results in a greater fetal demise although maternal outcomes are similar to cardiac surgery in nonpregnant women. CPB is associated with uteroplacental hypoperfusion and sustained forceful uterine contractions reduce uterine blood flow and intervillous perfusion causing fetal hypoxia. Metabolic acidosis due to low fetal cardiac output, develops 6–8 h after CPB is discontinued and results in fetal death. Therefore, alterations in routine CPB need to be done to provide adequate oxygenation to mother and maintain uteroplacental perfusion.

Pump blood flow and perfusion pressure: High blood flow during CPB with high mean pressure (75–85 mm Hg) is associated with better maternal and fetal outcome and normal fetal heart rate tracing.

Pulsatile vs nonpulsatile flow: Concrete data in terms of uteroplacental flow and fetal outcome is lacking regarding superiority of pulsatile flow over continuous flow during CPB. But, it has been observed in animals that the pulsatile flow preserves placental flow and oxygen delivery to peripheral muscles is reduced with continuous flow. Pulsatile flow prevents the drop in placental perfusion and limits the rise in placental vascular resistance that is observed with the nonpulsatile flow. It preserves endothelial nitric oxide synthesis and decreases the activation of the fetal renin-angiotensin pathway, resulting in improved blood flow to the fetoplacental unit. Lactate levels during pulsatile-flow CPB have been shown to remain stable, whereas a continuous increase is observed with nonpulsatile CPB.

Nevertheless, in order to preserve placental blood flow and avoid hypotension-induced increased vascular resistance in placenta, high blood flow rate with high perfusion pressure is recommended in pregnant patients.

Temperature: Hypothermia during CPB increases uterine tone and reduces uterine blood flow. This may lead to fetal bradycardia. Also, autoregulation of fetal heart is impaired below 35°C. Hypothermia decreases oxygenation capacity of placenta too. But still, occasional cases have been reported in which both maternal as well as fetal outcome was good despite hypothermic circulatory arrest during aortic arch aneurysm repair. Normothermia poses problems related to inadequate myocardial protection due to myocardial rewarming which needs to be corrected by using either continuous cold irrigation method or continuous warm cardioplegia.

Cardioplegia: High potassium content in cardioplegia solution raises maternal serum-potassium levels which ultimately diffuse into fetal circulation resulting in fetal hyperkalemia and fetal cardiac arrest may ensue. One simple solution to this problem is to prevent return of cardioplegia solution into venous reservoir by applying external suction to coronary sinus through which cardioplegia solution drains. Nevertheless, serum potassium level should be maintained less than 5 mmol/L and needs vigilant monitoring.

Alpha stat vs pH stat: Alpha stat strategy is favored because better CO₂ homeostasis is maintained. One needs to closely monitor arterial blood gases especially CO₂ level because hypocapnia decreases uterine blood flow by causing uteroplacental vasoconstriction.

Aortocaval compression: Enlarged uterus causes aortocaval compression which reduces uteroplacental perfusion and so uterine displacement is advisable during surgery. This can be done by keeping a wedge under right hip or leftward tilting of OR table to 15°.

Hematocrit during CPB must be kept >25% in order to facilitate adequate oxygen delivery to the fetus.

In addition, optimizing maternal oxygen saturation and avoiding maternal hypoglycemia are important for preventing fetal bradycardia.

Administration of maternal corticosteroids to initiate endothelial membrane stability and maturation of the fetal lungs must be considered as this can substantially improve fetal outcome, should delivery occur after CPB.

FETAL HEART RATE (FHR) MONITORING

Hypotension early in the initial period of commencing CPB is most commonly associated with fetal bradycardia due to compromised uteroplacental perfusion resulting from sudden hemodilution. Hence, CPB needs to be commenced gradually under continuous fetal heart rate monitoring because bradycardia is the most frequent fetal response to CPB and is usually reversible by increasing the perfusion pressure. Hence, fetal heart rate monitoring enables early detection and management of alterations in fetal heart function. Fetal heart rate monitoring can be done intermittently for fetuses at <24 weeks of gestation but should be continuously done for fetuses at ≥ 24 weeks of gestation. Postoperative monitoring of the fetus for at least 24 hours is also recommended due to risk, of preterm labor.

Fetal heart monitoring is commonly done using cardiometry, fetal echocardiography or Doppler transducer and aimed toward maintaining fetal heart rate between 110 and 160 beats/min and accordingly adjust the pump flow rate, perfusion pressure and temperature.

UTERINE MONITORING

Uterine contractions occur frequently during rewarming phase of CPB and may occur even postoperatively. This may cause placental insufficiency resulting into fetal demise and therefore, uterine contraction monitoring by using **tocodynamometry** becomes essential. When the uterus is relaxed, a mean arterial pressure of ≥ 70 mm Hg is required for adequate placental perfusion, however when uterine contractions occur, higher blood pressure is needed to ensure adequate placental perfusion. Tocolytic therapy with β_2 -agonists, NTG, magnesium, and progesterone supplementation has been successfully used to relax uterus during CPB.

ANESTHETIC DRUGS

Hemodilution during CPB alters pharmacokinetics and pharmacodynamics of anesthetic agents. Hence, these drugs often require more frequent dosing, without a change in the total daily maternal dosage. All inhaled anesthetics and most intravenous anesthetics being highly lipid soluble cross placenta freely. Volatile anesthetics are also potent uterine relaxants. All opioids appear to be safe for use. Although the evidence is weak, it would be prudent to avoid benzodiazepines in early pregnancy as it is associated with increased incidence of cleft palate. However, it should be remembered that most adverse maternal and fetal outcomes from cardiac surgery during pregnancy are attributed to the effects of CPB and the underlying cardiac status of the mother, and not the anesthetic agent used.

SUMMARY

Preoperative optimization of maternal cardiovascular status, appropriate perioperative fetal and uterine monitoring, judicious use of CPB, short surgical time and perioperative consultation with obstetrician/perinatologist improve outcome of both mother as well as the fetus.

PREGNANCY AND MYOCARDIAL INFARCTION

The approximate incidence of myocardial infarction (MI) in pregnancy is estimated to be 1:10000. It usually occurs in the antepartum period in multiparous women and postpartum in nullipara. High mortality occurs in the third trimester and if delivery occurs within 2 weeks of MI. Exaggerated hypercoagulability in the pregnancy poses high risk of MI in susceptible patients. Advanced maternal age and multiparity may increase the risk of coronary dissection probably due to hormonal imbalance. Also, it is normal to have raised CK-MB enzyme levels during pregnancy and in the postpartum period. Hence, these enzyme tests may not be a specific diagnostic test for MI. Certain things need to be kept in mind while treating acute MI in pregnancy. Use of beta blockers may result in premature labor. There is a possibility of maternal and fetal hemorrhage with thrombolytic therapy. Antifibrinolytics can have adverse effects on placental implantation. Urokinase is preferred over streptokinase in view of low systemic lytic effect and less allergic reactions but its effective dose may be increased due to placental release of various inhibitors.

PREGNANCY AND HEART TRANSPLANT

Literature suggests that pregnancy is well tolerated after cardiac transplantation though with increased incidence of complications like hypertension, infection, preeclampsia, and preterm labor. However, the risk of rejection (immunologic) and infection needs to be considered in this class of patients. Denervated transplanted heart is preload dependent and responds to direct catecholamines. Immunologic rejection results in systolic and diastolic dysfunction of the heart which may not tolerate pregnancy. Nevertheless, successful maternal and fetal outcomes after pregnancy have been reported in cardiac transplant patients.

SUGGESTED READING

1. Arnoni RT, Arnoni AS, Bonini RC, de Almeida AF, Neto CA, Dinkhuysen JJ, et al. Risk factors associated with cardiac surgery during pregnancy. *Ann Thorac Surg*. 2003;76:1605-8.
2. Buffalo E, Palma JH, Gomes WJ, et al. Successful use of DHCA in pregnancy. *Ann Thorac Surg*. 1994;58:1532.

3. Crowther CA, Harding JE. Repeat doses of prenatal corticosteroids for women at risk of preterm birth for preventing neonatal respiratory disease. *Cochrane Database Syst Rev.* 2007;3:CD003935.
4. Ginz B. Myocardial infarction in pregnancy. *J Obstet Gynaecol Br Commonw.* 1970;77:610.
5. Kapoor MC. Cardiopulmonary bypass in pregnancy. *Ann Card Anaesth.* 2014;17(1).
6. Lin TY, Chiu KM, Shieh JS, Chu SH. Emergency redo mitral valve replacement in a pregnant woman at third trimester: Case report and literature review. *Circ J.* 2008;72:1715-7.
7. Mahli A, Izdes S, Coskun D. Cardiac operations during pregnancy: Review of factors influencing fetal outcome. *Ann Thorac Surg.* 2000;69:1622-6.
8. Martin SR, Foley MR. Intensive care in obstetrics: An evidence-based review. *Am J Obstet Gynecol.* 2006;195:673-89.
9. Parry AJ, Westaby S. Cardiopulmonary bypass during pregnancy. *Ann Thorac Surg.* 1996;61:1865-9.
10. Reddy VM, McElhinney DB, Rajasinghe HA, Liddicoat JR, Hendricks-Munoz K, Fineman JR, et al. Role of the endothelium in placental dysfunction after fetal cardiac bypass. *J Thorac Cardiovasc. Surg.* 1999;117:343-51.
11. Scott JR, Wagoner LE, Olsen SL, et al. Pregnancy in heart transplant patients: management and outcome. *Obstet Gynecol.* 1993;82:324.
12. Vedrinne C, Tronc F, Martinot S, Robin J, Allevard AM, Vincent M, et al. Better preservation of endothelial function and decreased activation of the fetal renin-angiotensin pathway with the use of pulsatile flow during experimental fetal bypass. *J Thorac Cardiovasc Surg.* 2000;120:770-7.
13. Werch A, Lambert HM, Cooley D, Reed CC. Fetal monitoring and maternal open heart surgery. *South Med J.* 1977;70:1024.

Section 3

Thoracic Anesthesia

Section Outline

- Anesthesia for Thoracic Surgery

Chapter 13

Anesthesia for Thoracic Surgery

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INTRODUCTION

Presently the indications for thoracic surgery are predominantly malignant lesions of the lung, esophagus and mediastinum as opposed to infectious lesions in the past. Also, patients with a greater degree of morbidity are now considered 'operable' due to innovative surgical techniques, and improved anaesthesia management. An 'operable' patient is one who can tolerate the proposed lung resection with acceptable risk.

PREOPERATIVE CONSIDERATIONS

Preoperative evaluation aims to identify patients at 'high risk' for lung resection. The concept of 'lung-sparing' surgery entails preservation of an amount of functioning lung parenchyma that can ensure adequate postoperative respiratory function.

Preoperative assessment will follow the protocol for a supra major surgery with special emphasis on medical comorbidities, allergies, medications and upper airway.

Respiratory complications like atelectasis, pneumonia and respiratory failure figure high on the list of perioperative complications (15–20%). No less than 10–15% of the thoracic surgical population is vulnerable to cardiac complications like ischemia and arrhythmias.

Assessment of Respiratory Function

The most sensitive indicator of respiratory function is a history of patients quality of life.

The triad of prethoracotomy respiratory assessment consists of lung mechanics, gas exchange and cardiopulmonary interaction.

A. Lung Mechanics

The predicted postoperative FEV in 1 sec (ppoFEV₁%) is the most sensitive indicator for post-thoracotomy respiratory complications. It is calculated as:

$$\text{ppoFEV}_1\% = \frac{\text{Preop FEV}_1\% \times (1 - \% \text{ functional lung tissue removed})}{100}$$

The right upper and middle lobes combined are measured as equivalent to each of the other three lobes; the right lung is 10% larger than the left lung.

Low risk	=	ppoFEV1 > 40% predicted
Moderate risk	=	ppoFEV1 = 30–40% predicted
High risk	=	ppoFEV1 < 30% predicted

B. Pulmonary Parenchymal Function

Values of $P_aO_2 < 60$ mm Hg or $P_aCO_2 > 45$ mm Hg have been considered as cutoff values for pulmonary resection. The diffusing capacity for CO (DLCO) correlates with the total functioning surface area of alveolar-capillary interface. A ppoDLCO < 40% indicates increased risk of respiratory and cardiac complications.

C. Cardiopulmonary Reserve

Stair climbing is a convenient test in ambulatory patients. The ability to climb three flights or more is associated with decreased mortality. The “gold standard” for evaluation of cardiopulmonary function is laboratory exercise test. A highly reliable exercise predictor of post-thoracotomy outcome is maximal oxygen consumption ($VO_2\text{max}$).

This can be calculated by dividing the distance walked in meters in 6 min (6MWT) by 30 (e.g. 600 m/30 = 20 mL/kg/min).

Low risk	=	$VO_2\text{max} > 20$ mL/kg/min
Moderate risk	=	$VO_2\text{max} = 15-20$ mL/kg/min
High risk	=	$VO_2\text{max} < 15$ mL/kg/min

D. Ventilation Perfusion Scan

It is highly useful in pneumonectomy patients. A patient who has ppoFEV₁ less than 40% should preferably undergo this evaluation.

E. Prediction of Extubation

Patients with ppoFEV₁ > 40% can usually be extubated in the OR, if they are alert, warm and comfortable. In patients with ppoFEV₁ 30–40%, the associated comorbidities will determine the timing of extubation. Patients with ppoFEV₁ 20–30% will usually require ventilatory support.

Medical Comorbidities

□ Age:

- The rate of respiratory complications, is nearly doubled after the age of 80 years. The rate of cardiac complications is even higher.
- Pneumonectomy in patients older than 70 years carries a high rate of mortality (specially right pneumonectomy).

□ **Cardiac disease:**

- Pulmonary resection is usually considered an intermediate risk procedure for perioperative ischemia. Specialized tests for cardiac function are indicated in patients with active cardiac conditions like unstable angina, recent infarction, heart failure and arrhythmias. An optimal gap of 4–6 weeks is advisable in a patient who has to undergo urgent lung resection for malignancy, after a myocardial infarction.
- Atrial fibrillation is commonly seen after pulmonary resection surgeries (10–15%). The incidence of arrhythmias is determined by the amount of lung tissue resected, age, intrapericardial dissection and blood loss.

□ **COPD:**

FEV₁ percent predicted is used to grade the severity of COPD.

Stage I — FEV₁ >50%

Stage II — FEV₁ 35–50%

Stage III — FEV₁ < 35%

Certain factors in COPD should be taken into account.

- Respiratory drive: Stage II or III COPD patients should have an ABG evaluation preoperatively as in these patients, supplemental oxygen can lead to an increased PaCO₂ due to a blunted respiratory drive as well as increased dead space.
- Nocturnal hypoxemia: COPD patients tend to desaturate during sleep due to the rapid and shallow breathing pattern during REM sleep.
- RV dysfunction: The incidence of cor pulmonale in adult COPD patients with FEV₁ < 1 L is 40%. This incidence rises to 70% with FEV₁ < 0.6 lit. Supplemental oxygen is indicated in all COPD patients with resting PaO₂ < 55 mm Hg. However, the aim should be to maintain PaO₂ at 60–65 mm Hg. Echocardiography to assess RV function is indicated for pneumonectomy patients with ppoFEV₁ < 40%. Elevated right heart pressures will contribute to high risk.
- Respiratory optimization: Preoperative assessment should identify atelectasis, bronchospasm, chest infection and pulmonary edema as these are all sequelae of COPD. Preoperative optimization includes abstinence from smoking, treatment of infection and bronchospasm, liquefaction of secretions and chest physiotherapy.

