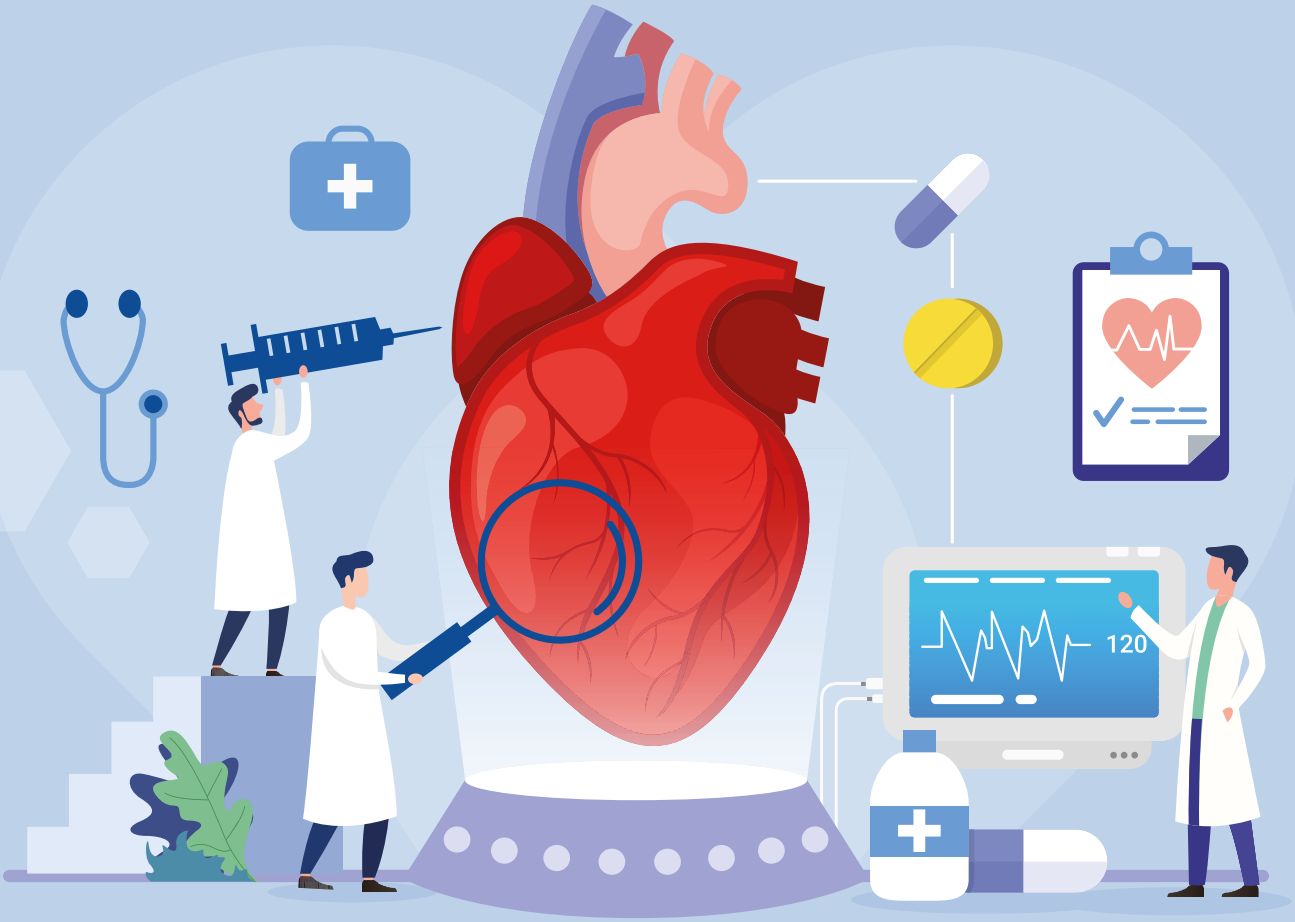

Student Section Cardiology Specialty Educational Booklet

January 2020

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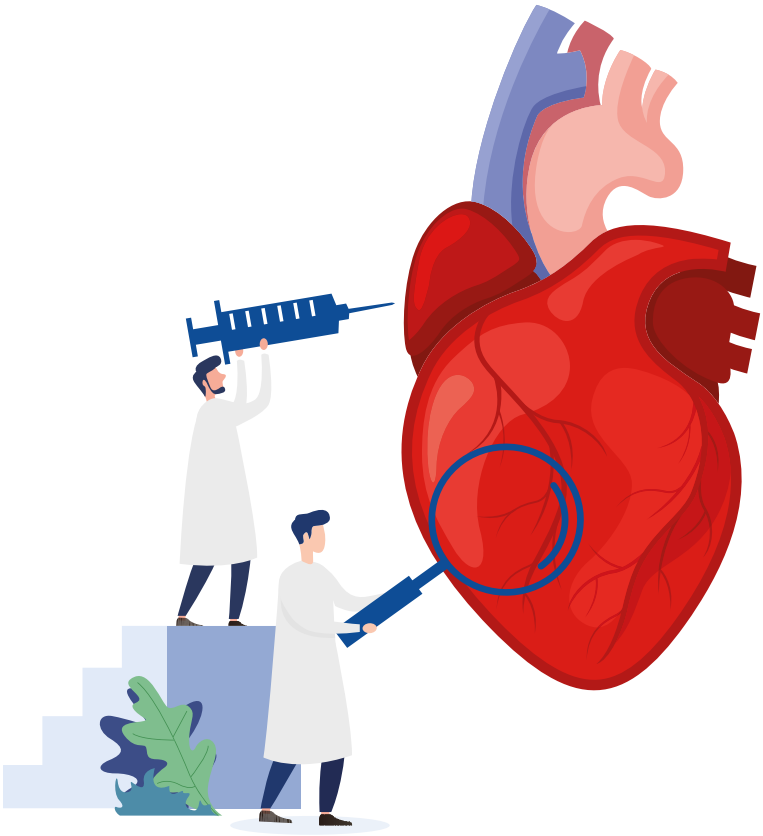


Note from the Editors

We are proud to publish our third Educational booklet, continuing to broaden our understanding of medicine and the opportunities which we are lucky enough to be presented with. Cardiology and cardiothoracic surgery are specialties which are challenging, and rewarding – given the prevalence of cardiovascular disease in this country and worldwide, we will be confronted to this burden of disease, if not as medical professionals, at least as relatives or friends.

