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Transoesophageal echocardiography (TOE/TEE) in cardiac patients is now almost routine. Its use in cardiac monitoring has also extended to include critically ill patients for non-cardiac surgery and the intensive care setting. Specific accreditation is required prior to practice of TOE/TEE involving a written examination and a documented logbook of experience. This book has been specifically designed to help candidates pass the written exam and has been structured around the syllabus. Providing a summary of all relevant information, this is an invaluable study aid. Lists of further reading material are provided with every topic, including guidelines and safety, cardiomyopathies, heart disease, haemodynamic calculations and many more. Each chapter ends with a series of exam-style questions for self-assessment. An extremely useful book for trainee anaesthetists, intensivists, trainee cardiologists and cardiac surgeons.

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<tr>
<td>A</td>
<td>amplitude</td>
</tr>
<tr>
<td>AC</td>
<td>attenuation coefficient</td>
</tr>
<tr>
<td>AF</td>
<td>atrial fibrillation</td>
</tr>
<tr>
<td>AI</td>
<td>aortic incompetence</td>
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<tr>
<td>A/L</td>
<td>antero-lateral</td>
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<tr>
<td>AMVL</td>
<td>anterior mitral valve leaflet</td>
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<td>AS</td>
<td>aortic stenosis</td>
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<td>ASD</td>
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<td>aortic valve area</td>
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<td>aortic valve closes</td>
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<td>AVN</td>
<td>atrio-ventricular node</td>
</tr>
<tr>
<td>AVO</td>
<td>aortic valve opens</td>
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<tr>
<td>BP</td>
<td>blood pressure</td>
</tr>
<tr>
<td>BSA</td>
<td>body surface area</td>
</tr>
<tr>
<td>BUR</td>
<td>beam uniformity ratio</td>
</tr>
<tr>
<td>CC</td>
<td>costal cartilage</td>
</tr>
<tr>
<td>CCF</td>
<td>congestive cardiac failure</td>
</tr>
<tr>
<td>CFD</td>
<td>colour flow Doppler</td>
</tr>
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<td>Cn</td>
<td>compliance</td>
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<td>CO</td>
<td>cardiac output</td>
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<td>CPB</td>
<td>cardiopulmonary bypass</td>
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<tr>
<td>CS</td>
<td>coronary sinus</td>
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<tr>
<td>CW</td>
<td>continuous wave</td>
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<tr>
<td>CWD</td>
<td>continuous wave Doppler</td>
</tr>
<tr>
<td>DBP</td>
<td>diastolic blood pressure</td>
</tr>
<tr>
<td>depT</td>
<td>depressurization time</td>
</tr>
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</table>
List of abbreviations

DF  duty factor
DT  deceleration time
EF  ejection fraction
ERO effective regurgitant orifice
ET  ejection time
$f_D$ Doppler frequency
FD  focal depth
FO  foramen ovale
FS  fractional shortening
HOCM hypertrophic obstructive cardiomyopathy
HV  hepatic vein
HVLT half value layer thickness
$I$ intensity
IAS  interatrial septum
ICU  intensive care unit
IHD  ischaemic heart disease
IPP intrapericardial pressure
IRC intensity reflection coefficient
ITC intensity transmitted coefficient
IVC  inferior vena cava
IVRT isovolumic relaxation time
IVS  interventricular septum
LA  left atrium
LAA  left atrial appendage
LAD  left anterior descending coronary artery
LAP  left atrial pressure
LARRD longitudinal resolution
LATA lateral resolution
LAX  long axis view
LBBB  left bundle branch block
LCA  left coronary artery
LCC  left coronary cusp
LCCA left common carotid artery
LCx  left circumflex coronary artery
LGC lateral gain compensation
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>LLPV</td>
<td>left lower pulmonary vein</td>
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<td>LPA</td>
<td>left pulmonary artery</td>
</tr>
<tr>
<td>LSCA</td>
<td>left subclavian artery</td>
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<td>LSE</td>
<td>left sternal edge</td>
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<td>LUPV</td>
<td>left upper pulmonary vein</td>
</tr>
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<td>LV</td>
<td>left ventricle</td>
</tr>
<tr>
<td>LVEDP</td>
<td>left ventricular end diastolic pressure</td>
</tr>
<tr>
<td>LVEDV</td>
<td>left ventricular end diastolic volume</td>
</tr>
<tr>
<td>LVESV</td>
<td>left ventricular end systolic volume</td>
</tr>
<tr>
<td>LVH</td>
<td>left ventricular hypertrophy</td>
</tr>
<tr>
<td>LVIDd</td>
<td>left ventricular internal diameter in diastole</td>
</tr>
<tr>
<td>LVIDs</td>
<td>left ventricular internal diameter in systole</td>
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<td>LVM</td>
<td>left ventricular mass</td>
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<td>LVOT</td>
<td>left ventricular outflow tract</td>
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<tr>
<td>LVP</td>
<td>left ventricular pressure</td>
</tr>
<tr>
<td>LVSP</td>
<td>left ventricular systolic pressure</td>
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<tr>
<td>MAPSE</td>
<td>mitral annular plane systolic excursion</td>
</tr>
<tr>
<td>MG</td>
<td>mean gradient</td>
</tr>
<tr>
<td>MI</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>MM</td>
<td>motion mode</td>
</tr>
<tr>
<td>MR</td>
<td>mitral regurgitation</td>
</tr>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
</tr>
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<td>MV</td>
<td>mitral valve</td>
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<tr>
<td>MVA</td>
<td>mitral valve area</td>
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<td>MVC</td>
<td>mitral valve closes</td>
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<td>MVL</td>
<td>mitral valve leaflet</td>
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<td>MVO</td>
<td>mitral valve opens</td>
</tr>
<tr>
<td>NCC</td>
<td>non-coronary cusp</td>
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<tr>
<td>P</td>
<td>power</td>
</tr>
<tr>
<td>PA</td>
<td>pulmonary artery</td>
</tr>
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<td>PADP</td>
<td>pulmonary artery diastolic pressure</td>
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<td>PAP</td>
<td>pulmonary artery pressure</td>
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<tr>
<td>PD</td>
<td>pulse duration</td>
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<tr>
<td>PDA</td>
<td>patent ductus arteriosus</td>
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<td>PE</td>
<td>pulmonary embolism</td>
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## List of abbreviations

<table>
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<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>P/E</td>
<td>piezo-electric</td>
</tr>
<tr>
<td>PFO</td>
<td>patent foramen ovale</td>
</tr>
<tr>
<td>PG</td>
<td>pressure gradient</td>
</tr>
<tr>
<td>PHT</td>
<td>pressure half-time</td>
</tr>
<tr>
<td>PI</td>
<td>pulmonary incompetence</td>
</tr>
<tr>
<td>PISA</td>
<td>proximal isovelocity area</td>
</tr>
<tr>
<td>PM</td>
<td>papillary muscle</td>
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<td>P/M</td>
<td>postero-medial</td>
</tr>
<tr>
<td>PMVL</td>
<td>posterior mitral valve leaflet</td>
</tr>
<tr>
<td>PRF</td>
<td>pulse repetition frequency</td>
</tr>
<tr>
<td>PRP</td>
<td>pulse repetition period</td>
</tr>
<tr>
<td>PS</td>
<td>pulmonary stenosis</td>
</tr>
<tr>
<td>PV</td>
<td>pulmonary valve</td>
</tr>
<tr>
<td>PVs</td>
<td>pulmonary veins</td>
</tr>
<tr>
<td>PW</td>
<td>pulse wave</td>
</tr>
<tr>
<td>PWD</td>
<td>pulse wave Doppler</td>
</tr>
<tr>
<td>PZT-5</td>
<td>lead zirconate titanate – 5</td>
</tr>
<tr>
<td>RA</td>
<td>right atrium</td>
</tr>
<tr>
<td>RAP</td>
<td>right atrial pressure</td>
</tr>
<tr>
<td>RBBB</td>
<td>right bundle branch block</td>
</tr>
<tr>
<td>rbc</td>
<td>red blood cell</td>
</tr>
<tr>
<td>RCA</td>
<td>right coronary artery</td>
</tr>
<tr>
<td>RCC</td>
<td>right coronary cusp</td>
</tr>
<tr>
<td>RF</td>
<td>regurgitant fraction</td>
</tr>
<tr>
<td>RLN</td>
<td>recurrent laryngeal nerve</td>
</tr>
<tr>
<td>RLPV</td>
<td>right lower pulmonary vein</td>
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<td>RPA</td>
<td>right pulmonary artery</td>
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<td>RSE</td>
<td>right sternal edge</td>
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<td>RUPV</td>
<td>right upper pulmonary vein</td>
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<tr>
<td>RV</td>
<td>right ventricle</td>
</tr>
<tr>
<td>RVH</td>
<td>right ventricular hypertrophy</td>
</tr>
<tr>
<td>RVOT</td>
<td>right ventricular outflow tract</td>
</tr>
<tr>
<td>RVP</td>
<td>right ventricular pressure</td>
</tr>
<tr>
<td>RVSP</td>
<td>right ventricular systolic pressure</td>
</tr>
<tr>
<td>RWMA</td>
<td>regional wall motion abnormality</td>
</tr>
</tbody>
</table>
SAM  systolic anterior motion
SAN  sino-atrial node
SAPA spatial average, pulse average
SATA spatial average, temporal average
SATP spatial average, temporal peak
SAX  short axis view
SBP  systolic blood pressure
SCA  sickle cell anaemia
SLE  systemic lupus erythematosus
SPL  spatial pulse length
SPPA spatial peak, pulse average
SPTA spatial peak, temporal average
SPTP spatial peak, temporal peak
STJ  sino-tubular junction
SV   stroke volume
SVI  stroke volume index
SVR  systemic vascular resistance
TA   truncus arteriosus
TAA  thoracic aortic aneurysm
TAPSE tricuspid annular plane systolic excursion
TAPVD total anomalous pulmonary venous drainage
TB   tuberculosis
Td   time delay
TDI  tissue Doppler imaging
TGA  transposition of great arteries
TGC  time gain compensation
TMF  transmirtal flow
TOE  transoesophageal echocardiography
TR   tricuspid regurgitation
TS   tricuspid stenosis
TTE  transthoracic echocardiography
TTF  transtricuspid flow
TV   tricuspid valve
TVA  tricuspid valve area
TVC  tricuspid valve closes
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>TVL</td>
<td>tricuspid valve leaflet</td>
</tr>
<tr>
<td>TVO</td>
<td>tricuspid valve opens</td>
</tr>
<tr>
<td>TX</td>
<td>transducer</td>
</tr>
<tr>
<td>U/S</td>
<td>ultrasound</td>
</tr>
<tr>
<td>Vcf</td>
<td>velocity of circumferential fibre shortening</td>
</tr>
<tr>
<td>VSD</td>
<td>ventricular septal defect</td>
</tr>
<tr>
<td>VTI</td>
<td>velocity–time integral</td>
</tr>
<tr>
<td>WPW</td>
<td>Wolfe–Parkinson–White syndrome</td>
</tr>
<tr>
<td>Z</td>
<td>impedance</td>
</tr>
</tbody>
</table>
Over the past decade there has been a dramatic increase in the use of transoesophageal echocardiography (TOE) in the perioperative setting among all disciplines caring for the cardiac patient. Where TOE used to be used mainly by cardiologists in the echocardiography laboratory, we now recognize its value in the operating theatre, cardiac catheter laboratory, and intensive care unit. TOE has become the gold standard perioperative cardiac monitor and diagnostic tool for certain cardiac surgical procedures. Its role has also been extended to critically ill or unstable patients for non-cardiac procedures and the general intensive care arena. The increasing involvement of anaesthetists and of other specialities at an advanced level has promoted the team approach to perioperative patient care. The rapid advances in the use of this technology have also resulted in a critical need for interdisciplinary training.

The development of training and certification in echocardiography has been a long and intensive process in Europe and the USA. Excellent comprehensive TOE courses have been available and working groups on TOE have published extensive practice and training guidelines on both sides of the Atlantic and in Japan. The American Society of Cardiovascular Anesthesiologists (SCA) developed the first formal examination in perioperative TOE in 1998. The SCA and the American Society of Echocardiography (ASE) then combined forces to establish the National Board of Echocardiography (NBE), which had the responsibility to further administer examinations and develop a certification process in clinical echocardiography. Europe followed a similar route with the Association of Cardiothoracic Anaesthetists (ACTA) joining forces with the British Society of Echocardiography
(BSE) to establish an accreditation process in TOE with its first examination held in the UK in 2003. Since then the European Association of Cardiothoracic Anaesthesiologists (EACTA) and the European Society of Echocardiography (ESE) produced its own European TOE examination and accreditation process in 2005. In 2004, the Japanese Society of Cardiovascular Anesthesiologists launched their first TOE competency examination.

The purpose of these accreditation processes is to enable recognition of special competence in perioperative echocardiography against an objective standard, and all of them consist of two parts. With the practical part, the candidate must demonstrate adequate training and competency through a supervised residency program or logbook. The theoretical part requires the successful completion of a multiple choice and image clip examination.

With his experience in learning, practicing and teaching perioperative echocardiography in North America and in the UK, the author fills a certain niche with this book. It is not intended to be a comprehensive reference book. In contrast to the vast amount of information on echocardiography already available both in print and online, this book provides the aspiring echocardiographer with a valuable summarized resource to prepare for any of the perioperative echocardiography examinations. It gives any examination candidate a convenient framework onto which further knowledge can be added. Both the American and the European perioperative TOE examination syllabus is well covered in a concise manner. The *Perioperative Transoesophageal Echocardiography Exam Notes* contains all the critical physics equations, standard values and plenty of diagrams in a highly absorbable way. Each chapter also concludes with a series of exam-style self-assessment questions to emphasize important facts and practice for the exam.

Cardiac surgery and anaesthesia have come a long way since the late 1970s when TOE was introduced into the perioperative arena. The development of many surgical procedures and the reduction in perioperative morbidity and mortality can be directly related to the use
of TOE. There rests a great responsibility on any clinician performing a diagnostic perioperative TOE. This book will certainly contribute not only to help preparation for the examinations, but also to raise the standard of our practice and patient care.

Steve Konstadt
Justiaan Swanevelder
Basic principles

Nature of ultrasound

Sound = longitudinal, mechanical wave
particles move parallel to direction of travel

Audible sound < 20 kHz
Ultrasound > 20 kHz
Sound cannot travel through a vacuum

Four acoustic variables

Density (g/l)
Pressure (kPa)
Temperature (K)
Particle motion (m)

Compressions: high density/pressure/temperature/motion +
Rarefactions: low density/pressure/temperature/motion
(Fig. 1.1)

Transthoracic echo (TTE) ∼ 2–5 MHz
Transoesophageal echo (TOE) ∼ 3.5–7 MHz

Sound is described by
Propagation speed (m/s)
Frequency (Hz)
Wavelength (m)
**Transoesophageal Echocardiography**

**Fig. 1.1**

- Period (s)
- Amplitude (kPa, g/l, K, m, dB)
- Power (W)
- Intensity (W/cm²)

**Propagation speed (v or c)**

\[ c = \text{speed of sound} \quad \text{Units} = \text{m/s or mm/µs} \]

Determined by the medium through which the wave travels

Soft tissue (heart) = 1540 m/s = 1.54 mm/µs

Speed affected by density and stiffness of medium

\[ \uparrow \text{density} \rightarrow \downarrow \text{speed} \]

\[ \uparrow \text{stiffness} \ (= \text{bulk modulus}) \rightarrow \uparrow \text{speed} \]

Elasticity and compressibility = opposite to stiffness

\[ \uparrow \text{elasticity/compressibility} \rightarrow \downarrow \text{speed} \]

All sound travels through a specific medium at the same speed

(Table 1.1)
Table 1.1 Speed of sound in different media

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Speed of sound (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>331</td>
</tr>
<tr>
<td>Lung</td>
<td>500</td>
</tr>
<tr>
<td>Fat</td>
<td>1450</td>
</tr>
<tr>
<td>Brain</td>
<td>1541</td>
</tr>
<tr>
<td>Liver</td>
<td>1549</td>
</tr>
<tr>
<td>Muscle</td>
<td>1585</td>
</tr>
<tr>
<td>Bone</td>
<td>&gt;3000</td>
</tr>
</tbody>
</table>

**Frequency** ($f$)

$f =$ number of cycles per second  Units = Hz

U/S $>20$ kHz

Determined by sound source

Affects penetration and axial resolution

**Period** ($T$)

$T =$ length of time to complete one cycle  Units = s

U/S $=0.1–0.5 \mu$s

Determined by sound source

Reciprocal of frequency  $T = 1/f$

**Wavelength** ($\lambda$)

$\lambda =$ distance occupied by a single cycle  Units = m

U/S $=0.1–0.8$ mm

Determined by sound source and medium

$\lambda$ influences axial resolution

Velocity ($v$), frequency ($f$) and wavelength ($\lambda$) associated by the equation

$$v = f\lambda$$
**Amplitude (A)**

\[ A = \text{max. variation in acoustic variable} \quad \text{Units} = \text{kPa, g/l, K, m, dB}, \]

i.e. difference between mean and max. values

(Fig. 1.2)

Decibel (dB) = logarithmic relative unit of measure of A

i.e. difference between two values

e.g. ↑ by 30 dB = ↑A by 10 \times 10 \times 10 \times 1000

Determined by sound source

Changed by sonographer

Amplitude decreases as sound wave travels = attenuation

(Fig. 1.3)

**Power (P)**

\[ P = \text{rate of work/rate of energy transfer} \quad \text{Units} = \text{W} \]
Fig. 1.4

Intensity ($I$)