□ **4 “M” with malignancy**

The anesthesiologist should assess lung cancer patients for the 4 “M”, namely, mass effects, metabolic abnormalities, metastases and medications.

Immediate Preoperative Period

The patient should be explained the risks and benefits of various modalities of postoperative analgesia. If epidural analgesia is planned, coagulopathy and sepsis should be ruled out. Special precautions need to be taken if patient is receiving anticoagulation. Thoracic epidural analgesia definitely aids earlier extubation.

Premedication

Most of the drugs like bronchodilators, β blockers and antihypertensives are continued. Mild sedation is usually required before placement of invasive monitoring lines and catheters. An anti-sialogogue is used to aid fiberoptic bronchoscopy (FOB) for placement of DLT.

Airway Concerns

The anesthetist needs to evaluate the potential for difficult endobronchial intubation. The chest X-ray film is the most reliable predictor of difficult endobronchial intubation. Significant tracheal or bronchial compression and/or distortion can be detected.

Chest CT scan will help to visualize distal airway problems. It is imperative for the anesthetist to examine the chest image before placement of DLT or bronchial blocker.

Prediction of Desaturation During OLV

Certain factors can be used to predict desaturation during OLV, the most important being a low PaO_2 during two lung ventilation in the lateral position.

As the right lung is larger, a greater shunt is created when the right lung is collapsed. Hence, right-sided surgery is also a risk factor.

Patients with more severe airflow limitation on preoperative spirometry tend to have a better PaO_2 during OLV due to development of auto-PEEP. A high percentage of ventilation or perfusion to the operative lung on preoperative V-Q scan also predisposes to desaturation during OLV.

INTRAOPERATIVE MANAGEMENT

Lung Separation

This can be achieved by DLTs (Left or right), single lumen endobronchial tubes (EBTs) and bronchial blockers (BB). Currently available PVC DLTs have high volume low pressure tracheal and bronchial cuffs.

Indications for Lung Separation

Absolute indications

- ❑ Prevention and protection of noninvolved lung from soiling due to pus or blood from an infected or bleeding lung
- ❑ Unilateral lung surgeries like bronchopleural or bronchopleural-cutaneous fistula
- ❑ Unilateral bronchopulmonary lavage in patients with pulmonary alveolar proteinosis.

Relative indications

- ❑ Pulmonary resection, including lobectomy and pneumonectomy
- ❑ Video-assisted thoracoscopic surgery (VATS), including wedge resection, biopsy, and pleurodesis
- ❑ Mediastinal surgery
- ❑ Minimally invasive cardiac surgery
- ❑ Thoracic vascular surgery
- ❑ Thoracic spine surgery
- ❑ Esophageal surgery

Surgical lesions of the chest in the pediatric age group: The common indications for thoracic surgery are different in the pediatric population. A variety of congenital intrathoracic lesions requiring surgery may present in the neonatal period or in infancy

➤ **Neonates and infants**

- Congenital cystic lesions (Bronchogenic cysts, dermoid cysts, cystic adenomatoid malformations)
- Congenital lobar emphysema
- Pulmonary sequestrations
- Pulmonary AV fistulas
- Congenital diaphragmatic hernia
- Tracheoesophageal fistula
- PDA, coarctation of aorta

➤ **Childhood**

- Neoplasms: Primary or metastatic malignancy of lung, pleura or mediastinum
- Infectious diseases: Empyema, lung abscess
- Musculoskeletal deformities: Pectus excavatum, scoliosis

Selection of Airway Device

Right-sided pulmonary resection

A left DLT is the first preference as it has the highest margin of safety in positioning. The advantages include access to non-ventilated lung for suctioning, fiberoptic monitoring of position and application of C-PAP. Other options include single lumen EBT or Univent Tube (BB).

Left-sided pulmonary resection (not pneumonectomy)

The choice between left DLT and a BB is a difficult one. Obstruction of tracheal lumen by lateral tracheal wall can be a problem when a left DLT is used for left thoracotomy.

❑ **Left-sided pneumonectomy**

- A right DLT is the first preference. It allows palpation of left hilum during OLV without interference from a tube or blocker in the left mainstem bronchus. Most currently available designs of right DLTs include a ventilatory side slot in the distal bronchial lumen for right upper lobe

ventilation. The alternatives in this situation include left DLT or BB, but both these will require repositioning before the left main bronchus is clamped.

□ **Upper airway abnormalities**

In patients with upper airway abnormalities, if DLT insertion is difficult the following alternative techniques can be considered:

- Fiberoptic guided intubation with a DLT
- Secure the airway with an ETT and then use a “tube exchanger” to place DLT
- Use a BB
- Use an uncut single lumen tube as an EBT.

□ **Chest trauma**

- A variable degree of hemoptysis from alveolar hemorrhage is common in both open and closed chest trauma.
- Management with bronchoscopy and suction is adequate in most of these cases without a need for lung isolation. The mortality in these patients is related to other injuries rather than airway hemorrhage or air embolus. A few cases can benefit from lung isolation. However, resuscitation of the patient remains the top priority.

□ **Iatrogenic airway injury**

The incidence of iatrogenic injury is 0.5–2.0 per 1000 cases with a DLT. Prevention of iatrogenic injury requires:

- Meticulous examination of chest X-ray film or CT scan to identify difficult endobronchial intubations.
- Use of an appropriate size DLT. A size that is too large for the patient is likely to cause trauma; too small a size will make lung isolation difficult.

Guidelines for DLT size in adult

- Female height < 1.6 m (63 inch) : 35 Fr. (Possibly 32 Fr if < 1.5 m)
- Female > 1.6 m : 37 Fr.
- Male < 1.7 m (67 inch) : 39 F (Possibly 37 Fr if < 1.6 m)
- Males > 1.7 m : 41 Fr.

- Optimal depth of insertion of DLT:

This is related to the height of the patient. The average depth of insertion from the tip for a cuffed DLT is 29 cm in an adult and varies +/- 1 cm for each 10 cm of patient height above or below 170 cm.

- Avoidance of nitrous oxide as it can increase bronchial cuff volume.
- Inflation of bronchial cuff to a minimal volume required for lung isolation.
This volume is usually <3 cc.
- Inflation of the bronchial cuff for minimum possible time.

□ **Malpositioning and airway resistance**

- It is mandatory to verify the position of DLT with FOB before initiation of OLV because migration of DLT during patient positioning is quite common. After the start of OLV, malpositioning due to dislodgement is more common with a BB rather than a DLT.
- Airflow resistance is in an acceptable range with a DLT for short periods. The flow resistance from a 37 Fr DLT is less than that of 8 mm ETT but more than that of 9 mm ETT.

Lung Isolation Techniques in Children

In the 20th century, nearly all thoracic surgeries in children were performed by thoracotomy. In the majority of cases, anesthesiologists used to ventilate both lungs with conventional endotracheal tube and surgeons retracted operative lung to gain exposure. But, during past decade, the use of video-assisted thoracoscopic surgery (VATS) has dramatically increased in which single-lung ventilation is extremely desirable. VATS can also be performed using a retractor to displace lung tissue in the operative field while ventilating both lungs simultaneously.

Currently, lung isolation technique in infants and children includes the use of single lumen endotracheal tube, balloon-tipped bronchial blockers such as Fogarty embolectomy catheter and Arndt endobronchial blocker, double lumen endotracheal tubes, and Univent tubes. The choice of airway gadgets for OLV still remains limited in children.

The smallest size (DLT) available is 26 F, which can be used for children above 8 years old. Balloon-tipped bronchial blockers remain 'technique of choice' in pediatric patients, below 6 years. This is because Univent 3.5 uncuffed version tube (recommended for 6–8 years old) and double-lumen ETT (recommended for 8–10 years old) has diameters too big for the aforementioned age group. The smallest Univent tube available has 3.5 mm internal diameter and is used in children above 6 years of age. The smallest sized Arndt pediatric endobronchial blocker (5 Fr) is suitable for children more than 2 years as it requires at least 4.5-mm endotracheal tube for insertion.

The other option for OLV in infants and smaller children is Marraro Pediatric Bilumen Tube. This comprises two uncuffed tubes attached to each other, bronchial tube being longer than the tracheal tube. This tube has been reported to be effective for OLV in children up to 3 years.

Positioning

Most thoracic surgical procedures are performed in the lateral position. It is usual to induce anesthesia in the supine position followed by lateral positioning. However, in certain situations like bronchiectasis or hemoptysis involving a single lung, induction in the lateral position may become imperative till lung isolation is achieved.

It is important to ensure that all lines and monitors are secured and functioning optimally after positioning.

A slight movement of DLT or BB is practically unavoidable during positioning. Hence, the anesthetist must recheck the position of DLT, or BB and the adequacy of ventilation after final position.

Care in Lateral Position

It is important to take care of certain vulnerable areas which are likely to suffer injury in the lateral position. Areas to be protected include the dependent eye and ear, brachial plexus in both the arms, sciatic nerve in the lower leg, peroneal nerve in the upper leg and the cervical spine.

Changes in Ventilation and Perfusion

In the lateral position, ventilation of dependent lung increases approximately 10% during spontaneous ventilation. However, after anesthesia and paralysis the ventilation of the dependent lung decreases by 15%. The applied PEEP preferentially goes to the more compliant upper lung and hyperinflates it. The dependent lung is likely to develop atelectasis.

There is a reduction of approximately 10% in the blood flow to the non-dependent area due to gravity. Thus, lateral position usually leads to an increase in a pulmonary arteriovenous shunt from 5% to 10–15%.

Effect of Position and One Lung Ventilation in Children

The overall effect of lateral decubitus position on V/Q match is different in infants as compared to older children. Oxygenation is optimal when the patient is placed in the lateral decubitus position with the healthy lung placed dependent or 'down'. This is due to the hydrostatic gradient due to gravity that causes an increase in blood flow to the healthy dependent lung, and a decrease in blood flow to the diseased lung which is nondependent. **In infants with asymmetric lung disease, oxygenation improves with the healthy lung in the nondependent, "up" position. There are several reasons for this difference.** The soft, compressible rib cage offers little support to the lung. In the lateral position, therefore, the FRC approaches the residual volume, causing airway closure in the dependent lung. The small size of the infant's chest reduces the hydrostatic pressure difference between the dependent and the nondependent lung. As a consequence the increase in perfusion to the dependent lung is not as much as in adults. The higher diaphragm on the dependent lung side, in adults, has a mechanical advantage due to the abdominal hydrostatic pressure gradient. This is lower in infants. As a result, infants are vulnerable to hypoxemia in the lateral decubitus position.

Monitoring

Standard ASA monitoring is mandatory for all cases.

- ❑ **Oxygenation:** Intermittent ABG monitoring of PaO₂ is required even if pulse oximetry is used, PaO₂ is a more sensitive indicator of the risk of desaturation than SaO₂ on pulse oximeter. Hence, PaO₂ should be measured before OLV and 20 minutes after the start of OLV. The more rapid the fall in PaO₂ after onset of OLV, the greater is the risk of subsequent desaturation.
- ❑ **Capnometry:** During OLV, Pa-etCO₂ gradient increases due to increased perfusion of the lower lung and increased dead space of the upper lung. Initially all the minute ventilation is transferred to the lower lung with a subsequent fall in PetCO₂ of lower lung. Collapse and pulmonary vasoconstriction of the upper lung improve the fractional perfusion of the lower lung and PetCO₂ rises. Maldistribution of perfusion between upper

and lower lungs will manifest as severe or prolonged falls in PetCO_2 . Such falls may serve as indicators for subsequent desaturation.

- ❑ *Invasive BP monitoring (IBP monitoring)*: Thoracic surgical procedures involve surgical compression of the heart or great vessels, leading to transient and severe hypotension. Thus IBP monitoring is usually indicated for beat to beat estimation of BP as well as ABG sampling.
- ❑ *CVP Monitoring*: With the chest open intraoperatively, CVP readings are not accurate. Hence, CVP lines are usually reserved for major procedures like pneumonectomy. One option is to use right IJV with USG guidance to minimize risk of pneumothorax.
- ❑ *PA catheter*: Noninvasive cardiac output monitoring systems have reduced the need for PA catheters. The risk of complications with PA catheters restricts their use to specific circumstances.
- ❑ *Spirometry*: Measurements of inspiratory and expiratory pressures, volume and flow interactions help to detect adequacy of lung isolation as well as air leaks. Changes in pressure volume loops can indicate change in position of DLT.

Anesthesia Technique

Most centers use combined thoracic epidural and general anesthesia for thoracic surgical procedures.

Certain special points need emphasis:

- ❑ Pulmonary edema of the dependent lung can be easily caused by excessive IV fluids hence IV fluids should be given only for maintenance and to replace volume deficits. No fluid administration is required for 3rd space loss.
- ❑ Nitrous oxide: Oxygen mixtures are avoided as they can lead to atelectasis in the poorly ventilated lung regions. Nitrous oxide can also increase PA pressures and is not recommended for patients with pulmonary hypertension.
Air-oxygen mixtures are recommended during both two lung ventilation and OLV with sufficient FiO_2 to prevent hypoxia.
- ❑ Temperature: Heat loss from an open hemithorax can lead to hypothermia, especially in the pediatric and geriatric population. Methods to prevent hypothermia should be adopted.
- ❑ Bronchospasm: Presence of reactive airways in COPD patients and manipulation of airway with placement of DLT or BB predispose to bronchospasm. Measures to prevent bronchospasm include use of bronchodilators, ketamine or propofol during induction, and volatile anesthetics during maintenance. Drugs that release histamine should be avoided.
- ❑ Coronary artery disease: Ischemic heart disease is quite common in this population due to old age and smoking. It is important to adopt measures, including thoracic epidural analgesia that help to optimize the myocardial oxygen supply/demand ratio.

Management of OLV

Factors important for maintaining PaO₂ during OLV:

- ❑ Hypoxic pulmonary vasoconstriction (HPV): Hypoxemia is a stimulus for HPV which prevents V/Q mismatch by directing blood away from the nonventilated lung. Nitric oxide and cyclooxygenase synthesis inhibition are implicated in HPV. All volatile anesthetics inhibit HPV in a dose-dependent manner. Vasodilators like NTG will decrease the extent of HPV.
- ❑ Cardiac Output: Increase in cardiac output will aid the maintenance of adequate PaO₂ during OLV.
- ❑ Ventilation: Recommended tidal volume (TV) is 5–6 mL/kg. Optimal tidal volume should maintain peak airway pressure below 35 cm H₂O and plateau airway pressure below 25 cm H₂O. Low level of PEEP (5 cm H₂O) is useful in patients with restrictive lung disease. However, in condition of reduced elastic recoil (COPD), auto PEEP develops and applied PEEP may have unpredictable effects.
Respiratory rate should be titrated to maintain normocapnia. Pressure control mode may be preferred in the presence of blebs and bullae to prevent the risk of lung injury. At the start of OLV FiO₂ should be 0.8–1.0 which can be titrated later to maintain adequate saturation.
- ❑ Normal pH and PaCO₂ help to maintain optimal efficacy of HPV.

Management of Hypoxemia During OLV

It is important to have a sequential approach to the management of hypoxemia.

- ❑ Increase inspired oxygen concentration to 1.0
- ❑ Check position of DLT with FOB
- ❑ Maintain adequate cardiac output
- ❑ Apply PEEP to the ventilated lung
- ❑ Apply CPAP to the nonventilated lung
- ❑ Adopt intermittent two lung ventilation
- ❑ Restrict pulmonary blood flow to the non-ventilated lung. (e.g. clamp PA)
Resume two lung ventilation if possible in case of sudden and severe desaturation.
Besides these measures, elimination of vasodilators like nitroglycerin will also help.