We have several perspectives to present to you in this booklet – Alicia Huang (p.4) shares her motivation to pursue the specialty, whilst Prof Berry (p.11) and Mr Unsworth-White (p.12) give us insight into their immensely rewarding careers. Gabrielle Sanders's personal experience (p.3) of coming close with cardiac disease reminds us of the necessity of being trained in order to perform our jobs well, but also to be able to help those directly around us. Nadeem Ahmed's elective experience might also inspire you (p.7).

We hope to help you to understand cardiothoracics in a practical manner, relevant to medical undergraduates, so do not miss James Walker's excellent study guide on congenital heart defects (p.5), and a handy revision checklist for cardiovascular examinations! (p.13) Emma Norton's visual summary of valve pathologies (p.9) will come in handy to refresh your mind before exams or a new placement.

To further this journey into the world of cardiothoracics, read Abdo Kamaledeen's review (p.7) for a taste of the greatest discoveries of the 20th century in the specialty. An example of a novel technique used currently is laid out in an engaging patient case on the use of intra-aortic balloon pump during percutaneous coronary intervention (p.8).

As ever, we hope that this compilation of student views, expert opinions and interesting study guides will be of use to students across the country – if you find this series helpful, please do share it with your friends and colleagues. We encourage submissions for articles, opinion pieces, study aids and will gladly consider original formats; get in touch at rsmstudentssection@gmail.com.

Wishing you all a very happy new year!

On behalf of the RSM Student Section committee,

Mary Goble, University of Sheffield

Editor

My Grandad's collapse

Gabrielle Sanders

It's a Monday morning in November in my Grandparents bungalow. Mum, a private hairdresser by trade, is cutting my Nan and Grandad's hair in the kitchen before returning back to London later that afternoon, wrapping up her weekend visit. Its Grandad's turn, and the three of them are chatting away, but Grandad starts to get very agitated: he's moving around a lot in his chair and holding his chest. Mum picks up on his odd behaviour and asks what is wrong, to which he replies he has some pain in his chest. He has lived with angina ever since I can remember, and Mum knew to fetch his GTN spray. This unfortunately had no effect and a few seconds later he slumps forwards off his chair with his hands clasping his chest and becomes unresponsive; he is dribbling.

My mum had undergone repeated training in BLS as she works part time at a primary school, where she is required to renew this qualification every 3 years, the most recent session taking place two years ago. However, she was panicked as in this case, the casualty was her father, and the situation was completely unexpected. They called for an ambulance immediately.

In the initial panic whilst the operators were taking the basic details from my Nan, the two generations were debating whether to put him into the recovery position or start CPR, trying to piece together what they had seen on TV and been taught during training programmes-which when unrehearsed for two years, was a challenge. Mum placed her hands on his chest and was about to begin compressions without feeling for a pulse first- never mind following the "DRs ABC" approach to recheck his level of consciousness. Luckily he groaned in pain and signalled to withdraw her hands before she pressed any harder. I would like to highlight this all happened within a matter of seconds, and the telephone operators did an excellent job of talking the team through step by step and reassuring them that they were doing a great job. However, it was as if time were moving in slow motion; it took 20 minutes for the ambulance to arrive, during which they were keeping my Grandad calm and encouraging him to breathe.

I am so proud of how quickly my mum and Nan responded to the incident and called the ambulance straight away. We find humour in my mum attempting chest compressions on my Grandad now, however the incident has made me reflect on the value of BLS qualifications and how renewal every 3 years is perhaps too long of an interval. It is so important for the general public, and healthcare professionals alike, to practice what they learn during training courses on their own friends and family members after sessions in their own homes. We must make effort to generalize our knowledge beyond the classroom, rather than keeping the memory of BLS protocols confined to plastic dummies and teaching environments.

Secondly, we must make the effort to continue to practice, every few months or so, to ensure these skills are engrained into our muscle memory. We must not fall for the false sense of security of having a qualification in BLS and assume we are competent, because when your casualty is a family member, it is so much harder to slow your mind down and ensure you are following a logical sequence, even if you are in good practice. In order to execute BLS effectively, the first aider must be confident in their approach and not be afraid to make a mistake or hurt the person. Although my mum is trained in BLS, she was doubting her knowledge and getting confused with the order due to the combination of lack of practice and the intense stress of the situation.

Thirdly, we should recognise the huge impact asking for help can have on the outcome. No matter your level of experience, it is so important to remain humble. As a healthcare professional, we may feel we are expected to have these situations under control and be embarrassed or ashamed to ask for help. As well as the added emotional turmoil of treating a family member, we are also biased towards their past medical history, and we may overlook warning signs, easily spotted by an outsider who has no connection to our loved ones, or a non-medical member of the family who has no biases towards red flag signs. It must become second nature to always seek help in emergency.

Beyond my own family experience, as a medical student during my A&E and ITU rotations, I have observed too many cases whereby a patient was unconscious and experienced prolonged "downtime", not because nobody was present on the scene, but due to ineffective chest compressions. Some common errors included performing chest compressions on a soft surface such as a bed, and failing to press deeply enough. This may be because of inadequate education; our media not helping this situation - I am sure we have all watched scenes of a Dr performing CPR in a soap opera and been shouting at the TV "that's not how you do it!" - or alternatively, adequate education which is not revised or inappropriately executed in a panic. It is vital we ensure that not only the general public receive adequate BLS education, but that we are continually revisiting the approach regularly to empower ourselves and believe that we are competent in the moment, taking confident action.

Why I Want to be a Cardiologist

Alicia Huang

Sometimes, you can be drawn to a specialty for such a long time that you forget quite how to verbalise the rationale behind your passion. However, there is always a beginning to each story, and I suppose my journey with cardiology began back in secondary school.

As a naïve 17-year-old from a small village in Somerset, I applied for work experience in Hammersmith Hospital, London. I was serendipitously placed into the Department of Cardiothoracic Surgery where for the first time, I saw the beautiful complexity of the human heart. My escape from theatre into the cardiac catheterization lab showed me both the diagnostic, elective side of cardiology, as well as acute cardiological emergencies. The former involved use of angiography to identify coronary artery stenosis, and the latter was a primary PCI for a ST elevation myocardial infarction (STEMI). In both cases, I was amazed that symptoms were so effectively relieved – one primarily by pharmacological intervention, and the other primarily by physical intervention. As I have personally never had a surgical mind (or should I say hands?), the idea of specialising in cardiology was something that started from that day.