$I = \text{concentration of energy/power in a sound beam}$

Units = W/cm$^2$

Determined by sound source

Changed by sonographer

$U/S \quad I = 0.1–100 \text{ mW/cm}^2$

$I = P/\text{area}$

Pulsed ultrasound

Pulse = collection of cycles travelling together

individual ‘cycles’ make up the ‘pulse’

‘pulse’ moves as one

‘pulse’ has beginning and end

Two components:

‘cycle’ or ‘on’ time

‘receive’ or ‘off’ or ‘dead’ time (Fig. 1.4)

Pulsed U/S described by:

pulse duration (PD)

pulse repetition frequency (PRF)

pulse repetition period (PRP)
spatial pulse length (SPL)
duty factor (DF)

**Pulse duration (PD)**

\[
PD = \text{time from start of one pulse to end of pulse} \quad \text{Units} = \text{s}
\]

\[
= \text{‘on’ time (Fig. 1.5)}
\]

Determined by:
- number of cycles in a pulse (‘ringing’)
- period of each cycle

Characteristic of transducer/not changed by sonographer

TOE PD = 0.5–3 µs

\[
PD = \text{number of cycles} \times T \quad PD = \text{number of cycles} / f
\]

**Pulse repetition frequency (PRF)**

\[
PRF = \text{number of pulses per second} \quad \text{Units} = \text{Hz}
\]

(Number of cycles per pulse *not* relevant)

Determined by sound source

Changed by sonographer by changing image depth

As image depth increases \( \rightarrow \) PRF↓

Sonographer ↑‘dead’ time by ↑image depth = ↓PRF

TOE \( \text{PRF} = 1–10 \text{ kHz} \)

\[
PRF(\text{kHz}) = 75 / \text{depth (cm)}
\]
Pulse repetition period (PRP)
PRP = time from start of one pulse to start of next pulse
Units = s
PRP = ‘on’ time (PD) + ‘off’ time (Fig. 1.5)
Changed by sonographer by changing ‘off’ time
TOE  PRP = 0.1–1 ms

\[
PRP \ (\text{µs}) = 13 \times \text{depth (cm)}
\]

Spatial pulse length (SPL)
SPL = length in distance occupied by one pulse  Units = m
Determined by sound source and medium
Cannot be changed by sonographer
TOE  SPL = 0.1–1 mm
Determines axial resolution
i.e. short SPL → better axial resolution

\[
SPL = \text{number of cycles} \times \lambda
\]

Duty factor (DF)
DF = percentage of ‘on’ time compared to PRP  Units = %
Changed by sonographer by changing ‘off’ time
TOE  DF = 0.1–1% (i.e. lots of ‘off’/listening time)

\[
DF = \frac{PD}{PRP}
\]

↑DF by:
↑PRF (more pulses/s)
↑PD (by changing transducer)
↓DF by:
↑PRP (by ↑‘off’ time)
↑image depth
DF = 100% = continuous wave (CW) U/S
DF = 0% = machine off
Properties of ultrasound

Intensity ($I$)
Described by:

1. Spatial – U/S beam has different $I$ at different locations (Fig. 1.6)
   - Peak $I = \text{spatial peak (SP)}$
   - Average $I = \text{spatial average (SA)}$

2. Temporal – U/S beam has different $I$ at different points in time
   (Fig. 1.7)
   - Peak $I = \text{temporal peak (TP)}, \text{i.e. ‘on’ time}$
   - Average $I = \text{temporal average (TA)}, \text{i.e. average of ‘on’ and ‘off’}$
   - For CW: TP = TA

3. Pulse – U/S beam has average $I$ for duration of pulse (‘on’)
   $= \text{pulse average (PA)}$
SPTA relevant to tissue heating
For CW: SPTP = SPTA and SATP = SATA
When PW and CW have same SPTP/SATP
    CW has higher SPTA/SATA
PA > TA for PW

**Beam uniformity ratio (BUR)**

BUR = SP/SA factor
No units
Scale 1–∞ (infinity)
Describes the spread of sound beam in space
TOE BUR = 5–50

**Attenuation**

Decrease in A/P/I as sound wave travels (Fig. 1.3)
Units = −dB
In soft tissue: ↑f → ↑attenuation
Three components:
1. absorption:
   energy transferred to cell in tissue by conversion to other form of energy
   sound → heat/vibration
2. reflection:
   energy returned to source when it strikes a boundary between two media
(i) Specular reflections

![Specular reflections diagram]

U/S Specular U/S with Specular reflection small SPL reflection Smooth surface Rough surface

(ii) Scatter

![Scatter diagram]

U/S with high SPL Scatter U/S with SPL >> rbc Rayleigh scattering Rough surface rbc

Fig. 1.8

(3) scatter:

sound beam hits rough surface → sound wave redirected in several directions

Rayleigh scattering = when reflector << SPL (e.g. red blood cells)
→ scattering equal in all directions

(Fig. 1.8)

Attenuation coefficient (AC)

Units = −dB/cm

In soft tissue: ↑f → ↑AC

\[ AC = 0.5 \times f \text{ (MHz)} \]

Total attenuation = AC × path length (cm)
↑AC in: bone (absorption and reflection)
air/lung (scatter)
Table 1.2 Effect of transducer frequency on attenuation coefficient (AC) and half-value layer thickness (HVLT)

<table>
<thead>
<tr>
<th>Transducer f (MHz)</th>
<th>AC (dB/cm)</th>
<th>HVLT (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>1.5</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>5</td>
<td>2.5</td>
<td>1.2</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

Half value layer thickness (HVLT)

\[
HVLT = \text{depth at which } I \text{ falls by } \frac{1}{2} = -3 \text{ dB} \quad \text{Units} = \text{cm}
\]
(also called penetration depth and half boundary layer)

TOE  \( HVLT = 0.25–2 \text{ cm (Table 1.2)} \)

\[HVLT = \frac{3}{AC} \quad HVLT = \frac{6}{f}\]

Impedance (Z)

\[Z = \text{resistance to sound propagation} \quad \text{Units} = \text{Rayls}\]

Calculated/not measured

Soft tissue = 1.25–1.75 MRayls

Reflection depends upon change in Z between two media

(Fig. 1.9)

\[Z = \rho \times v \text{ (density } \times \text{ velocity)}\]

Intensity reflection coefficient (IRC)

\[\text{IRC (\%)} = \frac{\text{reflected } I}{\text{incident } I}\]

Intensity transmitted coefficient (ITC)

\[\text{ITC (\%)} = \frac{\text{transmitted } I}{\text{incident } I}\]

Clinically: Soft tissue  \( \text{IRC} = 1\% \quad \text{ITC} = 99\% \)

Bone  \( \text{IRC} = 99\% \quad \text{ITC} = 1\% \)

With a 90° incident angle, reflection only occurs if \( Z_1 \neq Z_2 \)

Greater the difference between \( Z_1 \) and \( Z_2 \) → ↑IRC

\[\text{IRC (\%)} = \left(\frac{Z_2 - Z_1}{Z_2 + Z_1}\right)^2\]
Incident $I$ = transmitted $I$ + reflected $I$

**Fig. 1.9**

With an oblique angle of incidence $→$ reflection and refraction

Reflection: incident angle = reflected angle (Fig. 1.10)

Refraction: obeys Snell’s Law (Fig. 1.11)

Velocity of transmitted beam > incident beam if $t > i$

**Range**

= time taken for pulse to travel from transducer to reflector and back to transducer

= ‘go–return’ time

Distance to boundary (mm) = $v$ (mm/µs) × range (µs)/2

$D = 1.54 \times \text{range}/2$

$D = 0.77 \times \text{range}$

13 µs rule: range = 13 µs $→$ reflector depth = 10 mm

= 26 µs $→$ = 20 mm

= 39 µs $→$ = 30 mm
Transducers

Basic principles

Transducer (TX) = converts energy from one form to another
- acoustic → kinetic → electrical → heat

Piezoelectric (P/E) effect
- ability of a material to create a voltage when mechanically deformed
Reverse P/E effect = material changes shape when voltage applied
P/E materials = ferroelectric

Natural P/E materials
- quartz, Rochelle salts, tourmaline
Synthetic = Ba titanate, Pb titanate, Pb zirconate titanate (PZT)
U/S imagers – PZT-5 (also called ‘ceramic’)

Curie temperature
- temperature above which the P/E material loses its P/E effect because it depolarizes

Therefore: TX cannot be heated/sterilized/autoclaved
Ultrasound transducers (Fig. 1.12) composed of:

1. active element: P/E crystal (PZT-5)
2. case:
   - protects internal components
   - insulates patient from electrical currents
3. wire:
   - provides electrical contact with P/E crystal
   - voltage from U/S system → vibration → U/S wave
   - reception of signal → vibration → voltage to wire
4. matching layer:
   - has impedance ($Z$) in-between that of TX and skin to prevent large reflection at skin
   - $Z$ of TX $\approx$ 33 MRayls
   - $Z$ of skin $\approx$ 1.5 MRayls
   - $\rightarrow$ 96% IRC at skin
   - $Z$ of matching layer $\approx$ 7 MRayls
   - Thickness of matching layer = $\lambda/4$
   - Improves axial resolution
5. damping element:
   - material bonded to active element
   - epoxy resin impregnated with tungsten limits ‘ringing’
   - Improves axial resolution
‘Ringing’

\[ \text{P/E crystals have prolonged response to excitation} \]
\[ \rightarrow \uparrow \text{PD} \rightarrow \text{reduced axial resolution} \]

Length of ‘ringing’ response = ‘ringdown’
\[ = \text{number of half cycles required for oscillations of P/E crystal to decay} \]
\[ \text{to } 10\% \text{ (} \sim 20 \text{ dB} \text{) of the max peak-to-peak amplitude} \]

\[ \text{Damping} \rightarrow \downarrow \text{ringdown} \]
\[ \rightarrow \text{absorbs U/S emitted from back face of TX, which causes} \]
\[ \text{interference by reflecting within housing of TX} \]

**Transducer frequencies**

Resonant \( f \) of TX depends on thickness of P/E crystal

Max resonance occurs when thickness \( = \frac{\lambda}{2} \)

CW U/S: U/S \( f \) determined by and equal to \( f \) of voltage applied to P/E crystal

PW U/S: PRF determined by number of electrical pulses the machine delivers to P/E crystal

\( f \) of U/S determined by:

- thickness (\( \frac{\lambda}{2} \))
- \( c \) in P/E crystal (\( \sim 4-6 \text{ mm/} \mu\text{s} \))

\[ f (\text{MHz}) = \frac{c \text{ (mm/} \mu\text{s})}{2} \times \text{thickness (mm)} \]

**Sound beams**

Beam diameter:

- starts same size as TX
- converges to focus
- diverges away from focus

Focus = location at minimum diameter (Fig. 1.13)

Focal depth (FD) = distance from TX to focus
Near zone = Fresnel zone
Far zone = Fraunhofer zone

At focus: $d_2 = d_1 / 2$

**Fig. 1.13**

$\uparrow$TX diameter $\rightarrow$ $\uparrow$FD/$\downarrow$divergence

$\downarrow$TX diameter $\rightarrow$ $\downarrow$FD/$\uparrow$divergence

**Fig. 1.14**

Focal depth determined by:
- TX diameter
- $f$ of U/S

$$FD = TX\ diam^2 \times f/6$$

**Sound beam divergence**

$\uparrow$TX diameter $\rightarrow$ $\uparrow$FD/$\downarrow$divergence

$\downarrow$TX diameter $\rightarrow$ $\downarrow$FD/$\uparrow$divergence (Fig. 1.14)

**Focusing**
- $=$ changing FD
- $\downarrow$FD $\rightarrow$ $\downarrow$diameter of beam
- Lateral resolution improved by focusing
Types of focusing: (Fig. 1.15)

1. External focusing = lens
2. Internal focusing = curved P/E crystal
3. Focusing mirror
4. Electronic focusing = phased array
   → dynamic variable focusing
   → adjustable by sonographer
   → better resolution

**Arrays**

Array = collection of active elements in one TX  
(single slab of PZT-5 cut into small pieces)

Each active element is connected to its own electronic circuitry
Linear = elements in a line: linear switched array
    linear phased array

Annular = elements with a common centre in a ring

Convex (curved) = collection in curved manner
    convex switched array
    convex linear array

**Linear switched array (Fig. 1.16)**
Large TX with elements arranged in a line
Image no wider than TX with a rectangular image
P/E crystals fire in sequence to give 2-D image
No steering/fixed vertical focusing
Defective crystal causes vertical dropout

**Phased arrays (Fig. 1.17)**
Collection of electric pulses delivered to the active elements in various
    patterns, which focus and steer U/S pulse
Fan-shaped image
Many signals excite multiple crystals → one sound pulse
If one element breaks → erratic focusing/steering
Small time delays (nearly simultaneous) between electronic pulses
delivered to array elements
Time delays during reception applied to electrical signals returning from TX to machine
‘Reception zone’ focusing can be matched to depth of returning echoes and optimizes image quality
Electronic curvature → focusing
Electronic slope → steering (Fig. 1.18)

**Annular phased arrays**
Concentric rings cut from circular slab of PZT-5
Small diameter → shallow focus (↓FD) and rapid divergence
Large diameter → ↑FD

Selected focal zones:
- inner crystals → shallow focus
- outer crystals → deep focus

Fan-shaped image
Electronic focusing/mechanical steering
Defective crystal causes horizontal dropout (Fig. 1.19)

**Convex curved array (Fig. 1.20)**
P/E crystals in curve → natural sector shape
Steering = slope

Focusing = curvature

Fig. 1.18
Convex switched:
  - sequential (large TX)
  - no steering/fixed focusing
  - defective crystal $\rightarrow$ vertical dropout

Blunted-fan image
Convex phased (small TX): electronic steering and focusing
Imaging

Resolution

Longitudinal resolution

- Longitudinal
- Axial
- Range
- Radial
- Depth

LARRD resolution

Ability to distinguish two reflectors as separate entities parallel to U/S beam (Fig. 1.21)

Determined by source ($f$) and medium ($\lambda$)

$$LARRD = 0.05\text{–}0.5 \text{ mm}$$

Improve LARRD resolution (i.e. $\downarrow$LARRD distance) by:

- $\uparrow f \rightarrow \downarrow \lambda \rightarrow \downarrow \text{SPL} \rightarrow \downarrow \text{LARRD distance}$
- $\downarrow \text{ringing} \rightarrow \downarrow \text{SPL} \rightarrow \downarrow \text{LARRD distance}$

$$\text{LARRD (mm)} = \frac{\text{SPL}}{2}$$

$$\text{LARRD (mm)} = 0.77 \times \text{ringing/} f \text{ (MHz)}$$
Lateral resolution

Ability to distinguish two reflectors as separate entities perpendicular to U/S beam (Fig. 1.22)

LATA depends on beam width
LATA better when beam narrow
LATA optimal at FD (beam narrowest)
LATA varies with depth

When two reflectors are closer together than beam width, only one object is seen on image
LATA distance > LARRD distance (i.e. LARRD resolution is better than LATA resolution) because beam width > SPL
↑A/P/I → ↑LATA distance (i.e. degrades LATA resolution)

Temporal resolution

= frame rate, i.e. number of frames per second
1 pulse → 1 scan line → 1 image line
100 lines/frame = 100 pulses/frame → 1 picture
Not true for multiple focus beam systems and colour imaging because
multiple pulses needed per scan line

Factors affecting temporal resolution

(1) number of pulses/scan line
(2) max. imaging depth
(3) sector size
(4) line density (lines/angle of sector)

↑ frame rate (better temporal resolution) by

(1) single focus, i.e. 1 pulse/scan line
(2) shallower image depth
(3) reduce sector size
(4) reduce line density

↓ frame rate (worse temporal resolution) by

(1) multifocus, e.g. colour flow imaging
(2) increase image depth, e.g. 6 cm → 12 cm → \( \frac{1}{2} \) frame rate
(3) increase sector size
(4) increase line density

TOE temporal resolution = 30–60 frames/second on 2-D image
< 15 frames/second → ‘flickering’

Display modes

A Mode (Fig. 1.23)
= amplitude mode
U/S pulse emitted → ‘dot’ moves across screen at constant speed
Echo returns → upward deflection of ‘dot’ proportional to amplitude of echo
B Mode (Fig. 1.24)
= brightness mode
Returning echoes appear as ‘spots’ on line of travel of emitted U/S pulse
Brightness of ‘spot’ proportional to amplitude

M Mode (Fig. 1.25)
= motion mode
Dragging photosensitive paper across B mode creates lines instead of dots, giving motion of reflected surfaces occurring in time
High temporal resolution = 1000×/second
Ideal for imaging localized areas of heart and analysing time-related events

2-D imaging
Multiple narrow beams of pulsed U/S
B mode can be moved through path by sonographer to create 2-D picture, but slow and patient movement causes artefacts

Real-time imaging
U/S system steers beam through pathway
Multiple scan lines gives 2-D image at 30–60 frames/s

3-D echo
Requires:
- sequential acquisition of 2-D data from multiple planes
- digitization of data and off-line reconstruction
- Time-consuming

Instrumentation

Six components:
- Transducer (TX)
- Pulser
- Receiver
- Display
- Storage
- Master synchronizer (M/S)

Transducer
Transmission: electrical → acoustic energy
Reception: acoustic → electrical energy
**Pulser**

Controls electrical signals sent to TX for pulse generation
Receives signal from M/S

Determines:
- PRF/PRP
  - Amplitude (↑voltage → ↑A)
  - Firing pattern for phased array TX

CW: constant electrical sine wave signal
PW (single crystal): one electrical ‘spike’ → one pulse
PW (arrays): many ‘spikes’ → one pulse

**Receiver**

Signals returning back from TX are weak
Therefore, needs ‘boosting’, ‘processing’ and ‘preparing’ for display