Lobectomy

It is the most common resection to be done for lung malignancy. Lobectomy leads to transient dysfunction in the remaining lobes. Respiratory complications like pneumonia and collapse are major causes of mortality. Recovery of lung function after thoracotomy takes 6 weeks.

Pneumonectomy

It is indicated when lobectomy is not sufficient to remove the local lesion or when lymphnode secondaries are present. Pneumonia and collapse occur after pneumonectomy but the consequences are not as severe due to the absence of residual parenchymal dysfunction. However, mortality rate is higher for pneumonectomy as compared to lobectomy due to following complications :

- ❑ Post-pneumonectomy pulmonary edema: It occurs 2–3 days postoperatively due to increased pulmonary capillary permeability. It is not due to increased PA pressure and may be exacerbated by fluid overload. The mortality rate is very high (>50%)
- ❑ Atrial fibrillation: RV strain and increased sympathetic activity are perhaps the most important factors for the atrial fibrillation seen in a large percentage of postpneumonectomy patients. Atrial fibrillation definitely contributes to the mortality. It is not prevented by digoxin but may respond to diltiazem.
- ❑ Mechanical compression: Cardiac herniation through an incompletely closed pericardium presents with acute hypotension and is potentially fatal. Mediastinal shifts can compress great vessels or the airways.

Postoperative Analgesia

A variety of techniques including intercostals blocks, intrapleural infusions and epidural anesthesia have been described for postoperative analgesia. Of these, epidural block best facilitates intraoperative and postoperative analgesia in neonates and infants; an epidural catheter can be inserted through the caudal space and easily advanced to the thoracic level. In older children, a thoracic epidural catheter may be directly inserted at the mid thoracic level.

Use of thoracic epidural analgesia, offers several benefits.

- ❑ The ability to cough and clear secretions is maintained, reducing the chances of atelectasis
- ❑ Airway resistance is lowered
- ❑ Phrenic nerve function is maintained
- ❑ Coronary endothelial function is optimized with better myocardial perfusion.

Adequate postoperative analgesia is important to reduce the perioperative complications. The sensory afferents for pain arise from:

- ❑ The incision (intercostals nerves T4–T6)
- ❑ Chest drains (T7–T8)
- ❑ Mediastinal pleura (Vagus N)
- ❑ Central diaphragmatic pleura (Phrenic N)
- ❑ Ipsilateral shoulder (brachial plexus).

All these pain afferents cannot be blocked by a single analgesic technique. Hence a multimodal approach, using a combination of thoracic epidural analgesia with systemic opioids and NSAIDs is ideal.

SUGGESTED READING

1. Choudhry DK. Single-lung ventilation in pediatric anesthesia. *Anesthesiol Clin North America*. 2005;23(4):693-708.
2. Golianu B, Hammer GB. Pain management for pediatric thoracic surgery. *Curr Opin Anaesthesiol*. 2005;18(1):13-21.
3. Slinger P, Triolet W, Wilson J. Improving arterial oxygenation during one lung ventilation. *Anesthesiology*. 1988 68:291-5.
4. Slinger PD, Campos JH. Anesthesia for Thoracic Surgery. *Miller's Anesthesia*. In: Miller RD (Ed). 7th edition. Churchill Livingstone Elsevier, Philadelphia.
5. Slinger PD, Sullivan E. Anesthesia Management for Surgery of Lungs and Mediastinum. A practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravelee GP (Eds). 5th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia.

Section 4

Cardiac Patients for Noncardiac Surgery

Section Outline

- Anesthesia for Cardiac Patients for
Noncardiac Surgery

Chapter 14

Anesthesia for Cardiac Patients for Noncardiac Surgery

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MANAGEMENT OF PATIENTS WITH CONGENITAL HEART DISEASE FOR NONCARDIAC SURGERY

Madhulata Garasia, Priyanka Karnik

The incidence of congenital heart disease worldwide is 0.3 to 1.2% in live neonates. In India, the incidence is 6–8 per 1000 live births. Nearly 1,80,000 children are born with heart defects each year in India. Bicuspid aortic valve is the most common congenital defect occurring in about 1% of population. Ventricular septal defect is the most common congenital lesion followed by atrial septal defects. Tetralogy of Fallot is the most common cyanotic congenital heart disease accounting for 5–6% of CHDs.

The possible adverse events in a case of CHD are:

- ❑ Infective endocarditis
- ❑ Arrhythmias including complete heart block
- ❑ Systemic or pulmonary hypertension
- ❑ Thromboembolism
- ❑ Coagulopathy
- ❑ Sudden death.
- ❑ **Salient Features of Management**
 - Thorough history and physical examination with special emphasis on cardiovascular signs and symptoms, medications, and any cardiac intervention done. Investigations include ECG, 2D echo, cardiac catheterization to know pressure gradients, ABG, serum digoxin levels, serum electrolytes, etc. along with the routine investigations. The cardiovascular physiology after correction of complex defects may pose a challenge for the anesthesiologists, as evident in patients who undergo Fontan repair or cardiac transplantation.
 - Premedication decreases the likelihood of excitement and sympathetic stimulation that may predispose the child to various types of cardiovascular compromise that may eventually result in cyanosis or congestive heart failure. The dose may need to be decreased or given in a titrated manner especially in children with some degree

of compromised function. Midazolam and fentanyl are relatively cardiostable and can be used for this purpose. Hypoventilation should be prevented as the resultant hypoxia and hypercarbia may increase the pulmonary vascular resistance (PVR).

- Patients undergoing surgical and dental procedures or instrumentations involving mucosal surfaces or contaminated tissues commonly suffer from transient bacteremia. Those lesions associated with turbulent flow and vortex shedding, cyanotic heart disease and prosthetic valves have a higher propensity to develop infective endocarditis than septal defects. Hence prophylactic antibiotics should be given to all patients at risk of developing endocarditis.
- Routine standard of care monitoring should be done as in any other pediatric case with special attention to:
 - BP cuff: Previous Blalock-Taussig shunt or subclavian flap angioplasty, repair of coarctation of the aorta prohibits the use of the affected arm for accurate blood pressure determination. Cannulation at the dorsalis pedis or posterior tibial arteries is preferable to that of the radial arteries in patients awaiting definitive repair.
 - EtCO₂: In children with cyanotic heart disease, the PETCO₂ is not always constant, and it cannot be used during surgery to estimate reliably the PaCO₂ due to diminished pulmonary blood flow.
 - Precordial stethoscope, although not specific for cardiac patients is an important tool for clinical assessment intraoperatively.
 - Two pulse oximeters may become necessary (one on right upper and other on left lower extremity) to monitor preductal and postductal oxygen saturation, e.g. PDA with preductal coarctation, PDA with pulmonary hypertension.
 - Prolonged fasting should be avoided to prevent hypovolemia and hypotension during induction, as also hypoglycemia and hemoconcentration, especially in cyanotic heart disease.
 - Intravenous access should be preferably taken prior to surgery to maintain hydration and for antibiotic prophylaxis. Air filters should be used in intravenous tubings.
 - Care should be taken to avoid air bubbles while injecting through the IV line as this can lead to paradoxical embolization in case of right-to-left shunts.
- **General Considerations**
 - The possibility of a difficult airway due to narrow subglottis should be considered.
 - The risk of stroke is high when polycythemia is present along with right-to-left shunt.
 - When the hematocrit >65%, the increased viscosity leads to tissue hypoxia. Both SVR and PVR rise leading to an increased risk of stroke and cardiac ischemia. Hence, adequate hydration and a reduction in RBC mass is protective.
 - Use of diuretics can lead to hypochloremic hypokalemic metabolic alkalosis.

- ❑ **Specific Considerations for Left-to-right Shunt**
 - The common left-to-right shunts encountered are VSD, ASD and PDA.
 - The problems due to this shunt include pulmonary congestion due to increased pulmonary blood flow. Volume overload of right ventricle can lead to early failure. Also, progressive rise in PVR leads to pulmonary hypertension.
 - The onset of action of intravenous induction agents is delayed due to dilution in pulmonary circulation. However, the rate of onset of inhalational agents is not affected unless the cardiac output is reduced significantly.
 - Induction should be cautious as the degree of RV overload and failure may be underestimated.
 - Measures to reduce SVR and augment PVR are adopted to reduce the shunt.
 - SVR is reduced by the use of volatile agents, thiopentone, propofol, beta-blockers and systemic vasodilators.
 - PVR can be augmented by the use of PPV and PEEP.
- ❑ **Specific Considerations for Right-to-left Shunts**
 - The right-to-left shunts include tetralogy of Fallot, Eisenmenger's syndrome, Ebstein's anomaly, and tricuspid atresia.
 - As $PVR > SVR$, there is pulmonary oligemia. Cyanosis and hypoxemia result from the entry of deoxygenated blood into the systemic circulation. Pressure overload of the right ventricle can lead to failure in the long run.
 - Measures that reduce PVR and augment SVR are adopted to reduce the shunt. The lung volumes and oxygenation should be maintained. Hypoxia and hypercapnea should be strictly avoided. Low mean airway pressures are preferable. Nitric oxide and other pulmonary vasodilators can be used.
 - SVR can be augmented by the knee, chest or the squatting position, sympathetic stimulation, ketamine and vasoconstrictors.
 - Pulmonary blood flow can be improved by reducing the viscosity with the help of adequate hydration. PGE1 infusion to be continued while pain and crying should be avoided to maintain adequate pulmonary blood flow. Inhalation induction would be slower than intravenous induction due to reduced pulmonary blood flow.

INDUCTION

- ❑ Inhalational induction with sevoflurane followed by placement of intravenous access and administration of muscle relaxant.
- ❑ If IV access is present, either a potent opioid+amnesic agent+muscle relaxant combination (fentanyl + midazolam + vecuronium).
OR
Ketamine (2 mg/kg) in case of cyanotic heart disease due to its ability to increase the systemic vascular resistance.
OR
Etomidate (0.3 mg/kg) due to its cardiostability.

- Regional anesthesia has been demonstrated to be effective and relatively safe in patients with CHD. However, it should be avoided in severe valvular stenosis and critically ill patients. Caudal block can be given safely using lower dilution of local anesthetic and an adjuvant like clonidine. Penile block, ilioinguinal block, etc. can also be given. This reduces the requirement of anesthetic drugs, provides good analgesia, reduces nausea and vomiting and promotes faster postoperative recovery. Adjuvants like clonidine may help to attenuate the sympathetic outflow and stress response associated with surgery.
- During emergence from anesthesia, there is tachycardia and hypertension which can be detrimental in certain lesions like TOF, MS, etc. Removal of supraglottic airway device should be preferably done when the patient is relatively deep with spontaneous respiration rather than fully awake. Desflurane and even sevoflurane are useful agents due to rapid recovery profile. Extubation of endotracheal tube, however, is preferred when the patient has regained the protective reflexes.
- Postoperative care includes prevention of tachycardia, hypothermia, shivering, hypoxia, pain and any other factor that can induce a sympathetic response. Hypoventilation leading to hypoxia and hypercarbia should be avoided due to risk of increase in PVR, especially in patients with pulmonary hypertension and right-to-left shunts.

RECENT ADVANCES

Short-acting opioids like remifentanyl can be used as an infusion along with inhalational anesthetics as maintenance with minimal changes in heart rate, blood pressure or oxygen saturation for short duration procedures where endotracheal intubation may not be required. Laryngeal mask airway can also be used. The time to recovery after discontinuation of infusion is 2–4 minutes. Dexmedetomidine is also being increasingly used in pediatric population due to its sedative, analgesic and opioid sparing effect with less incidence of apnea. Single shot caudal or intrathecal morphine with or without local anaesthetic or placement of epidural catheter can be done to provide analgesia intra-as well as postoperatively. However, coagulation parameters should be within the normal limits prior to these procedures.

Recently detailed echocardiographic techniques have been used to evaluate ventricular function in response to anesthetics in patients with congenital heart disease. Myocardial performance index (MPI) has been validated as an accurate measurement for all ventricular configurations including single ventricle. The smaller the index, the better is the ventricular function.

$$\text{MPI} = \text{ICT} + \text{IRT}/\text{ET}$$

Where, ICT is isovolumetric contraction time

IRT is isovolumetric relaxation time and

ET is ejection time.

MANAGEMENT OF PATIENTS WITH ISCHEMIC HEART DISEASE FOR NONCARDIAC SURGERY

Sunil Gvalani, Manish Kela

The possible complications that should be anticipated are:

- ❑ Myocardial ischemia
- ❑ Arrhythmia: Ventricular tachycardia, ventricular fibrillation and heart block
- ❑ Pulmonary edema
- ❑ Heart failure
- ❑ Cardiac arrest.

PREOPERATIVE EVALUATION AND IDENTIFICATION OF RISK FACTORS

The major goal is to assess the risk of myocardial infarction (MI), heart failure (HF), or both, which are the most common causes of morbidity and mortality.

The Goldman Cardiac Risk Index attempts to quantify the risk of adverse perioperative cardiac events (Table 1). The index scores each of a range of various conditions including cardiac disease, age and the nature and urgency of the proposed surgery. The total score predicts the likelihood of complications and death. However, revised cardiac risk index is more commonly used nowadays.

Table 1: Goldman cardiac risk index

3rd heart sound / elevated JVP	11 points
MI within 6 months	10 points
Ventricular ectopic beats >5/min	7 points
Age >70 years	5 points
Emergency operation	4 points
Severe aortic stenosis	3 points
Poor medical condition	3 points
Abdominal or thoracic operation	3 points

Score	Incidence of death	Incidence of severe CVS complications
0–5	0.2%	0.7%
6–12	2%	5%
13–25	4%	17%
>25	56%	22%

INTRAOPERATIVE MANAGEMENT

Anesthetic goals:

- ❑ Maintain stable hemodynamics
- ❑ Avoid drug-induced myocardial depression
- ❑ Prevent myocardial ischemia by optimizing myocardial oxygen supply and demand

- ❑ Maintain normothermia
- ❑ Maintain sinus rhythm
- ❑ Avoid anemia
- ❑ Avoid hypoxia, hypercarbia and acidosis
- ❑ Maintain balance between myocardial oxygen demand and supply.

Factors that influence myocardial oxygen supply and demand:

Increased demand

- ❑ Sympathetic stimulation
- ❑ Tachycardia
- ❑ Hypertension
- ❑ Increased contractility
- ❑ Increased preload
- ❑ Increased afterload

Decreased supply

- ❑ Decreased coronary blood flow
- ❑ Tachycardia
- ❑ Diastolic hypotension
- ❑ Anemia (Decreased O₂ content)
- ❑ Hypoxemia

REGIONAL ANESTHESIA AND IHD

Advantages:

- ❑ Reduces stress response
- ❑ Reduces thromboembolic complications
- ❑ Avoids pulmonary complications of general anesthesia
- ❑ Provides excellent pain relief that can be continued in the postoperative period.

Disadvantages:

- ❑ Sudden uncontrolled hypotension, which can cause myocardial ischemia, may occur
- ❑ Catheter-related hematologic complications.

MONITORING

The degree of invasive monitoring required depends upon the complexity of the surgery and the severity of the ischemic heart disease.

1. ECG: Leads II, V₃-V₅ are most sensitive to detect ischemia. Myocardial ischemia is indicated by at least 1 mm ST segment horizontal or down sloping depression.
2. Pulmonary artery catheter: Ischemia is manifested by acute increases in pulmonary artery occlusion pressure (PCWP >18 mm Hg)
3. TEE: Development of new regional wall motion abnormality is the most sensitive method for detecting ischemia.