The most appealing aspect of cardiology to me is that it makes a tangible difference to so many people's lives. Unfortunately, cardiovascular diseases are the biggest cause of death globally[1]. Not only can cardiologists improve patients' quality of life, but also their life expectancy - surely medicine can't get much more rewarding than that! Furthermore, in cardiology you experience a balance between attending acute emergencies and taking care of patients with chronic conditions. It is also a very broad specialty - you will see cardiology in every single specialty; be it an aortic dissection in ED, a congenital heart defect in paediatrics, or heart failure in geriatrics.

Career-wise, the unique aspect about cardiology is the ability to combine aspects of general medicine, radiology, and surgery into one whole package. Many cardiologists can also subspecialise in aspects such as interventional cardiology, structural heart disease, electrophysiology, imaging, and paediatrics. Being a speciality with such a solid evidence base also gives way to a dynamic range of research opportunities, and evolving advances in pharmaceutics and device technologies will further expand these opportunities.

I am often wary of the challenges that I might face as a woman pursuing a career in cardiology. However, these societal barriers are ones to be broken, so long as our passions can drive us through. I believe that with commitment to the field, the right mentors and role models, and a strong genuine interest in the field, nothing should stop us from pursuing our dream careers.

References

1. Who.int. (2019). Cardiovascular diseases. [online] Available at: https://www.who.int/health-topics/cardiovascular-diseases/#tab=tab_1 [Accessed 14 Dec. 2019].

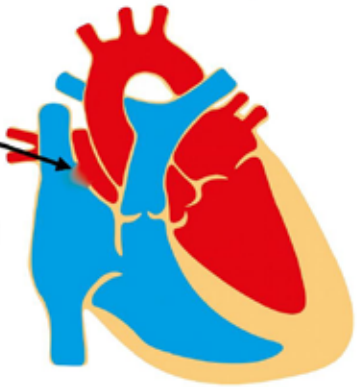
Congenital Heart Disease: *an overview*

This guide will outline some of the most common congenital heart diseases (CHDs). In **bold** are facts that commonly appear in medical school examinations. CHDs can be divided into cyanotic and acyanotic defects as described below.

Acyanotic: deoxygenated blood does not enter the systemic circulation, so there is no cyanosis on inspection.

Atrial Septal Defect (ASD)

A hole in the septum separating the left and right atria

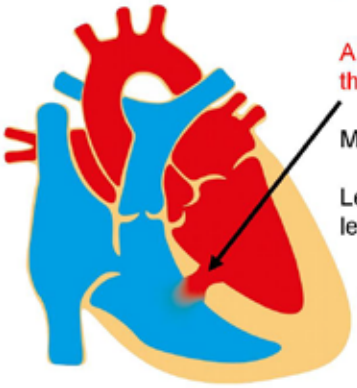


Left-to-right shunt at atrial level due to higher left-sided pressures

Hx	Children are often asymptomatic - murmur usually detected incidentally (mean age 4.5 years) May present at 20-40 years old with arrhythmias/dyspnoea
Ex	**Wide fixed-splitting of S2*** with **ejection systolic murmur** loudest at ULSB**
Ix	ECG: normal/right atrial enlargement, RVH, RAD Echo: visualise defect
Mx	Small: may close spontaneously Large: surgical repair

*Left-to-right shunt increases blood flow to right ventricle, increasing duration of RV ejection resulting in delayed pulmonary valve closure
**due to increased blood flow through the pulmonary valve resulting from the left-to-right shunt at the atrial level

Ventricular septal defect (VSD)



A hole in the septum separating the left and right ventricles

Most common (20% of CHDs)

Left-to-right shunt at ventricular level

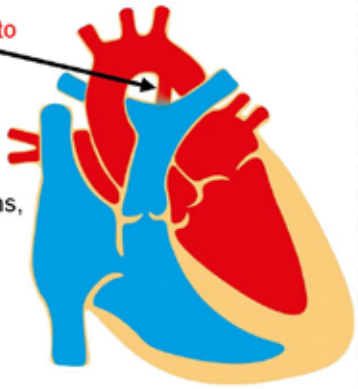
Associations: Down's syndrome

Clinical features depend on the size of the defect:

	Small	Large
Hx	No symptoms	HF symptoms (dyspnoea, difficulty feeding, failure to thrive) at 4-6 weeks
Ex	**Pan-systolic murmur** loudest at LLSB	Quiet/no murmur
Ix	ECG + CXR normal Echo: visualise defect	ECG: LVH, RVH CXR: cardiomegaly Echo: visualise defect
Mx	Typically close spontaneously by 5 years old	Diuretics Surgical repair

Patent Ductus Arteriosus (PDA)

The ductus arteriosus (DA) fails to close



Left-to-right shunt from aorta to pulmonary artery

DA is kept open by prostaglandins, most close within 24h of birth

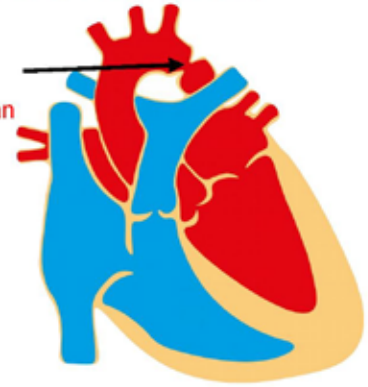
Commoner in preterm infants

Hx	Asymptomatic/HF symptoms
Ex	**Continuous* 'machinery' murmur** loudest at ULSB **Bounding pulse, wide pulse pressure, collapsing pulse****
Ix	ECG: normal/LVH Echo: visualise defect
Mx	Preterm: most close spontaneously Indomethacin (NSAID, inhibits prostaglandin synthesis) +/- trans-catheter occlusion/surgical ligation

*as aortic pressure>pulmonary artery pressure in systole *and* diastole
**due to run-off of blood from aorta into pulmonary artery in diastole

Coarctation of the aorta

A narrowing of the descending aorta usually just distal the left subclavian artery