1. **Amplification**
   - ↑Gain → every signal amplified (Fig. 1.26)
     Changed by sonographer
2. **Compensation**
   - Attenuation proportional to image depth
     Deep image → ↓A

Changed by sonographer

1. **Time-gain compensation (TGC) = ‘depth’ compensation**
   - Amplifies signal from deeper objects (Fig. 1.27)
(2) Lateral gain compensation (LGC) = ‘lateral’ compensation
   Allows application of gain to selected sectors

(3) Compression = dynamic range manipulation (Fig. 1.28)
   Process of reducing total range of received echo amplitudes
   Keeps signal within operating range
   Does not alter relationship between voltages
   Converts linear scale to log scale → uniformity of signals

(4) Demodulation
   Changes signal into form suitable for display
   ‘Rectification’ = negative to positive voltage
   ‘Enveloping’ = ‘smoothing’ of signal
   ‘Leading edge enhancement’ = narrower and brighter image

(5) Rejection = filtering (Fig. 1.29)
   Low A signals associated with ‘noise’ rejected
Display
Cathode ray tubes (CRT) = TV screens (525 horizontal lines)
Electron beam strikes phosphor coating on screen → light
(1) interlaced: odd number lines filled in first, then even
(2) non-interlaced: lines filled in sequentially

Storage
Cine memory – captures short sequences in digital memory
Videotape – analog format
DVD – 1 frame = 1 Mbyte, large memory needed

Master synchronizer
Communicates with all components and organizes

Doppler
Principles
Doppler effect:
The frequency of a sound wave reflected by a moving object is different from that emitted
= frequency shift/Doppler frequency ($f_D$)
The magnitude and direction of $f_D$ is related to the velocity and direction of the moving object (Fig. 1.30)

$$f_D = 2 \nu v f_0 \cos \theta/c$$
Objects moving towards TX:  $f_0 \uparrow \lambda$

Positive above baseline

Objects moving away from TX:  $f_0 \downarrow \lambda$

Negative below baseline

\[ v = \text{velocity of rbc} \]
\[ c = 1540 \text{ m/s} \]
\[ f_D = \text{frequency shift} \]
\[ f_0 = \text{emitted frequency} \]
\[ \theta = \text{angle of incidence} \]

Parallel beam (0° and 180°) → $\cos \theta = 1$

Perpendicular beam (90° and 270°) → $\cos \theta = 0$

Angle of incidence $< 20°$ → $< 6\%$ error

Measured velocity = true velocity $\times \cos \theta$

\[ v = c \frac{f_D}{2} f_0 \cos \theta \]

Unidirectional Doppler measures presence of moving rbc by Doppler shift, but cannot distinguish +ve or –ve, i.e. unidirectional
Bidirectional Doppler distinguishes +ve from −ve

TOE $f_D = 20–20000 \text{ Hz (i.e. audible)}$

**Pulse wave Doppler**

PW: one crystal emits and receives at specific PRF
   blood flow parameters at specific point (sample volume)

(1) mechanical sector scanners: TX stopped to record signal
(2) phased array:
   uses missing signal estimator (MSE)
   Doppler ‘on’ for 10 ms $\rightarrow$ Doppler signal
   2-D image ‘on’ for 20 ms $\rightarrow$ 2-D image
   total time = 30 ms $\rightarrow$ 30 frames/second
   MSE gives synthesized signal during 2-D 20 ms

Pulsed Doppler ‘interrogates’ target once per PRP
Time delay ($T_d$), from emission of U/S beam to reception of signal,
determines depth at which flow is sampled

\[
\text{Depth} = \frac{c T_d}{2}
\]

PWD = good for velocities $< 2 \text{ m/s}$
Velocities $> 2 \text{ m/s}$ $\rightarrow$ ‘aliasing’ artefact

**High pulse repetition frequency**
= modification of PWD
2–5 samples simultaneously

Allows:
\[\uparrow f \text{ because TX does not wait for return of signals before sending next pulse}\]
\[\uparrow \text{max velocity before ‘aliasing’ occurs}\]
BUT – ‘range ambiguity’, i.e. do not know exactly where along pathway
   signal is returning from
Transoesophageal Echocardiography

Fig. 1.31

‘Aliasing’
When \( f_D \) exceeds certain limit, ‘aliasing’ (wraparound) occurs
High velocities appear negative (Fig. 1.31)
\( f_D \) at which aliasing occurs = Nyquist limit (frequency) = \( f_N \)

\[
f_N = \frac{PRF}{2}
\]

When \( f_D > f_N \) → ‘aliasing’/wraparound artefact

Reduce aliasing by
- Use TX with \( \downarrow f \)
- Shallower depth (D) → \( \uparrow PRF \)
- Use CW
- Baseline shift

Max velocity (\( V_{max} \)) before aliasing occurs is given by:

\[
V_{max} = \frac{c^2}{8 f_o}
\]

\( \downarrow f_0 \) → \( \uparrow V_{max} \) → \( \downarrow \) aliasing
\( \downarrow \) Depth → \( \uparrow V_{max} \) / \( \uparrow PRF \) → \( \uparrow f_N \) → \( \downarrow \) aliasing

Continuous wave Doppler

CW uses two crystals:
1. transmitter
2. receiver
Allows high $V_{\text{max}}$ (up to 9 m/s) without aliasing
BUT → ‘range ambiguity’

<table>
<thead>
<tr>
<th>PW</th>
<th>vs.</th>
<th>CW</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) one crystal</td>
<td></td>
<td>two crystals</td>
</tr>
<tr>
<td>(2) range resolution</td>
<td></td>
<td>range ambiguity</td>
</tr>
<tr>
<td>(3) $V_{\text{max}} &lt; 2$ m/s</td>
<td></td>
<td>$V_{\text{max}}$ up to 9 m/s</td>
</tr>
</tbody>
</table>

**Colour flow imaging**

‘Real-time’ blood flow as colour on 2-D image
→ location, direction, velocity and laminar or turbulent flow

Based on multi-gated PWD, therefore:
- range resolution
- subject to aliasing

Multiple pulses → one Doppler packet → mean velocity of rbc
↑no. of pulses/packet → ↑accuracy of velocity
BUT ↑pulses/packet → ↓frame rate

 Colour assigned to velocity depends on direction/flow type

Traditionally –
- red = towards TX
- blue = away from TX
- green hue (variance mode) = turbulence

**LARRD vs. velocity resolution**

Short SPL → better LARRD
Long SPL → better velocity resolution

**Depth vs. PRF**

Depth inversely proportional to PRF
Velocity resolution/depth/line density/frame rate
Many pulses down each line, averaged to give mean velocity

\[ n \times \text{PRP} \times N \times F = 1 \]

\[ n = \text{pulses/line} \]
\[ \text{PRP} = 1/\text{PRF} \]
\[ N = \text{lines/frame} \]
\[ F = \text{frame rate} \]

Therefore, increase in one parameter leads to decrease in others

Tissue Doppler imaging (TDI)

Three modalities:
- Pulse wave-TDI (PW-TDI)
- 2-dimensional-TDI (2-D-TDI)
- M mode-TDI (MM-TDI)

Sample volume placed on myocardium or A–V valve annulus
High frequency, low amplitude signals from blood filtered out
Measures peak velocities of a selected region
Mean velocities calculated to give colour velocity maps

PW-TDI
Good temporal resolution

Wave pattern:
- S wave (ventricular systole)
- IVRT
- E wave (rapid diastolic filling)
- Diastasis
- A wave (atrial contraction)

Tissue Doppler velocities \( \approx 5–15 \text{ cm/s} \)
2-D-TDI
Poor temporal resolution/good spatial resolution
Uses colour flow imaging
Low velocity myocardium coded with dark colours
High velocity myocardium coded with lighter colours

MM-TDI
Excellent temporal resolution
Uses colour flow imaging with M mode

Artefacts

Reverberations
Secondary reflection along the path of the U/S pulse due to the U/S ‘bouncing’ between the structure and another strong reflector or the transducer
Creates parallel irregular lines at successively greater depths from the primary target
Two types (Fig. 1.32)
(i) linear reverberation
(ii) ring down = solid line directed away from TX due to merging of reverberations

Ghosting
Type of reverberation artefact when using colour flow Doppler (Fig. 1.33)
Amplitude of ‘ghost’ > A of initial reflector if target is moving

Mirror images
Occurs with Doppler (CW and PW)
↑↑A of fD spectrum → signal in opposite direction (normally below threshold, therefore filtered out) exceeds threshold (Fig. 1.34)
Transoesophageal Echocardiography

(a) 
Descending aorta

Linear reverberations

(b) 
Descending aorta

‘Ring down’

Fig. 1.32

Colour flow Doppler in descending aorta

‘Ghosting’ CFD in false image

Fig. 1.33

Aliasing

= ‘wraparound’

With PWD, when $f_0$ exceeds Nyquist limit (Fig. 1.35)

$$f_0 > \frac{\text{PRF}}{2}$$

Usually $> 2 \text{ m/s}$

Reduced by:

1. $\downarrow f_0$
2. $\uparrow \text{PRF (\downarrow \text{depth})}$
3. use CWD
4. baseline shift
Low A, therefore removed by ‘rejection’/filtering, i.e. seen as noise

‘Mirrors’ seen as threshold is exceeded

Fig. 1.34

Shadowing

U/S beam hits a strong reflector (e.g. mechanical valve)
→ ↓↓A of beam distal to reflector
→ ‘fallout’, i.e. no image seen beyond reflector

‘Enhancement’ = reverse shadowing
U/S beam hits very weak reflector with minimal attenuation
→ ↑reflection from distal tissue
→ brighter image (corrected using TGC)
Near field clutter

In the ‘near field’ strong signals are received from reflectors, which dominate the image

Amplitude of near field echoes reduced by: near field gain control

Refraction

U/S beam is deflected from its path
Creates falsely perceived object (Fig. 1.36)
TX assumes reflected signal originated from original scan line

Range ambiguity

With CWD: unsure of exact site of peak velocity/f_D along the U/S beam path
With high PRF: unsure from which of the several sites the signal may be returning.

Side lobes

TX emits several side beams with the main central beam
Reflection from side beam appears as object in main beam
Usually, multiple side lobes create a curved line, with the true reflector the brightest (Fig. 1.37)
Fig. 1.37

Have common radius from TX
Cross anatomical planes

**Beam width**

= spatial resolution problem occurring with Doppler
↑ beam width → poor LATA
↑ beam width → inappropriate spatial localization
i.e. strong flow signals at margin of beam appear to arise from central part of beam

‘Crying’

\( f_D \) in audible range (20–20 000 Hz)
TX acts as a microphone
External noise (e.g. patient talking) with high \( A \) is detected by TX, causing oversaturation of amplifier
‘Noise’ displayed on spectral image

**Multiple choice questions**

1. The speed of sound through the heart is approximately
   - A. 330 m/s
   - B. 1450 m/s
   - C. 1540 m/s
D. 14.5 mm/µs
E. 1.54 cm/µs

2. Audible sound has a frequency of
   A. 2–20 Hz
   B. 20–20 000 Hz
   C. 20–20 000 kHz
   D. 2–20 MHz
   E. >20 MHz

3. The speed of sound through a medium is increased with
   A. increased transducer frequency
   B. increased medium density
   C. reduced medium stiffness
   D. increased medium bulk modulus
   E. increased medium elasticity

4. The following are all acoustic variables except
   A. density
   B. force
   C. temperature
   D. pressure
   E. particle motion

5. The intensity of an ultrasound wave is
   A. measured in watts
   B. the concentration of power in a beam
   C. amplitude multiplied by power
   D. amplitude squared
   E. usually less than 100 mW

6. In pulsed ultrasound, pulse duration is
   A. determined by the period of each cycle
   B. analogous to wavelength
   C. 0.5–3 seconds in TOE
   D. number of cycles multiplied by frequency
   E. altered by the sonographer
7 At a depth of 10 cm, the pulse repetition frequency is
   A 3.75 Hz
   B 7.5 Hz
   C 3.75 kHz
   D 7.5 kHz
   E 7500 kHz

8 When the pulse repetition period is 0.104 seconds, the depth of the image is
   A 4 cm
   B 5 cm
   C 6 cm
   D 7 cm
   E 8 cm

9 Spatial pulse length
   A influences axial resolution
   B influences lateral resolution
   C is usually 0.1–1 µm in TOE
   D is determined only by the medium
   E is changed by the sonographer

10 The following are true regarding attenuation except
   A it occurs by absorption
   B it can be measured in decibels
   C it increases with reducing transducer frequency
   D it occurs by scattering
   E it occurs by reflections

11 With a 6 MHz ultrasound transducer, the half value layer thickness is
   A 1 mm
   B 0.5 cm
   C 1 cm
   D 1.5 cm
   E 3 cm

12 All the following statements are true except
   A in soft tissue acoustic impedance is 1.25–1.75 Rayls
   B reflections depend upon changes in acoustic impedance
C acoustic impedance is density multiplied by velocity
D specular reflections occur at smooth boundaries
E acoustic impedance is resistance to sound propagation

13 The intensity reflection coefficient of a sound wave traveling from medium 1 ($Z = 20$ Rayls) to medium 2 ($Z = 80$ Rayls) is
A 30–40%
B 40–50%
C 50–60%
D 60–70%
E 70–80%

14 With regard to ultrasound transducers
A TOE transducers have a frequency of 3–6 Hz
B each piezoelectric crystal is supplied by four electrical wires
C most ultrasound crystals are made from quartz
D the damping element improves temporal resolution
E the matching layer has a lower impedance than the crystal

15 The following statements about sound beams are true except
A the focus is the position of minimum diameter
B the Fresnel zone is the near zone
C smaller diameter transducers have a shorter focal depth
D higher frequency transducers have a shorter focal depth
E smaller diameter transducers have greater divergence

16 Axial resolution is
A improved by reduced ringing
B worsened by increasing transducer frequency
C improved by increasing spatial pulse length
D worsened by shortening wavelength
E the ability to separate two objects perpendicular to the beam

17 Temporal resolution can be improved by
A increasing image depth
B adding colour flow Doppler to the image
C adding pulse wave Doppler to the image
D reducing sector size
E increasing line density
18 Motion (M) mode imaging
A requires sequential acquisition from multiple planes
B has low temporal resolution
C has velocity on the y-axis
D is poor for analysing time-related events
E is developed from B mode imaging

19 Pulse wave Doppler
A suffers from ‘range ambiguity’ artefact
B requires one crystal to emit and a second crystal to receive
C is used in colour flow Doppler imaging
D is accurate with velocities up to 9 m/s
E suffers from ‘aliasing’ at velocities above 2 cm/s

20 The following statements regarding ‘aliasing’ are true except
A it is reduced by imaging at a shallower depth
B it is worsened by increasing transducer frequency
C it can be removed by changing to pulse wave Doppler
D it is reduced by increasing pulse repetition frequency
E it occurs when the Doppler frequency exceeds the Nyquist limit
Indications

Category I

TOE useful in improving clinical outcomes

(1) Pre-operative
   (a) suspected TAA, dissection or disruption in unstable patient
(2) Intra-operative
   (a) life-threatening haemodynamic disturbance
   (b) valve repair
   (c) congenital heart surgery
   (d) HOCM repair
   (e) endocarditis
   (f) AV function in aortic dissection repair
   (g) evaluation of pericardial window procedures
(3) ICU setting
   (a) unexplained haemodynamic disturbances

Category II

TOE may be useful in improving clinical outcomes

(1) Pre-operative
   (a) suspected TAA, dissection or disruption in stable patient
(2) Intra-operative
   (a) valve replacement
(b) cardiac aneurysm repair
(c) cardiac tumour excision
(d) detection of foreign bodies
(e) detection of air emboli during cardiac/neuro procedures
(f) intracardiac thrombectomy
(g) pulmonary embolectomy
(h) suspected cardiac trauma
(i) aortic dissection repair
(j) aortic atheromatous disease/source of aortic emboli
(k) pericardial surgery
(l) anastomotic sites during heart/lung transplant
(m) placement of assist devices

(3) Peri-operative
   (a) increased risk of haemodynamic disturbances
   (b) increased risk of myocardial ischaemia

Category III

TOE infrequently useful in improving clinical outcomes

(1) Intra-operative
   (a) evaluation of myocardial perfusion, coronary artery anatomy, or graft patency
   (b) repair of non-HOCM cardiomyopathies
   (c) endocarditis in non-cardiac surgery
   (d) monitoring emboli in orthopaedic surgery
   (e) repair of thoracic aortic injuries
   (f) uncomplicated pericarditis
   (g) pleuropulmonary disease
   (h) monitoring cardioplegia administration

(2) Peri-operative
   (a) placement of IABP, ICD or PA catheters
Safety

Contraindications and complications

Absolute contraindications
(1) patient refusal
(2) patient has had oesophagectomy
(3) recent major oesophageal surgery
(4) oesophageal atresia, stricture, tumour

Relative contraindications
(1) oesophageal diverticulum
(2) oesophageal varices
(3) Barrett’s oesophagus
(4) recent oesophageal/gastric radiotherapy
(5) hiatus hernia
(6) unexplained upper gastrointestinal bleed
(7) in awake patient where tachycardia undesirable

Complications
Minor < 13%  Serious < 3%
Mortality 0.01–0.03%

(1) direct trauma to:
mouth: lip, dental injuries
pharynx: sore throat
larynx: RLN injury, tracheal insertion (!)
oesophagus: dysphagia, tear, burn
stomach: haemorrhage
(2) indirect effects:
tachycardia, causing myocardial ischaemia
bradycardia
arrhythmias
bacteraemia
(3) equipment damage
Biological effects

Dosimetry = science of identifying/measuring characteristics of ultrasound fields causing biological effects

