UNIVERSITY of NICOSIA | MEDICAL SCHOOL

MED-501
Cardiology, Cardiothoracic and Vascular Surgery Course Handbook

2024-2025

University of Nicosia Medical School Academic and Administrative Contacts:

Chair of Clinical Education:
Professor Joseph Joseph
E-mail: joseph.j@unic.ac.cy

Year 5 Lead:
Dr Aris Tourvas
E-mail: tourvas.a@unic.ac.cy

Cardiology Curriculum Lead:
Professor Joseph Joseph
E-mail: joseph.j@unic.ac.cy

Surgery Curriculum Lead:
Professor Panos Economou
E-mail: economou.p@unic.ac.cy

Course Lead of MED-501 Cardiology, Cardiothoracic and Vascular Surgery:
Dr Constantinos Kyriakou
E-mail: cnkyriakou@cytanet.com.cy

Senior Clinical Placements Administrator:
Ms Katerina Tsiamezi
E-mail: tsiamezi.k@unic.ac.cy

Limassol General Hospital Academic and Administrative Contacts:

Academic Lead at Limassol General Hospital:
Dr Christos Nicolaou
E-mail: c.nikolaou@shso.org.cy

Clinical Lead at Limassol General Hospital:
Dr Andreas Tryfonos
E-mail: a.tryfonos@shso.org.cy

Deputy Clinical Lead at Limassol General Hospital:
Dr Andreas Andreou
Email: a.y.andreou@shso.org.cy

Administrative Contact:
Ms Helen Sophroniou - LGH Senior Site Administrator
E-mail: sophroniou.h@unic.ac.cy

Ms Aristea Bati - LGH Site Administrator
E-mail: bati.a@unic.ac.cy

Paphos General Hospital Academic and Administrative Contacts:

Academic Lead at Paphos General Hospital:
Prof Joseph Moutiris
E-mail: moutiris.j@unic.ac.cy

Clinical Lead at Paphos General Hospital:
Prof Joseph Moutiris
E-mail: moutiris.j@unic.ac.cy

Administrative Contact:
Ms Stella Naziri - PGH Senior Site Administrator
E-mail: naziri.s@unic.ac.cy

Klinik Hirslanden Academic and Administrative Contacts:

Academic Leads at Klinik Hirslanden:
Prof. Dr. med. Stefan Russmann
E-mail: stefan.russmann@hirslanden.ch

KD Dr. med. Tanja Volm
E-mail: t.volm@himed.ch

Clinical Co-Leads at Klinik Hirslanden:
Prof. Dr. med. Diana Reser (HerzKlinik)
E-mail: diana.reser@hirslanden.ch

Prof. Dr. med. Maurizio Taramasso (HerzZentrum)
E-mail: taramasso@herzzentrum.ch

Prof. Dr. med. Lyubov Chaykovska (Vascular Surgery)
E-mail: lyubov.chaykovska@hirslanden.ch

Administrative Contact:
Ms Barbara Etzensberger
E-mail: barbara.etzensberger@hirslanden.ch

Ms Michelle van Haaften
E-mail: michelle.vanhaaften@hirslanden.ch

Lewisham & Greenwich NHS Trust Academic and Administrative Contacts:

Academic Lead at Lewisham & Greenwich NHS Trust:
Dr George Dervenoulas
E-mail: georgios.dervenoulas@nhs.net

Clinical Lead at Lewisham & Greenwich NHS Trust:
Name: Shoaib Hamid
E-mail: shoaibhamid@nhs.net

Site Administrator: Nikola Hewitt
E-mail: nikola.hewitt@nhs.net

Site Administrator: Carly Raven
E-mail: carly.raven@nhs.net

Site Administrator: Suzanne Faulkner
E-mail: suzanne.faulkner@nhs.net

Hellenic Healthcare Group Academic and Administrative Contacts:

Academic Lead at Hellenic Healthcare Group:
Prof Panagiotis Skandalakis
E-mail: skantakis@yahoo.gr

Deputy Academic Leads at Hellenic Healthcare Group:
Prof Dimitrios Karatzas
E-mail: dimitriskaratzas@hotmail.com

Prof Stergios Douvetzemis
E-mail: stergiosdouvetzemis@yahoo.gr

Clinical Lead at Hellenic Healthcare Group:
Prof Dimitrios Karatzas
E-mail: dimitriskaratzas@hotmail.com

Administrative Contact:
Ms Vickie Mastrogianni
E-mail: vicmast@yahoo.gr

Ms Angeliki Petrou
E-mail: agpetrou@mitera.gr

Swedish Hospital Academic and Administrative Contacts:

Academic Lead: Dr Iryna Aleksandrova
Email: ialeksandrova@schosp.org

Clinical Lead at Swedish Hospital:
Dr Frederman Concepcion
Email: FConcepcion@northshore.org

Administrative Contact:
Ms Jeanette Maatouk
E-mail: JMaatouk@schosp.org

Course Handbook introduction by the Chair of Clinical Education

Dear Student,

Your course handbook is an invaluable resource. Reading it before starting your placement will make a huge difference to your learning experience. You will find outlines of the course, the objectives of your learning, the assessment requirements, learning resource suggestions, contact numbers and reporting structures.

You can use the learning outcomes and objective list to guide you through your learning of the subject and even to discuss with your tutors. The requirements, whether these are workplace - based assessments or tutorials, will allow smooth running of your clinical block and avoid unnecessary last-minute hardship.

The medical school has a course lead in place, with knowledge of the specific block, including its learning, revision, and assessment. And the department of clinical education ensures smooth running and delivery of the clinical training, through a number of Curriculum Leads (one for each main specialty) who work with the course leads and the local teams at the placement, to ensure the curriculum is delivered as planned.

The nature of clinical learning is such that, whilst your experiences will be comparable, it is not possible to have identical clinical exposure across all students; a case of disease X may be available today, whereas when the next student comes along it may be disease Y. It is desirable that we see all cases as far as possible but your handbook with its checklist of conditions and objectives will allow you to not fall behind and fill any gaps that may appear during your clinical exposure, and help guide your preparation for assessments.