INDUCTION

Aim: To prevent persistent excessive changes in the heart rate and blood pressure. Maintain heart rate and blood pressure within 20% of baseline value.

Prevent excessive sympathetic stimulation during induction and laryngoscopy and intubation.

Methods to Prevent Sympathetic Response

The sympathetic effects of laryngoscopy can be blunted using:

- ❑ Opioids (fentanyl 2 µg/kg IV)
- ❑ Bolus of lidocaine (1.5 mg/kg IV), and/or spraying the vocal cords with lignocaine spray
- ❑ β-blockers (esmolol 0.5 mg/kg IV)
- ❑ Short duration laryngoscopy (less than 15 sec).

MAINTENANCE OF ANESTHESIA

Anesthesia is usually maintained with a combination of oxygen and medical air (50%:50%) with either inhalation agents or opioids depending upon the LV ejection fraction. Patients with poor LV function are better maintained on opioids whereas inhalation agents are better for those with good LV function.

Muscle relaxant: Vecuronium, cisatracurium and rocuronium are preferred as they have minimal or no effect on hemodynamics.

Advantages of inhalational anesthetic agents are:

- ❑ Reduction of myocardial O₂ requirement
- ❑ Ischemic preconditioning properties.

INTRAOPERATIVE ISCHEMIA

Treatment

- ❑ Maintain diastolic blood pressure to ensure coronary perfusion (mean BP > 65 mm Hg is acceptable)
- ❑ Increase FiO₂ to 100%
- ❑ Decrease myocardial oxygen demand by adequate pain control and/or beta-blockers
- ❑ Improve coronary blood flow by nitroglycerin/nicorandil (coronary vasodilators)
- ❑ Vasopressors to maintain blood pressure if hemodynamic instability occurs
- ❑ Consider aspirin.

POSTOPERATIVE MANAGEMENT

Most adverse cardiac events occur in first 48 hours. Therefore, intensive monitoring should be continued.

During the recovery period, factors which can cause ischemia include: tachycardia, pain, hypothermia, shivering, hypoxia, hypercarbia and anemia. These should be treated in the immediate postoperative period. Supplemental oxygen for 24 hours postoperatively is advisable. Adequate analgesia is vital. Ischemic events in the early postoperative period indicate long-term ischemia.

MANAGEMENT OF VALVULAR HEART DISEASE FOR NONCARDIAC SURGERY

Sunil Gvalani, Sunil Chaphane

Optimal management of patients with valvular heart disease for noncardiac surgery requires a meticulous understanding of hemodynamic alterations and measures to maintain an adequate cardiac output.

MITRAL STENOSIS

Introduction

- ❑ Preoperative evaluation aims to identify the severity of stenosis, presence of a prosthetic valve, and presence of complications like pulmonary hypertension, right heart failure and atrial fibrillation.
- ❑ Evaluation in the presence of prosthetic valve:
 - Assess prosthetic valve function by echocardiography and /or MRI and/or cardiac catheterization study
 - The complications that can be anticipated with a prosthetic valve are mechanical dysfunction, paravalvular leak, valve thrombosis (choked valve), systemic embolization and endocarditis
 - Patients with prosthetic valve will be on warfarin anticoagulation. Conversion to heparin is required for major surgery. The risk of arterial embolization is high during pregnancy, thus anticoagulation therapy is vital. Since warfarin is associated with fetal defects and fetal death during first trimester, warfarin is discontinued during the first trimester and prior to delivery
 - Prevention of endocarditis is vital in the presence of a prosthetic valve.
- ❑ Mitral stenosis (MS) in pregnancy

The common complications of MS in pregnancy are pulmonary edema, atrial fibrillation, and thromboembolic phenomena. It is usually diagnosed in the antenatal period. To recapitulate the hemodynamic goals in MS:

 - Rate: The crucial step is LV filling. Tachycardia reduces the diastolic time and LV filling.
The optimal rate is 60–80 beats per minute. Bradycardia also can reduce the cardiac output.
 - Rhythm: Atrial fibrillation negates the atrial pump mechanism which is important for LV filling. Hence whenever possible, AF should be aggressively corrected before induction.
 - Preload: Fluid overload can lead to pulmonary edema. However, adequate LV diastolic filling is essential, hence precise CVP monitoring is required.
 - Contractility: LV contractility may be impaired in 25% of cases.
 - Afterload: Reduction in SVR will lead to severe hypotension as stroke volume cannot increase. Hence, SVR should be maintained. Due to

RV dysfunction, PVR should be lowered. Hypoxemia and hypercarbia can worsen pulmonary HT and precipitate right heart failure.

Pregnancy aggravates the symptoms of mitral stenosis due to certain significant changes:

- Intravascular volume increases by 40–50%.
- HR rises by 20–30%
- SVR reduces
- Gravid uterus reduces the venous return and decreases preload.

Cardiac decompensation during pregnancy is likely to occur in late third trimester, and second stage of labor due to sympathetic stimulation and autotransfusion of blood.

The main predictors of adverse cardiac events are:

- ❑ Prior cardiac events like failure, TIA, stroke and arrhythmias
- ❑ LVEF <40%
- ❑ NYHA class II or higher.

Labor analgesia is important to prevent decompensation. It helps by preventing tachycardia secondary to labor pain. This tachycardia can increase flow across the mitral valve and the sudden rise in LAP can result in acute pulmonary edema.

Preoperative Period

Echocardiography helps to estimate the valve area, detect the presence of thrombus in LA and calculate Wilkin score.

Preoperative optimization includes the use of diuretics and beta-blockers. Anticoagulation is often required as pregnancy is a hypercoagulable state.

BMV is done in the second trimester of pregnancy without the risk of teratogenesis. It is the procedure of choice in pregnancy in cases of severe to critical MS due to increased risk of surgery. It is usually done under local anesthesia with appropriate protection from radiation. Adequate anticoagulation must be maintained as pregnancy is a prothrombotic state. Surgical intervention should be reserved for those patients who have symptoms refractory to medical therapy in whom valvuloplasty is contraindicated.

Intraoperative Management of LSCS

Choice of Anesthesia Technique

Epidural anesthesia should be titrated carefully with incremental doses. Sudden hypotension can occur which can be difficult to treat. However, regional anesthesia reduces the incidence of thromboembolic events.

GA is usually chosen as it provides better hemodynamic stability. It can be rapidly administered in fetal distress. High FiO₂ reduces PVR and controlled ventilation prevents hypercarbia. The pressor response to laryngoscopy and intubation needs to be attenuated. Positive pressure ventilation can reduce venous return.

Drugs to control atrial fibrillation should be continued. Patients on warfarin should be switched to heparin.

Combination of drugs is chosen to prevent tachycardia and maintain SVR. Short acting beta-blockers and opioids can be used to blunt the pressor response. Invasive monitors may be required to assess PA pressure, intravascular fluid status, ventilation and oxygenation. Hypercarbia, hypoxemia and acidosis should be treated to prevent pulmonary hypertension. In case of RV failure, inotropic support of RV and pulmonary vasodilation will be necessary.

As oxytocin reduces SVR and increases PVR, it should be used carefully. Methylergometrine and PGF-2-alpha produce severe hypertension, tachycardia and increase in PVR.

Postoperative Period

It is important to prevent pain and hypoventilation as it may lead to respiratory acidosis and hypoxemia. This in turn can lead to a rise in heart rate and PVR. Ventilatory support may be required due to reduced pulmonary compliance and increased work of breathing.

MITRAL REGURGITATION

A. Introduction

Mitral regurgitation (MR) of rheumatic origin is usually associated with MS. Severe acute MR can occur during BMV. It can also be associated with ischemic heart disease.

The crucial step is a reduction in the forward left ventricular stroke volume and cardiac output. The regurgitant fraction leads to left atrial volume overload and pulmonary congestion.

The hemodynamic goals to maintain a normal cardiac output are:

- Rate: Bradycardia should be avoided. A normal to high rate is preferable.
- Rhythm: Atrial fibrillation will reduce the LV filling and should be treated
- Preload: Fluid overload is poorly tolerated and sudden acute MR presents as pulmonary edema. However, hypovolemia should be avoided
- Contractility: Drugs that depress the myocardium should be avoided as left ventricular contractility is vital to maintain normal cardiac output
- Afterload: An increase in SVR will aggravate the regurgitant fraction and should be avoided.

B. Preoperative

Clinically, MR is characterized by a holosystolic apical murmur radiating to the axilla. ECG will show LA and LV hypertrophy. Echocardiography will confirm the presence of MR, LV ejection fraction, LA size, LV dimensions and pulmonary artery pressure. The presence of a thrombus can also be detected.

Medical management involves the use of vasodilators, ACE inhibitors, beta-blockers and biventricular pacing. Patients with severe MR and compromised LV function may require mitral valve surgery before elective noncardiac surgery

C. Intraoperative

The aim of anesthesia technique will be to achieve the hemodynamic goals mentioned earlier. Regional anesthesia can be safely used as it reduces SVR and preserves forward flow.

General Anesthesia

Induction: IV induction is acceptable. Selection of drugs should be to prevent bradycardia or an increase in afterload.

Maintenance and Monitoring

Volatile anesthetics help to reduce the afterload and can be safely used. Opioids are acceptable as long as bradycardia is prevented. Intravascular fluid volume should be strictly maintained in the normal range.

Severe MR demands use of invasive monitoring. This will help to estimate the adequacy of cardiac output, monitor fluid replacement and gauge the response to anesthetic drugs.

MITRAL VALVE PROLAPSE

A. Introduction

MVP is defined as prolapse of one or both mitral leaflets into the left atrium during systole with or without mitral regurgitation.

It is more common in young women and can be associated with Marfan's syndrome, thyrotoxicosis, rheumatic fever and SLE.

Complications: MVP can lead to:

- Infective endocarditis
- Severe MR
- Cerebral embolic events
- Arrhythmias
- Sudden death.

B. Preoperative period

- The diagnosis is conclusively established by echocardiogram
- Betablockers to control arrhythmias should be continued. Patients with AF and/or LA thrombus are likely to be on anticoagulants.

C. Intraoperative period

The basic principles of management are the same as for MR. However, a smaller left ventricle leads to a greater degree of prolapse. Thus, it is important to avoid perioperative events leading to left ventricular emptying like:

- Upright posture
- Decreased SVR

- Increased sympathetic activity with a corresponding rise in myocardial contractility.

Conversely, vasoconstriction, myocardial depression and volume resuscitation will increase LV volume and reduce the degree of prolapse.

Both general and regional anesthesia can be safely used. However, the reduction in SVR due to regional anesthesia should be prevented by adequate volume loading.

For GA, a combination of drugs to reduce sympathetic stimulation should be selected. Adequate anesthetic depth and analgesia are important. Ventricular arrhythmias are more likely to occur in sitting position due to augmented LV emptying. These arrhythmias usually respond to lidocaine and beta-blockers.

AORTIC STENOSIS

A. Introduction

Several studies have demonstrated that patients with aortic stenosis have increased risk of perioperative mortality. Myocardial oxygen demand rises due to concentric LVH and increased myocardial work. On the other hand, myocardial oxygen delivery reduces due to compression of subendocardial vessels by increased LV pressure. Both these factors result in angina in spite of normal coronary function.

B. Preoperative period

These patients typically have angina, syncope and exertional dyspnea. Syncope is probably due to exercise-induced decrease in SVR which is not compensated due to fixed output. Exertional dyspnea indicates CHF, which can be due to systolic and/or diastolic dysfunction.

Echocardiography will assess the severity of stenosis (aortic valve area), measure transvalvular pressure gradients and detect LVH with systolic and diastolic dysfunction. Some patients may require cardiac catheterization study and coronary angiography.

Severely symptomatic patients may require percutaneous balloon valvulotomy or aortic replacement for an elective noncardiac surgery.

C. Intraoperative period

The hemodynamic goals are-

- Rate: An optimal heart rate of 60–80/min should be maintained. Tachycardia will reduce LV filling and also reduce the time available for coronary perfusion. Bradycardia will cause overdistention of LV.
- Rhythm: LV end diastolic volume significantly depends on the atrial pump mechanism. Hence, correction of AF which can produce a fall in stroke volume is vital.
- Preload: A normal intravascular volume should be maintained. Hypovolemia will reduce venous return and LV filling.
- Contractility: It is prudent to avoid myocardial depression in presence of concentric LVH.
- Afterload: Sudden fall in SVR can cause hypotension.

GA is usually preferred: Sympatholysis due to regional anesthesia can lead to severe hypotension which may be difficult to treat. For GA, a combination of drugs that maintain SVR and myocardial depression should be selected. Beta-blockers, lignocaine and defibrillator should be available to treat supraventricular tachycardia and ventricular arrhythmias. Monitoring should be planned to detect arrhythmias and myocardial ischemia.

AORTIC REGURGITATION

A. Introduction

AR commonly results from rheumatic fever, infective endocarditis, bicuspid aortic valve, aortic dissection and Marfan's syndrome.

The decrease in cardiac output is a result of regurgitation from the aorta back in the left ventricle during diastole. LV is subjected to pressure and volume overload. Angina may occur due to rise in oxygen requirement of hypertrophic LV and the reduction of coronary blood flow due to reduced aortic diastolic pressure. Acute AR due to aortic dissection or endocarditis results in coronary ischemia and heart failure.

B. Preoperative period

Echocardiography is used to detect the presence and grade the severity of AR. Anatomic abnormality of aortic valve can also be detected. Medical therapy usually involves a combination of a vasodilator and inotrope to improve forward LV stroke volume. The need for surgical replacement of a diseased valve will be decided by the degree of LV dysfunction.

C. Intraoperative period

The hemodynamic goals are:

- Rate: Heart rate should be maintained above 80 bpm since the degree of regurgitation increases with longer diastolic time.
- Rhythm: Normal sinus rhythm is optimal.
- Preload: Hypervolemia should be avoided in the presence of LV volume overload
- Contractility: Myocardial depression should be avoided.
- Afterload: Any increase in SVR will lead to a corresponding rise in the regurgitant fraction.

GA is usually preferred. A combination of drugs to prevent bradycardia or an increased afterload should be selected. Monitoring should be aimed at detecting arrhythmias and myocardial ischemia. Severe AR may necessitate monitoring with a PA catheter or TEE. This will help to assess myocardial dysfunction, evaluate the response to vasodilators and guide fluid replacement.

SUGGESTED READING

1. Frankville D. Anesthesia for noncardiac surgery in children and adults with congenital heart disease, pediatric cardiac anesthesia. In: Lake CL, Brooker PD (Eds). 4th edition. Wolter Kluwer/Lippincott Williams and Wilkins, Philadelphia; 601-32.
2. Herrera A. Valvular heart disease. Stoelting's anaesthesia and coexisting disease. In: Hines RL, Marschall KE (Eds), 5th edition. Saunders Elsevier; 27-40.

Section 5

Specialty-based Management of a Cardiac Patient

Section Outline

- Cardiovascular Concerns in Neurosurgical Procedures
- Cardiovascular Concerns in Urosurgical Procedures
- Cardiovascular Concerns in Major Orthopedic Surgeries
- Cardiovascular Concerns in Plastic Surgeries
- Cardiac Disease and Liver Surgery
- Cardiovascular Concerns During Liver Transplantation
- Cardiovascular Concerns in General and Emergency Abdominal Surgery

Chapter 15

Cardiovascular Concerns in Neurosurgical Procedures

Anita Shetty, Shrikanta Oak, Viraj Shah, Swarada Pardeshi

Management of high intracranial pressure (ICP), vasospasm after subarachnoid hemorrhage (SAH), neurogenic shock, methods used for prevention of cerebral injury and unusual positioning can aggravate cardiovascular instability in patients with coexisting cardiac disease. In addition, interpretation of ECG in presence of intracranial pathology, perioperative complications due to neurosurgical procedures will be discussed.