Associations: Turner's syndrome

Hx	Severe obstruction: HF and circulatory shock following ductus arteriosus closure in first few days of life Mild obstruction: hypertension/murmur in adolescents + adults
Ex	**Ejection systolic murmur** at ULSB, often **loudest at the back between the scapulae** **Weak femoral pulses** **Radio-femoral delay**
Ix	ECG: infants – RVH*, older patients - LVH Echo: visualise defect
Mx	Diuretics for HF symptoms Balloon dilation +/- prostaglandin to maintain DA

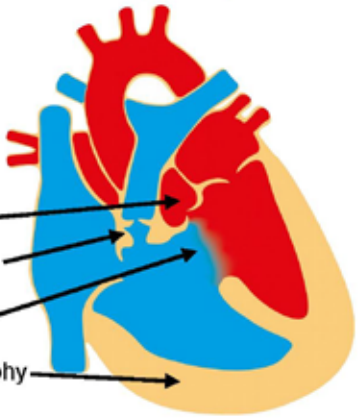
*DA is patent, therefore increased RV afterload

James Walker, King's College London

Cyanotic: deoxygenated blood enters the systemic circulation via a 'right-to-left shunt' and mixes with oxygenated blood, resulting in cyanosis. The cyanotic CHDs can be remembered as those beginning with the letter 'T'.

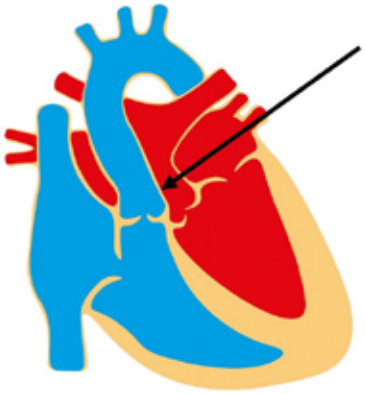
Tetralogy of Fallot (TOF)

Congenital cardiac malformation characterised by 4 features



Hx	Cyanotic spells - timing of presentation depends on degree of right ventricular outflow tract (RVOT) obstruction
Ex	**Ejection systolic murmur** (RVOT obstruction) VSD often too large to cause murmur
Ix	ECG: RVH CXR: **'boot-shaped' heart** (but may be normal) Echo: visualise defect
Mx	Blalock-Taussig shunt (branch of subclavian or carotid artery connected to pulmonary artery) initially Prostaglandin E1 to keep DA open Definitive surgical repair at around 6m

Transposition of the Great Arteries (TGA)



Aorta arises from RV and pulmonary artery arises from LV (switched sides)

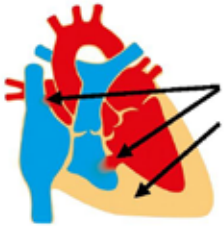
Systemic and pulmonary circulations do not mix (in parallel rather than series)

Not compatible with life without PDA/VSD/ASD enabling deoxygenated blood to reach the lungs

Hx	**Cyanotic from birth**
Ex	Usually no murmur (unless VSD present)
Ix	CXR: heart 'egg on a string/its side' sign Echo: visualise defect
Mx	Prostaglandin E1 to maintain DA patency Atrial septostomy to enable mixing Arterial switch operation in early weeks

Tricuspid valve abnormalities

Tricuspid valve atresia



The tricuspid valve is missing/abnormally developed

No direct communication between RA and RV

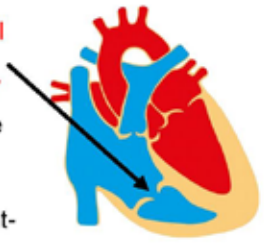
ASD and VSD usually present

Underdeveloped RV

Mx: all pts ultimately need Fontan procedure

Ebstein's anomaly

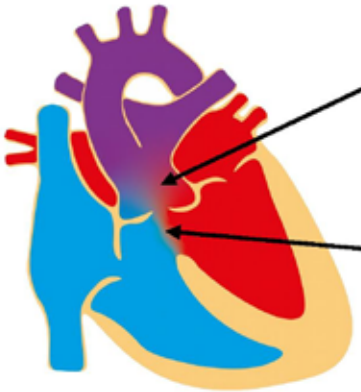
Downward displacement of the septal and posterior tricuspid valve leaflets, leading "atrialization" of the upper RV



Associated with maternal **lithium** use in early pregnancy

Most patients also have an ASD and can become cyanotic if there is a right-to-left shunt

Truncus arteriosus



Only one artery arises from the heart, which then divides into the aorta, pulmonary arteries and coronary arteries

Large VSD usually present

Associations: DiGeorge syndrome

Hx	HF symptoms, cyanosis
Ex	ESM at left sternal border
Ix	CXR: cardiomegaly, increased pulmonary markings Echo: visualise defect
Mx	Diuretics for HF Surgical repair at 2-3m

Eisenmenger syndrome: acyanotic CHDs may also result in cyanosis later in life. A left-to-right shunt (as seen in ASD, VSD, PDA) may eventually cause pulmonary hypertension. This results in increased right-sided pressures and eventual reversal of the shunt into a right-to-left shunt, resulting in cyanosis.

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- Khambadkone S. Congenital heart disease. BMJ Best Practice. March 2018. https://bestpractice.bmj.com/topics/en-gb/1308/details. Last accessed 21 July 2019.
- Sun R, Liu M, Lu L, Zheng Y, Zhang P. Congenital heart disease: causes, diagnosis, symptoms, and treatments. *Cell Biochem Biophys*. 2015;72:857– 860.

Abbreviations: CHD, congenital heart disease; Hx, history; Ex, examination; Ix, investigations; Mx, management, ASD, atrial septal defect; VSD, ventricular septal defect; PDA, patent ductus arteriosus; TOF, tetralogy of Fallot; TGA, transposition of the great arteries; ULSB, upper left sternal border; CXR, chest x-ray; Echo, echocardiography; RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy; LRAD, right axis deviation; RV, right ventricle; HF, heart failure; NSAID, non-steroidal anti-inflammatory drug; ULSB, upper-left sternal border; DA, ductus arteriosus); RVOT, right ventricular outflow tract; ESM, ejection systolic murmur