If there are academic or administrative problems, if you need help and if you need to report anything back to your medical school, the reporting structures ensure that you should not feel even for one minute that you do not have someone to go to when needed. Please contact the team if you need any help, we are all here with our contact details available if you need us.

Please ensure that you are present and available for learning, opportunities will come when you least expect them, the amount we can learn from patients is huge and endless and therefore sitting with, talking to and examining patients is one of your strongest tools.

I hope you gain a lot of knowledge and clinical experience.

Enjoy the next few weeks!

Professor Joseph Joseph
Chair of Clinical Education

Table of Contents

1. Welcome

The **MED-501 Cardiology, Cardiothoracic and Vascular Surgery course/ clinical rotation** offers students the opportunity to develop an understanding of the presentation, signs and symptoms, physical examination findings, investigations, diagnosis, treatment (medical and/or surgical as appropriate) and management plan for common cardiovascular diseases.

Every effort has been made to provide a structured and well organised learning experience that will ensure adequate exposure to the diagnostic and therapeutic approaches that take place in a large Academic Hospital.

On behalf of all the clinicians, nurses and administrative staff of the Cardiology, Cardiothoracic and Vascular Surgery departments, I would like to warmly welcome you as an integral part of our team and wish you an effective and pleasant learning experience. Our education team stands ready to assist you with any questions you may have.

Kind Regards,

Dr Constantinos Kyriakou
Course Lead for MED-501 Cardiology, Cardiothoracic and Vascular Surgery

2. MED-501 Cardiology, Cardiothoracic and Vascular Surgery course/ Clinical rotation Course Outline

Course Title	**Cardiology, Cardiothoracic and Vascular Surgery**						
Course Code	**MED-501**						
Course Type	Required						
Level	Undergraduate						
Year / Semester	Year 5/ Semester 9 (Fall)						
Teacher's Name	**Course Lead:** Dr Constantinos Kyriakou						
ECTS	6	Lectures / week	4	Laboratories / week	0	Clinical Practice	36
Course Purpose and Objectives	The main objectives of the last two years of the six year medical course are to provide students with extensive experience in the clinical environment, mainly in hospitals but also in the community, so that they can utilise their learning over the previous 4 years to practise their clinical, communication, diagnostic and reasoning skills on real patients, and to learn about the management of patients, from a medical, therapeutic, surgical, psychosocial and caring perspective. In this course, students will spend four weeks working primarily with patients with heart conditions. They will develop an understanding of the presentation, signs and symptoms, physical examination findings, investigations, diagnosis, treatment (medical and/or surgical as appropriate) and management plan for common cardiovascular diseases. The students will learn how to take detailed histories from, carry out systematic clinical examination of, and interpret laboratory and imaging data on patients with disorders of the cardiovascular system. They may also spend time in theatre observing cardiothoracic / vascular surgery.						

Learning Outcomes	After the completion of the course the students should be able to: 1. Take a history from a patient, or relative of a patient, presenting with a cardiovascular condition, in a sensitive and caring manner 2. Carry out a physical examination of patients so presenting 3. Come up with a differential diagnosis for the presenting complaint 4. Identify appropriate investigations, including blood and urine tests and imaging, to assist in the diagnosis of the presenting complaint and to interpret the results from such tests 5. Prepare a treatment management plan for the patient to present to the responsible clinician to include medical, pharmacological, surgical options as appropriate 6. Observe, and where appropriate carry out or assist with, the following procedures: ECG, cardiac stress testing, catheterization, angiography, echocardiographs, MRI, CT & PET scans, angioplasty, coronary artery bypass, stent insertion, pacemaker insertion and other surgical procedures, planned and opportunistic.

Prerequisites	None	Required	None
Course Content	• Hypertension • Angina • Acute coronary syndrome/ Myocardial infarction • Arrhythmias and conduction defects (atrial fibrillation/flutter, complete heart block, ventricular tachycardia) • Cardiac Failure • Rheumatic fever and rheumatic heart disease • Cardiac valve defects • Infective endocarditis • Pericardial disease • Myocarditis • Cardiomyopathy • Atrial and ventricular septal defects • Patent ductus arteriosus • Fallot's tetralogy • Coarctation of the aorta • Acute circulatory failure / shock • Cardiorespiratory arrest • Acute & chronic limb ischaemia • Limb ulceration and gangrene • Arterial aneurysms • Vascular disease in other sites (renal, mesenteric, carotid) • Varicose Veins		

<table>
<tr><td>Teaching Methodology</td><td>The course is delivered by clinical placements, lectures, tutorials, case studies and group discussions.</td></tr>
<tr><td>Bibliography</td><td>Recommended Textbooks/Reading:
<table>
<tr><th>Authors</th><th>Title</th><th>Publisher</th><th>Year</th><th>ISBN / E-book</th></tr>
<tr><td>Punit Ramrakha and Jonathan Hill</td><td>Oxford Handbook of Cardiology, 2nd ed.</td><td>OUP Oxford</td><td>2012</td><td>978-0199643219
E-book</td></tr>
<tr><td>Brian P Griffin</td><td>Manual of Cardiovascular Medicine, 5th ed.</td><td>Lippincott Williams and Wilkins</td><td>2018</td><td>978-1496312600
E-book</td></tr>
<tr><td>Neil Herring, David J. Paterson.</td><td>Levick's introduction to cardiovascular physiology, 6th ed.</td><td>CRC Press</td><td>2018</td><td>978-1498739849</td></tr>
</table>
E-book resources for Clinical Placements Support Resources:
https://libguides.unic.ac.cy/placementresources</td></tr>
<tr><td>Assessment</td><td>Final year exam (EYEs) and final year OSCE.</td></tr>
<tr><td>Language</td><td>English</td></tr>
</table>

3. MED-501 Cardiology, Cardiothoracic and Vascular Surgery Course/ Clinical Rotation Requirements

Please take note of the following assessments that students will need to complete and submit via **'MyProgress Health'**, **no later than one week** after the completion of the MED-501 Cardiology, Cardiothoracic and Vascular Surgery course/ clinical rotation. You must also cross-reference the above with the Year 5 PVB assessment domain handbook, which takes precedence to any other handbook.