CARDIOVASCULAR CONCERNS DURING MANAGEMENT OF RAISED INTRACRANIAL PRESSURE

It is important to maintain normal intracranial pressure for minimizing ischemic injury and facilitating surgical exposure. Brain relaxation can be achieved with use of diuretics like mannitol, furosemide, hyperventilation, glucocorticoids, barbiturates and ventriculostomy.

1. **Diuretics:** Mannitol reduces ICP by creating an osmotic gradient across the blood-brain barrier and dehydrating the brain. It is advocated that mannitol (0.25–1.0 g/kg) be given over 30 minutes. Rudehill et al observed that this rate of administration increases serum osmolarity from 292 to 310 mOsm/L. This change was associated with a significant increase in cardiac output (CO), stroke volume, and pulmonary capillary wedge pressure (PCWP), and a decrease in hematocrit. This increase in CO returned to basal levels by 30 minutes after the infusion of mannitol; however, the increase in PCWP did not return to basal levels for 3 hours. These effects can precipitate left ventricular failure in patients with decreased cardiac reserve. Mannitol may also reduce preload. In patients, who are dependent on high preload, e.g. valvular heart disease may require meticulous monitoring to assess preload with the help of pulmonary artery catheter or transesophageal echocardiography. Mannitol can cause electrolyte imbalance, which decreases threshold for arrhythmia. Diuresis can cause hypotension due to volume contraction, leading to myocardial or cerebral ischemia. Mannitol and furosemide in combination may aggravate hypovolemia and electrolyte imbalance.

2. Hyperventilation: In Ischemic heart disease, hypocapnia due to hyperventilation can precipitate coronary vasospasm.
3. Glucocorticoids: Glucocorticoids are used to reduce tumor-induced vasogenic edema. Continuous use of glucocorticoids can cause hypokalemia and hyponatremia, which may precipitate cardiac arrhythmias.

CARDIOVASCULAR CONCERNS DURING PREVENTION OF CEREBRAL INJURY

Cerebral protective measures like drug-induced metabolic suppression or other techniques employed can affect cardiovascular system.

Metabolic Suppression

Drugs used for metabolic suppression are propofol, barbiturate and etomidate.

Thiopentone sodium is the most commonly used drug for brain relaxation and cerebral protection. It causes hypotension with tachycardia, which can be detrimental in patients with coronary artery disease. In patients with cardiac disease requiring high preload or those with impaired ventricular function, thiopentone should be used cautiously.

Etomidate, in comparison with thiopental, produces less hypotension. Etomidate, also decreases cerebral metabolic rate for oxygen (CMRO₂). Tyler-Frizzell et al. found that induction dose of 0.2 to 0.3 mg/kg of etomidate given to patients with cardiac disease undergoing noncardiac surgery produced no change in measured or derived cardiovascular parameters. It is a safer CMR depressant and putative cerebral protective agent in the patient with cardiac disease.

Propofol is also cerebral protective agent that depresses CMR and cerebral blood flow (CBF) at burst-suppressive doses. In equipotent doses, propofol produces less cardiovascular effects than thiopental.

Hypothermia

Mild hypothermia (in the range of 34°C) is one of the modalities for prevention of cerebral injury. Hypothermia increases systemic vascular resistance, blood viscosity, hematocrit and afterload. Hypothermia decreases left ventricular contractility and cardiac output and impairs diastolic relaxation. Hypothermia shifts potassium in the intracellular compartment and increases catecholamine release, which may result in arrhythmias, hypertension, and myocardial ischemia.

When hypothermia is exercised in patient with coronary artery disease, extreme caution is advised. Deeper plane of anesthesia and prevention of shivering is important in patients with coexisting cardiac disease. Shivering causes increase in CO₂ production and oxygen consumption predisposing patients to myocardial ischemia and arrhythmias.

CARDIOVASCULAR CONCERNS IN INTRACRANIAL PATHOLOGY

Intracranial pathology such as subarachnoid hemorrhage, colloid cyst of third ventricle, Cushing's syndrome and acromegaly affect the cardiovascular system.

Subarachnoid Hemorrhage

ECG changes in patients with subarachnoid hemorrhages are due to sympathetic outflow from lateral and posterior hypothalamic regions. *T-wave inversion* is a common finding after SAH and should not delay the treatment due to the unnecessary fear of cardiac problem. The circulating catecholamines result in hypertension, tachycardia, dysrhythmias, myocardial ischemia and pulmonary edema.

Excessive norepinephrine release from myocardial sympathetic nerve terminal is seen, independent of plasma catecholamine levels, which lead to neurogenic stunned myocardium (NSM).

Alpha-blockers prevent peripheral and cerebral arterial spasm. Beta-blockers are used to treat ventricular arrhythmias and prevent formation of microscopic cardiac lesions due to hyperstimulation.

These patients have propensity to develop pulmonary edema. Morphine as an analgesic can increase pulmonary vessel capacitance and prevent extravascular fluid shift.

Cerebral vasospasm: In patients who survive a subarachnoid hemorrhage, cerebral vasospasm leading to ischemia or infarction is common cause of morbidity and mortality.

Nimodipine: It appears to act predominantly upon the cerebral vasculature, so significant systemic hypotension is not associated with its use. It is a mild myocardial depressant. In patients with decreased cardiac reserve, nimodipine at clinically relevant doses (60 mg) can administered safely. It is unlikely to cause congestive cardiac failure in patients with coexisting cardiac disease, due to its mild myocardial depressant action. Exacerbation of angina is rare, in patients with ischemic heart disease, with its use, due to its anti-ischemic properties.

Triple H therapy: Hypervolemia, hemodilution and arterial hypertension is most effective regimen for prevention and treatment of ischemic neurologic deficit due to cerebral vasospasm. After subarachnoid hemorrhage (SAH), autoregulation of cerebral perfusion is hampered and cerebral blood flow depends on cerebral perfusion pressure, mean arterial pressure and intravascular volume. But, at the same time, this intervention may not be tolerated well in patients with limited cardiac reserve.

Colloid Cyst of Third Ventricle

Hypothalamic structures are located close to the walls of the third ventricle. Catecholamines release due to prolonged bilateral hypothalamic nuclei stimulation can increase cardiac preload, afterload and contractility leading

to neurogenic cardiac stunning and death. This explains sudden death in patients with colloid cysts when signs and symptoms of brain herniation or hydrocephalus are absent.

Acromegaly

In acromegaly, IGF-1 can cause cardiomyopathy, abnormalities of cardiac rhythm and valves.

In the initial stages, there is increased heart rate and systolic output which leads to concentric biventricular hypertrophy and diastolic dysfunction. If left untreated, it can lead to heart failure. Once treatment is initiated to suppress GH/IGF-1, left ventricular mass can decrease and cardiac function can also improve. Careful preoperative evaluation of cardiac function is mandatory in these patients.

Cushings Syndrome

It is associated with cardiovascular complications such as premature atherosclerosis, coronary artery disease and congestive heart failure, which mainly occur due to metabolic complications leading to cardiac structural and functional changes.

Structural changes include increased LV mass index and relative wall thickness causing LV diastolic dysfunction. These changes are reversible upon normalization of corticosteroid excess.

These patients have a sympathovagal imbalance characterized by increased parasympathetic activity which can lead to silent arrhythmias.

Evaluation and management of these patients should be focused on identifying not only cardiovascular risk but also other associated risk factors like obesity, glucose intolerance, dyslipidemia, insulin resistance and prothrombotic state.

Important features that strongly suggest neurogenic causes of cardiovascular dysfunction are:

- ❑ No history suggestive of pre-existing cardiac disorder
- ❑ Isolated ECG changes
- ❑ Temporal relation between cardiac dysfunction and intracranial pathology
- ❑ Regional wall motion abnormalities not corresponding to vascular territories
- ❑ Inconsistence between the findings of 2D-Echo and ECG
- ❑ Spontaneous and early resolution of abnormalities.

CARDIAC EMERGENCIES DURING NEUROSURGICAL PROCEDURES

In neurosurgical patients, position and procedure have important effects on the occurrence and management of cardiovascular complications. Intra-operatively, cardiac events such as bradycardia, hypotension, hypertension and arrhythmias are common and usually transient in nature. However, more

severe events such as ventricular fibrillation, pulseless electrical activity and asystole do occur in some patients.

The incidence of intraoperative cardiac arrest is about 1.1 to 7.2 per 10,000 anesthetics. Its management during neurosurgical procedures is not well defined and is further complicated due to patient positioning and intracranial pathology.

Appropriate management of such events in neurosurgical patients requires immediate attention to the underlying cause rather than routine application of Advanced cardiac life support (ACLS) algorithms.

Supratentorial Surgery

- ❑ Most common cause of hemodynamic disturbance in patients undergoing supratentorial surgeries is trigeminocardiac reflex (TCR). It occurs due to stimulation of sensory branches of trigeminal nerve causing bradycardia, hypotension, apnea and asystole. The trigeminal nerve forms the afferent limb and vagus nerve carries the efferent fibers. Stimulation of the vagus nerve can lead to modulation of ACh receptors on the epicardium causing coronary artery spasm (CAS). Atropine administration or local anesthetic infiltration may help if there is repeated TCR-related disturbances. IV adrenaline might be required in refractory episodes. Cardiac arrest during elective supratentorial surgery has been attributed to the stimulation of insular cortex, limbic and amygdala.
- ❑ Sudden increase in intracranial pressure due to anesthetic causes (coughing, gagging, light plane of anesthesia, hypercarbia and hypoxia) or surgical causes (bleeding, seizures or aneurysm rupture) can lead to severe hemodynamic compromise via Cushing's reflex.
- ❑ Ventriculoperitoneal shunt insertion and extradural drain placement can lead to intracranial hypotension. This predisposes to severe cardiac disturbances due to either rapid drainage of CSF or suction application to the drains causing reverse herniation of the brain.
- ❑ During endoscopic ventriculostomy, irrigation and manipulation of the floor of the third ventricle can stimulate the midbrain cardiovascular centers and lead to bradycardia and arrhythmias.

Skull-base Surgery

Complications such as severe bradycardia and asystole have been reported in transsphenoidal pituitary surgeries and are attributed to the TCR.

Anterior hypothalamic stimulation can also lead to cardiac arrest due to parasympathetic stimulation.

Posterior Fossa Surgery

Posterior fossa contains vital structures like the brainstem, floor of fourth ventricle and cranial nerves. Even minimal manipulation in this area can lead to variety of cardiac complications. Cardiac arrest can be due to trigemino-

cardiac reflex, glossopharyngeal vagal reflex (GVR) or venous air embolism. The reflex arc of GVR is formed by the glossopharyngeal nerve carrying afferent fibers and the vagus nerve carrying efferent. It stimulates the carotid sinus causing bradycardia, hypotension and syncope. Brainstem manipulation leads to arrhythmia, hypotension/hypertension and tachycardia/bradycardia.

NEUROPHARMACOLOGICAL INTERVENTIONS AND CARDIOVASCULAR CONCERNS

Dexmedetomidine, remifentanyl and phenytoin are commonly used in patients for neurosurgery. Overdose or rapid infusion of these drugs can produce bradycardia, hypotension or even cardiac arrest during elective procedures. Careful cardiac monitoring is essential during and after infusion of these drugs.

Dexmedetomidine: Bradycardia and hypotension may precipitate LV failure in patients with compromised LV function.

Phenytoin: It acts by altering sodium, potassium and calcium conductance, which often leads to hypotension if given rapidly.

MANAGEMENT OF BRADYCARDIA AND ASYSTOLE DURING NEUROSURGERY

If episodes of bradycardia occur during neurosurgical procedure, then it is important to rule out reflex mediated causes due to surgical stimulation and the stimulus need to be withdrawn immediately. If bradycardia persists, IV atropine should be administered. Procedure can be resumed if the heart rate returns to normal. Causes such as venous air embolism, brainstem injury and hypothalamic injury are to be considered in refractory cases and CPR to be initiated.

Considerations for CPR in neurosurgical patients with skull pin fixation and non-supine position:

- ❑ Change to supine position as soon as possible
- ❑ Defibrillate immediately, if necessary.
- ❑ Remove skull pins if feasible: Performing cardiopulmonary resuscitation and defibrillation with skull pins in situ may have serious implications such as cervical spine injury due to body jerks. It can also cause burns at the site of skull pin fixation.
- ❑ Place neck in neutral position and provide in-line stabilization, if required.
- ❑ Prone: Apply chest compressions between shoulders with fist beneath sternum.
- ❑ Lateral: Apply chest compressions to sternum while stabilizing back (two-person technique).

SUGGESTED READING

1. Alfrey D, Shapiro H, Gagnon R. Cardiac arrest following rapid drainage of cerebrospinal fluid in a patient with hydrocephalus. *Anaesthesiology*. 1980;52:443-4.
2. Chatterjee S. ECG Changes in Subarachnoid Haemorrhage: A Synopsis. *Neth Heart J*. 2011;19(1):31-4.
3. Emanuela T, Stefano B, Margherita N, Irene R, Vittorio F. Colloid cyst of the third ventricle, hypothalamus, and heart: A dangerous link for sudden death. *Diagn Pathol*. 2012;7:144.
4. Furay C, Howell T. Paediatric neuroanaesthesia. *Cont Edu Anaesth Crit Care Pain*. 2010;10:172-6.
5. Geraldine S. Electrophysiologic changes and intracranial pathology. *J Am Asso of Nurse Anesth*. 1991;59(3).
6. Micheal P, Ronald AK. Neurosurgery for the patient with cardiac disease. *Anaesth clinics of North Am*. 1997;15:159-70.
7. Tamsin G, Martin S. Cardiovascular complications of brain injury. *Continuing Education in Anaesthesia, Critical Care & Pain*; Dec 2011.
8. Tumul C, Andrea P, Ronald C. Cardiac Emergencies in Neurosurgical Patients. *BioMed Research International* 2015, Article ID 751320.
9. Tumul C, Cyrill M, Nora S, Ronald C, Bernhard S. Coronary Spasm in Neurosurgical Patients and Role of Trigemino-cardiac Reflex. *Neurology Research International*. Volume 2014, Article ID 974930.
10. Vitale G, Pivonello R, Lombardi G, Colao A. Cardiovascular complications in acromegaly. *Minerva Endocrinologica*. 2004;29(3):77-88.

Chapter 16

Cardiovascular Concerns in Urosurgical Procedures

Aarti Kulkarni, Nalini Jadhav

GENERAL

- ❑ Patients with chronic renal disease need careful preoperative evaluation and optimization including volume and electrolyte correction.
- ❑ Another issue that should be considered is the high serum creatinine level. Conventional coronary angiography cannot be done in these patients. So other imaging modalities like MR angiography should be considered.

NEPHRECTOMY/PYELOPLASTY

Physiological changes in lateral decubitus position during these surgeries include reduced venous return and preload; this may cause excess hypotension leading to myocardial ischemia in compromised patients. Also, hypotension may be exaggerated in cardiac patients who are on continuous diuretic therapy.