High A/P/I causes damage (SPTA related to tissue heating)
SPTA < 100 mW/cm² unfocused = safe
SPPA < 1 W/cm² focused = safe

Thermal

Tissue absorption (bone) of U/S → heat
Localized scattering → heat
TOE exam causing < 1 °C rise in temperature = safe
> 41 °C → harmful
Tightly focused beams → ↑temperature elevation as heat is dissipated
Unfocused beams → ↓temperature elevation
Fetal ↑temperature a concern (effects on fetal bone)
Thermal index = quantification of tissue heating

Cavitation

Bodies of gas/microbubbles are excited by U/S → vibration → tissue and heat injury

(1) stable cavitation

oscillating bubbles: intercept reradiate absorb \{ acoustic energy \\
→ shear stresses/microstreaming in surrounding fluid

(2) transient cavitation

bubbles expand and burst → highly localized violent effects
mechanical index = quantification of cavitation effects

Electrical hazards

Uncommon
Patient susceptible to electrical injury from:

1) frayed/worn cables
2) damaged U/S TX
3) damaged case/housing
4) damaged electrical circuitry/plug

**Infection**

Incidence of bacteraemia is up to 4%

*but* no evidence for clinical consequences
Antibiotic prophylaxis only recommended in high risk patients

Infectious complications reduced by:

1) use of mouth guard
2) careful insertion/removal of probe
3) gross decontamination
4) Hibiscrub wash
5) soak in Metiricide > 20 min
6) rinse in water

**Multiple choice questions**

1. **The following are category I indications for TOE except**
   - A mitral valve repair
   - B congenital heart surgery
   - C life-threatening haemodynamic disturbances
   - D evaluation of pericardial window procedures
   - E cardiac tumour excision

2. **An absolute contraindication to perioperative TOE is**
   - A oesophageal atresia
   - B Barrett’s oesophagus
   - C hiatus hernia
D unexplained upper gastrointestinal bleed
E oesophageal diverticulum

3. The following statements relating to the biological effects of ultrasound are true except
   A tightly focused beams cause less of a temperature rise
   B TOE is considered safe if temperature rises less than 1 °C
   C in transient cavitation, bubbles expand and burst
   D thermal index is the quantification of tissue heating
   E focused beams are considered safe if the intensity is less than 1 kW/cm²

4. With regard to complications of TOE
   A bacteraemia occurs in 15% of patients
   B serious complications occur in 5–10% of patients
   C indirect complications include tachyarrhythmias
   D mortality from TOE is 0.1%
   E antibiotic prophylaxis is recommended for all patients
Chambers

Left atrium (Fig. 3.1)
LA area = 14.0 cm² ± 3 cm²
LA pressure = 2–10 mmHg
LA SaO₂ = 97%

LA appendage
Seen at 30°–150°
Single or multiple lobes
May contain pectinate muscles
Common site for thrombus

Doppler velocities:
  contraction (emptying) and filling
  low velocities associated with thrombus

Right atrium (Fig. 3.2)
RA area = 13.5 cm² ± 2 cm²
RA pressure = 1–5 mmHg
RA SaO₂ = 75%

Left ventricle (Fig. 3.3)
LV pressure = 120/10
LV SaO₂ = 97%
LV FS% (Mmode) ≈ 30–45%
Four-chamber view

Fig. 3.1

Four-chamber view

Fig. 3.2

(a) Four-chamber view

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<tr>
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<th>Systole</th>
<th>Diastole</th>
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<td>4.7</td>
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(b) Short axis view

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<td>Mid-pap (cm)</td>
<td>3.5</td>
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**Fig. 3.3a, b (cont.)**

Vol of disc = $H(D_1/2 - D_2/2)$

Total vol = $vol_1 + vol_2 + \ldots$

**Fig. 3.4**

**LV volume**

LVEDV index = 50–60 ml/m$^2$

Calculated using Simpson’s method = sum of volume of discs (Fig. 3.4)

**LV segments**

Midoesophageal views (Fig. 3.5)
Transgastric short axis views (Fig. 3.6)

**Right ventricle (Fig. 3.7)**

RV pressure = 25/5 mmHg

RV SaO$_2$ = 75%

RV FS% = 45–50%

**RV volume**

Determined by Simpson’s method
Valves

Mitral valve

Two leaflets:
  - anterior (AMVL)
  - posterior (PMVL)
Attachment of PMVL > AMVL
Size of AMVL > PMVL (Fig. 3.8)
Normal MV area (MVA) = 4–6 cm²

Composed of:
- leaflets
- chordae tendineae
- papillary muscles (PMs)
- fibromuscular annulus
Four-chamber

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<tr>
<td>Area (cm²)</td>
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</tbody>
</table>

Fig. 3.7

Postero-medial (P/M) commissure

Antero-lateral (A/L) commissure

From each PM – $1\degree/2\degree/3\degree$ chordal structures subdivide and attach to ventricular surface and free edge of AMVL and PMVL. Fibromuscular annulus supports PMVL. AMVL continuous with membranous ventricular septum, aortic valve, and aorta. AMVL attaches to fibrous skeleton of heart. All aspects of AMVL and PMVL seen on midoesophageal views (Fig. 3.9)
Transoesophageal Echocardiography

(a) Four-chamber (0°)

(b) Commissural (40–60°)

(c) Two-chamber (90°)
(d) Three-chamber (110–140°)

(c) Five-chamber (0° and anteflex)

Fig. 3.9a, b, c, d, e (cont.)

MVL motion (Mmode) (Fig. 3.10)
D → E = early diastole/passive rapid LV filling
E → F = ↓LA pressure prior to LA contraction
F → A = atrial systole
A → C = LV pressure (LVP) > LA pressure (LAP) → trivial MR
LV systole → LVP >> LAP → MV closes (MVC)

Factors affecting MVL motion

(1) LAP: LVP
(2) volume/velocity of blood flow across MV
(3) annulus/PM motion
(4) LA/LV compliance (Cn)
(5) LV systolic function
Transmitral flow (TMF)

PW Doppler at MVL tips (Fig. 3.11)

- $E =$ passive LV filling: $a_t$ due to LAP $> LVP$
  - $d_t$ due to inertia of flow
- $L =$ pulmonary veins (PVs) filling $LA \rightarrow LAP > LVP$
  - $L$ incorporated into $E$ as HR increases
- $A =$ atrial systole

Doppler velocities
- $E =$ 50–80 cm/$s$ (decreases with increasing age)
- $A =$ 30–50 cm/$s$ (increases with age/diastolic dysfunction)
- $E/A = 1–2.2/1$ (ratio decreases with age)
- $VTI_E/VTI_A = 2.5/1$
E wave (Fig. 3.12)

\[ a_m = \text{flow acceleration} \]

determined by rate of ↑pressure gradient (PG) when MVO secondary to: initial LAP
rate of LV relaxation
MV resistance (MV area)

\[ d_m = \text{determined by rate of equalization of LAP:LVP} \]
related to LA/LV Cn
i.e. ↓LV Cn → ↑rate of \( d_m \) (↓\( d_t \))

\[ d_t (\text{deceleration time DT}) = \text{due to flow inertia} \]
reduced MVA (e.g. MS) → ↑\( d_t \)

\( V_{\text{max}} \) determined by: initial LAP:LVP
LA/LV Cn

\( V_{\text{max}} \) with ↑LAP
\( V_{\text{max}} \) with ↓LV Cn

Aortic valve

Three leaflets:
left coronary cusp (LCC)
right coronary cusp (RCC)
non-coronary cusp (NCC)
with associated sinuses of Valsalva (Fig. 3.13)
Transoesophageal Echocardiography

(a) AV SAX (30–60°)

(b) AV LAX (110–140°)

Fig. 3.13a, b

Leaflet = crescent-shaped
    thickening at leaflet tip = node of Arantius (↑ with age)
    two ridges from node to lateral margins = coaptation line
    above ridges = lunula (fenestrated)
Lambl’s excrecescences = filamentous lesions on free edge of leaflet
    connective tissue
    degenerative change
    ? nidus for infection/thrombus

Doppler flow (Fig. 3.14)
Normal flow = systolic
    laminar (some turbulence at peak systole)
    rapid acceleration
    peak at mid-systole
    slow deceleration
    AV closes
Flow velocity depends on:
- CO
- SVR
- AV area

\[ AV \, V_{\text{max}} = 1.35 \, \text{m/s} \, (1.0–1.7 \, \text{m/s}) \]
\[ LVOT \, V_{\text{max}} = 0.9 \, \text{m/s} \, (0.7–1.1 \, \text{m/s}) \]

**Tricuspid valve**

Three leaflets: anterior (largest)
- posterior
- septal (Fig. 3.15)

PMs: anterior (largest) from moderator band
- posterior and septal (small)

TVL = continuous veil of fibrous tissue
- indentations = commissures

Septal TVL insertion infero-apical compared to anterior TVL
Transttricuspid flow (TTF)
TV opens before MV because:
- peak RVP < LVP
- RAP > RVP before LAP > LVP
TV closes after MV because:
- LV activation before RV
- LVP > LAP before RVP > RAP

RA systole before LA systole (activated from SA node in RA)

*TTF vs. TMF (Fig. 3.16)*

\(a_m\) determined by:
- initial RAP
- rate of RV relaxation
- TV resistance (TVA)

\(d_m\) determined by:
- RA/RV Cn
- \(\downarrow\) RV Cn \(\rightarrow\) \(\uparrow\) rate of \(d_m\)

TTF E \(V_{max}\) < TMF because RAP < LAP
TTF E \(a_m\) < TMF because RAP < LAP
TTF E \(d_m\) < TMF because RV Cn > LV Cn
Respiration
Greater influence on TTF compared to TMF
On inspiration → TTF increases
↑E \( V_{\text{max}} \) and A \( V_{\text{max}} \) by \( \approx 15\% \)
E/A ratio remains constant

Pulmonary valve
Three leaflets: anterior
right posterior
left posterior

Lies anterior/superior/to the left of AV
PV area > AV area

Flow
Systolic
Laminar
Mid-systolic peak \( V_{\text{max}} \)
PV \( V_{\text{max}} = 0.6–0.9 \text{ m/s} \)

Vessels

Aorta
Thick musculoelastic wall – thin intima
thick media, multiple elastic sheets
thin adventitia

Ascending aorta (Fig. 3.17)
From AV to aortic arch ≈ 5 cm
Commences at AV at LSE third CC
Passes anterior/superior/to the right
Joins proximal aortic arch at RSE second CC
Branches:
LCA from LC sinus
RCA from RC sinus
Transoesophageal Echocardiography

Sinus of Valsalva

![Fig. 3.17](image)

Fig. 3.17

Aortic arch (Fig. 3.18)

Runs from ascending aorta to descending aorta
Commences at RSE second CC
Initially passes superior/posterior/lateral in front of trachea
Passes inferior/to the left
Joins descending aorta at anterior aspect of T4

Branches:
- innominate artery
- left common carotid artery
- left subclavian artery
Fig. 3.19

**Descending aorta**
Commences at distal aortic arch  
Runs from arch to iliac bifurcation at L4  
Divided into thoracic and abdominal by diaphragm at T12  
Thoracic aorta diameter $\approx 20$ mm

**Pulmonary artery**
Runs from PV to bifurcation into LPA and RPA  
Approximately 2–3 cm in length (Fig. 3.19)  
LPA passes posteriorly/to the left, to left hilum  
RPA passes to the right beneath aorta, superior branch passes to right hilum

**Doppler flow**
Laminar flow with flat velocity profile  
Normal PA = 0.6–0.9 m/s  
PA flow: ↑15% on inspiration  
$\uparrow$30% post-Fontan’s procedure  
$\uparrow$50% with tamponade
Pulmonary veins

Four veins: 2 right–upper and lower (RUPV and RLPV)  
  2 left–upper and lower (LUPV and LLPV)  
2% population have > 2 PVs from right lung  
Doppler flow composed of S, D and A waves (Fig. 3.20)

S wave (PV₅)
Systolic antegrade flow due to low LAP  
S1 = atrial relaxation  
S2 = mitral annular plane systolic exclusion (MAPSE), due to the  
  descent of MV annulus with LV systole  
Affected by:  
  LA Cₙ  
  MR  
Normal PV₅ = 40 cm/s

D wave (PV₀)
Diastolic antegrade flow due to drop in LAP when MV opens  
Determined by PG from PV:LA
Fig. 3.21

Peak PV_D occurs 50 msec after peak E V_max
Normal PV_D = 30 cm/s

A wave (PV_A)
Diastolic retrograde flow due to atrial contraction
Reversal of flow back into PV depends on LV C_n
i.e. ↓LV C_n → ↑PV_A reversal
Normal PV_A = 20 cm/s
Atrial fibrillation (AF):
   no PV_S1
   no PV_A
   PV_S2 < PV_D

Coronary sinus
Venous return of heart
Posterior aspect of heart in A–V groove
Covered by LA wall and pericardium
Normal CS < 10 mm diam

Doppler flow composed of S, D and A waves (Fig. 3.21)

CS dilated with:
   RV dysfunction
   increased RAP
   increased volume flow, e.g. persistent left SVC
Vena cavae/hepatic veins

**IVC**
From common iliac veins at L5 to RA
Passes through diaphragm at T8/11–25 mm diameter
Doppler flow composed of S, D and A waves (Fig. 3.22(a))

**SVC**
From R and L innominate veins to RA at third CC

**HVs**
Insert into IVC proximal to diaphragm (at \(\sim 30^\circ\))/5–11mm diam
Doppler flow composed of S, SR, D and A waves (Fig. 3.22(b))

S wave: ↓RAP due to: atrial relaxation
TAPSE

SR wave: slight reversal of flow at end of RV systole
D wave: ↓RAP as TV opens
A wave: RA contraction → small reversal of flow
Coronary arteries

From sinuses of Valsalva
LCA = 10 mm long/3–10 mm diam
  bifurcates into LAD and LCx
LAD supplies ant LV/ant $\frac{2}{3}$ IVS
PWD of LAD during diastole = 40–70 cm/s
LCx supplies lat LV/SAN (40%)/AVN (15%)/post $\frac{1}{3}$ IVS
RCA supplies RA/RV/SAN (60%)/AVN (85%)/post $\frac{1}{3}$ IVS
Post $\frac{1}{3}$ IVS from post. desc. artery = RCA (50%)
  LCx (20%)
  RCA + LCx (30%)

Septa

Interatrial septum

Thin muscular membrane separating RA and LA
Depression in mid portion = fossa ovalis (foramen ovale in fetus)

Development (Fig. 3.23)
Downward growth of septum primum
Septum primum separates from superior atrium and continues
downward growth
Downward growth of septum secundum to right of septum primum
  creates flap = foramen ovale (FO)
Fetus: RAP > LAP: FO open
Birth: LAP > RAP: FO closes
25% of population have patent FO (PFO)

IAS motion
Reflects RAP vs. LAP
Predominantly reflects LAP because LA less compliant than RA,
  therefore increase in volume increases LAP > RAP
Fig. 3.23a, b, c

(1) movement to LA = RA contraction before LA systole
(2) movement to RA = LA filling
(3) continued movement to RA = TV opens before MV opens
(4) movement back to LA = MV opens, rapid LV filling

**Interventricular septum**

Thick, triangular muscular wall except small membranous part at superior border below AV (RCC and NCC)
Functional component of LV (=$\frac{1}{3}$ of LV muscle)
Concave to LV
Normal IVS = 7–12 mm thick (= LV free wall thickness)
(measured in mid-diastole)
Thin septum = post-MI scar tissue
<7 mm
high echogenicity
30% thinner than surrounding myocardium

**IVS motion**
Contracts with LV inwards towards centre of LV (SAX view)

---

**Multiple choice questions**

1. The normal left atrial area is
   - A 4 mm²
   - B 1.4 cm²
   - C 4 cm²
   - D 10 cm²
   - E 14 cm²

2. Normal right atrial oxygen saturation is
   - A 55%
   - B 65%
   - C 75%
   - D 85%
   - E 95%

3. From the transgastric short axis view of the left ventricle, normal fractional shortening at basal level is
   - A 20%
   - B 35%
   - C 50%
   - D 65%
   - E 80%
4. The left ventricular walls seen from the standard two chamber view (at 90°) are
   A inferior and lateral
   B anterior and lateral
   C posterior and anteroseptal
   D inferior and anterior
   E septal and lateral

5. Normal right ventricular systolic and diastolic pressures are approximately
   A 20/10 mmHg
   B 25/5 mmHg
   C 35/15 mmHg
   D 25/15 mmHg
   E 40/0 mmHg

6. The following statements about the normal mitral valve are all true except
   A the posterior leaflet is continuous with the membranous ventricular septum
   B the anterior leaflet is larger than the posterior leaflet
   C there is an anterolateral and a posteromedial commissure
   D chordal structures arise from the papillary muscles and attach to the ventricular surface of both the anterior and posterior leaflets
   E the anterior leaflet attaches to the fibrous skeleton of the heart

7. The following parts of the mitral valve can be observed from the standard commissural view (at 40–60°)
   A A1, A2, P1
   B A2, P1, P3
   C A1, A3, P2
   D A1, P1, P2
   E A3, P1, P3

8. Normal mitral valve area is
   A 1–2 cm²
   B 2–4 cm²
   C 4–6 cm²
9. Regarding transmitral flow, a normal E wave velocity in a healthy 50-year-old is
   A 3 cm/s
   B 6 cm/s
   C 30 cm/s
   D 60 cm/s
   E 3 m/s

10. The following statements regarding transmitral flow are all true except
    A the E wave represents passive left ventricular filling
    B the L wave occurs in late passive diastole
    C the E wave duration is affected by left ventricular compliance
    D the A wave velocity increases with increasing age
    E the E wave velocity increases with increasing age