Required PVB Assessments

1 x Clinical Placement and Professionalism Certificate (CPPC)
2 x Mini Clinical Education Exercise (Mini-CEX) - 2 Cardio **or** 1 Cardio + 1 Vascular
2 x Case Based Discussion (CBD) - 2 Cardio **or** 1 Cardio + 1 Vascular
3 x ECSA (1 Drug initiation +1 Wells' score calculation + 1 Interpreting an ECG)
Daily Attendance Logbook (submitted via 'MyProgress Health' by uploading the attendance weekly sheets)
1 x Learning Outcomes Record (LOR)
Evidence of completing online student feedback survey

Floating WPBA and On-line Feedback Survey

Submit the above forms together with any floating WPBAs completed during the MED-501 Cardiology, Cardiothoracic and Vascular Surgery course/clinical rotation. You also need to ensure that you have completed the online feedback survey and upload evidence of this via '**MyProgress Health'**, by the deadline.

Logbook

The logbook should be completed on a daily basis and submitted at the end of your course/ clinical rotation by uploaded on **'MyProgress Health'.**

Attachment sign-off process

PLEASE NOTE: It is your responsibility to ensure that your tutor completes your Clinical Practice Portfolio Certificate (CPPC) as well as WPBA forms for you.

You should follow these steps:

- You should contact your Clinical Lead in advance to agree a mutually convenient date to conduct the completion and sign-off of the **CLINICAL PLACEMENT & PROFESSIONALISM CERTIFICATE (CPPC)** using the final week of your attachment. It may take around 15-20 minutes so please bear that in mind when arranging a suitable time slot. You should check in advance if the Clinical Lead will be absent at any time towards the end of the attachment.

- Your CPPC must be completed by the relevant tutor/lead clinician no later than one week after the completion of the attachment (recommended to be completed by the last day of the clinical attachment). Your tutor will be able to access all the WPBAs you have completed on your device during the attachment, as well as your logbooks and other evidence of your attendance.
- If you have not been told who will be signing you off you should contact your local administrator for guidance.
- Attend the meeting with your clinical tutor and bring your device to present all required WPBA forms and logbooks, as well as any other evidence of your attendance.
- It is your responsibility to ensure that the CPPC certificate and all WPBA forms are properly completed prior to their submission. In addition, you are required to use your logbooks to record activities and attendance and ask for a signature from your supervisor prior to submitting these.

DON'T FORGET:

☑ You should use the diaries to record activities and attendance and ask for a signature from supervising Consultant or Registrar each week.

☑ You need to achieve submission of all assessment forms and attendance logbook/diaries via **'MyProgress Health'** within one week of completing an attachment. If you are using the *'Email for later'* function it is your responsibility to chase up the clinician and have your WPBAs completed and submitted by the deadline. **If you anticipate any delays please always inform the PVB administrator. No communication and late submissions are considered serious and recorded under Professional Behaviour of the PVB domain.**

☑ You must check that all section of WPBA forms have been completed appropriately and fully before you submit. Otherwise, your forms will not be accepted by the system.

☑ If you are marked with a 'Fail' on the CPPC form for any of the elements, the assessor must give specific detailed feedback explaining reasons for the grade. Feedback should be focused and specific. In such cases or in cases where you fail to complete the required number of WPBAs, you will normally be required to redo part or the whole of the course/clinical rotation (after discussion with your local PVB Domain Lead). **Once remediation is complete a new CPPC form needs to be completed and signed off by the Clinical Lead or the remediation supervisor or the remediation supervisor along with a new Logbook that shows remediation dates.**

☑ Check that your course/clinical rotation specific and floating WPBAs have been supervised and signed off by the correct assessor level. If you are unsure, please check before you undertake the WPBA to avoid your forms being rejected and having to return to your placement to repeat the WPBA with the correct assessor.

☑ Ensure you have completed the online feedback survey and upload evidence of this via '**MyProgress Health'**, by the submission deadline.

Note that late submissions will be recorded under PVB Professional Behaviour Element.
Your clinical Site Administrator is here to help!
If you are unclear or unsure about any aspect of the PVB Domain ask your Clinical Site Administrator.

In case of illness or absence

Students must notify their Clinical Site Administrators via E-mail, and their Clinical Tutor prior or on the day of absence.

4. MED-501 Cardiology, Cardiothoracic and Vascular Surgery course/ Clinical rotation material on Moodle

1. Heart Failure
2. Acute Coronary Syndromes
3. Arrhythmias
4. Deep Venous thrombosis and limb ischaemia
5. Assessment of a patient with Chest Pain / Dyspnea
6. Chest X Rays and Echo Basics
7. Basic Clinical CV Examination
8. Electrocardiography in Acute Coronary Syndromes
9. Pacemaker and Defibrillator Basics
10. Valvular Heart Disease

5. Cardiovascular History Taking

Presenting complaints

Chest pain

S site
O onset
C character
R radiation
A alleviating factors
T tempo
E exacerbating factors
S symptoms associated

Remember angina can present as heavy/stabbing/burning/aching chest pain, shoulder pain, epigastric pain, isolated neck/jaw/arm pain, or without pain.

Canadian Cardiovascular Society functional classification angina
Class I No angina with normal activity (strenuous exertion)
Class II Slight limitation of normal activity (walking up-stairs briskly)
Class III Marked limitation of normal activity (walking up-stairs)
Class IV Angina on minimal exertion or at rest

SOB

- Precipitants: exertion, lying flat
- Relieving factors: rest, GTN, inhalers (to differentiate from respiratory cause)
- Associated symptoms

New York Heart Association functional classification heart failure
Class I No SOB with normal activity
Class II Slight limitation with normal activity
Class III Marked limitation of normal activity
Class IV SOB with minimal exertion or at rest

Palpitations

- Onset: gradual or sudden
- Rhythm: fast, slow, regular or irregular (patient can tap out rhythm)
- Precipitants: exertion, alcohol
- Associated symptoms: particularly SOB, dizziness or collapse
- Relieving factors (many patients learn vagal manoeuvres to control a SVT)

Dizziness / syncope

- Differentiate from vertigo
- Onset: exertional, postural
- Associated symptoms: palpitations, SOB
- Recovery time and state (may help differentiate from neurological cause)

Past Medical History

'Angina'
- Who made the diagnosis: GP, hospital doctor, cardiologist
- Regular clinic follow-up: with whom, where, next appointment, planned interventions
- Usual pattern of symptoms: how does this presentation differ

(This to be used for every diagnosis the patient says they have been given.)