RADICAL NEPHRECTOMY

Due consideration should be given towards the IVC extension and intracardiac involvement of the tumor. Patients should be prepared for open heart surgery in such cases. Major complications of this surgery are tumor embolism and bleeding. Transesophageal echocardiography may be performed if there is suspicion of tumor embolism. Risk of pneumothorax is increased if the surgery is extensive and one should be more vigilant as postoperative hypoxia may deteriorate cardiac function in compromised patients.

RADICAL CYSTECTOMY AND ILEAL CONDUIT

These patients require bowel preparation which increases the risk of dehydration, acidosis and hypokalemia. Often these patients present with bleeding and the resultant anemia in cardiac patients should be corrected to avoid cardiac failure.

TRANSURETHRAL RESECTION OF PROSTATE (TURP)

Use of glycine during resection increases risk of cardiac failure due to fluid overload and hyponatremia.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL)

High energy vibration can cause closure of reed switch causing asynchronous pacing in patients with pre-existing pacemaker. Rate responsive pacemaker can be affected due to damage caused by piezoelectric crystals by ESWL. The shock waves can produce ventricular extrasystole if not synchronized with R-wave. Thus, pacemaker malfunction can occur in patients undergoing ESWL. Focal point of lithotripter should be kept at least 6 inches away from pacemaker. Patient with abdominally placed pacemaker generator should not be treated with ESWL. Dual chamber demand pacemaker is especially sensitive to shock wave and should be reprogrammed to a simple mode (VOO, VVI) preoperatively.

RENAL TRANSPLANT

During this surgery, high preload maintenance may not be tolerated in patients who have developed uremic dilated cardiomyopathy and the anesthetic-induced myocardial depression should be minimized.

Chapter 17

Cardiovascular Concerns in Major Orthopedic Surgeries

Yogita Patil, Navin Pajai

CHOICE OF ANESTHESIA

Regional anesthesia is the anesthesia of choice in a majority of orthopedic surgeries. But, in patients having compromised cardiac function, spinal anesthesia may result in detrimental hemodynamics and cardiac failure may be precipitated. Hence, nerve blocks and/or general anesthesia may be preferred in these patients for minimal disturbance/ better control of hemodynamics.

Spine surgeries are highly invasive procedures of long duration ranging from two to more than ten hours. Excessive blood loss is anticipated. Major fluid shifts are possible depending upon the blood loss and duration of surgery. *Hypothermia* is very likely if not actively prevented. Patients with known cardiac conditions have a greater risk of adverse events with increased postoperative morbidity.

Hypotensive anesthesia required for spine surgeries is not well tolerated in cardiac patients because of the risk of decreased coronary blood supply.

Multiple blood transfusions result in acid-base and electrolyte imbalance, while hypothermia has an exaggerated effect in these patients.

Arthroplasty: There is a heightened risk of complications during *cementing* process in TKR/THR due to fat/debris embolism.

Tourniquet release has the risk of eliciting increased reperfusion injury.

Shoulder arthroscopy: During these procedures, strict vigilance is needed pertaining to the amount of normal saline used intraoperatively which is later on absorbed in the intravascular compartment. The risk of sudden fluid overload culminating into pulmonary edema is increased in patients with compromised cardiac function.

Chapter 18

Cardiovascular Concerns in Plastic Surgeries

Shweta Salgaonkar, Sanjeevani Zadkar

INTRODUCTION

Pediatric patients coming for congenital deformity/defect correction procedures have a higher incidence of associated congenital heart disease. The most common procedures performed are cleft lip and cleft palate. The most common syndromes associated with a cleft lip and a cleft palate are:

- ❑ Velocardiofacial (Shprintzen's) syndrome
- ❑ van der Woude's syndrome
- ❑ Stickler's syndrome
- ❑ Pierre Robin syndrome: micrognathia/retrognathia
- ❑ Fetal alcohol syndrome
- ❑ Goldenhar's syndrome (facio-auriculo-vertebral syndrome or hemifacial microsomia)
- ❑ Treacher Collins syndrome: Hypoplastic cheeks, zygomatic arches, and mandible, microtia with possible hearing loss, high arched or cleft palate, macrostomia (abnormally large mouth) and small oral cavity and airway with normal-sized tongue
- ❑ Nager syndrome
- ❑ Down syndrome
- ❑ Catel–Manzke syndrome: Micrognathia (Robin sequence), ventricular septal defect, and talipes equinovarus
- ❑ Apert syndrome: Craniosynostosis and syndactyly of the hands and the feet, amblyopia cleft palate, tracheal stenosis, VSD, abnormalities of the cervical vertebrae.

During induction extra precaution needs to be taken as cardiovascular reserve will be low. In severe cases of Treacher Collins syndrome and Pierre Robin syndrome, even sevoflurane can cause enough respiratory depression to cause desaturation as even ventilation is difficult in these cases.

It is a very common practice in plastic surgery to infiltrate the surgical site with adrenaline with or without local anesthetic (1:200,000 or 1:500,000). It should be avoided in patients with IHD, cyanotic heart disease, stenotic lesion and limited cardiac reserve because inadvertent intravascular entry may produce a catastrophe.

Reconstructive Flap Procedures

The guiding principle of anesthesia for free flap surgery is to maintain optimal blood flow to the flap. Goals of anesthesia include vasodilatation, maintenance of good perfusion pressure and low viscosity. Hemodilution is preferred to improve microvascular circulation. Sympathetic blockade (epidural, plexus) causes dilatation of vessels.

All these manipulations may be detrimental in patients with limited cardiac reserve because vasodilation is not well-tolerated in valvular and severe ischemic pathology. Hemodilution decreases oxygen supply to the heart and prolonged duration of surgery leads to hypothermia which increases cardiac work load.

LIPOSUCTION AND ABDOMINOPLASTY

In these patients, there is a higher incidence of diabetes, obstructive sleep apnea and coronary artery disease along with other obesity-related problems. Patients undergoing liposuction surgery are at high risk for developing pulmonary fat embolism syndrome due to fat particles being dislodged during surgery. The amount of lignocaine injected may be very large, approximately 35–55 mg/kg, and this increases the risk of local anesthetic toxicity. The intraoperative fluid management has to be carefully titrated along with hemodynamic monitoring and temperature control. Assessment of blood loss is difficult, as it is mixed with the aspirated fat. Absorption of a large amount of tumescent solution from subcutaneous to the intravascular compartment can cause pulmonary edema and overhydration. This is commonly seen with large-volume liposuction. Large doses of lignocaine in the tumescent solution can impair cardiac contraction and conduction, resulting in fatal arrhythmias. Addition of epinephrine to the wetting solution can cause arrhythmias if the circulating levels are high. Maintaining adequate fluid balance is challenging in large-volume liposuction due to absorption of fluid in third space and later on into the intravascular compartment. In patients with coronary artery disease, central venous pressure monitoring should be done.

BURNS

In compromised patients, propofol during induction may lead to *hemodynamic instability*. Development of tolerance can be a problem with opioids. These patients are very prone for *hypothermia* due to loss of protective skin layer. This can be prevented by warming operating room before surgery, using warm skin preparation solutions, decreasing amount of air exchanges during surgery, using fluid warmers and humidifiers. These procedures can cause massive blood loss. *ECG monitoring can be difficult* sometimes because of burns involving arms, chest and back. *Fluid* contraction in the initial period is followed by redistribution into the intravascular compartment which along with an overzealous fluid replacement may precipitate cardiac failure. *Hyperkalemia* due to burns can lead to ventricular arrhythmias which may not be well-tolerated in patients with compromised cardiac function.

SUGGESTED READING

1. Donald M. Anaesthesia for microvascular surgery. *Br J Anaesth.* 1985;57:904-21.
2. Longnecker DE, Tinker JH, Morgan GE. Principles and practice of Anesthesiology 2nd Edn. Mosby; 1998.
3. Lubin MF. Medical Management of the Surgical Patient, 4th edition. Cambridge, UK. 2004;813.
4. Pollard I. Handbook of Clinical Anaesthesia, 2nd edition Churchill Livingstone Edinburgh. UK; 2004. pp. 821.
5. Sigurdsson GH. Anaesthesia and microvascular surgery: Clinical practice and research. *Eur J Anaesthesiol.* 1995;12:101-22.

Chapter 19

Cardiac Disease and Liver Surgery

Prerana Nirav Shah

INTRODUCTION

With the increase in life span and better medical care, the number of patients needing surgery and thereby anesthesia is increasing. Cardiac disease in these patients is not uncommon. Unlike corrective cardiac surgery for cardiac ailment, these patients will continue to have the cardiac disease even post-operatively.¹ This adds to the already existing challenges in managing these patients. The incidence of cardiac morbidity is high.¹ Also, patients in whom cardiac complications occur there are high chances of developing noncardiac complications.¹

Patients coming for liver surgery may have associated cardiac diseases. Heart and liver are related.² As liver receives 25% of cardiac output, it is sensitive to reduction in blood flow.² Cardiac hepatopathy is setting of heart failure. Acute and chronic heart failure correlate with changes in liver. Outcome of cardiac disease is often influenced by liver damage and often recovers with improving cardiac function. In addition they may have alcohol-induced cardiomyopathy, portopulmonary hypertension, hypertrophic cardiomyopathy. Liver disease causes high resting cardiac output and low systemic vascular resistance.

Liver transplant per se is a complex surgery, more so with associated cardiac disease. Cardiovascular evaluation is an important step in a recipient especially if the patient has diabetes, coronary artery disease or peripheral vascular disease.

Cardiac disease could be valvular, congenital or ischemic heart disease. Liver surgery could be segmental, hemi-, wedge, extended liver resection or liver transplant. Liver surgery being intraperitoneal surgery comes in intermediate risk with 1–5% risk of cardiac death or nonfatal myocardial infarction.

Preoperative assessment should include history, physical examination, basic hematological tests, 12 lead ECG and chest radiograph to identify the presence of heart disease, severity, stability and previous treatment of the disease.¹ Additional cardiac investigations should be done for elective surgery depending on the functional capacity of the patient and presence of comorbid conditions. Recent cardiac evaluation may not be further investigated if their symptoms and activity levels have not deteriorated.

The oxygen supply and demand ratio must be maintained in the perioperative period to avoid ischemia. The type of anesthesia is governed by the experience and skill of the anesthesiologist and familiarity with the techniques and drugs. Etomidate has the least cardiovascular effects.

Monitoring is done with a five lead ECG with V5 the most sensitive lead. Invasive monitoring by arterial and pulmonary artery catheters can be useful in recent myocardial infarction with cardiac failure. The pulmonary artery catheter is useful in monitoring volume status and cardiac performance. Transesophageal echocardiography can be used to assess volume status and valvular disease, and is the best way to detect ischemia early (segmental wall motion abnormalities). Information received from the monitors must be acted on promptly. Normothermia with adequate glucose control is important.

Postoperatively, all patients should receive humidified oxygen along with adequate analgesia. Anemia should be treated and thromboprophylaxis continued. The patient's normal cardiac medication should be restarted as soon as permissible.

CONGENITAL CARDIAC DISEASE

Advances in pediatric cardiac surgery, have made several infants born with severe congenital heart defects living near normal growth and development and good quality of life. This is especially in children born with single ventricle malformations, such as tricuspid or pulmonary atresia and hypoplastic left heart syndrome. Surgery diverts blood from the right atrium to the pulmonary arteries and is palliative but not curative because significant long-term complications are known to arise. The ensuing cavopulmonary anastomosis results in increased CVP 3 to 4 times normal, and passive hepatic congestion leading to fibrosis and cardiac cirrhosis is frequently observed. Moreover, reduction in cardiac index and bradycardia leads to ischemic and hypoxic injury to the liver. As a result, these children often develop the similar findings, including abnormal liver function tests, ascites and coagulopathy, as seen in adult patients with biventricular heart failure.

VALVULAR HEART DISEASE

Significant valvular heart disease increases cardiac risk for patients undergoing noncardiac surgery. Patients with suspected valvular heart disease should undergo echocardiography to quantify the severity of stenosis or regurgitation, calculate systolic function, and estimate right heart pressures. Evaluation for concurrent coronary artery disease (CAD) with electrocardiography exercise testing, stress echocardiographic or nuclear imaging study, or coronary angiography, is needed as appropriate. Emergency noncardiac surgery may occur in the presence of uncorrected significant valvular heart disease. The risk of noncardiac surgery can be minimized by knowing the type and severity of valvular heart disease, choosing an appropriate anesthetic approach and considering a higher level of perioperative monitoring (e.g. arterial pressure,

pulmonary artery pressure, and transesophageal echocardiography), as well as managing the patient postoperatively in an intensive care unit (ICU) setting. Replacement of the valve before noncardiac surgery may be needed in those with severe symptomatic disease, especially aortic stenosis. Patients with regurgitant valves benefit from after load reduction, faster heart rates and maintenance of preload. Antibiotic prophylaxis is necessary for all cases and should be governed by the type of surgery and institutional protocols.

ISCHEMIC HEART DISEASE

Elective surgery must be postponed for 6 months after a myocardial infarction.⁴ Patients who have had a myocardial infarction 6 weeks prior with no evidence that further myocardium is at risk can proceed with urgent surgery with perioperative cardiac risk-reduction strategies. After 6 months have elapsed and those who have resumed normal daily activity and have no post-infarction angina should not need further testing, unless the risk of surgery or the functional capacity warrants it.¹ The anesthesia plan may be decided after evaluating respiratory, renal, endocrine especially diabetes, skeletal, airway systems. It is essential to optimize the patient's condition and medication prior to surgery.

Patients who have undergone coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) with or without stent insertion in the previous 5 years, and who have had no recurrence of symptoms with a return to an active lifestyle, may not need further testing. Stoppage of antiplatelet drugs is not recommended within 4–6 weeks of bare-metal stent implantation or within 12 months of drug-eluting stent implantation. Hence, it is a challenge in prevention of blood loss.⁴

LIVER TRANSPLANT

Cardiovascular assessment in liver transplantation candidates must be able to reveal whether a patient can be expected to survive the operation and immediate postoperative period and if the patient has such severe cardiopulmonary disease that transplantation would be futile. Active cardiac conditions include unstable coronary syndromes (e.g. unstable angina, severe angina, or recent MI), decompensated heart failure, significant arrhythmias, and severe valvular disease. The presence of one or more of these conditions is associated with high rates of perioperative cardiovascular morbidity and mortality and may require delay or cancellation of surgery. Accurate preoperative cardiac evaluation is essential to detect coronary artery disease and to treat it before OLT. Coronary angiography is done if there is abnormal noninvasive test or high probability of coronary artery disease (CAD) based on the presence of two or more classical risk factors, such as age above 50 years, diabetes mellitus, smoking, family history of CAD, arterial hypertension and hyperlipidemia.³ Echocardiography with agitated saline contrast is often recommended to detect pulmonary hypertension and/or intrapulmonary arteriovenous shunt. Echocardiography is also recommended to evaluate ventricular and valvular functions and left

ventricular outflow tract obstruction. Control of lipid abnormalities and medical management of atherosclerotic risk factors are not high priorities during pretransplantation phase of most liver transplantation recipients. Many liver transplantation candidates have a coagulopathy with prolonged prothrombin times, thrombocytopenia from hypersplenism, and esophageal and gastric varices. Hence, any form of anticoagulation is generally avoided unless the patient has well-documented coronary artery disease (CAD). Patients with severe portopulmonary hypertension (PPH) and significant right ventricle dysfunction must be excluded for OLT.⁵

Preoperative therapy using vasodilator drugs to reduce PPH and right ventricular dysfunction may improve the clinical status and make orthotopic liver transplant (OLT) feasible. Nonselective beta-blockers are started in liver transplantation candidates with large esophageal varices. Advanced intra-operative and postoperative hemodynamic monitoring plus TEE is needed in such surgery.⁵ OLT imposes a severe challenge to the cardiovascular system due to hemorrhage, clamping of major vessels reducing venous return, reperfusion syndrome, aggressive fluid replacement and electrolyte and acid-base disturbance.³

SUMMARY

Patients needing liver surgery with cardiac disease pose a challenge to the anesthesiologist in the perioperative period.