11. The normal aortic valve comprises the following three coronary cusps
    A left, right and anterior
    B left, right and posterior
    C anterior, posterior and non-
    D superior, inferior and non-
    E left, right and non-

12. The normal maximum velocity measured by Doppler through the left ventricular outflow tract is
    A 9 cm/s
    B 90 cm/s
    C 1.35 m/s
    D 9 m/s
    E 13.5 m/s

13. The following statements regarding the normal tricuspid valve are all true except
    A it is composed of anterior, posterior, and septal leaflets
    B the anterior leaflet insertion is infero-apical compared to the septal leaflet insertion
C the tricuspid valve opens before the mitral valve opens
D the tricuspid valve closes after the mitral valve closes
E transtricuspid blood flow increases on inspiration

14. The normal diameter of the ascending aorta at the sino-tubular junction is
   A 14–26 mm
   B 17–34 mm
   C 21–35 mm
   D 25–41 mm
   E 26–41 mm

15. Regarding pulmonary venous Doppler flow waves
   A S2 is due to mitral annular plane systolic excursion
   B normal S wave velocity is 4 cm/s
   C D wave is due to atrial systole
   D normal D wave velocity is 30 m/s
   E A wave velocity decreases with reduced left ventricular compliance

16. Normal interventricular septum thickness measured in mid-diastole is
   A 1–2 mm
   B 2–5 mm
   C 5–7 mm
   D 7–12 mm
   E 12–17 mm
Ventricular function

**LV systolic function**

**Quantitative echo**

**LV volume**
Normal LVEDV = 50–60 ml/m²
Calculated using Simpson’s method (Fig. 3.4)

**LV mass**
LV adapts to increases in pressure and volume with muscular hypertrophy
Eccentric hypertrophy due to ↑ chamber volume (volume overload)
Concentric hypertrophy due to ↑ wall thickness (pressure overload)

\[
\text{LV mass (LVM)} \approx \text{V}_{ep} - \text{V}_{end} = \text{V}_m
\]
(i.e. LVM = total within epicardium – total within endocardium)

\[
\text{LVM} = \text{V}_m \times 1.05 \text{ (specific gravity for myocardium)}
\]
LVH is > 134 g/m² for men
> 120 g/m² for women

**Ejection indices**
(1) Stroke volume \( SV = \text{LVEDN} - \text{LVESV} \)
SV index (SVI) = 40–50 ml/m²
(2) Ejection fraction  \[ EF = \frac{(LVEDV - LVESV)}{LVEDV} \times 100 \]
\[ EF = \frac{(SV/LVEDV) \times 100}{\times 100} \]
\[ EF = 50–70\% \]

(3) Fractional shortening
\[ FS = \left[ \frac{(LVIDd - LVIDs)}{LVIDd} \right] \times 100 \]
\[ LVIDd = LV \text{ internal diameter in diastole} \]
\[ LVIDs = LV \text{ internal diameter in systole} \]
\[ FS = 28–45\% \]

(4) Velocity of circumferential fibre shortening (Vcf)
\[ Vcf = \frac{(LVIDd - LVIDs)}{(LVIDd \times ET)} \]

\[ ET = \text{ejection time} \]
Reflects amplitude and rate of LV contraction
\[ V_{cf} > 1.1 \text{ circumferences/s} \]

**Global LV function**

Contractility = thickening and inward movement of LV wall during systole

Quantitative assessment:
- LV volume
- LV mass
- EF
- FS
- Vcf

Qualitative assessment:
- normal
- hypokinesia
- akinesia
- dyskinesia
Non-TOE assessment

(1) MRI: high resolution, 3-D images
   LV function, extent of ischaemia
(2) Nuclear imaging: myocardial scintigraphy (Tec-99)
   = ‘hot-spot’ imaging
   perfusion scintigraphy (Th-201)
   = ‘cold-spot’ imaging
   radionuclide angiography (Tec-99)
   = assesses LV function, CO, EF, and LVEDV
(3) CT scan: with Th-201
   perfusion defects, MI size
(4) Angiography: LV function
   coronary artery assessment

Effect of altered physiology/pathophysiology

(1) Exercise
   ↑HR ↑SV → ↑CO ↑EF ↑BP
   with LVESV↓/LVEDV↔
(2) AI
   ↑LVEDV/↑LVESV → ↑LVM (eccentric hypertrophy)
   EF remains normal until late (due to ↓SVR)
   Poor prognosis if LVIDs > 50 mm
(3) AS
   ↑LVM (concentric hypertrophy)
   ↑EF/↑Vcf
   ↓EF late in disease
(4) MR
   ↑LVEDV/↑LVESV → ↑LVM (eccentric hypertrophy)
   EF preserved until late in disease
   Poor prognosis if: LVIDs > 50 mm
   LVIDd > 70 mm
   FS < 30%
(5) Hypertension
   ↑wall stress
Transoesophageal Echocardiography

Fig. 4.1

↑LVM (concentric hypertrophy)
Diastolic dysfunction with ↑IVRT

(6) HOCM
Diagnosis: septum/post wall thickness > 1.3/1
This occurs in:
- 12% of normal population
- 32% of LV hypertrophy
- 95% of HOCM

Segmental LV function

Regional wall motion abnormality (RWMA)
Occurs 5–10 beats after coronary artery occlusion
Precedes ECG changes

Adjacent area asynergy = hypokinesia due to:

(1) mechanical tethering by ischaemic tissue
(2) ATP depletion
(3) metabolic abnormalities

Region of hypokinesia depends on blood supply (Fig. 4.1)
Other causes of RWMA:
(1) LBBB  
(2) RBBB  
(3) pacing  
(4) WPW syndrome  
(5) post-CPB  

Chronic ischaemia  
(1) Fixed RWMA: varies in size/distribution  
(2) Scar: post-MI = dense and thin (<7 mm)  
(3) Aneurysm: post-MI, traumatic, congenital  
  (a) True: gradual expansion  
  thinning of myocardium  
  wide neck (> 1/2 diam of aneurysm)  
  assoc. with thrombus, arrhythmias, CCF  
  (b) Pseudo:  
  due to myocardial rupture  
  blood contained by parietal pericardium  
  narrow neck (< 1/2 diam of aneurysm)  
  assoc. with thrombus, rupture, arrhythmias, CCF  
(4) VSD: post-MI IVS rupture with poor prognosis  
(5) PM rupture: P/M PM more common than A/L PM causes severe MR  
(6) Thrombus:  
  common after large MI  
  assoc. with LV aneurysm  
  echo dense speckled mass  
  interrupts LV contour  
  common in apical aneurysms  

Stress echo  
Designed to induce RWMA by:  
  exercise (treadmill)  
  pharmacology (Dobutamine)  
  pacing (transoesophageal)
ECG

Fig. 4.2

Normal response = hyperkinesis/↑EF%/↑aortic VTI
Abnormal = new RWMA/worsening of existing RWMA/↓EF%

LV diastolic function

Phases of diastole (Fig. 4.2)

Isovolumic relaxation time (IVRT)
= 70–90 ms
From AVC – MVO
Aortic pressure > LVP → AV closes
LVP > LAP so MV remains closed
LV volume constant
LV relaxes → LVP
IVRT ends when LAP > LVP & MV opens
Early rapid filling

= E wave on TMF
LAP >> LVP with continued LV relaxation
As LV fills → ↑LV vol → ↑LVP
As LAP LVP → ↓filling rate
As LAP = LVP → filling stops

Diastasis/late filling

= L wave on TMF
LAP LVP → little filling
PVs contribute to LV filling

Atrial systole

= A wave on TMF
↑LAP → LV filling (10–30% of total)

Indices of relaxation

IVRT

AVC – MVO
↓relaxation → ↑IVRT > 90 ms
  Affected by: aortic diastolic pressure (aortic DBP)
  LAP
i.e. ↓Aortic DBP/↑LAP → ↓IVRT

−dP/dt

Negative rate of change of LVP (Fig. 4.3)
Occurs soon after AVC
Affected by aortic systolic pressure (aortic SBP)
i.e. ↑Aortic SBP → ↑−dP/dt

Time constant of relaxation (τ)

τ = −1/A
Fig. 4.3

Fig. 4.4

\[ \tau = -\frac{1}{A} \]

= 25–40 ms

\( \downarrow \) LVP during IVRT = exponential decay (Fig. 4.4)

**Chamber stiffness**

Passive property of myocardium

Reciprocal of compliance, i.e. \( \frac{dP}{dV} \)

Affected by:

- LV volume
- LV mass
- RV pressure
- pericardial pressure
- pleural pressure
Diastolic dysfunction

IVRT
Impaired relaxation $\rightarrow$ ↑IVRT $> 90$ ms
Restrictive pathology $\rightarrow$ ↓IVRT $< 70$ ms

Transmitral flow (Fig. 4.5)
LV filling depends on:
(1) LAP:LVP gradient
   LAP – LA Cn/LA contractility
   LVP – LV Cn/LV relaxation/LVESV
(2) MV area
Impaired relaxation:
   $\downarrow E_{V\text{max}} / \uparrow A_{V\text{max}}$
   $\downarrow E_{\text{VTI}} / \uparrow A_{\text{VTI}}$
   $\downarrow E_{am} / \uparrow E_{at}$
   $\downarrow E_{dm} / \uparrow E_{dt}$
   $\downarrow E/A / \downarrow E_{\text{VTI}} / A_{\text{VTI}}$
Restrictive pathology:
   $\uparrow E_{V\text{max}} / \downarrow A_{V\text{max}}$
   $\uparrow E_{\text{VTI}} / \downarrow A_{\text{VTI}}$
   $\uparrow E_{dm} / \downarrow E_{dt}$
   $\uparrow E/A$
Transoesophageal Echocardiography

**Pulmonary vein flow**
Impaired relaxation → ↑PVS/↓PV_D
→ ↑PV_A duration
Restrictive pathology → ↓PVS/↑PV_D

**Physiological effects**
(1) Respiration: inspiration causes ↑TTF $E_{V_{\text{max}}}$ / ↓TMF $E_{V_{\text{max}}}$
(2) Heart rate:
   ↑HR causes ↓$E_{V_{\text{max}}}$ / ↑$A_{V_{\text{max}}}$
   ↑↑HR causes $A$ on $E$ (A incorporated into $E$)
(3) Age:
   ↑age causes ↓$E_{V_{\text{max}}}$ / ↑$A_{V_{\text{max}}}$
   ↑IVRT
   ↑$E_{dt}$
(4) AV interval:
   prolonged PR interval delays LV contraction
   → delays $E$ wave
   → $E$ and $A$ fuse

**Pathological states**
(1) LV hypertrophy: ↓$E/A$
(2) Ischaemia: ↓$E/A$
   ↑$E_{dt}$
(3) RVP: pulmonary ↑BP → ↓$E/A$ and ↑IVRT
   volume overload → ↑$E/A$ and IVRT↔
(4) Tamponade: exaggerated TTF ↑$E_{V_{\text{max}}}$ on inspiration
(5) Pericardial constriction: ↑IVRT/↓$E_{V_{\text{max}}}$ on inspiration
Table 4.1 Diastolic dysfunction summary

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Impaired relaxation</th>
<th>Pseudo-normal</th>
<th>Restrictive pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>DT (ms)</td>
<td>160–240</td>
<td>&gt;240</td>
<td>160–200</td>
<td>&lt;160</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>70–90</td>
<td>&gt;90</td>
<td>&gt;90</td>
<td>&lt;70</td>
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<tr>
<td>$E/A$</td>
<td>1–2</td>
<td>&lt;1</td>
<td>1–1.5</td>
<td>&gt;2</td>
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<tr>
<td>$A_{dur}/P_{V_{A_{dur}}}$</td>
<td>$A&gt;P_{V_{A}}$</td>
<td>$A&gt;P_{V_{A}}$</td>
<td>$A&lt;P_{V_{A}}$</td>
<td>$A&lt;&lt;P_{V_{A}}$</td>
</tr>
<tr>
<td>$P_{V_{S}}/P_{V_{D}}$</td>
<td>$P_{V_{S}}&gt;&gt;P_{V_{D}}$</td>
<td>$P_{V_{S}}&lt;P_{V_{D}}$</td>
<td>$P_{V_{S}}&lt;&lt;P_{V_{D}}$</td>
<td>$P_{V_{S}}&lt;&lt;P_{V_{D}}$</td>
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<tr>
<td>$E_{VII}/A_{VII}$</td>
<td>$E&gt;A$</td>
<td>$E&lt;A$</td>
<td>$E&gt;A$</td>
<td>$E&gt;&gt;A$</td>
</tr>
<tr>
<td>Valsalva $E: A$</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↓↑</td>
<td>↓↓</td>
</tr>
</tbody>
</table>

Summary of diastolic dysfunction (Table 4.1)

**RV function**

**Normal RV function**

RV = triangular/crescent-shaped
Contains muscle ridges = trabeculae carneae
Moderator band: large muscle bundle from low IVS to ant RV wall

Velocity of RV ejection:
- ↑ gradually
- peaks later than LV
- persists longer than LV

RV volume determined by Simpson’s method

**RV dysfunction**

**Volume overload**
Dilated RV
Flattening of IVS (moves to left)
Pressure overload

(1) Chronic:
   e.g. pulmonary hypertension
   RV hypertrophy (RV free wall thickness > 5 mm)
   progresses to RV dilatation/free wall hypokinesia

(2) Acute:
   e.g. PE
   RV can compensate to PAP < 40 mmHg
   RV dilatation with TR
   RV free wall hypokinesia
   IVS flattening in diastole
   RA/IVC dilatation

Multiple choice questions

1. For an adult female, left ventricular hypertrophy is defined as a left ventricular mass of greater than
   A 12 mg/m²
   B 120 mg/m²
   C 12 g/m²
   D 120 g/m²
   E 1.2 kg/m²

2. If the left ventricular internal diameter in systole is 30 mm and the left ventricular internal diameter in diastole is 45 mm, the fractional shortening is approximately
   A 15%
   B 25%
   C 33%
   D 50%
   E 66%

3. In mitral regurgitation
   A ejection fraction is preserved until late in the disease
B there is commonly left ventricular concentric hypertrophy
C prognosis is poor if fractional shortening is less than 42%
D there is a reduction in left ventricular end diastolic volume
E there is a reduction in left ventricular end systolic volume

4. The following statements regarding segmental left ventricular function are all true except
A regional wall motion abnormality occurs 5–10 beats after coronary occlusion
B right coronary artery supplies the inferior wall
C pacing can cause a regional wall motion abnormality
D left circumflex coronary artery supplies the lateral wall
E post-myocardial infarction scarring often causes wall thickening greater than 9 mm

5. A normal isovolumic relaxation time is
A 7–9 µs
B 70–90 µs
C 0.7–0.9 ms
D 7–9 ms
E 70–90 ms

6. Isovolumic relaxation
A commences when the mitral valve closes
B involves a 35% reduction in left ventricular volume
C terminates when left atrial pressure exceeds left ventricular pressure
D terminates when the mitral valve closes
E commences when the aortic valve opens

7. Chamber stiffness is affected by all of the following except
A left ventricle volume
B right ventricle pressure
C pericardial pressure
D pleural pressure
E ascending aortic compliance
8. Regarding impaired relaxation, there is
   A an increase in $E$ wave maximum velocity
   B a decrease in $A$ wave maximum velocity
   C an increase in the $E/A$ ratio
   D an increase in pulmonary vein flow $A$ wave duration
   E an increase in pulmonary vein flow $D$ wave velocity

9. Regarding transmitral flow
   A impaired relaxation causes shortening of the $E$ wave acceleration time
   B restrictive pathology causes an increase in $E$ wave deceleration time
   C inspiration causes increased $E$ wave velocity
   D increasing heart rate causes reduced $E/A$ ratio
   E restrictive pathology causes increased $A$ wave velocity

10. Regarding restrictive pathology
    A isovolumic relaxation time is often greater than 90 ms
    B deceleration time is usually less than 160 ms
    C $E/A$ ratio is greatly reduced
    D transmitral $A$ wave duration greatly exceeds pulmonary vein flow $A$ wave duration
    E pulmonary vein flow $S$ wave velocity greatly exceeds $D$ wave velocity

11. Increasing age causes
    A increase in isovolumic relaxation time
    B increase in $E$ wave maximum velocity
    C decrease in $E$ wave deceleration time
    D decrease in $A$ wave maximum velocity
    E increase in $E/A$ ratio

12. The following statements about the right ventricle are all true except
    A the normal right ventricle is crescent shaped
    B it contains muscle ridges called trabeculae carneae
    C in right ventricular hypertrophy the free wall is usually thicker than 15 mm
    D its volume can be determined using Simpson’s method
    E acute pulmonary embolism can cause right ventricular free wall hypokinesia
Hypertrophic obstructive cardiomyopathy

Definition and epidemiology

Unexplained hypertrophy of non-dilated LV
Prevalence ∼ 1–2% of population
Familial autosomal dominant ≈ 55%
Sporadic ≈ 45%

Features

Asymmetric septal hypertrophy
(1) Type I: anteroseptal
(2) Type II: panseptal
(3) Type III: extensive, sparing only posterior wall
(4) Type IV: apico-septal

IVS: posterior wall thickness ratio > 1.3:1

Systolic anterior motion (SAM) of anterior MV leaflet (AMVL)
  = functional subaortic stenosis
  Common with large, redundant AMVL
  Anterior motion of antero-lateral papillary muscle
  Venturi effect causes suction of AMVL into LVOT
  LVOT PG > 36 mmHg (velocity > 3 m/s)
  CW Doppler → ‘dagger-shaped’ pattern with late peaking
Mitral regurgitation
Magnitude of MR greatest in mid- to late-systole