Myocardial infarction
To clarify past history it is essential to ask about admission, length of inpatient stay, intervention, investigation and planned follow-up.

Cardiac surgery
Details are important when planning further investigation and intervention, so ask when, where and who operated. Also ask about angiograms or angioplasty.

IHD risk factors
- Diabetes mellitus
- Smoking history
- Family history (1st degree relative age <60yrs is significant)
- Hypertension/previous IHD
- Cholesterol
- Obesity

Valve lesions
- Past history of rheumatic fever
- If suspect endocarditis enquire about risk factors (i.e. recent dental or invasive procedure, IVDU)

Medication
- Current medication

Not every patient brings a list, but do not accept 'I can't remember Doc' and run through some possible names. Think of all the drugs a patient ideally should be on (i.e. post MI: ACE inhibitor, statin, aspirin, and beta-blocker)
- OTC and herbal remedies
- Allergies: what was the reaction?

Family History
Ask about premature coronary disease
Enquire if any unexplained sudden death

Social History
- Alcohol
- Illicit drug use: cocaine can cause coronary artery spasm
- Work: MI and arrhythmias will affect driving licences (taxi, bus, HGV)
- Housing and support: cardiac symptoms can severely limit mobility

Review of Symptoms

Respiratory	SOB can be respiratory in origin. What limits patients' mobility more: their COAD or angina.
Gastro-intestinal	Heartburn symptoms may be confused with angina. GORD, peptic ulcer disease and GI bleeds are important to identify in the context of the anticoagulation a patient receives (aspirin, clopidogrel, heparin, thrombolysis.)
Musculo-skeletal	Arthritic patients make take NSAIDs, which worsen fluid retention and cause gastric irritation. What limits patients' mobility more: their arthritis or angina.
Neurology	TIA /CVA secondary to embolism or carotid atherosclerosis. History of haemorrhagic stroke is important in planning anticoagulation.
Psychiatric	Anxiety, fear of loss of independence and depression can occur post-MI.

6. Cardiovascular Examination

As with all examinations:

- Introduce yourself.
- Explain what you will do.
- Gain consent.
- Position patient – maximal exposure ideally, 45º
- One step back- general inspection:
  Comfortable/ distressed, cyanosed, scars, mechanical sounds, de Musset's sign, oxygen, GTN, cardiac monitor, Marfan's
  (Scars:- *median sternotomy, lateral thoracotomy, mid-CAB, vein + artery harvest*)

Next to the hands:

- Ask about pain before touching patient
- Look at **both** hands together
- Clubbing:
  - *proliferation + increased vascularity of nail bed*
  - *Loss of angle, boggy & fluctuant, increased curvature in all directions, expansion of end of digit (drumstick)*
  - **Cardiac causes:** **Subacute** (not acute) endocarditis (SBE)
    cyanotic heart disease (e.g. Fallot's)
    atrial myxoma (polypoid, gelatinous, pedunculated)
- Tar staining (Rothman's sign)
- Peripheral cyanosis- due to central cyanosis or peripheral vasoconstriction
- Splinter haemorrhages: due to trauma/ SBE.
- Janeway lesions: erythematous non-tender blanching macules, mainly on thenar & hypothenar eminences. Due to SBE.
- Osler's nodes: hard, painful papules (subcut swellings) in fingers, toes, palms, soles. Due to SBE.
  (N.B. macule = change in colour; papule = change in contour)

The arms:

- Radial pulses: *Do both together (radio-radial delay + equality)*
  Rate (count for 15s)
  Rhythm (regular, regularly irregular, irregularly irregular)
  Vessel wall
- Waterhammer pulse: *Named after Victorian toy*
  Feel brachial pulse with 4 fingers with arm hanging down
  Ask about pain in shoulder + lift arm up
  When above level of heart, pulse felt travelling up and down fingers
  Due to AR, PDA or high cardiac oputput states

- Tendon xanthomas (hyperlipidaemia)

- Say you would do the BP *at this point*.
  Remember: *pulse pressure = systolic bp – diastolic bp*
  Narrow pulse pressure in AS
  Wide pulse pressure in AR, PDA

The face:

- Eyes: Arcus senilis- sign of hyperlipidaemia if under age 60
  Anaemia seen in the palpebral conjunctiva
  Xanthelasma- yellowish plaques around the eyes (hyperlipidaemia)

- Malar flush: *most commonly linked with mitral stenosis*
  Does not bridge nose (cf butterfly rash)

- Mouth: Poor dentition- source of endocarditis
  Central cyanosis: *"Stick your tongue up to the roof"*
  5 g/dl deoxy-Hb
  Circulatory/ resp pathology
  More likely in polycythaemia (more Hb)
  N.B. lips are peripheral, not central

The neck:

- Jugular venous pressure (JVP):
  "Look away from me". Make sure neck relaxed.
  Use **internal** jugular vein (no valves, passes through less planes)
  Measured as **vertical** distance from manubriosternal joint (angle of Louis)
  <3cm H_2O normally
  Differentiate from carotid pulse: Double pulse
  Fills from above
  Obliterated with pressure
  Hepatojugular **reflux**
  Hepatojugular reflux- abdominal pressure (ask about pain)
  Temporary rise in venous return
  Causes JVP to temporarily rise
  Use to exclude v. high JVP

  "a" wave (**atrial** systole), "c" wave (RV contraction before tricuspid valve **closes**) and "v" wave (atrial filling by **venous** return against closed tricuspid valve)

  Large a waves- resistance to ventricular filling
  Pulmonary hypertension, pulmonary stenosis
  Rare in tricuspid stenosis (as usually AF)

Cannon waves- atria/ ventricles contracting together
Complete heart block, VT

Giant v waves-vent contraction not stopped by tricuspid valve
Tricuspid regurgitation

Raised JVP- fluid overload or **right** heart failure (not LVF)

- Carotid pulse- **Never palpate both together**
  Palpate with thumb (rest of hand on chest)
  Feel for **character** mainly (& volume):