Cardiology Elective

Nadeem Ahmed

My elective in cardiology took place at the Golden Jubilee National Hospital in Clydebank, Scotland. I decided to undertake an elective in cardiology as I am particularly interested in this specialty as a career. The elective involved a wide variety of clinics, daily ward rounds and occasional theatre sessions, providing plenty of clinical exposure and patient contact. Ward rounds allowed me to see a spectrum of patients presenting with acute myocardial infarction (AMI) to patients with arrhythmias, such as Wolff Parkinson White (WPW) syndrome. It also allowed me to practise my chest x-rays (CXR) and electrocardiograms (ECGs) interpretation skills, a key investigation in cardiology. Furthermore, it was also on the ward were I first began to appreciate the different types of murmurs, something which cannot be learned from a book. The GJNH also happened to be a busy primary percutaneous coronary intervention (PCI) centre performing more that 700 primary PCIs per annum. This made my elective an insightful period into not just cardiology but interventional cardiology too. With this opportunity, I was able to spend time in the catheterisation labs watching primary and elective PCIs. This was extremely rewarding since I was able to see the immediate impact of such an intervention. Overall, I would like to thank the staff and individuals who took the time to teach me during my elective. This was an insightful and richly rewarding experience. For those wishing to carry out an elective in cardiology try to identify a supportive and enthusiastic supervisor, this makes all the difference during your stay. Secondly, try to come up with a set of objectives to meet before you start your elective. This will provide you with some direction at the beginning of your elective. Finally, an elective in an opportunity to do something that you enjoy so choose wisely.

Article Review:

Cardiology's 10 Greatest Discoveries of the 20th Century by Nirav J. Mehta, MD and Ijaz A. Khan

Abdo Kamaledeen

Spanning over 200 years this paper explores the origins and developments of the 10 most important cardiology discoveries today. Without these, cardiology as we know it today would not be possible. From Augustus Waller, the pioneer of electrocardiograms (ECGs), stating that ECGs would not be extensively used in hospitals, to Werner Forssmann performing the first human cardiac catheterisation on himself, this literature review succinctly summarises the key developments.

Mehta NJ, Khan IA. Cardiology's 10 greatest discoveries of the 20th century. Texas Heart Institute Journal 2002;29(3):164-71.

Available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC124754/

The use of an Intra Aortic Balloon Pump (IABP) During a Percutaneous Coronary Intervention (PCI)

Balrik Kailey

Patient history and examination

A 67 Caucasian gentleman with known Coronary Artery Disease (CAD) presented following typical anginal chest pain, which occurred whilst he was having lunch. It was described as constant and tight in nature, with a pain severity of eight out of ten, lasting 30 minutes, associated with breathlessness and diaphoresis. He described similar episodes of pain occurring infrequently over the preceding year, though these episodes were stable in nature, brought on by exertion and alleviated by rest. His past medical history shows a high cardiovascular risk profile including essential hypertension, type 2 diabetes and hypercholesterolemia. He had a myocardial infarction (MI) four years previously and received percutaneous coronary intervention (PCI) subsequent to this. On examination, the pain had subsided and the patient looked comfortable at rest. He had a BMI of 32.3. His pulse was 84bpm and regular. His BP was elevated at 169/98mmHg. His apex beat was laterally displaced to the mid-axillary line.

Investigations

The ECG showed sinus rhythm with no acute ischaemic changes. A Troponin assay, 12 hours after symptom onset, suggested no acute infarction. A transthoracic echocardiogram was performed which confirmed severe hypokinesis in the anterior wall with severe impairment of overall left ventricular (LV) function with an ejection fraction (LVEF) of 25%. In view of this, a coronary angiogram was performed. This showed two vessel coronary disease including a significant left anterior descending (LAD) stenosis in addition to moderate stenosis of the left

Treatment

The intention was to treat the LAD stenosis with PCI. The angiographic BCIS myocardial jeopardy score (BCIS JS) was used to calculate the severity of CAD. The BCISJS in this case was 8 out of 12 and in conjunction with severe LV dysfunction, he was deemed to be at high-risk of procedural complications during the intended PCI. To ameliorate this intraprocedural risk, additional mechanical haemo-dynamic support with an intra-aortic balloon pump (IABP) was utilised (see Info Box). The PCI was successfully performed via a femoral approach, with the contralateral femoral artery being used to deploy the IABP at the outset of the case.

The patient was discharged the following day and remains well at three months with an improvement in his anginal symptoms and an LVEF of 35-40%.

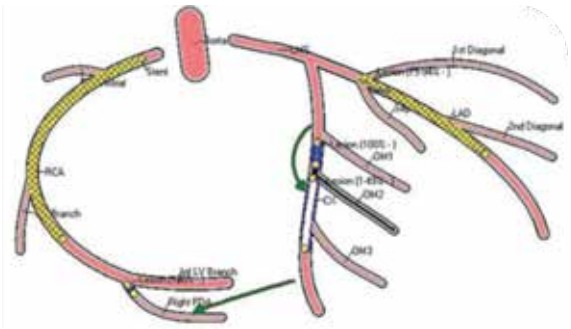
Research on IABP

Two key studies in the field both offered conflicting views. The recently presented BCIS-1 long-term follow up study suggested that those treated with adjunctive IABP therapy had improved long term survival at a median of 4.8 years, though the mechanism underlying this is unclear1. Perera et al. looked at two cohorts of patients undergoing elective PCI: one cohort underwent PCI without an IABP in situ and the other cohort had IABP assistance2. There

was a crossover arm where 12% of non IABP patients required 'bail out' IABP. Despite this there were no statistically significant differences in mortality rates.

Patient Angiogram

An angiogram report showing severe (75-94%) stenosis of the LAD as well as moderate stenosis of the circumflex artery. Green arrows signify collaterals. The stent in the RCA is from a previous PCI procedure.



When and why is IABP used?

High risk PCI in multivessel disease and impaired LV function is increasingly being performed as an alternative to coronary artery bypass surgery. PCI in those with impaired left ventricular function is associated with significant mortality and morbidity, particularly when a large proportion of the remaining viable myocardium is supplied by vessels sustaining a large burden of coronary disease. This burden may result in a deleterious downward spiral of haemo-dynamic compromise, culminating in cardiogenic shock or death.

How does an IABP work?

Intraaortic balloon counterpulsation simultaneously increases coronary blood flow, by augmentation of the diastolic aortocoronary pressure gradient, and decreases myocardial oxygen, demand by reducing the end-diastolic pressure and therefore the afterload. This makes it an attractive means of ameliorating ischaemia and consequently enhancing cardiac output.