Early AV closure
Mid-systolic AV closure

Dilated cardiomyopathy

Definition
Four-chamber enlargement with impaired RV and LV systolic function

Aetiology
Idiopathic
IHD
Post-partum
Post-CPB
Toxins – alcohol, cobalt, adriamycin, snake bites
Metabolic – acromegaly, thiamine, and selenium deficiency
Infection – post-viral, Chagas’ disease
Inherited – Duchenne’s muscular dystrophy, SC anaemia
Systemic disease – haemoachromatosis: Fe deposition within myocytes in epicardial region → fibrosis

Features
Four-chamber dilatation
RV and LV systolic dysfunction +/- diastolic dysfunction
Normal wall thickness
Increased LV mass
LV inflow directed postero-laterally
May have predominantly RV dilatation (Coxsackie B infection)

Restrictive cardiomyopathy

Causes

Idiopathic
Amyloid
Sarcoid
Storage diseases
Carcinoid
Endocardial fibroelastosis
Endomyocardial fibrosis

Features

Biatrial dilatation
Normal ventricular size and systolic function
Restriction to RV and LV filling
Echo-dense RV and LV walls

Amyloidosis

Deposition of abnormal proteins between myocardial fibres, in
PMs, in conductive tissue and in pericardium
Increased RV and LV wall thickness
‘Speckled’/granular appearance
RV/LV size and systolic function normal
Biatrial dilatation
Diffuse valvular thickening (MV and TV)
Small/moderate effusion
Sarcoidosis
Non-caseating granulomas
Affects LV free wall, IVS (conduction tissue), PMs causing MR and LV dilatation with RWMA

Storage diseases
Accumulation of abnormal metabolites

(1) Glycogen (Pompe’s/Cori’s): LVH +/- SAM
(2) Lipid (Fabry’s) ≡ amyloidosis
(3) Mucopolysaccharide (Hurler’s, Sanfilipo etc.): MV thickening

Carcinoid
Malignant tumour with hepatic metastases
Endocardial injury due to hormones (serotonin, kinins)
RA wall/TV/PV thickening
Usually TR + PS
Primary bronchogenic tumour can cause left-sided lesions

Endocardial fibroelastosis
Diffuse endocardial hyperplasia
Increased chamber size and wall thickness
AV/MV fibrosis

Endomyocardial fibrosis (Loeffler’s endocarditis)
Assoc. with:
- idiopathic hypereosinophilic syndrome, acquired hypereosinophilia

Fibrosis affecting:
- MV/TV
- subvalvular apparatus
- apex
- MR/MS
- TR/TS
Increased risk of thrombus formation
Preserved LV systolic function

**Multiple choice questions**

1. **Regarding hypertrophic obstructive cardiomyopathy**
   - A the prevalence is 0.1%
   - B type II septal hypertrophy is limited to the apex
   - C more than 65% of cases are sporadic
   - D type III septal hypertrophy is limited to the posterior wall
   - E the interventricular septum : posterior wall thickness ratio is usually greater than 1.3

2. **Systolic anterior motion of the anterior mitral valve leaflet**
   - A creates a functional sub-aortic stenosis
   - B is common with a small, redundant anterior leaflet
   - C is associated with posterior motion of the antero-lateral papillary muscle
   - D is associated with a fall in the pressure gradient across the left ventricular outflow tract
   - E creates a ‘dagger-shaped’ pattern with early peaking on application of continuous wave Doppler

3. **The following statements about dilated cardiomyopathy are all true except**
   - A it may be caused by cobalt toxicity
   - B there is an increase in left ventricular mass
   - C left ventricular inflow is directed antero-laterally
   - D left ventricular wall thickness is normal
   - E left ventricular diastolic dysfunction may occur

4. **Features typical of restrictive cardiomyopathy include**
   - A right ventricular dilatation in amyloidosis
   - B aortic and mitral valve fibrosis in endocardial fibroelastosis
   - C reduced left atrial size in sarcoidosis
   - D reduced left ventricular systolic function in endomyocardial fibrosis
   - E echolucent ventricular walls in amyloidosis
Valvular heart disease

Mitral valve

Mitral stenosis

Aetiology
Rheumatic
Degenerative calcification
Congenital
Vegetations
Parachute MV (chordae attached to single PM)
Infiltrative (fibrosis, amyloid)
Ergot, hypereosinophilia, non-valvular (myxoma, thrombus)

Features
M Mode
  ↓E-F slope of AMVL
  Anterior motion of PMVL
2-D
  Reduced leaflet motion
  Leaflet thickening
  Reduced orifice size
  AMVL ‘hockey stick’ appearance
  ‘diastolic doming’ – body of leaflets more pliable and receive some of
  blood flowing from LA to LV
LA – enlarged/’smoke’/thrombus/AF
LAA – ‘smoke’/thrombus/reduced Doppler velocities
LV – small/underfilled
Signs of pulmonary hypertension (RA/RV enlarged)
Table 6.1 Assessment of mitral stenosis by mean pressure gradient (MG) and mitral valve area (MVA)

<table>
<thead>
<tr>
<th>Severity</th>
<th>MG (mmHg)</th>
<th>MVA (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0</td>
<td>4–6</td>
</tr>
<tr>
<td>Mild</td>
<td>&lt;6</td>
<td>2–4</td>
</tr>
<tr>
<td>Moderate</td>
<td>6–12</td>
<td>1–2</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;12</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

Fig. 6.1

Rheumatic MS
Calcification of MV and subvalvular apparatus
Fusion of commissures and chordae
‘Fish-mouth’ MV orifice

Assessment of MS severity
(1) Planimetry:
  trace ‘fish-mouth’ in transgastric basal SAX view
  affected by plane and gain
  TOE underestimates degree of MS

(2) Transvalvular gradient: uses modified Bernoulli equation

\[ P = 4V^2 \]

Use mean pressure gradient (MG) (Table 6.1)
Trace around E and A waves (Fig. 6.1)
Underestimates degree of MS if AI present
(3) Continuity equation:

\[
\text{Flow} = \text{Velocity} \times \text{Area} \\
V_1 A_1 = V_2 A_2 \\
A_2 = V_1 A_1 / V_2 \\
\text{MVA} = V_{LVOT} \times A_{LVOT} / V_M \\
\text{MVA} = V_{TILVOT} \times A_{LVOT} / V_{TIVM}
\]

Inaccurate with AI (affects VTILVOT)

(4) Colour flow Doppler area (Fig. 6.2)

\[
\text{MVA} = (\pi/4) (ab) \\
\text{MVA} = 0.785 (ab)
\]

(5) Pressure half time (PHT) (Fig. 6.3)

Time taken for pressure to fall by \(1/2\)

Inaccurate with AI: AI \(\rightarrow\) ↓PHT \(\rightarrow\) overestimates MVA

\[
\text{MVA} = 220 / \text{PHT}
\]

(6) Depressurization time (DepT) (Fig. 6.3)

\[
\text{MVA} = 750 / \text{DepT}
\]

(7) Proximal isovelocity surface area (PISA). Flow converges uniformly and radially towards a small orifice, creating concentric isovelocity layers
When orifice is very small compared to region of acceleration the isovelocity surfaces are hemispheric (Fig. 6.4)

Flow through isovelocity surface = velocity × area

Conservation of mass: flow at each surface = orifice flow

If orifice flow known, orifice area calculated: area = flow/velocity

At first aliasing boundary: flow = area \( (2\pi r^2) \times V_{\text{alias}} \)

Correction for funnel-shaped inflow angle \( \alpha \)

\[
\text{MVA} = 2\pi r^2 \times \frac{\alpha}{180} \times \frac{V_{\text{alias}}}{V_{\max}} \\
\text{MVA} = 6.28r^2 \times \frac{\alpha}{180} \times \frac{V_{\text{alias}}}{V_{\max}}
\]
Transoesophageal Echocardiography

(8) Gorlin formula: used in cardiac catheter lab

\[ MVA = \frac{CO}{(DFT \times HR) \left( 44 \times C \times \sqrt{MG} \right)} \]

- \( CO \) = cardiac output
- \( DFT \) = diastolic filling time
- \( HR \) = heart rate
- \( C \) = orifice constant (for MV = 0.85)
- \( MG \) = mean gradient

\[ MVA = \frac{CO}{(DFT \times HR) \left( 37.5 \times \sqrt{MG} \right)} \]

Mitral regurgitation

Aetiology
(1) Congenital
   - Cleft MV
   - Double-orifice MV
   - Mitral arcade
(2) Acquired
   - Rheumatic
   - Ischaemic
   - MV prolapse
   - PM dysfunction/rupture
   - Chordal dysfunction/rupture
   - Vegetation
(3) Other
   - MV aneurysm
   - Annular calcification
   - Fibrosis
   - Tumours

Carpentier classification
I: normal leaflet motion:
   - dilated annulus
   - leaflet perforation
II: excessive leaflet motion:
  myxomatous disease
  PM/chordal rupture
  MV prolapse
  flail

III: restricted leaflet motion:
  rheumatic disease
  chordal tethering (ischaemia)

Assessment of MR severity

(1) Jet length
  Trivial < 1.5 cm
  Mild 1.5–3 cm
  Moderate 3–4.5 cm
  Severe > 4.5 cm

(2) Jet length/LA length
  Trivial < 25%
  Mild 25–50%
  Moderate 50–75%
  Severe > 75%

(3) Jet area
  Trivial < 1.5 cm²
  Mild 1.5–4 cm²
  Moderate 4–7 cm²
  Severe > 7 cm²

(4) Jet area/LA area
  Mild < 20%
  Moderate 20–40%
  Severe > 40%

(5) Qualitative
  Signal strength with CW Doppler
  i.e. large volume MR gives strong CW signal

(6) Regurgitant volume (RV)
  Difference between MV diastolic flow and AV systolic flow,
  assuming no AI
Severe >60 ml

\[ \text{RV} = \text{MV vol} - \text{LVOT vol} \]
\[ \text{RV} = (\text{Area}_{\text{MV}} \times \text{VTI}_{\text{MV}}) - (\text{Area}_{\text{LVOT}} \times \text{VTI}_{\text{LVOT}}) \]

(7) Regurgitant fraction
   Trivial <20%
   Mild 20–30%
   Moderate 30–50%
   Severe >50%

(8) Effective regurgitant orifice (ERO): from PISA
   Mild <0.2 cm²
   Moderate 0.2–0.4 cm²
   Severe >0.4 cm²

\[ \text{ERO} = 6.28r^2 \times \frac{V_{\text{alias}}}{V_{\text{MR}}} \]

(9) Pulmonary venous flow (Fig. 6.5)
   Moderate PV_S blunting
   Severe PV_S reversal

(10) Vena contracta
   Narrowest portion of jet downstream from orifice
   >0.5 cm \(\equiv\) ERO >0.4 cm²
Valvular heart disease 101

Fig. 6.6

**Diastolic MR**
Retrograde flow from LV to LA during diastole (Fig. 6.6)
Causes include AV block, atrial flutter, severe AI, high LVEDP

**Mitral valve prolapse**
Displacement of MV leaflet >3 mm above level of annulus
Occurs mid/end systole as annulus moves towards apex
- Bilateral leaflet prolapse: 75–90%
- Posterior leaflet prolapse: 10–20%
- Anterior leaflet prolapse: 3–5%
Associated with infective endocarditis, MR, sudden death from ventricular arrhythmias

**Aortic valve**

**Aortic stenosis**

**Aetiology**
(1) Congenital
- Uni-/bi-/quadricuspid valve
(2) Acquired
   Rheumatic
   Degenerative calcification
   Amyloid

Features
Thick, immobile, calcified AV leaflets
Commissural fusion (rheumatic)
‘Doming’ of AV leaflets
Reduced AV opening
Associated LVH +/- dilated aortic root

Assessment of AS severity
(1) Planimetry: severe AS suggested if AV area <0.7 cm²
(2) Continuity equation
   \[ AVA = A_{LVOT} \times V_{TI_{LVOT}} / V_{TI_{AV}} \]
   \[ AVA = A_{LVOT} \times V_{LVOT} / V_{AV} \]

(3) Gorlin formula
   \[ AVA = \frac{CO}{HR} \times ET \times 44 \sqrt{MG} \]
   \[ CO = \text{Cardiac output} \]
   \[ HR = \text{Heart rate} \]
   \[ ET = \text{Ejection time} \]
   \[ MG = \text{Mean gradient} \]

(4) Doppler pressure gradients: normal \( V_{\text{max}} < 1.5 \text{ m/s} \) (Table 6.2)

Peak PG vs. ‘Peak-to-peak’ PG (Fig. 6.7)
\( P_1 \) = peak PG by Doppler
Instantaneous
Maximum difference between aorta and LV pressures during systole at one instant in time
\( P_2 \) = ‘peak-to-peak’ pressure in cardiac catheter lab
Table 6.2 Assessment of aortic stenosis by peak pressure gradient (PG), mean PG and aortic valve area (AVA)

<table>
<thead>
<tr>
<th></th>
<th>Peak PG (mmHg)</th>
<th>Mean PG (mmHg)</th>
<th>AVA (cm²)</th>
</tr>
</thead>
<tbody>
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<td>Normal</td>
<td>-</td>
<td>-</td>
<td>2–4.5</td>
</tr>
<tr>
<td>Mild</td>
<td>&lt;40</td>
<td>&lt;20</td>
<td>1–2</td>
</tr>
<tr>
<td>Moderate</td>
<td>40–80</td>
<td>20–50</td>
<td>0.7–1.0</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;80</td>
<td>&gt;50</td>
<td>&lt;0.7</td>
</tr>
</tbody>
</table>

Non-simultaneous difference between peak aortic and peak LV pressures

\[ P_1 > P_2 \]

Mean PG measured with Doppler and cardiac catheter usually equal

**Aortic insufficiency**

**Aetiology**

1. Congenital
   - Uni-/bi-/quadricuspid valve
2. Acquired
   - (a) Leaflet pathology:
     - Degeneration
     - Rheumatic
Infective endocarditis
Trauma
(b) Annulus pathology:
Infection (syphilis)
Thoracic aortic aneurysm
Ascending aortic dissection

Features
Premature closure of MV
Poor coaptation of AV leaflets
Dilated aortic root

Assessment of AI severity
(1) Jet length (inaccurate)
   Mild < 2 cm
   Moderate 2 cm papillary muscles
   Severe beyond papillary muscles
(2) Perry index = jet height/LVOT diameter
   Mild < 25%
   Moderate 25–60%
   Severe > 60%
(3) Regurgitant fraction/volume
   \[ RF = \left( \frac{Vol_{AI}}{Vol_{LVOT}} \right) \times 100 \]
   Mild < 30%
   Moderate 30–50%
   Severe > 50%
   Regurgitant volume > 60 ml = severe AI
(4) Pressure half-time (PHT)
   Mild > 550 ms
   Moderate 300–550 ms
   Severe < 300 ms
(5) Flow reversal
   Mild ascending aorta
   Moderate descending thoracic aorta
   Severe abdominal aorta
Table 6.3 Assessment of aortic incompetence using Perry index, pressure half-time (PHT), regurgitant fraction (RF) and aortic flow reversal (AoFR)

<table>
<thead>
<tr>
<th>Category</th>
<th>Perry index (%)</th>
<th>PHT (ms)</th>
<th>RF (%)</th>
<th>AoFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;25</td>
<td>&gt;550</td>
<td>&lt;30</td>
<td>Ascending aorta</td>
</tr>
<tr>
<td>Moderate</td>
<td>25–60</td>
<td>300–550</td>
<td>30–50</td>
<td>Desc. thor. aorta</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;60</td>
<td>&lt;300</td>
<td>&gt;50</td>
<td>Abdominal aorta</td>
</tr>
</tbody>
</table>

Summary of AI assessment (Table 6.3)

Tricuspid valve

Tricuspid stenosis

Aetiology
(1) Congenital
   TV atresia associated with RV hypoplasia
(2) Acquired
   Rheumatic
   Carcinoid
   Endocardial fibroelastosis
   Endomyocardial fibrosis

Features
Scarred, thickened leaflets/chordae
Commissural fusion (rheumatic)
Reduced leaflet opening
‘Doming’ of ant. leaflet (rheumatic)

Assessment of TS severity
(1) Planimetry: inaccurate due to position of TV attachments
(2) Doppler pressure gradient (Table 6.4)
(3) Continuity equation:

\[ TVA = MVA \times \frac{VTI_{MV}}{VTI_{TV}} \]

Inaccurate with TR
Table 6.4 Assessment of tricuspid stenosis by mean pressure gradient (PG)

<table>
<thead>
<tr>
<th></th>
<th>Mean PG (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Moderate</td>
<td>3–6</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;6</td>
</tr>
</tbody>
</table>

(4) Pressure half-time (PHT):

\[ TVA = \frac{190}{PHT} \]

**Tricuspid regurgitation**

Mild–moderate TR common in normal population

\[ \uparrow \] TR with age

\[ \uparrow \] TR with physical fitness

**Aetiology**

(1) Congenital

- TV dysplasia
- Ebstein’s anomaly

(2) Acquired

- Rheumatic
- Annular dilatation due to RV dilatation
- TV prolapse
- Carcinoid
- Infective endocarditis
- Tumours
- Trauma

**Features**

Ebstein’s

- apical attachment of TV leaflets (usually septal leaflet)
- atrialization of RV
- dilated RA/small RV
- septal TVL attaches to IVS >8 mm/m² (BSA) below anterior MVL
RV dilatation/pulmonary ↑BP (annular dilatation)
RA/IVC dilatation
TV prolapse assoc. with MV prolapse/Marfan's syndrome
Infective endocarditis assoc. with IV drug use/alcoholism
Thick, short TV leaflets with reduced motion (carcinoid)