  Collapsing: wide pulse pressure
  AR, PDA & high output states
  May be *visible* as pulsation of the neck
  (Corrigan's sign) or head nodding
  (de Musset's sign)

  Slow-rising: Also known as "plateau", "anacrotic"
  Aortic stenosis

  Pulsus alternans: Regular, but alternate weak then strong beats
  Due to poor cardiac function

  Pulsus bigeminus: Irregular. Two beats then a pause, repeated.
  Due to ectopic after every normal beat

  Pulsus bisferiens: Double- headed pulse
  "Feels how a JVP looks"
  HOCM or mixed aortic valve disease

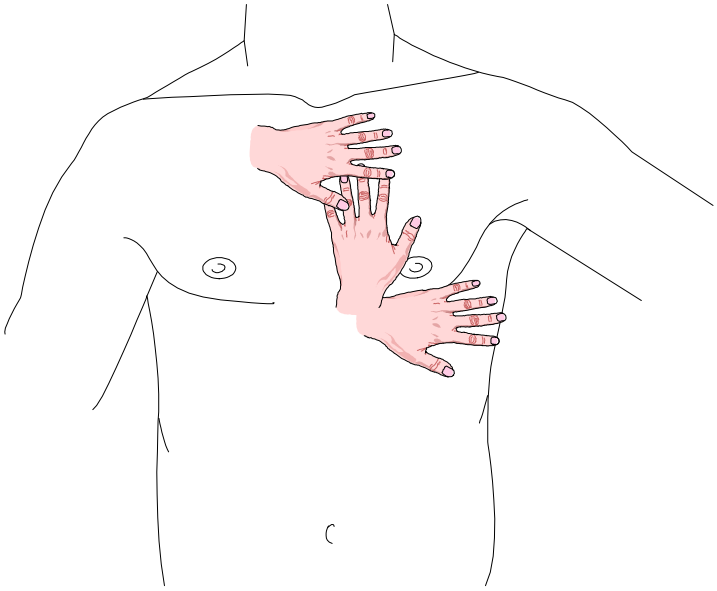
Finally onto the praecordium:

- Inspect initially, especially for: Lateral thoracotomy
  Visible heaves
  Audible prosthetic valves

- Palpate **apex beat** (most inferior & lateral point at which the heart beat may be felt)
  Start laterally and move medially
  When located, note location (inferiorly from manubriosternal joint (2nd intercostal space), and in which vertical plane e.g. mid-clavicular, anterior axillary, posterior axillary, mid-axillary).
  Normal site is 5th intercostal space, mid-clavicular line (5ICS MCL)

- Feel for heaves and thrills (Z manouvre)
  Heave- hand lifted off chest wall by strenuous ventricular activity.

Thrill- palpable component of a murmur. A vibrating sensation.



The Z manoeuvre

- No percussion in CVS exam

- **Auscultation:** the aim is to move the patient and change between bell and diaphragm *as little* as possible.

  Bell- for low pitched sounds (mitral stenosis, 3rd and 4th heart sounds) and auscultating on irregular surfaces (e.g. neck)

  Diaphragm- for high pitched sounds (pretty much everything else)

  Normal sounds- First heart sound (lub) then second (dub)
  (Ventricular) systole is between heart sounds 1 & 2
  The carotid pulse is felt between heart sounds 1 & 2

  The four areas- Mitral area @ apex
  Tricuspid @ lower left sternal edge (LLSE)
  Pulmonary @ 2nd intercostal space left sternal edge
  Aortic @ 2nd intercostal space right sternal edge

  Respiration- **In inspiration**, you suck air + blood into chest (negative pressure)
  More blood returns to R heart. Therefore, more flow/ turbulence
  Therefore R-sided murmurs louder in inspiration

  In expiration, positive pressure in chest
  Pulmonary vessels squeezed- more blood pushed into left heart
  Therefore, in expiration, L-sided murmurs louder