References

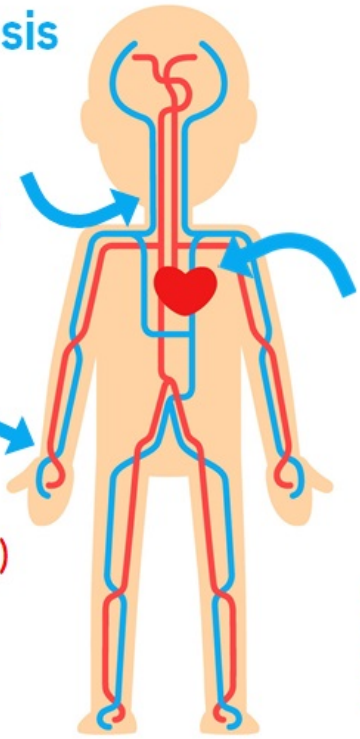
1. De Silva K, Morton G, Sicard P, Chong E, Indermuehle A, Clapp B, Thomas M, Redwood S, Perera D. *The BCIS-1 Myocardial Jeopardy Score Predicts mortality Following PCI.* Heart 2011:97
2. Perera D, Stables R, Thomas M, Booth J, Pitt M, Blackman D, de Beider A, Redwood S; BCIS-1 Investigators. *Elective intra-aortic balloon counterpulsation during high-risk percutaneous coronary intervention: a randomized controlled trial.* JAMA 2010:304(8):867-74.

Valve Pathologies At A Glance

Emma Norton

Mitral Stenosis

If pulmonary HTN:
Face: malar flush
JVP: large a waves



Opening snap
Presystolic accentuation
MDM

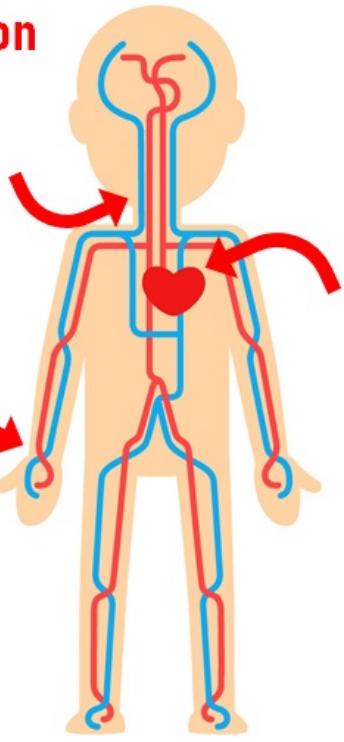
Palpation: 'tapping' apex
Auscultation: mid-diastolic murmur (MDM), loud S1

Pulse: atrial fibrillation (AF)

Aetiology: rheumatic heart disease (mostly), rarely congenital, calcification & fibrosis in the elderly

Mitral Regurgitation

If endocarditis:
Face: vasculitic lesions/Roth's spots



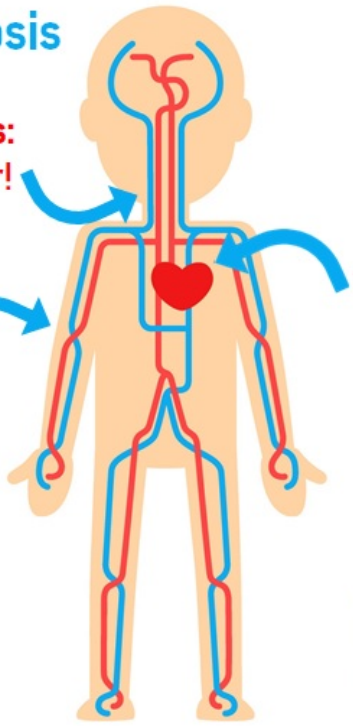
PSM

Palpation: displaced, thrusting apex
Auscultation: pan systolic murmur (PSM) radiating to the axilla, thrill

Pulse: atrial fibrillation (AF)

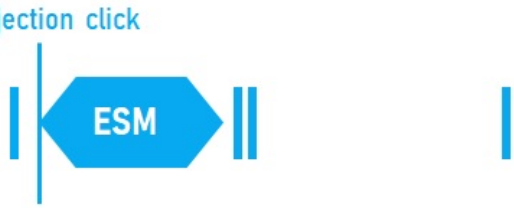
Aetiology: rheumatic heart disease, valve prolapse, post-MI, left ventricular dilation, infective endocarditis, degeneration, collagen disorders

Aortic Stenosis



Carotids: murmur!

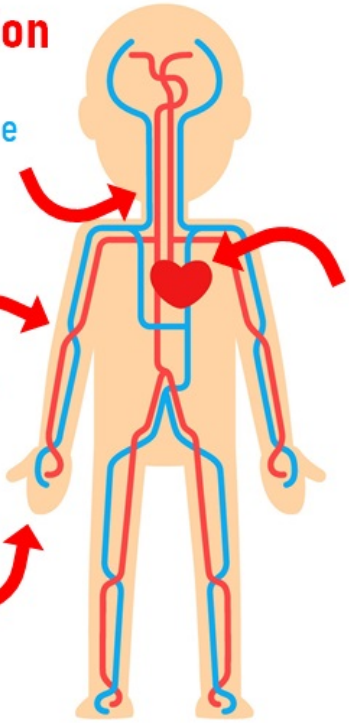
BP: narrow pulse pressure



Palpation: heaving apex, thrill
Auscultation: ejection systolic murmur (ESM), soft S2

Aetiology: rheumatic heart disease, congenital (bicuspid valve), age-related degeneration/ calcification