**Assessment of TR severity**

1. **Jet length**
   - Trivial < 1.5 cm
   - Mild 1.5–3 cm
   - Moderate 3–4.5 cm
   - Severe > 4.5 cm

2. **Jet area**
   - Trivial < 2 cm²
   - Mild 2–4 cm²
   - Moderate 4–10 cm²
   - Severe > 10 cm²

3. **Jet length/RA length**
   - Mild < 33%
   - Moderate 33–66%
   - Severe > 66%

4. **Jet area/RA area**
   - Mild < 33%
   - Moderate 33–66%
   - Severe > 66%

5. **Systolic flow reversal in IVC/hepatic vein** = severe TR

**Pulmonary valve**

**Pulmonary stenosis**

**Aetiology**

1. Congenital
   - Uni-/bi-/quadricuspid valve
   - Fallot’s tetralogy
Table 6.5 Assessment of pulmonary insufficiency by regurgitant fraction (RF)

<table>
<thead>
<tr>
<th></th>
<th>RF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;40</td>
</tr>
<tr>
<td>Moderate</td>
<td>40–60</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;60</td>
</tr>
</tbody>
</table>

(2) Acquired
- Carcinoid
- Rheumatic

**Features**
- Thickened leaflets
- ‘Doming’ of leaflets
- $V_{\text{max}} > 1 \text{ m/s}$

**Pulmonary insufficiency**

**Aetiology**
1. Congenital
   - Uni-/bi-/quadricuspid valve
2. Acquired
   - Carcinoid
   - Infective endocarditis

**Assessment of PI severity**
1. Regurgitant fraction (Table 6.5)

**Valve surgery**

**Mitral valve repair**

Repair:
- reduced morbidity and mortality
Valvular heart disease

better durability
preserves tensor apparatus
avoids anticoagulation

BUT: 6–8% inadequate

Better for:
PMVL
annular dilatation
no calcification

(1) Carpentier I (normal leaflet motion)
Ring annuloplasty

(2) Carpentier II (↑leaflet motion)
Quadrangular resection of PMVL (usually P2)
Shortening of AMVL chordae
Transposition of PMVL chordae to AMVL
Secondary chordae transposition from AMVL body to leaflet tips
Partial resection of AMVL + ring annuloplasty

(3) Carpentier III (↓leaflet motion)
Commissurotomy
Resection of secondary chordae/fenestration of primary chordae
Resection of fused chordae
Balloon valvuloplasty

Valve replacement

Homografts
From cadaveric human hearts/cryopreserved

(1) Unstented:
usually AV
avoids anticoagulation
good durability
(2) Stented:
   usually MV
   duration ∼ 5 yrs

Bioprostheses
(1) Porcine:
   Hancock/Carpentier–Edwards
   premounted porcine AV
   leaflet degeneration/calcification
   duration ∼ 5–10 yrs
(2) Bovine:
   Ionescu–Shiley
   bovine pericardium
   calcification/abrasions → stenosis and regurgitation
   duration ∼ 5–10 yrs

Mechanical valves
(1) Ball-and-cage:
   Starr–Edwards
   Double cage with silastic ball
   Haemolysis occurs in AV position
   Duration ∼ 20 yrs
(2) Single tilting disc:
   Bjork–Shiley/Medtronic
   Single-hinged mobile disc
   Eccentric attachment
   Good durability
(3) Bileaflet tilting disc:
   St Jude
   Equal-sized semicircular leaflets with midline hinge

Normal valve replacement gradients (Table 6.6)
Table 6.6a Mean pressure gradients (PG) measured across different mitral valve replacements (MVR)

<table>
<thead>
<tr>
<th>MVR</th>
<th>Mean PG (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpentier–Edwards</td>
<td>6.5 ±/– 2.1</td>
</tr>
<tr>
<td>Hancock</td>
<td>4.3 ±/– 2.1</td>
</tr>
<tr>
<td>Starr–Edwards</td>
<td>4.5 ±/– 2.4</td>
</tr>
<tr>
<td>St Jude</td>
<td>3.5 ±/– 1.3</td>
</tr>
<tr>
<td>Bjork–Shiley</td>
<td>2.9 ±/– 1.6</td>
</tr>
</tbody>
</table>

Table 6.6b Mean and peak pressure gradients (PG) measured across different aortic valve replacements (AVR)

<table>
<thead>
<tr>
<th>AVR</th>
<th>Mean PG (mmHg)</th>
<th>Peak PG (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpentier–Edwards</td>
<td>12 ±/– 6</td>
<td>23 ±/– 8</td>
</tr>
<tr>
<td>Hancock</td>
<td>11 ±/– 2</td>
<td>22 ±/– 10</td>
</tr>
<tr>
<td>Starr–Edwards</td>
<td>24 ±/– 4</td>
<td>39 ±/– 12</td>
</tr>
<tr>
<td>Bjork–Shiley</td>
<td>14 ±/– 5</td>
<td>24 ±/– 9</td>
</tr>
<tr>
<td>St Jude</td>
<td>13 ±/– 6</td>
<td>26 ±/– 5</td>
</tr>
</tbody>
</table>

Multiple choice questions

1. Typical features of mitral stenosis include all of the following except
   A dilated left ventricle
   B thrombus in the left atrial appendage
   C commissural fusion
   D atrial fibrillation
   E ‘hockey stick’ appearance of the anterior leaflet

2. Regarding the assessment of mitral stenosis severity, the following statement is correct
   A pressure half-time is reduced with aortic incompetence
B transvalvular gradient overestimates the degree of mitral stenosis in the presence of aortic incompetence
C the continuity equation is accurate in the presence of aortic incompetence
D planimetry often overestimates the degree of mitral stenosis
E a depressurization time of 550 ms equates to severe mitral stenosis

3. Mitral regurgitation
A cannot be caused by myocardial ischaemia
B is classified as severe if the effective regurgitant orifice is greater than 0.4 cm²
C is classified as severe if the regurgitant volume is greater than 40 ml
D due to excessive leaflet motion is classified as Carpentier I
E due to myxomatous disease is usually classified as Carpentier III

4. In moderate mitral regurgitation
A the jet length is typically 1–2 cm
B the jet area is 4–7 cm²
C the regurgitant fraction is 50–75%
D there is reversal of pulmonary vein flow S wave
E the vena contracta is 0.5–0.75 cm

5. Causes of aortic stenosis include all of the following except
A congenital unicuspid valve
B congenital bicuspid valve
C degenerative calcification
D amyloidosis
E myocardial ischaemia

6. A mean pressure gradient of 40 mmHg across the aortic valve equates to
A aortic valve area of 2–4.5 cm²
B mild aortic stenosis
C moderate aortic stenosis
D a peak pressure gradient of 100 mmHg
E aortic valve area of 4–6 cm²

7. Features of mild aortic valve incompetence include
A Perry index greater than 60%
B regurgitant fraction greater than 60%
C regurgitant volume greater than 60 ml
D pressure half-time greater than 600 ms
E diastolic flow reversal in the abdominal aorta

8. In aortic incompetence, a Perry index of 50% is consistent with
   A pressure half-time of 550 ms
   B regurgitant fraction of 25%
   C diastolic flow reversal in the descending thoracic aorta
   D diastolic flow reversal in the abdominal aorta
   E pressure half-time of 750 ms

9. In the assessment of tricuspid stenosis severity
   A planimetry is the most accurate method
   B mean pressure gradient of 9 mmHg is severe stenosis
   C the continuity equation is accurate in the presence of tricuspid regurgitation
   D pressure half-time of 220 ms is mild stenosis
   E pressure half-time of 110 ms gives an approximate tricuspid valve area of 2.2 cm²

10. The following statements regarding tricuspid regurgitation are all true except
    A Ebstein’s anomaly results in a small right atrium with a dilated right ventricle
    B carcinoid disease is a cause
    C a jet length of 7 cm is considered to be severe
    D a jet area of 11 cm² is severe
    E mild regurgitation is common in the normal population

11. The maximum velocity across a normal pulmonary valve is
    A 1–2 cm/s
    B 6–9 cm/s
    C 10–20 cm/s
    D 60–90 cm/s
    E 1–1.2 m/s

12. Regarding heart valve surgery
    A St Jude valve is an example of a bileaflet tilting disc
B ring annuloplasty is usually not suitable for Carpentier I mitral regurgitation

C the mean pressure gradient across a Hancock mitral valve replacement is approximately 11–12 mmHg

D the advantage of valve replacement is avoidance of anticoagulation treatment

E commissurotomy is suitable for Carpentier II mitral regurgitation
Cardiac masses

Tumours

Primary tumours

Myxoma
A myxoid matrix of acid mucopolysaccharide and polygonal cells

Benign
25% of all primary cardiac tumours
75% in LA/20% in RA/5% other sites in heart
LA myxomas: 90% on IAS (fossa ovalis)
Usually present between 30 and 60 years of age
May be part of a syndrome (Carney’s complex)
Homogenous echo appearance
May contain calcium, haemorrhage or secondary infection
Soft, friable, gelatinous, and pedunculated

Features:
- disruption of MV function
- emboli
- systemic symptoms (fever, malaise)

Lipoma
Occur throughout the heart
Subepicardial: large, smooth, and pedunculated
Subendocardial: small and sessile
Less mobile/more echodense than myxomas
May cause arrhythmias/conduction defects
May present with pericardial effusion

**Papillary fibroelastoma**
Small (usually < 1 cm)
Attached to valve surfaces/supporting valvular apparatus
Round/oval tumour with well-demarcated border
Homogeneous texture
May cause systemic embolization

**Rhabdomyoma**
Common paediatric primary tumour
Assoc. with tuberous sclerosis
90% multiple/nodular masses
Associated with outflow tract obstruction
May resolve spontaneously

**Fibroma**
Solitary
Occur in LV/RV myocardium
Firm with central calcification
May appear as localized irregular myocardial hypertrophy
May be mistaken as thrombus at the apex of the heart
Cause dysrhythmias and congestive cardiac failure

**Haemangioma**
Solitary and small
Occur in RV/IVS/AV node
Cause complete heart block

**Cysts**
Mesotheliomas: primary malignant tumour of pericardium
Teratomas: intrapericardial or intracardiac
Benign cysts: fluid-filled recesses of parietal pericardium
Echinococcal cyst: secondary to echinococciosis
Malignant tumours
25% of all primary cardiac tumours are malignant
Angiosarcomas
Rhabdomyosarcomas
Lymphosarcomas

Secondary tumours
Cardiac metastases reported in up to 20% of patients with malignant tumours.
Metastases by
(1) direct extension
(2) lymphatic spread (carcinoma)
(3) haematogenous spread (melanoma/sarcoma)
Common primary malignancy metastasizing to the heart include
(1) lung
(2) breast
(3) melanoma
(4) leukaemia
(5) lymphoma
(6) ovary
(7) oesophagus
(8) kidney
Most common spread to heart via IVC includes
(1) renal cell carcinoma
(2) Wilms’ tumour (paediatric)
(3) uterine leiomyosarcoma
(4) hepatoma

Carcinoid syndrome
Patient with carcinoid tumour of ileum with hepatic metastases
Right-sided heart lesions
Left-sided lesions with bronchial carcinoid/ASD/PFO
Endocardial thickening causing fixation of TV and PV
TR universal finding, usually with PS

**Thrombus**

Found in setting of
  - Blood stasis
  - AF
  - Reduced CO states
  - MV disease
  - Prosthetic MV
  - Post-MI
  - RWMA

**Features**

Round/oval masses
‘Speckled’ with ↑echodensity compared to LA/LV wall
Interrupts normal endocardial contour
Posterior and lateral walls of LA/LAA
Apex of LV
Associated with ‘smoke’ in LA

**Effects**

Mechanical disruption of valve function
Causes emboli

**Pseudomasses**

**Trabeculations**

Muscle bundles on endocardial surfaces
More common in RA/RV than LA/LV
Accentuated by RVH
May occur in LAA

**False tendons**
Fine filamentous structures in LV
No clinical significance

**Pectinate muscles**
Parallel ridges across anterior endocardium of LA (LAA) and RA
No clinical significance

**Moderator band**
Prominent muscle band in apical third of RV
Involved with conduction system
Confused with thrombus/tumour

**Lipomatous hypertrophy of IAS**
Lipomatous thickening of IAS > 1 cm
Benign
‘Dumb-bell’ appearance of IAS
Lack of involvement of fossa ovalis

**Eustachian valve**
= Remnant of valve of sinus venosus
Occurs in 25% of individuals
At junction of IVC and RA
Elongated, membranous undulating structure

**Chiari network**

? Remnant of sinus venosus derived structures
Mobile, filamentous, thin structure in RA
Highly mobile/random movement in RA
? Associated with PFO/IAS aneurysm
Crista terminalis
Remnant of valve of sinus venosus
At junction of SVC and RA

Thebesian valve
Thin piece of tissue guarding coronary sinus
May inhibit retrograde coronary sinus cannulation

Warfarin ridge
Atrial tissue separating LAA from LUPV

Vegetations

TTE sensitivity ~ 80%
TOE sensitivity ~ 95% (reduced with prosthetic valves)

Features
Classic triad
changing murmur
fever
positive blood cultures
Variable appearance
discrete sessile mass
pedunculated friable clump
elongated strand
Occur on low pressure side of valves
Usually at leaflet tips
Right-sided vegetations usually larger than left-sided
Fungal vegetations larger than bacterial
Chronic, healed vegetation = fibrotic and echodense
Multiple choice questions

1. Atrial myxomas
   A comprise 75% of all primary cardiac tumours
   B usually arise from the appendage in the left atrium
   C are usually malignant
   D cause systemic symptoms of fever and malaise
   E occur in the right atrium in 5% of cases

2. Features of cardiac thrombus include all of the following except
   A association with ‘smoke’ in the left atrium
   B association with reduced cardiac output states
   C ‘speckled’ oval mass in the left atrial appendage
   D reduced echodensity compared to the ventricular wall
   E mechanical disruption of valve function

3. The following statements regarding cardiac pseudomasses are all true except
   A false tendons occur in the left ventricle
   B trabeculations are muscle bundles on epicardial surfaces
   C the Eustachian valve is the embryological remnant of the valve of the sinus venosus
   D the crista terminalis occurs at the junction of the right atrium and the superior vena cava
   E a thebesian valve may inhibit retrograde coronary sinus cannulation

4. Regarding cardiac vegetations
   A transthoracic echocardiography is more sensitive than transoesophageal echocardiography for diagnosis
   B transoesophageal echocardiogram sensitivity is increased in the presence of prosthetic heart valves
   C they usually occur on the high pressure side of valves
   D right-sided vegetations are usually larger than left-sided
   E bacterial vegetations are usually larger than fungal ones
Congenital heart disease

Valve defects

Mitral valve

Parachute MV
Normal leaflets attach to single, large papillary muscle
Reduced leaflet motion → MS

Cleft mitral valve
‘Clefts’ in ant MV leaflet
Accessory chordae attach to cleft margins, holding leaflets anteriorly
during systole → MR

Mitral arcade
Fibrous bridge between papillary muscles with poor commissural development
Arcade prevents closure of AMVL → MR

Aortic valve

Unicuspid
Acommissural with central orifice
Commissural with eccentric orifice → AS

Bicuspid
Most common congenital cardiac defect (1–2% of population)
AS + AI
Congenital heart disease

Common site for bacterial endocarditis
Associated with coarctation/PDA/ascending aortic aneurysm

**Quadricuspid**
AI
Associated with truncus arteriosus

**Tricuspid valve**

**Atresia**
Large RA/hypoplastic RV
VSD present
Treatment: Fontan/Glenn procedures
= conduit from IVC/SVC to PA

**Ebstein’s anomaly**
Apical displacement of TV leaflets (usually septal TVL)
Atrialization of RV → large RA/small RV
Diagnosis: septal TVL attaches to IVS > 8 mm/m² below ant MVL
AMVL – LV apex/STVL – RV apex > 1.8
Associated with TR/ASD

**Pulmonary valve**

Uni-/bi-/quadricuspid valve → PS
Congenital absence of PV
Fallot’s tetralogy: PS

**Ventricular defects**

**Univentricle**
Two atria → one ventricle
Second ventricle hypoplastic/absent
TOE assessment
(1) Accessory chamber
   Hypoplastic or absent
(2) Atrio-ventricular valve function
   2 AV valves 65%
   1 AV valve 35%
(3) Great vessel orientation
   Aorta or PA may arise from either
   Hypoplastic or functioning ventricle
   Associated with TGA
(4) RVOT/LVOT obstruction
   Hypoplastic PA common
(5) Univentricle function
   Response to volume/pressure overload
(6) Venous return
   Associated with TAPVD

Treatment
Aorto-pulmonary shunt:
   Waterson = asc. aorta → PA
   Potts = desc. aorta → LPA
Blalock–Taussig shunt:
   R subclavian artery → RPA
Septation:
   creation of artificial IVS

Great vessels

Fallot’s tetralogy
(1) PS: usually infundibular with PA hypoplasia
(2) VSD: perimembranous
(3) Overriding aorta
(4) Concentric RV hypertrophy
Congenital heart disease

Associated with
  Abnormal coronary anatomy (2–5%)  
  Secundum ASD  
  PDA  
  Right-sided aortic arch

Treatment
(1) Unobstructed PV:
  valvulotomy
(2) Two-stage:
  initial aorto-pulmonary shunt  
  later valved conduit from RV to PA (Rastelli)

Transposition of great arteries (TGA)
Aorta from RV/PA from LV
Associated with
  VSD  
  Secundum ASD  
  Abnormal atrio-ventricular (A–V) valves  
  LVOT/RVOT obstruction  
  PDA  
  Abnormal coronary anatomy