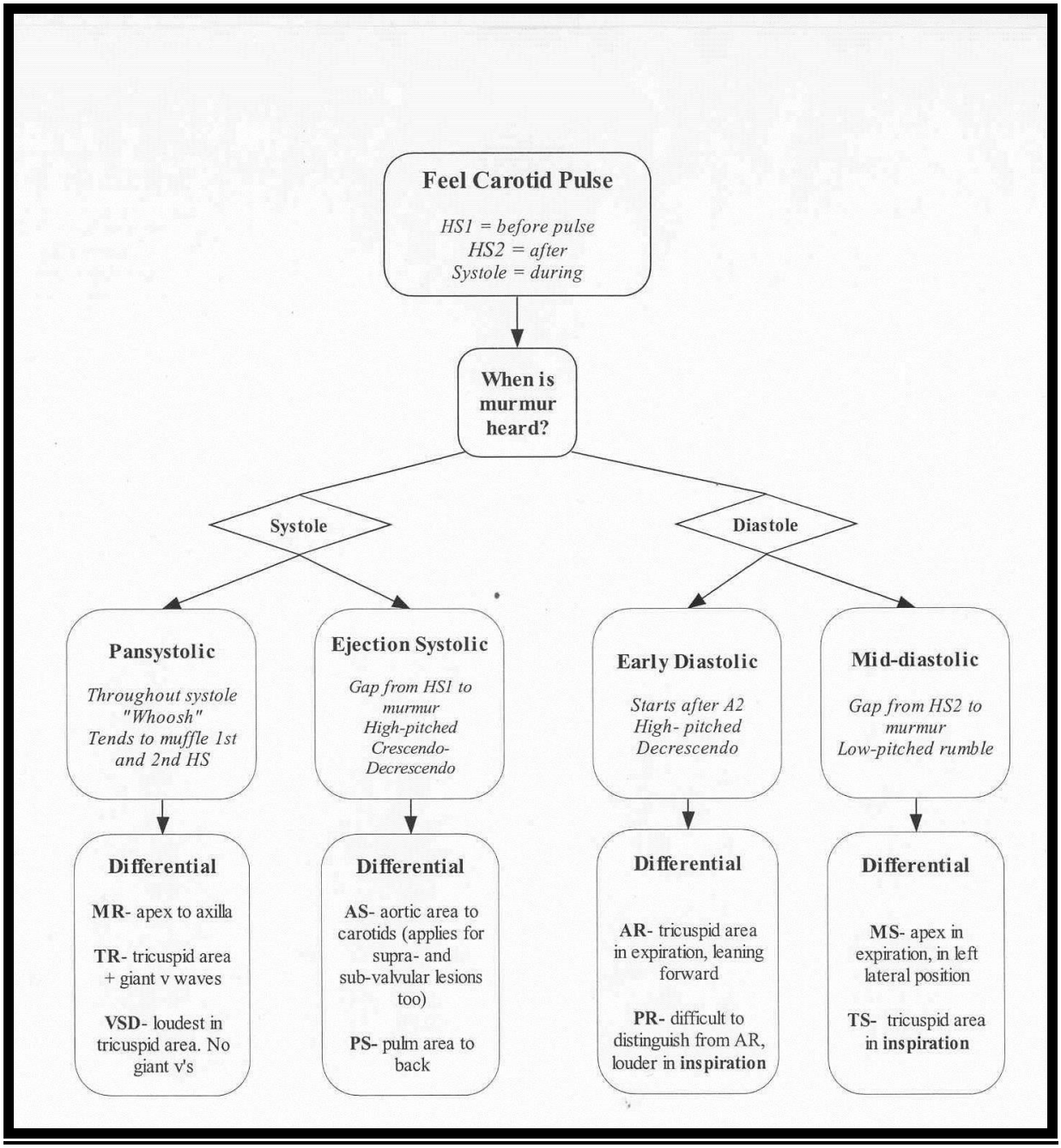
Order- Feel carotid pulse while listening (tells you when systole is)
Bell @ apex. "*Deep breath in.. and out.. and stop breathing"*. For mitral stenosis & extra heart sounds.
Turn patient to left lateral position. Palpate apex and listen over with bell.
Diaphragm @ apex. Listen in expiration for mitral regurgitation
Diaphragm in axilla. For radiation of MR
Diaphragm @ tricuspid area. In inspiration for TS/TR
Diaphragm @ pulmonary area. In inspiration for PS/PR
Diaphragm @ aortic area. In expiration for AS (note not AR)
Bell @ both carotids. "*Deep breath in.. and out.. and stop breathing".* For radiation of AS or carotid bruits.
Sit patient forward
Diaphragm @ tricuspid area. "*Deep breath in.. and out.. and stop breathing".* For aortic regurgitation (note, we are listening in the tricuspid area)
Diaphragm @ lung bases. For pulmonary oedema (LVF, fluid overload)
Feel sacrum for oedema
Lie patient back
Feel for ankle oedema

To Finish:

- Thank the patient
- Say you would also like to do the following:
  - Examine the peripheral pulses
  - Feel for hepatomegaly (RVF and TR) and splenomegaly (SBE)
  - Dip the urine for blood and look at a temperature chart (SBE)
  - Perform fundoscopoy (Roth spots in SBE, hypertensive and diabetic changes)

- Present your patient

7. Murmur Interpretation



8. Heart Sounds and Valve Lesions

Normal Sounds

- Normal valves do not make opening sounds
- Heart sounds 1 + 2 are due to **closure** of valves.
    - 1 = mitral + tricuspid valves
    - 2 = aortic + pulmonary valves

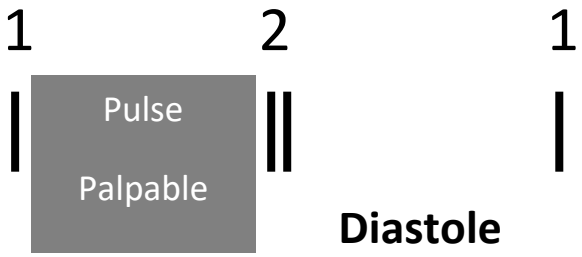
- Heart sound 1 is not split.
- Heart sound 2 is split in inspiration- A_2 occurs before P_2 (see later)

Systole v diastole

- Systole = contraction
- Diastole = relaxation

Note: these terms usually refer to ventricular **systole / diastole. If referring to atrial activity, they will make this apparent e.g**. "atrial systole"

- Systole occurs between heart sounds 1 and 2. The pulse is therefore also palpable between heart sounds 1 and 2.



- Whilst listening to the praecordium, feel the carotid (not radial, as too peripheral) pulse to orientate oneself in the cardiac cycle i.e. when you feel the pulse, this is systole, the preceding sound = 1, and the sound after the pulse = 2

Blood Flow in the normal heart

- Blood returns from the systemic circulation into the vena cavae
- Into the right atrium (no valve)
- Into the right ventricle (through Tricuspid valve)
- Into the pulmonary artery (through pulmonary valve)
- Through the lungs and into the pulmonary veins (no valve)
- Into the left atrium (no valve)
- Into the left ventricle (through mitral valve)
- Into the aorta (through aortic valve)
- To the rest of body

The cardiac cycle in a normal heart

- **Diastole:**
    - Starts when aortic/ pulmonary valves shut
    - Ventricles relax
    - Mitral/ tricuspid valves open after very short period (when ventricular pressure has dropped sufficiently)
    - Blood then flows across mitral/ tricuspid valves from atria to ventricles
    - No flow across aortic/ pulmonary valves, as shut during systole

- **Systole:**
    - Starts when mitral/ tricuspid valves shut
    - Ventricles contract
    - Aortic/ pulmonary valves open after very short period (when ventricular pressure has risen sufficiently)
    - Blood then flows across aortic/ pulmonary valves from ventricles
    - No flow across mitral/ tricuspid valves, as shut during diastole

When do murmurs occur?