Aortic Regurgitation



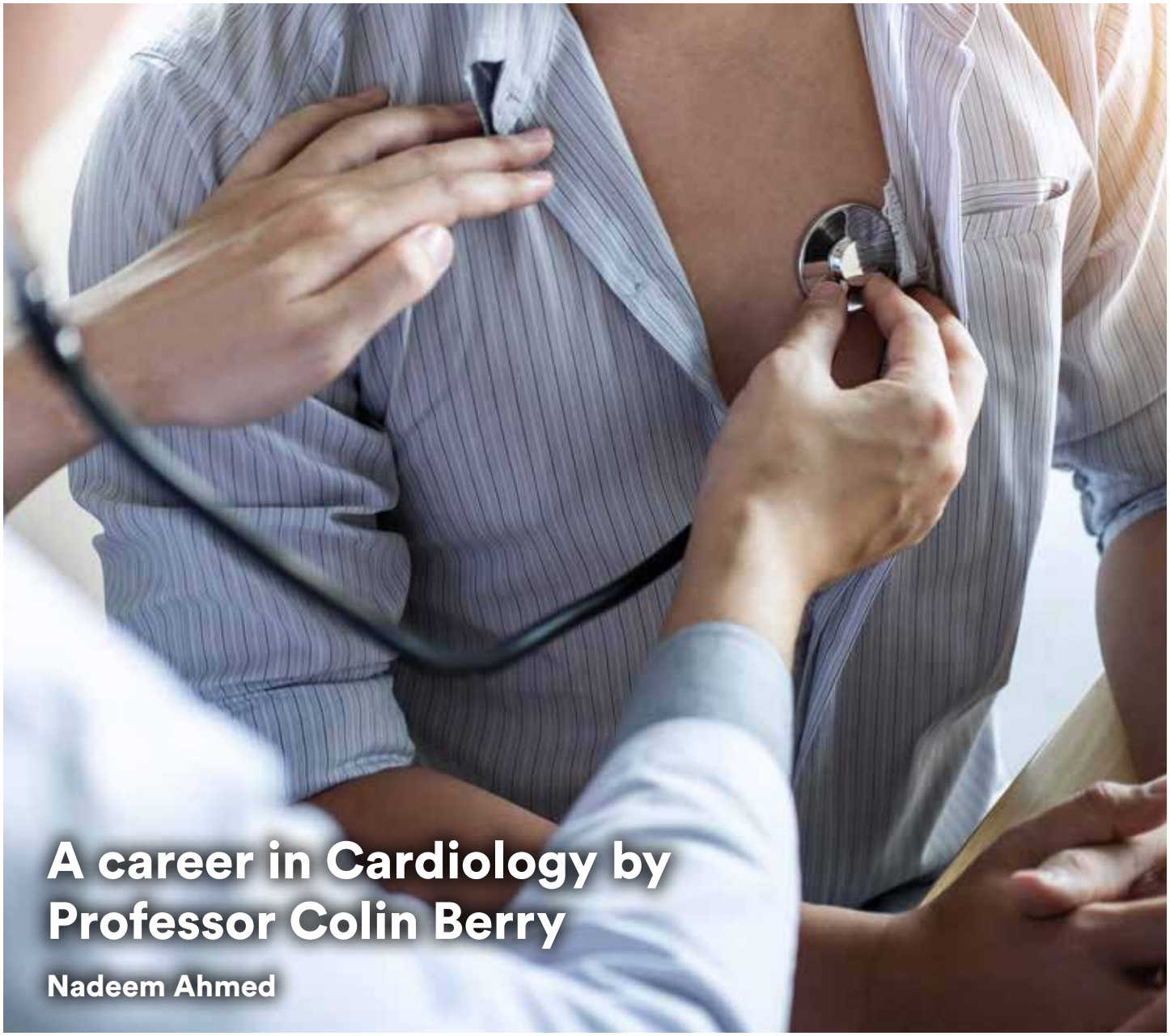
Carotids: visible pulse ('Corrigan's sign')

BP: wide pulse pressure
Pulse: 'waterhammer pulse'

Nails: Quincke's sign (visible pulsation when nail bed pressed)

Palpation: displaced, thrusting apex
Auscultation: ejection diastolic murmur (EDM)

Aetiology: rheumatic heart disease, infective endocarditis, syphilis, rheumatoid, collagen disorders, aortic dissection (and many more!)



A career in Cardiology by Professor Colin Berry

Nadeem Ahmed

The rigors and rewards of cardiology and advice on how to pursue it.

Cardiology is an exciting and rewarding specialty that requires a good grasp of clinical and practical skills as well as a firm understanding of the key pathophysiological concepts. Patients with heart disease may have acute and chronic health problems and the cardiologist has an important role in both the primary and secondary care settings. The specialty of cardiology is centred on evidence based medicine with research having pivotal importance to create new evidence and change guidelines.

Cardiology is an increasingly competitive training specialty with a limited number of ST3 training posts available across the UK. For those who are successful, there are three years of core cardiology training in echocardiography, pacing and angiography followed by a further two years of subspecialisation. The subspecialties on offer range from heart failure (including palliative care), devices, congenital, intervention, imaging and electrophysiology. Furthermore, research training represents an opportunity to focus on an area of interest, with a clinical research fellowship working towards an MD or PhD are always encouraged. My passion in cardiology led me to undertake a PhD at the University of Glasgow in Cardiovascular Science which was supported by the Medical Research Council Clinical Training fellowship. Building on this success, I then trained in cardiology first in Glasgow and then at the Montreal Heart Institute, supported by a British Heart Foundation International Fellowship and an international Exchange Award from the Royal Society of Edinburgh. Cardiology always offers opportunities and during my time in Canada, I became the first British cardiologist to take part in transcatheter aortic valve implantation (TAVI). My current research interest is coronary heart disease (CHD) and our team leads on a number of successful projects which aim to provide new knowledge and improve patient's health and wellbeing. For any budding cardiologist, try to seize any opportunities that may come your way. I would consider carrying out case reports, audits or research as well as attending accredited courses and national conferences. The more experience the better as this will put you in good stead as you try to secure a cardiology specialty training post.

A career in cardiothoracic surgery by Mr Jonathan Unsworth-White

Saleh Khwaja

I am a cardiothoracic surgeon and I usually love my job. Rigors and rewards? The scrutiny of results, published for each surgeon, is tough. A “bad run” of results can leave a surgeon feeling nervous, risk averse, and threatened. Proponents of the system point to the year on year reduction in mortality rates. However, this is at the expense of surgeons being permanently on call for their own patients to maintain close supervision on events, and may lead to the suspicion that rosy results may be due to cherry picking patients of cherry picking patients when results are rosy. Against this is the surgery itself. Cardiac and thoracic operations are technically demanding and mistakes are cruelly punished, yet the satisfaction of a challenging job well done is immense and patients are fantastically grateful. The relief they experience in regaining their quality of life and longevity is infectious.