Treatment
Early arterial switch procedure  
Palliative balloon atrial septostomy with later repair (Mustard)

Truncus arteriosus (TA)
Single trunk from heart provides aorta/PA/coronary arteries
Associated with
  Large VSD  
  Abnormal truncal valve
Right-sided aortic arch
Abnormal coronary anatomy

**Treatment**
Close VSD
Repair/replace truncal valve
Conduit from RV to PA

**Patent ductus arteriosus (PDA)**
Normal in fetus/closes by third day after birth
Causes L $\rightarrow$ R shunt with ↑PA flow
Abnormal diastolic flow in PA seen with TOE

**Coarctation**
Localized defect of media with eccentric narrowing of lumen
Adult type = postductal narrowing
Infantile type = preductal coarctation

**Venous return**

**Total anomalous pulmonary venous drainage (TAPVD)**
(1) Supracardiac: PVs $\rightarrow$ SVC/innominate vein
(2) Cardiac: PVs $\rightarrow$ RA/coronary sinus
(3) Infracardiac: PVs $\rightarrow$ IVC/portal vein
(4) Mixed

**ASD**

**Primum ASD**
20% of ASDs
Due to incomplete fusion of septum primum
Low in septum (Fig. 8.1)
Secundum ASD

70% of ASDs
Due to incomplete development of septum secundum
In region of fossa ovalis (Fig. 8.1)

Patent foramen ovale (PFO)

Present in ~ 25% of population
Incomplete closure of foramen ovale at birth

Sinus venosus ASD

6–8% of ASDs
Superior sinus venosus: high in septum by SVC
Inferior sinus venosus: low in septum by IVC
Associated with TAPVD (Fig. 8.1)
Coronary sinus (CS) ASD

At site of origin of CS (Fig. 8.1)
Associated with unroofed CS/persistent left SVC

Endocardial cushion defects

Due to A–V canal defects

Complete
Large primum ASD
Inlet of IVS deficient with large VSD

Partial
Primum ASD
Cleft MV

VSD

Supracristal

Above level of crista supraventricularis (Fig. 8.2)
Immediately inferior to PV and AV (LCC and RCC)
= infundibular VSD

Infracristal

Inferior and posterior to crista supraventricularis (Fig. 8.2)

(1) Membranous: beneath AV (RCC/NCC)
(2) Muscular: occur post-MI
(3) Inlet VSD
Multiple choice questions

1. The following statements regarding the bicuspid aortic valve are all true except
   A it is associated with ascending aortic aneurysm
   B it is a common site for bacterial endocarditis
   C it occurs in approximately 1–2% of the population
   D aortic incompetence does not occur
   E it is associated with coarctation of the aorta

2. In Ebstein’s anomaly
   A there is apical displacement of the mitral valve leaflets
   B diagnosis is made when the septal tricuspid valve leaflet attaches to the interventricular septum more than 8 mm above the anterior mitral valve leaflet
   C tricuspid regurgitation is not a feature
   D there is an association with atrial septal defect
   E atrialisation of the left ventricle occurs
3. Regarding congenital ventricular defects,
   A the accessory chamber is usually hypertrophied
   B there is an association with total anomalous pulmonary venous drainage
   C two atrioventricular valves occur in 35% of cases
   D echocardiographic assessment of the right ventricular outflow tract is not important
   E it can be treated by the Rastelli procedure

4. Fallot’s tetralogy
   A includes a muscular ventricular septal defect
   B has abnormal coronary anatomy in 50% of cases
   C is treated by the Mustard procedure
   D usually includes eccentric right ventricular hypertrophy
   E can be initially managed with an aorto-pulmonary shunt

5. The following statements regarding congenital heart defects are all true except
   A transposition of the great arteries is associated with secundum atrial septal defect
   B truncus arteriosus is associated with abnormal coronary anatomy
   C patent ductus arteriosus causes a right to left shunt
   D adult type coarctation involves postductal narrowing
   E in total anomalous pulmonary venous drainage, pulmonary veins may drain into the coronary sinus

6. Regarding atrial septal defects (ASDs)
   A 70% are primum ASDs
   B 20% are secundum ASDs
   C 17% are sinus venosus ASDs
   D secundum ASDs occur low in the interatrial septum
   E primum ASDs are due to incomplete fusion of the septum primum

7. Endocardial cushion defects (ECDs)
   A involve aortic valve defects
   B in complete ECDs there is usually a small ventricular septal defect
   C partial ECDs are associated with cleft mitral valve
   D complete ECDs have a small secundum atrial septal defect
   E partial ECDs have a large secundum atrial septal defect
8. Regarding ventricular septal defects (VSDs)
   A supracristal VSDs include membranous VSDs
   B membranous VSDs usually occur beneath the right and non-coronary cusps of the aortic valve
   C infracristal VSDs include infundibular VSDs
   D infracristal VSDs do not occur post-myocardial infarction
   E infundibular VSDs are best seen on a mid-oesophageal four-chamber view
Extracardiac anatomy

Pericardium

Effusion
Normal pericardial sac contains 20–30 ml of fluid from subepicardial lymphatics

Causes
(1) Idiopathic
(2) Cardiac: CCF, post-MI, post-cardiac surgery
(3) Metabolic: hypoalbuminaemia, uraemia, hypothyroidism
(4) Infective: bacterial, TB, viral, fungal
(5) Trauma
(6) Connective tissue disease: SLE, rheumatoid arthritis
(7) Neoplasm
(8) Drugs: hydralazine
(9) Radiotherapy

Size
(1) Small: < 100 ml
   localized behind posterior LV
(2) Moderate: 100–500 ml
(3) Large: > 500 ml
   swinging of heart in fluid
   electrical alternans on ECG
Chronic effusion causes fibrinous exudates on pericardial surface
Fibrin strands appear as ‘soap-suds’ on visceral pericardium
Tamponade

Impairment of diastolic filling caused by raised intrapericardial pressure (IPP)
Due to

(1) rapid accumulation of small amount of fluid
(2) gradual collection of large volume of fluid

IPP dependent on compliance of pericardium and volume within pericardium
As intra-pericardial volume increases, IPP increases (Fig. 9.1)
As IPP↑ cardiac volume is maintained by increasing venous pressure to maintain venous return
When IPP = venous pressure (volume ~ 60–80 ml) → steep part of compliance curve
When IPP > venous pressure → stroke volume falls
RV filling pressure = LV filling pressure

Effect of respiration
(1) Normal

Inspiration → Fall in intrapleural pressure
→ This fall transmitted to IPP
→ Expansion of RA and RV into pericardial space
→ ↑Venous return to right side
(2) Tamponade

Inspiration → Fall in IPP less than normal
→ RV fills
→ RV unable to expand into pericardial space
→ RV expands to the left
→ IVS shifts to the left
→ LV filling compromised
→ ↓LVEDV
→ ↓CO and ↓SBP during inspiration

Onset of systole: ↓RAP = ‘x’ descent
Onset of diastole: no fall in RAP = no ‘y’ descent
Right-sided filling becomes monophasic (confined to systole)
Transient pressure gradient reversal: IPP > RAP/RVP
→ RV wall inversion in diastole
→ RA wall inversion in late diastole/early systole
↑venous return to right side → ↑RV volume
→ LV compromise
→ ↑TTF by 80% / ↓TMF by 40%

Pericarditis

Pericardium becomes rigid due to
  Inflammation
  Fibrosis
  Calcification
  Neoplasms
Impedes diastolic filling

Causes
(1) Hereditary
(2) Metabolic: uraemia
(3) Infection: bacterial, viral, parasitic
(4) Trauma
(5) Connective tissue disease: polyarteritis nodosa, SLE
Table 9.1 Constrictive vs. restrictive pathophysiology

<table>
<thead>
<tr>
<th>Constrictive</th>
<th>Restrictive</th>
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<tbody>
<tr>
<td>Thickened calcified pericardium</td>
<td>Normal pericardium</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td></td>
</tr>
<tr>
<td>Normal PA pressures</td>
<td>↑PA pressures</td>
</tr>
<tr>
<td>MAPSE preserved</td>
<td>MAPSE reduced</td>
</tr>
<tr>
<td>Large respiratory variation in TTF and TMF</td>
<td>Minimal (&lt;5%) respiratory variation in TTF and TMF</td>
</tr>
<tr>
<td>Inspiration → ↑TTF/↓TMF</td>
<td>Inspiration → ↓RVSP/↓LVSP</td>
</tr>
<tr>
<td>Respiratory variation in pulmonary venous flow</td>
<td>Hepatic vein flow → ↑SR</td>
</tr>
<tr>
<td>Inspiration → ↑RVSP/↓LVSP</td>
<td></td>
</tr>
<tr>
<td>Hepatic vein flow → ↓D/↑DR</td>
<td></td>
</tr>
</tbody>
</table>

(6) Neoplasms
(7) Post-cardiac surgery
(8) Radiotherapy

Diagnosis:
- normal ventricular size
- ↓diastolic function
- IVC/HV dilated
- pericardial thickening

Early rapid ventricular filling (rapid ‘y’ descent), which stops abruptly as limit of ventricular expansion achieved
- Respiratory variations in intrapleural pressure not transmitted to heart due to dense pericardial encasement

Constrictive vs. restrictive physiology (Table 9.1)
Limitation to diastolic ventricular filling occurs earlier in CONstrictive pathology because of fixed volume within the pericardial sac
- Myocardial relaxation prolonged in restrictive disease
- Variation of IVRT on inspiration with CONstrictive pathology
Aortic disease

Atherosclerosis

Severe disease of descending aorta increases likelihood of aortic arch disease

Grading
I: Minimal intimal thickening
II: Extensive, widespread intimal thickening
III: Sessile atheroma
IV: Atheroma protruding into aortic lumen
V: Mobile, protruding atheroma

Aneurysm

Dilatation of all layers of aortic wall

Causes
Atherosclerosis
Cystic medial necrosis
Trauma
Congenital (Marfan’s)
Syphilis

Affects ascending aorta/aortic arch/thoracic and abdominal aorta

Dissection

Degeneration/destruction of media
Associated with
  Hypertension
  Connective tissue disease
  Turner’s syndrome
  Coarctation
Table 9.2 Comparison of Stanford type A and B dissection

<table>
<thead>
<tr>
<th></th>
<th>Stanford A</th>
<th>Stanford B</th>
</tr>
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<tbody>
<tr>
<td>Frequency (%)</td>
<td>70</td>
<td>30</td>
</tr>
<tr>
<td>Age</td>
<td>50</td>
<td>70</td>
</tr>
<tr>
<td>Male : female</td>
<td>2:1</td>
<td>3:1</td>
</tr>
<tr>
<td>Associated ↑BP (%)</td>
<td>50</td>
<td>80</td>
</tr>
<tr>
<td>AI (%)</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Acute mortality (%)</td>
<td>90</td>
<td>40</td>
</tr>
</tbody>
</table>

Classification
(1) Stanford (Table 9.2)
   A: proximal tear
   B: distal tear

(2) De Bakey
   I: proximal tear, extending distally
   II: proximal tear
   IIIA: distal tear, extending proximally
   IIIB: distal tear

Management
Stanford A → surgery
Stanford B → medical therapy

Multiple choice questions
1. Causes of pericardial effusion include all of the following except
   A  Wilson’s disease
   B  neoplastic disease
   C  trauma
D rheumatoid arthritis
E radiotherapy

2. Regarding intrapericardial pressure (IPP)
   A when IPP increases to equal venous pressure, right ventricular filling pressure will equal left ventricular filling pressure
   B IPP is independent of intrapericardial volume
   C when IPP exceeds venous pressure stroke volume increases
   D IPP equals venous pressure at a volume of 500 ml
   E IPP is independent of pericardial compliance

3. In adults, cardiac tamponade
   A is caused by an intrapericardial volume of 20 ml
   B is due to a gradual accumulation of a small amount of fluid
   C causes a rapid ‘y’ descent on the central venous waveform
   D causes right atrial wall eversion in diastole
   E causes right ventricular wall inversion in diastole

4. The following statements about pericarditis are all true except
   A it is caused by systemic lupus erythematosus
   B late ventricular filling occurs due to high intraventricular pressure
   C it impedes diastolic filling
   D respiratory variations in intrapleural pressure are not transmitted to the heart
   E the hepatic vein is usually dilated

5. In constrictive cardiac pathology
   A mitral annular plane systolic excursion is reduced
   B pulmonary hypertension is common
   C right ventricular systolic pressure decreases on inspiration
   D left ventricular systolic pressure decreases on inspiration
   E transmitral flow increases on inspiration

6. In restrictive cardiac pathology
   A the pericardium appears thickened and calcified
   B left ventricular systolic pressure decreases on inspiration
   C pulsus paradoxus is a feature
   D isovolumic relaxation time varies on inspiration
   E there is increased respiratory variation in pulmonary venous flow
7. All of the following may cause thoracic aortic aneurysm except
   A cystic medial necrosis
   B syphilis
   C gonorrhoea
   D Marfan’s syndrome
   E atherosclerosis

8. The following statements about thoracic aortic dissection are all true except
   A it is associated with coarctation of the aorta
   B Stanford type A has a higher acute mortality than type B
   C De Bakey type II involves a proximal aortic dissection
   D surgery is indicated in Stanford type A
   E aortic valve incompetence is more common in Stanford type B than type A
Haemodynamic calculations

**Doppler equation**

\[
\text{velocity} = \frac{c f_D}{2 f_0 \cos \theta} \\
\frac{f_D}{2} = \frac{2 v f_0 \cos \theta}{c}
\]

**Bernoulli equation**

\[
P_1 - P_2 = \left[ \frac{1}{2} \rho \left( V_2^2 - V_1^2 \right) \right] + \left[ \rho^2 \frac{dV}{dt} \frac{ds}{ds} \right] + \left[ RV^2 \right]
\]

Convective Flow Viscous acceleration acceleration friction

Modified Bernoulli \[ \Delta P = 4V^2 \]

**Intracardiac pressures**

\[
\begin{align*}
RVSP &= RAP + 4V^2 \text{ (TR)} \\
PADP &= RAP + 4V^2 \text{ (PI)} \\
LAP &= SBP - 4V^2 \text{ (MR)} \\
LVEDP &= DBP - 4V^2 \text{ (AI)}
\end{align*}
\]
Flow

Flow = Area × Velocity
SV = Area × VTI

Aortic valve

Aortic stenosis

\[ \Delta P = 4V^2 \]
AVA = Area_{LVOT} × \( V_{max_{LVOT}} / V_{max_{AV}} \)
AVA = SV_{AV} / VTI_{AV}

Aortic incompetence

RF% = SV_{LVOT} - SV_{MV}/SV_{LVOT}

Mitral valve

Mitral stenosis

\[ \Delta P = 4V^2 \]
MVA = 220/PHT
MVA = Area_{LVOT} × VTI_{LVOT}/VTI_{MV}
MVA = 6.28r^2 × \( \alpha/180 \times V_{alias} / V_{max_{MV}} \)

Mitral regurgitation

RV = (Area_{MV} × VTI_{MV}) − (Area_{LVOT} × VTI_{LVOT})
RF = SV_{MV} − SV_{LVOT} / SV_{MV}
ERO = 6.28r^2 × V_{alias} / V_{max_{MR}}
Multiple choice questions

1. A peak Doppler velocity of 4 m/s across the aortic valve equates to a peak pressure gradient of
   A 4 mmHg  
   B 16 mmHg  
   C 32 mmHg  
   D 64 mmHg  
   E 80 mmHg

The following data apply to Questions 2–4
   Right atrial pressure = 10 mmHg
   Left ventricular end diastolic/left atrial pressure = 18 mmHg
   Tricuspid regurgitation jet peak velocity = 3 m/s
   Mitral regurgitation jet peak velocity = 5 m/s
   Pulmonary insufficiency jet peak velocity = 1 m/s
   Aortic incompetence jet peak velocity = 4 m/s
   Mean arterial pressure = 94 mmHg

2. The right ventricular systolic pressure is
   A 46 mmHg  
   B 36 mmHg  
   C 26 mmHg  
   D 16 mmHg  
   E 12 mmHg

3. The systemic systolic pressure is
   A 82 mmHg  
   B 100 mmHg  
   C 118 mmHg  
   D 130 mmHg  
   E 146 mmHg

4. The systemic diastolic pressure is
   A 56 mmHg  
   B 64 mmHg  
   C 72 mmHg  
   D 82 mmHg  
   E 88 mmHg
The following data apply to Questions 5–6
Left ventricular outflow tract area = 3 cm²
Left ventricular maximum velocity = 1.5 m/s
Aortic valve maximum velocity = 4.5 m/s
Aortic valve VTI = 40 cm

5. Aortic valve area is
   A 0.5 cm²
   B 1.0 cm²
   C 1.2 cm²
   D 1.5 cm²
   E 2.0 cm²

6. Aortic valve stroke volume is
   A 40 ml
   B 50 ml
   C 60 ml
   D 70 ml
   E 80 ml

The following data apply to Questions 7–8
Mitral valve area = 5 cm²
Mitral valve VTI = 16 cm
Mitral regurgitation jet peak velocity = 4 m/s
Left ventricular outflow tract stroke volume = 50 ml

7. Mitral valve regurgitant volume is
   A 14 ml
   B 24 ml
   C 30 ml
   D 38 ml
   E 50 ml

8. Mitral valve regurgitant fraction is approximately
   A 25%
   B 38%
   C 48%
   D 60%
   E 80%
# MCQ answers

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3. A 7. E 11. A

Chapter 5

1. E 3. C
2. A 4. B

Chapter 6

2. A 6. C 10. A

Chapter 7

1. D 3. B
2. D 4. D

Chapter 8

1. D 5. C
2. D 6. E
3. B 7. C
4. E 8. B
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