REFERENCES

1. Bill MK. Anaesthesia for patients with cardiac disease undergoing non-cardiac surgery. *Anaesthesia and Intensive Care Medicine*. 10; 9: 446- 50.
2. Moller S, Henriksen JH. The heart and other organs—interactions of the heart and the liver. *Gut*. 2008; 57 (2):268-78.
3. Moller S, Bernardi M. Interactions of the heart and the liver. *Eur Heart J*. 2013; 34 (36):2804-11.
4. Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA Guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;64(22):e77-e137.
5. Rocca G, Costa MG, Pompei L, Rocca CP. The liver transplant recipient with cardiac disease. *Transplant Proc*. 2008;40(4):1172-4.

Chapter 20

Cardiovascular Concerns During Liver Transplantation

Manjula Sudeep Sarkar

PREOPERATIVE CARDIAC INVESTIGATIONS

Detection of ischemic heart disease in the preoperative period has assumed greater significance as the option of liver transplantation is now being offered to older patients. These patients cannot complete stress test due to end stage liver disease. Also, contrast-induced nephropathy precludes the use of angiography in these patients. Hence pharmacologic stress test, stress echocardiography or myocardial perfusion scan have to be used for the diagnosis. Nonischemic cardiomyopathy is also frequently seen in these patients.

PATHOPHYSIOLOGICAL CHANGES IN THE CARDIOVASCULAR SYSTEM RELATED TO LIVER FAILURE

Hyperdynamic circulation: High cardiac output, afterload reduction, increased left atrial size and cardiac index, mild LVH, cardiomyopathy, autonomic neuropathy and portopulmonary hypertension are seen. Portopulmonary hypertension is associated with right heart failure and increased perioperative mortality.

Intraoperative

Routine monitoring consists of electrocardiography, pulse oximetry, capnography and temperature monitor. Invasive monitoring including direct arterial BP, central venous and pulmonary artery catheter is obligatory.

Cirrhotic patients are relatively hypovolemic and hence adequate volume infusion improves tissue perfusion in these patients.

CVP cannulation is better performed after induction of anesthesia because these patients cannot tolerate Trendelenburg position due to tense ascites, and pleural effusion.

TEE helps to monitor valvular and ventricular function and diagnose intraoperative embolism.

Careful titration of drugs, considering coagulation profile, volume status and general hemodynamic status of the patient is important.

Isoflurane is preferred due to minimal effects on the cardiovascular system, vasodilatory effect on hepatic circulation (advantageous for the reperfused graft) and better preservation of splanchnic blood flow.

Preanhepatic Phase

The primary issue is the high risk of surgical bleeding and hemodynamic instability resulting from drainage of large volume of ascitic fluid, transection of varices and surgical manipulation of the liver. The low CVP approach maintaining the pressure at or below 5 cm H₂O to reduce blood loss and liver congestion may not be a good choice in patients with valvular heart disease. Complications arising from blood transfusion like fluid overload, hyperkalemia, acid-base disturbances can easily result in cardiac decompensation. The risk of air embolism is high during the manipulation of vena cava.

Anhepatic Phase

This phase carries the risk of reduced venous return and marked decrease in cardiac output with hypotension and cardiovascular collapse. In cardiac patients, these changes may not be tolerated and venovenous bypass may need to be initiated for hemodynamic stability. Noradrenaline and vasopressin maintain renal perfusion without impairing mesenteric blood flow.

Hypocalcemia from citrate intoxication should be corrected promptly as it results in myocardial depression.

Neohepatic Phase

It carries the risk of reperfusion syndrome characterized by bradycardia, hypotension, reduced SVR and augmented pulmonary vascular resistance. Use of inotropes is usually needed to maintain hemodynamics.

Postoperative

Extubation

Risk factors for delayed extubation and prolonged ventilatory support include encephalopathy, BMI >34, cardiovascular failure, graft dysfunction, neurologic impairment, and transfusion of more than 12 units of PRBC.

SUGGESTED READING

1. Anesthesia in Liver Transplantation, Meral Kanbak, AyseHeves Karagoz, Filiz Uzumcugill. North Am Clin 2012 Liver Transplantation—Basic issues ISBN 978-953-51-00164

Chapter 21

Cardiovascular Concerns in General and Emergency Abdominal Surgery

Manish Kela, Priti Pednekar

PACEMAKER AND ANESTHESIA MANAGEMENT

Pacemaker (PM) has an impulse generator and leads to carry impulse to the patient's heart. These are inserted via transvenous or epicardial route.

Types: There are unipolar, bipolar and multipolar pacemakers. Amongst them, unipolar are more sensitive to the effect of EMI. While bipolar pacemakers are resistant from muscle artifact and EMI effect. They are more commonly used nowadays. Bipolar PM is identified as ring electrode 1–3 cm proximal to lead tip on X-ray. It can be programmed to unipolar for pacing and sensing or both.

As per 2002 NBG, PM codes are as follows:

	Position I Pacing chamber	Position II Sensing chamber	Position III Response to sensing	Position IV Programmability	Position V Antitachycardia functions
O (None)	O	O	O	O (None)	o
A (Atrium)	A	A	I (Inhibited)	(R) Rate modulation	Pace
V (Ventricle)	V	V	T (Triggered)	R	Shock
D (Dual)	D	D	D (I+T)	R	D (Pace and Shock)

Indications for PM Insertion

- ❑ Symptomatic bradycardia, third and second degree AV block
- ❑ Sinus node disease
- ❑ AV node diseases
- ❑ Long QT syndrome
- ❑ HOCM, DCM
- ❑ Post MI with LBBB, bifascicular and trifascicular heart block

Preanesthetic Evaluation in Patients with PM

No special tests are required. But patient with coexisting cardiac diseases or other systemic disease should be monitored thoroughly. 12 lead ECG, 2 D Echo, chest X-ray should be done. Chest X-ray is done to know the position of coronary sinus lead, if IJV cannulation is planned.

ACC guidelines recommend timely preoperative interrogation with PM programmer. It gives idea about battery longevity, battery impedance, lead performance and adequacy of current setting. Appropriate reprogramming is safer to avoid intraoperative problems.

In case of unipolar electrocautery use, reprogramming to asynchronous mode helps to avoid oversensing during EMI. But sometimes internal damage or reset caused by EMI may ignore APCs and VPCs during asynchronous mode. This can lead to hemodynamic instability. A prospective study has shown that unipolar devices are far more susceptible than bipolar devices to electrocautery interference.

Diathermy: it may interfere with PM activity. Use it only if needed and away from site of generator.¹

External defibrillator paddles should be placed at least 10 cm away from PM location. For generators positioned in the right pectoral region, apex and posterior positions are more appropriate. It is always advisable to make the appropriate programmer available.²

In MRI, electromagnet may convert PM mode to asynchronous mode and can lead to hemodynamic embarrassment. So MRI in such patients should be avoided as far as possible, and if needed should be done with careful monitoring with cardiac support.²

Magnets can be helpful to protect the pacemaker-dependent patient during diathermy, electrocautery or other sources of pulsed EMI. After applying magnet over the pacemaker, it avoids inhibition by such pulsed interference. But manufacturer will give better idea about this. So he should be contacted for the same.^{1,2}

Anesthesia Concerns^{1,2}

- ❑ Preoperative assessment includes identification, type and functioning of PM, type of surgery, use of type of cautery and patient cardiopulmonary status
- ❑ 12 lead ECG may give idea about rhythm and pacing. 2D echo for heart functioning
- ❑ Reprogramming is indicated in some situation like AV delay, HOCM and heart failure. Antitachycardia mode should be disabled to avoid tachycardia response to mechanical ventilation
- ❑ During use of electrocautery, close proximity to PM may increase the risk of interference. In such conditions magnet or temporary cardiac transvenous pacing wire before surgery is recommended

- ❑ During surgery, use of bipolar cautery should be insisted. Anode pad should be as far as from position of PM. Use of cut is preferred over coag mode on bipolar
- ❑ ECG, pulse oximetry, and arterial pressure monitoring should be done vigilantly
- ❑ Postoperatively restoration of pacing mode and antitachycardia mode should be done by programmer. ICU observation for 24 hours is better.

In our hospital, we have come across around 13 cases in last year. All were elderly patients with PM for 3–10 years period for inguinal hernia repair. Our anesthesia management were as follows:

- ❑ For 4 cases, we have given general anesthesia i/v/o recurrent and large hernia. Programmer with magnet was present in perioperative period. Monitoring were 5 lead ECG, SpO₂, ABP monitoring. Judicious use of iv fluids, anesthesia agents were done. Anesthesia agents we used were fentanyl, midazolam, and etomidate, rocuronium as muscle relaxant. All four cases done uneventfully and observed in ICU for 24 hours. mode of PM was restored postoperatively immediately.
- ❑ 5 cases of unilateral inguinal hernia were done in inguinal field block with local anesthetic agents bupivacaine and lignocaine. We just gave monitored anesthesia care. Need of magnet application was not required. These cases were uneventful because of patients cooperativeness and inguinal field block.
- ❑ 4 cases were successfully done under USG-guided TAP block. Lignocaine and bupivacaine were used.

Vigilant monitoring, proper assessment of PM functioning, use of bipolar ESU, regional blocks for surgery as possible, PM magnet and programmer stand by, minimal surgery period, and postop ICU care will help such patients for successful outcome.

Rheumatic heart disease: It is a complication of rheumatic fever, mainly affecting heart valves. Mitral stenosis is most common presentation, but in mixed form, along with mitral valve, other valves are also affected. In India, most of the patients are incidentally diagnosed during workup for some other illness. Due to advanced medical and surgical management, prognosis for such patients has improved and their lifespan is also prolonged. They are presenting for variety of noncardiac general surgeries from fibroadenoma to incisional hernia. Anesthesia management of such patients is challenging. We have to take into consideration severity of cardiac lesion, preoperative optimization, type of surgery and perioperative monitoring.

Management of such patients starts with preoperative assessment. The cardiac evaluation is done to:

- ❑ Assess the significance of the cardiac lesion for the proposed surgery
- ❑ Plan anesthesia according to the hemodynamic status

Intraoperative monitoring: ECG, pulse oximeter, ETCO₂, NIBP for major surgery, radial arterial and IJV cannulation for invasive monitoring.

Infective Endocarditis Prophylaxis for Gastrointestinal Procedures

- ❑ It is indicated in cases of heart valve lesions (mainly stenotic), prosthetic valve or previous endocarditis: Amoxicillin 1g plus gentamicin, 120 mg IV at induction, then amoxicillin 500 mg, orally 6 hours later. If urine infected, prophylaxis should cover causative organism)
- ❑ If allergic to penicillin, or have received more than one dose of penicillin in preceding month
- ❑ Vancomycin 1g IV over 100 minutes plus gentamicin 120 mg IV at induction, or Teicoplanin 400 mg IV plus gentamicin 120 mg IV at induction.
The incidence of cardiac cases in general surgery OT is approximately 25 to 30%.
- ❑ Minor procedures like fibroadenoma excision: Sedation + LA
- ❑ Hernia and hydrocele: Sedation + LA, in selected cases—titrated epidural
- ❑ Laparoscopic surgery involves the creation of a pneumoperitoneum with its attendant hemodynamic disturbances. The raised intra-abdominal pressure leads to sympathetic stimulation via several pathways resulting in tachycardia and hypertension. This can be of concern in a patient with compromised cardiac function.

Laparoscopic cholecystectomy, two patients had mild mitral regurgitation with tricuspid regurgitation and one patient had mitral stenosis with pulmonary hypertension. To avoid pressor response of intubation and extubation, beta- blockers were used and LMA ProSeal was used instead of endotracheal tube. CO₂ was insufflated slowly to maintain intra-abdominal pressure <10 mm Hg. IJV was cannulated under local anesthesia. Fourth patient who underwent mitral valve replacement, was on warfarin. So it was converted to unfractionated Heparin. It was stopped 6 hours before surgery and restarted 4 hours postoperatively. Infective endocarditis prophylaxis was given.

Anesthetic Implications of Emergency Abdominal Surgery²

- ❑ Full stomach with risk of aspiration
- ❑ Hypovolemia with hemodynamic instability
- ❑ Acid base and electrolyte imbalance
- ❑ Coexisting medical illness
- ❑ Hypothermia
- ❑ Limited time for patient optimization.

Preoperative Optimization

Resuscitation of the patient prior to induction of anesthesia is important. Patients with abdominal pathology are generally hypovolemic which should be corrected under CVP guidance. Electrolyte abnormality, particularly related to potassium should be corrected preoperatively. Acid-base imbalance, particularly metabolic acidosis should be corrected. Antacid prophylaxis to reduce risk of regurgitation/aspiration should be given.

Preoperative Preparation

A fully equipped anesthesia workstation with airway and intubation equipment and emergency cardiorespiratory resuscitative and antiarrhythmic drugs should be available. Pacemaker with external pacing pads and a defibrillator with the appropriate sized paddles should be confirmed.

A chart that will enumerate the calculated dosages, volume and the infusion rate of resuscitative and vasoactive agents in a closed system using programmable syringe pumps should be kept ready. Availability of blood and blood products should be confirmed.

ANESTHETIC MANAGEMENT

Choice of anesthesia for emergency intra-abdominal surgery with cardiovascular disease is difficult. The choice of anesthesia and anesthetic agents should be judicious and balanced to maintain optimum cardiac output and to avoid myocardial depression for a successful postoperative outcome.

Anesthetic Goal

- ❑ Maintain stable hemodynamics
- ❑ Avoidance of drug-induced myocardial depression,
- ❑ Prevent myocardial ischemia by optimizing myocardial oxygen supply and demand
- ❑ Maintain normothermia
- ❑ Maintain sinus rhythm
- ❑ Avoid anemia
- ❑ Avoid hypoxia, hypercarbia and acidosis.

The anesthesiologist usually has to deal with several problems of the patient, such as hypertension, hypotension, heart failure, coronary artery disease, rhythm disturbances, hemodynamic changes, bleeding, perioperative fluid imbalance and metabolic disturbances.

Both general as well as regional anesthesia can be used for emergency abdominal surgery. Most commonly used technique is a combination of general anesthesia with epidural for postoperative pain relief.

Induction

Rapid sequence induction of general anesthesia is mandatory. Cricoid pressure should be applied prior to onset of loss of consciousness. Induction agents that minimally affect the hemodynamics are most suited for the induction of anesthesia. Usually, the induction of anesthesia in patients with known coronary artery disease, chronic heart failure, and perioperative hypertension, must be gentle, avoiding tachycardia and hypotension. The sympathetic effects of laryngoscopy can be blunted using an opioid (fentanyl 2 $\mu\text{g}/\text{kg}$ IV), a bolus of lidocaine (1.5 mg/kg), and/or a β -blocker agent (Esmolol 0.5 mg/kg). Hypotension is not preferred because it can decrease the coronary artery perfusion. Normal/mildly elevated diastolic pressures,

without tachycardia are the main hemodynamic goals in patients with a known coronary occlusion.