The future for cardiothoracic surgery is bright. Prospective randomised trials continue to show a firm place for coronary bypass surgery. Valve surgery is an increasing proportion of our practice as the population ages. Innovations include thoracoscopic lobectomy and new approaches to the cardiac valves. Surgeons work closely with cardiologists and chest physicians in a multidisciplinary approach to cardio-thoracic disease. Team work is pivotal. National recruitment this year includes twelve ST1 training numbers, and a further six at ST3. ST1‘s will follow a themed core program to ST3. ST6 trainees take the part three (FRCS CTh) examination and will spend their final two years in subspecialisation. Most consultant positions in the future will be either cardiac or thoracic. Paediatric, grown up congenital (GUCH) and transplantation surgery will be restricted to a smaller number of super-specialty units.

As with all specialty training schemes, trainees that work hard, covering all the domains of research, audit, teaching, reading and, of course, operating, will do well. Training is monitored by the Specialty Advisory Committee (SAC) which sends representatives to all Annual Reviews of Competence Progression (ARCP). The curriculum is tightly mapped within the Intercollegiate Surgical Curriculum Project (ISCP) which forms the core of a trainee’s portfolio. The SAC works with the work force planning unit to match training numbers to the future demand for consultants. To this end, the SAC maintains a register of CCT holders who have yet to obtain a consultant position.

A career in cardiothoracics? Competition for training and for consultant positions is no worse than for any other surgical specialty and may even be less. Remember, most of us will spend more than 20 years in our consultant posts. It is vital to choose a specialty which is going to continue to stimulate, stretch and excite right up to retirement and cardiothoracic surgery ticks those boxes with ease!

Tickbox CVS exam

Elishba Chacko

- ☐ Wash hands (Ayliffe technique)
- ☐ Introduction, identification and consent
- ☐ General inspection (looking for cachexia, signs of distress, pallor)

Inspect the hands for cardiac signs:

- ☐ Tar staining
- ☐ Temperature
- ☐ Pallor of the palmar creases (anaemia)
- ☐ Peripheral cyanosis
- ☐ Clubbing (congenital heart disease) (figure 1: patient with no clubbing)
- ☐ Splinter haemorrhages (infective endocarditis)

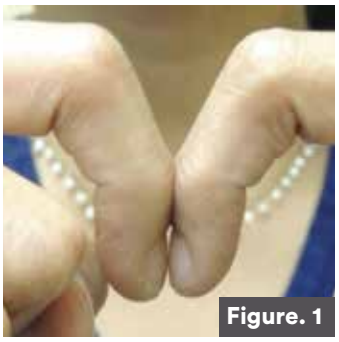


Figure. 1

- ☐ Osler's nodes and Janeway lesions (infective endocarditis)
- ☐ Tendon xanthomas (hyperlipidaemia)

Check the pulses:

- ☐ Check for capillary refill time. Should be <2 seconds.
- ☐ Check for radial pulses, assess rate and rhythm (irregularly, irregular in atrial fibrillation).
- ☐ Radio-radial delay, radio-femoral delay suggests co-arctation of the aorta.
- ☐ Assess character and volume of the brachial pulse. Check whether the patient has any pain in their arm before checking for water hammer pulse (aortic regurgitation) (Figure 2).

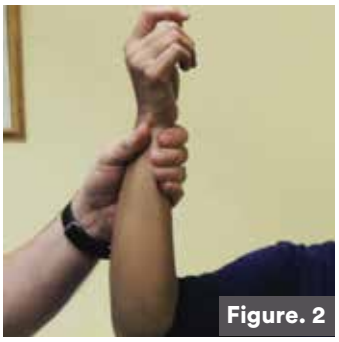


Figure. 2

Examine the neck and face:

- ☐ Assess the JVP with the patient positioned at an angle of 45 degrees (Figure 3)

  -The JVP should normally be <3cm above the sternal angle.

  - An increased JVP indicates rightventricular failure, tricuspid stenosis/regurgitation, fluid overload
- ☐ Assess the carotid pulse, palpable medial to the sternocleidomastoid.
- ☐ Assess the conjunctiva for pallor (anaemia)
- ☐ Look at the eyelids for xanthelasma (hyperlipidaemia) Check for malar flush (in mitral stenosis and pulmonary hypertension)

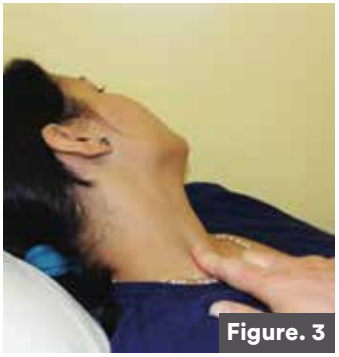


Figure. 3

- ☐ Look at the lips and tongue (central cyanosis)
- ☐ Check the mouth for dental caries, a risk factor for infective endocarditis.
- ☐ Look inside the mouth for a high arched palate (Marfan's syndrome)

Examine the thorax:

- ☐ Inspect the precordium for scars indicating recent coronary artery bypass graft or valve replacement, insertion of a pacemaker.
- ☐ Look for any abnormalities like pectus excavatum or carinatumas this can cause a displacement of the apex beat.

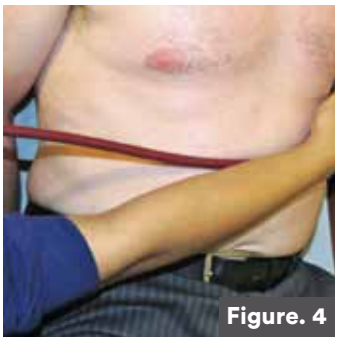


Figure. 4

- ☐ Place your hand over the apex to check for any displacement of the apex beat, heaves and thrills. Often found in volume overload due to valve failure.
- ☐ Auscultate aortic, pulmonary, mitral and tricuspid areas. Switch to the bell and with the patient rolled over to their left auscultate the apex for mitral stenosis. (Figure 4)
- ☐ Switching back to the diaphragm, sit the patient forward and auscultate the 4th - 5th intercostal space on held expiration for aortic regurgitation.
- ☐ Listen with your bell for any carotid bruits. Heard best with the patient holding their breath in inspiration. Typically, aortic stenosis radiates to the carotids.
- ☐ Auscultate the lung bases for any presence of inspiratory crackles or pleural effusion

Examine the peripheries

- ☐ Check for sacral oedema which occurs in heart failure
- ☐ Check for pitting oedema in both ankles, congestive cardiac failure. (Figure 5)
- ☐ Mention that you will carry out lying and standing blood pressure and ophthalmoscopy for hypertensive retinopathy.


Figure. 5

- ☐ Thank the patient and wash your hands.