- Murmurs are due to turbulent flow
- Turbulence occurs when there is:
    - More blood/ faster flow
    - Obstruction to flow (stenosis)
    - Blood flowing in the wrong direction (regurgitation)

- **Stenotic** murmurs therefore occur in the part of the cycle where the valve is supposed to be open
- **Regurgitant** murmurs occur in the part of the cycle where the valve is supposed to be closed
- Endocarditis tends to cause **regurgitant** murmurs (the vegetations are usually not big enough to interfere with forward flow, but do not allow the valve to shut properly)

Variations with respiration

- Blood enters right heart from vena cava
- Input to left heart is from pulmonary vessels
- **During inspiration:**
    - Negative pressure created in thorax to draw in air- also draws in blood from systemic circulation via vena cava (i.e. into right heart)
    - More blood in right heart = more turbulence. **Therefore, right sided murmurs** (pulmonary/ tricuspid) get **louder in inspiration**

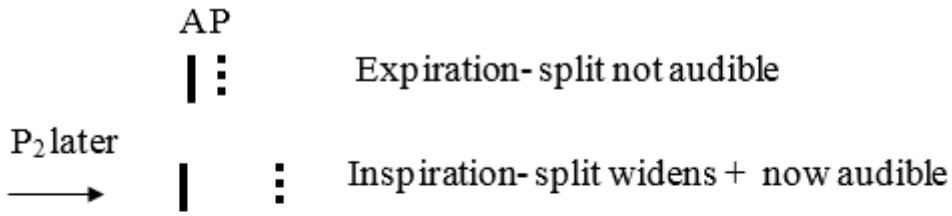
- **During expiration:**
    - Positive pressure created in thorax to push out air- this squeezes the pulmonary capacitance vessels, and the blood is pushed into the left heart
    - More blood in left heart = more turbulence. Therefore, **left sided murmurs** (aortic/ mitral) get **louder in expiration**

- To summarise **RILEs** (**R**ight **I**nspiratory, **L**eft **E**xpiratory)

- Heart sound 2 also varies with respiration. During expiration, it is not audibly split as P_2 closely follows A_2. As stated above, the venous return to the right heart increases during inspiration- this delays right heart emptying, and therefore P_2 occurs later. This leads to an audible split in the second sound.

-

Added Heart Sounds



Heart sounds 3 & 4 occur after heart sound 2

- 3 is usually heard as a sound just after heart sound 2
- 4 is usually heard as a sound just before the next heart sound 1
- **Heart sound 3:**
    - Due to rapid filling of ventricle e.g. mitral regurgitation (large volume of blood in atrium (venous return + regurgitated blood) flows into ventricle)
- **Heart sound 4:**
    - Due to filling against a stiff ventricle e.g. hypertension and aortic stenosis cause LVH (stiffens ventricle)

LVF v RVF

- **LVF (left ventricular failure):**
    - Pulmonary oedema

- **RVF (right ventricular failure):**
    - Peripheral oedema (sacral if patient bed-bound, legs if mobile)
    - Hepatomegaly (due to hepatic vein congestion)
    - Raised JVP
- **CCF (congestive cardiac failure):**
    - LVF + RV

Things to bear in mind

- Endocarditis tends to cause **regurgitant** murmurs.
- Mitral and tricuspid valve lesions can lead to atrial dilatation. This makes one prone to **atrial fibrillation**
- Pulse pressure and pulse character are influenced by **aortic** (and other left ventricular outflow) lesions only, unless the patient is compromised
- The JVP reflects **R-sided** failure or valve lesions. It will not be raised in left-sided lesions unless there is:
    - Fluid overload
    - Secondary R-sided failure (CCF)
    - Dual pathology (i.e. concurrent R lesion)

- The apex beat is only displaced if the ventricle is dilated, not hypertrophied (as hypertrophy only causes a small change in ventricular dimensions). Therefore, it **will be displaced in AR and MR** when the ventricle contains large volumes of blood, but **not in AS** when the volume is normal but the muscle has hypertrophied
- The apex beat reflects left ventricular activity. If the ventricle has to work hard, the apex beat becomes **hyperdynamic** (heaving or thrusting). A heaving apex is felt when the ventricle has to work against a constant resistance during systole (e.g. AS). A thrusting apex is felt when a large volume has to be expelled (e.g. AR, MR)
- Left ventricular overwork is felt as an apical heave. Right ventricular is felt as a parasternal heave (lower left sternal edge)
- Thrills are felt best at the site at which they are heard best
- **Stenotic** murmurs occur in the part of the cycle where the valve is supposed to be open
- **Regurgitant** murmurs occur in the part of the cycle where the valve is supposed to be closed
- Diastolic murmurs are much quieter than systolic murmurs. Therefore, special manoeuvres are used to bring the murmur out (turn to L lateral for MS, sit forward for AR)
- Right sided murmurs are similar in timing to their equivalent L-sided murmur (e.g. TR + MR). The two are distinguished by:
    - Site at which best heard and radiation
    - R- sided murmurs louder in inspiration, L- sided louder in expiration (RILEs)
- Stenotic murmurs tend to occur a short while after the start of the systole or diastole (MDM, ESM) as they only begin when the valve opens. Regurgitant murmurs tend to happen earlier as the valves just don't shut properly (and so leak straight away) – the only exception is in prolapse as the valve is initially competent.

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- Right Sided murmurs are similar in timing to their equivalent L-sided murmur (e.g. TR + MR). The two are distinguished by:

- Site at which best heard and radiation
- R- Sided murmurs louder in inspiration, L- sided louder in expiration (RILEs)

Important Valve Lesions

- The following pages explain the findings in the most common valve lesions in finals exams
- Imagine that you are examining the patient- the findings are listed in such an order. For mitral stenosis, all findings (positive & negative) are listed. For the other lesions, only positive findings are listed.
- Remember the rules stated in the previous section ("Things to bear in mind") to make sense of the findings

Mitral Stenosis

- *Rare to get this in finals unless obvious murmur*

- Hands: nil specific

- **Radial pulse:** AF *(this can be a big clue)* or SR

- BP: nil specific

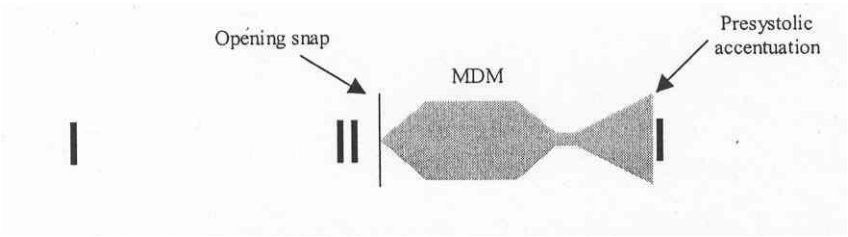
- **Face:** malar flush (differentiate from butterfly rash as malar flush does not affect bridge of nose) *when pulmonary hypertension*

- JVP: nil specific (large a waves when pulmonary HT)

- Carotid pulse: nil specific

- **Apex:** Tapping *(palpable first heart sound)*
  Not displaced

- Auscultation:



Low rumbling mid-diastolic murmur (MDM i.e. valve opens a short while *after* the start of diastole- murmur then ensues)- heard best in expiration, L lateral position. No radiation

Loud first heart sound (as valve closes late)

''Opening snap'' as abnormal valve opens

Pre-systolic accentuation (murmur loudens as atrial contraction occurs) - not present if patient in AF, as no coordinated contraction occurs.