Maintenance of Anesthesia

The anesthesiologist must be careful to choose the right anesthetic drugs. Anesthesia is usually maintained with a combination of oxygen and medical air (50%:50%) with either inhalation agents or opioid depending upon left ventricular ejection fraction. Patients with poor LV function are better maintained on opioids whereas inhalation agents are better for those with good LV function. Nitrous oxide is generally avoided particularly in patient with intestinal obstruction for fear of impairing oxygenation of patients.

Pancuronium is generally contraindicated in patients at risk for postoperative renal and hepatic failure. Cisatracurium and atracurium are the preferred muscle relaxants.

Nephrotoxic drugs such as aminoglycosides, vancomycin, and non-steroidal anti-inflammatory drugs must be avoided in patients with preoperatively deranged renal function.

Anesthetic drugs cause vasodilation and inhalational agents are important for avoiding intraoperative hypertension. Recent literature has indicated that inhalational anesthetics are superior to others in providing a specific myocardial protection.

Intraoperatively, it is important to take care of factors that affect myocardial oxygen supply and demands.

Monitoring

The standard ASA monitoring including ECG, noninvasive blood pressure, pulse oximetry, and capnography, CVP, temperature and urine output monitoring are essential for all procedures, monitoring three ECG leads (II, V4,V5 or V3,V4,V5) improves recognition of ischemia. Transesophageal echocardiography is useful in evaluation of fluid responsiveness, and cardiac monitoring. Inspired oxygen concentration, airway pressure and expired tidal volume to monitor the integrity of breathing system and adequacy of ventilation.

Intraoperative Fluid, Electrolyte and Acid-Base Balance

Intraoperatively metabolic acidosis, hypernatremia, hypokalemia, and dehydration can occur. The pre-existing hypovolemia must be corrected because of severe hypotension after the anesthesia induction. Intraoperative fluids should be given with caution as per CVP monitoring particularly in patient with actual or impending cardiac decompensation. Excessive fluid should be avoided. Measurement of urine output is important for assessing fluid status. Urine output greater than 0.5 mL/kg/h is regarded as an indicator of adequate renal perfusion.

Both hyper- and hypokalemia induces premature ventricular beats as well as other arrhythmias and need to be corrected. Metabolic acidosis is very common in intra-abdominal surgery and should be corrected.

Blood loss should be estimated during the surgery and the volume replaced with blood to maintain adequate oxygen carrying capacity. The anesthesiologist must maintain adequate hemoglobin level and optimal oxygenation. Hemoglobin levels of less than 9–10 g/dL predispose the patient to new ischemic episodes.

Temperature Management

Hypothermia is a major problem during intraabdominal surgery which delays the recovery from anesthesia and increases oxygen consumption.

Heat loss occurs via radiation, conduction and evaporation. As heat loss causes decreased organ perfusion and metabolic acidosis, and cannot be avoided, all fluids including skin preparation, irrigation and intravenous fluids should be warmed. Heated mattress should be used. Anesthesia circuits should be humidified. Low-flow or closed circuit technique is recommended.

Reversal and Recovery

Reversal of muscle relaxation with a combined anticholinesterase/anti-muscarinic causes tachycardia, and extubation in itself is a stressor. Before extubation, patient should be fully awake and able to protect the airways.

Problems in the recovery phase which can cause ischemia include; tachycardia, pain, hypothermia, shivering, hypoxia, and anemia. These should be treated not just in the immediate postoperative period, but throughout the hospital admission. The use of supplemental oxygen in the postoperative period is one of the simplest, yet most effective measures in preventing myocardial ischemia.

Postoperative events which cause death include myocardial infarction (MI), arrhythmias, and multiple organ failure secondary to low cardiac output.

POSTOPERATIVE PAIN MANAGEMENT

Neuraxial analgesia with local anesthetics, or opioids and/or alpha2-agonists, and intravenous opioids, alone or in combination with nonsteroidal anti-inflammatory drugs, seem to be the most effective regimens.

Nonsteroidal anti-inflammatory drugs and cyclo-oxygenase-2 inhibitors have the potential for promoting heart and renal failure, as well as thrombo-embolic events, and should be avoided in patients with myocardial ischemia or diffuse atherosclerosis. These drugs should be avoided in cases of renal and heart failure, or in patients who are elderly, on diuretics, or those with unstable hemodynamics.

INTERESTING CASE

Amiodarone induced thyrotoxicosis for thyroidectomy—45-year-old male patient with arrhythmic right ventricular dysplasia (ARVD) was treated with Amiodarone. Long-term treatment with amiodarone led to thyroiditis, so it

was stopped. He was treated with antithyroid drugs, e.g. carbimazole along with prednisolone, metoprolol and frusemide. But patient was not relieved of symptoms so total thyroidectomy was advised. Amiodarone-induced thyrotoxicosis is a high-risk for anesthesia management. It is the standard practice to make thyrotoxic patient euthyroid before surgery but this was not possible in this case. For managing such a patient, we have to take into consideration underlying cardiac condition, possibility of thyroid storm, and the effects of amiodarone on other systems of body.

Patient was advised to continue Tab. Metoprolol 100 mg till morning of surgery with serum potassium levels a day prior to surgery. Inj. Dexamethasone 4 mg IV, injected on morning of surgery.

Before induction, BP was 150/100 mm Hg with HR 100/min. To make patient comfortable, Inj midazolam 1 mg and IV, inj Fentanyl 150 µg IV was injected as sedative and analgesic. For preemptive analgesia,USG-guided B/L superficial and deep cervical plexus block was performed with 20 mL of 0.25% bupivacaine was infiltrated to avoid stimulation due to thyroid handling.

Induction was done with inj propofol 140 mg IV with inj vecuronium 6 mg. LMA ProSeal No. 4 was placed to avoid stress response of laryngoscopy and airway instrumentation. Anesthesia was maintained with O₂ + N₂O+ Isoflurane.

Right radial artery cannulation was done for invasive BP monitoring. Left sided antecubital vein was cannulated for CVP monitoring. IV fluid was restricted to 15 mL/kg of lactated Ringer solution.

Vitals were stable throughout the procedure with BP within a range of systolic 130–150 mm Hg and diastolic 80–90 mm Hg and pulse 70–90/min. During intraoperative period, there was one episode of displacement of LMA cuff during manipulation of trachea by the surgeon.

Duration of surgery was 1.25 hours. At the end of surgery, patient was reversed with neostigmine and glycopyrrolate. To avoid stress response Inj. xylocard was given and LMA was removed in deep plane of anesthesia.

In postoperative period, patient was shifted to cardiac ICU for further monitoring and after 24 hours, he was shifted to endocrinology ward. Tab metoprolol 100 mg OD was continued along with tapering doses of steroid.

REFERENCES

1. Rozner MA. Implantable Cardiac Pulse Generator: Pacemakers and Cardioverter-Defibrillators. Miller's Anesthesia. In: Miller RD (Ed). 7th edition. Churchill Livingstone.
2. Saluke TV, Dob D, Sutton R. Pacemakers and defibrillators: Anesthetic Implications, Brit Anesth. 2004;93(1):95-104.

Section 6

Pain Management in Cardiac Surgical Patient

Section Outline

- Postoperative Analgesia for Cardiac Surgical Patient

Chapter 22

Postoperative Analgesia for Cardiac Surgical Patient

Sunil Gvalani, Shamala Yashod

Optimal postoperative analgesia is vital to reduce the morbidity and mortality after cardiac surgery. Severe pain is associated with a stress response that needs to be attenuated with the help of multimodal analgesia.

PATHOPHYSIOLOGY OF CARDIAC SURGICAL PAIN

Acute Postoperative Pain

Excruciating pain following cardiac surgery is common. Inciting factors are sternotomy, pericardiotomy, thoracotomy, intercostal drain insertion and venous graft incisions. Nociceptor stimulation, release of inflammatory mediators and catecholamines affect all major organ systems thereby increasing morbidity of postsurgical patients. Cardiac surgical patients in whom internal mammary artery is harvested are particularly susceptible to increased postoperative pain.

Chronic Pain After Cardiac Surgery

Chronic pain after cardiac surgery is observed to be more common in younger patients and those requiring higher doses of analgesics in the postoperative period. Intercostal nerve injury, sternomyelitis following IMA graft, brachial plexus neuropathy, sternal wire sutures and saphenous neuralgia predispose to chronic pain.

BENEFICIAL EFFECTS OF ADEQUATE POSTOPERATIVE ANALGESIA

- ❑ Aggressive control of postoperative pain will prevent the adverse hemodynamic, immunologic, hemostatic and metabolic effects.
- ❑ Adequate analgesia helps to attenuate the increased levels of stress hormones released intraoperatively during initiation of CPB.
- ❑ It inhibits the stress response caused by intraoperative cardiac sympathetic stimulation.
- ❑ It maintains the balance between coronary blood flow and myocardial oxygen demand and helps prevent perioperative myocardial ischemia.

- ❑ It may potentially decrease morbidity and enhance health-related quality of life during the pivotal immediate postoperative period

MODALITIES FOR POSTOPERATIVE ANALGESIA

The available modalities are:

- ❑ Opioids
- ❑ Nonsteroidal anti-inflammatory agents
- ❑ Alpha-adrenergic agents
- ❑ Local anesthetic infiltration
- ❑ Nerve blocks
- ❑ Epidural techniques
- ❑ Multimodal analgesia.

ASA Guidelines

- ❑ In general, the American Society of Anesthesiologists Task Force on Acute Pain Management in the Perioperative Setting reports that the existing literature supports the efficacy and safety of three techniques used by anesthesiologists for perioperative pain control: (a) Regional analgesic techniques (including but not limited to intercostal blocks, plexus blocks, and local anaesthetic infiltration of incisions), (b) Patient-controlled analgesia (PCA) with systemic opioids, and (c) Intrathecal/epidural opioid analgesia.
- ❑ In regional analgesic techniques, the existing literature supports the analgesic efficacy of peripheral nerve blocks and postincisional infiltration with local anesthetics for postoperative analgesia, yet is equivocal regarding the analgesic benefits of preincisional infiltration.
- ❑ Regarding PCA with systemic opioids, the existing literature supports its efficacy (compared with intramuscular techniques) for postoperative pain management, yet is equivocal regarding the efficacy of PCA techniques compared with nurse or staff administered intravenous analgesia.
- ❑ In addition, the existing literature is equivocal regarding the comparative efficacy of epidural PCA versus intravenous techniques.

Opioids

Opioids provide excellent analgesia in cardiac surgical patients. But side effects like nausea, vomiting, pruritus, respiratory depression, constipation and tachyphylaxis need to be kept in mind. Opioids can be given as intravenous bolus, or infusion in the form of patient controlled analgesia (PCA).

Nonsteroidal Anti-inflammatory Agents

Nonsteroidal anti-inflammatory drugs (NSAID's) act by inhibition of cyclo-oxygenase enzyme. These help to reduce the dose of opioids and their side effects.

Selective COX-2 inhibitors are devoid of side effects like gastric mucosal bleeding, renal injury and platelet inhibition. But specific COX-2 inhibitors might impede reparative inflammatory responses and increase susceptibility to sternal wound infection.

Commonly used non-selective NSAIDs are diclofenac, ibuprofen, ketorolac, indomethacin, while selective COX-2 inhibitors are celecoxib, parecoxib valdecoxib, and etoricoxib.

However, current literature does not provide conclusive data on the efficacy of NSAID's when used alone. But a combination of NSAID with traditional intravenous opioid provides adequate postoperative analgesia with a beneficial reduction of opioid consumption and the associated side-effects.

Alpha-2 Adrenergic Agonists

Dexmedetomidine and clonidine are the most commonly used alpha-2 adrenergic agonists. Currently, these drugs are used for preoperative sedation, intraoperative supplementation during anesthesia to reduce sedative and analgesic requirements, and postoperative sedation and analgesia. These act via stimulation of alpha-2 receptors within the locus coeruleus and the spinal cord.

They have the advantage of providing cardiovascular stability but are associated with side effects like sedation, bradycardia and hypotension.

Local Anesthetic Infiltration

The anterior and posterior branches of the intercostal nerves innervate the sternum. Sternotomy pain can be treated with local anesthetic infiltration. It is a very simple technique to provide reliable postoperative analgesia. The commonly used drugs are lidocaine, bupivacaine, levobupivacaine and ropivacaine. A continuous infusion of local anesthetic is also used. But concerns regarding tissue necrosis and sternal wound infection have been raised. This technique also has risk of side effects related to local anesthetics.

Nerve Blocks

Nerve block provide adequate analgesia after minimally invasive cardiac surgery. Thoracotomy causes greater postoperative pain due to costal cartilage trauma, and tissue damage to ribs, muscles and peripheral nerves. Adequate analgesia after thoracotomy is important because pain restricts adequate respiratory function after surgery. This may lead to pulmonary complications and hypoxemia, with resultant myocardial ischemia, cerebrovascular accident, thrombo-embolism and delayed wound healing. The most commonly used techniques are intercostal nerve blocks, intra-pleural infiltration of local anesthetics and thoracic paravertebral block. Epidural technique is also effective in controlling post-thoracotomy pain. Continuous catheter infusion technique is also used. But, local anesthetic toxicity can be a hazard.

Epidural Techniques

Epidural techniques with local anesthetics and opioids provide reliable analgesia. It attenuates the stress response. Also, it allows for earlier tracheal extubation as compared to IV opioids. TEA with local anesthetics blocks cardiac sympathetic nerve activity and improves myocardial oxygen supply/demand ratio. However, epidural techniques carry the risk of hematoma formation.

“Multimodal” or “Balanced” Analgesia

The purpose of multimodal analgesia is to improve analgesia with minimum side effects. The American Society of Anaesthesiologists Task Force on Acute Pain Management in the Perioperative Settings reported that the literature supports the administration of two analgesic agents that act by different mechanisms via a single route for providing superior analgesic efficacy with equivalent or reduced adverse effects. NSAID, COX-2 inhibitors, and acetaminophen administration has a dose sparing effect for systemically administered opioids. It is also suggested that two routes of administration, when compared with a single route, maybe more effective in providing postoperative analgesia. The various examples of multimodal analgesia are: (a) epidural opioids administered with epidural local anesthetics or clonidine, (b) intravenous opioids in combination with ketorolac or ketamine and (c) intravenous opioids combined with oral NSAIDs, COX-2 inhibitors, or acetaminophen.

CONCLUSION

Quality of postoperative analgesia is one of the factors that determine the outcome after cardiac surgery. Intravenous systemic opioids form the basis of postcardiac surgery analgesia. NSAIDs are effective but their side effects are a concern. The American Society of Anaesthesiologists Task Force on Acute Pain Management in the Perioperative Setting recommends that anaesthesiologists who manage perioperative pain use analgesic therapeutic options only after thoughtfully considering the risks and benefits for the individual patient. The therapy (or therapies) selected should reflect the individual anesthesiologist's expertise, as well as the capacity for safe application of the chosen modality in each practice setting. This includes the ability to recognize and treat adverse effects that emerge after initiation of therapy. Whenever possible, multimodal pain management therapy should be implemented. Dosing regimens should be administered to optimize efficacy and minimize the risk for adverse events. The choice of medication, dose, route and duration of therapy should always be individualized.

SUGGESTED READING

1. American Society of Anaesthesiologists Task Force on Acute Pain Management: Practice guidelines for acute pain management in the perioperative setting: An updated report by the American Society of Anaesthesiologists Task Force on Acute Pain Management.
2. Smith MS, Mcloughlin T Jr. Pain management for cardiothoracic procedures—a practical approach to cardiac anesthesia. In: Hensley FA, Martin DE, Gravelee GP (Eds). 5th edition. Wolter Kluwer Lippincott Williams and Wilkins, Philadelphia; 786-99.

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