Graham Steell murmur - pulmonary regurgitation due to pulmonary hypertension (buildup of pressure) causing pulmonary valve incompetence

Loud P_2 if pulmonary hypertension
Lung bases: creps if LVF only
Peripheral oedema: nil

- Aetiology: Majority due to rheumatic heart disease
  Rarely congenital
  Calcification! fibrosis in elderly

Mitral Regurgitation

- Hands: Signs of endocarditis if underlying cause
  Arachnodactyly (Marfan's), hypermobility (Ehlers-Danlos)
- Pulse: AF (or SR)
- Eyes: Vasculitic lesions/ Roth spots if endocarditis
- Face: Malar flush if pulmonary HT
- Apex: Displaced thrusting {depending on severity). Thrill if severe
- Auscultation:

Soft 1st heart sound (incomplete mitral closure)

Pansystolic murmur (starts at the beginning of systole as valve does not shut properly and leaks straight away) @ apex radiating to axilla

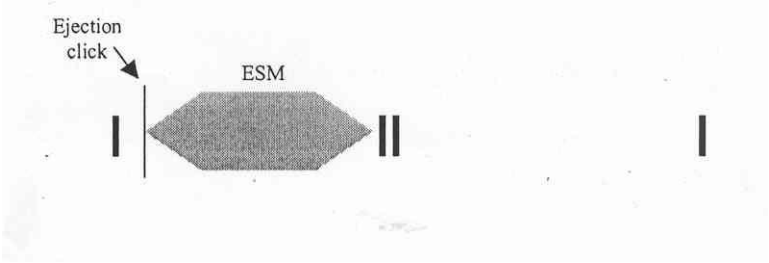
Third heart sound if severe

- Aetiology: Rheumatic heart disease (50%)
  Mitral valve prolapse (note, this causes mid-systolic murmur (after valve prolapses))
  Functional (due to LV dilatation causing mitral valve ring stretching such that valve cusps no longer meet in the middle)

Post-MI (papillary muscle dysfunction or rupture of the chordae tendinae)
Infective endocarditis
Collagen disorders causing floppy valves (Marfan's, Ehlers-Danlos)
Degeneration of valve (age-related)
Failure of prosthetic valve

Aortic Stenosis

- BP: narrow pulse pressure (narrower if more severe)
- Carotid: Small volume, slow-rising
- Apex: Heaving. Not displaced unless decompensated (failing heart)
- Palpation: Thrill in aortic area if severe
- Auscultation:



Ejection systolic murmur (ESM- begins once the valve opens) @,aortic area radiating to **both** carotids. It is crescendo- decrescendo in nature (i.e. gets louder, then softer- see diag).
Note, carotid bruit may be due to radiating aortic stenosis murmur or due to stenosis of the carotid artery itself- therefore check **both** carotids for bruits.

An ''ejection click'' due to opening of an abnormal valve
(compare with ''opening snap'' in mitral valve stenosis)

Soft second heart sound due to immobile valve (note compare this to *loud* heart sound 1 in mitral stenosis)

Fourth heart sound if severe

- Aetiology: Rheumatic heart disease
  Congenital (usually bicuspid, and most are in men)
  Age-related degeneration/ calcification
  Note:- can also have supra- and sub-valvular stenosis

Aortic Regurgitation

- The lesion with countless eponymous signs (all due to large pulse pressure)

- Hands: Signs of endocarditis/ Marfan's
  Quincke's sign- visible capillary pulsation in nail bed (push down on nail gently so that a border of red and white is seen- this border will move proximally/ distally with the pulse)

- **Water hammer pulse**: aka Corrigan's pulse.

- BP: large pulse pressure

- Eyes: Signs of endocarditis
  Becker's sign (retinal artery pulsation)

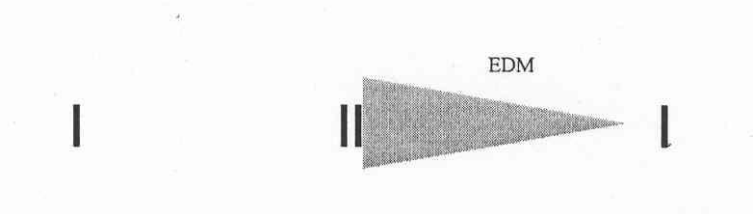
- Mouth: **Muller's sign** (dancing uvula)

- Carotids: Collapsing pulse

- Neck: **Corrigan's sign** (visible gross carotid pulsation)
  De Musset's sign (head nodding due to v. large pulse pressure)

- Apex: Displaced thrusting (depending on severity).

- Auscultation:



  High pitched early diastolic murmur (EDM- starts at the beginning of diastole as valve does not shut properly and leaks straight away) heard best in expiration, with patient leaning forward. No radiation.

- Femorals: These would be sought at the end of the examination
  Duroziez's sign (diastolic bruit when stethoscope lightly applied to femoral artery)
  Traube's sign (aka pistol shot femoral- sharp bang over the femoral arteries in time with each heart beat)

- Aetiology: Rheumatic heart disease
  Infective endocarditis

Syphilis
Rheumatoid arthritis
Seronegative arthritides (e.g. Reiter's)
Collagen disorders (Marfan's and Ehlers-Danlos)
Aortic dissection
Failure of prosthetic valve
Osteogenesis imperfecta

Tricuspid regurgitation

- Similar to MR
- More common in IVDAs (R-sided endocarditis) + carcinoid syndrome
- Signs: AF
  Giant v waves
  RV (parasternal) heave
  Pan-systolic murmur
  Third heart sound if severe
  Pulsatile hepatomegaly

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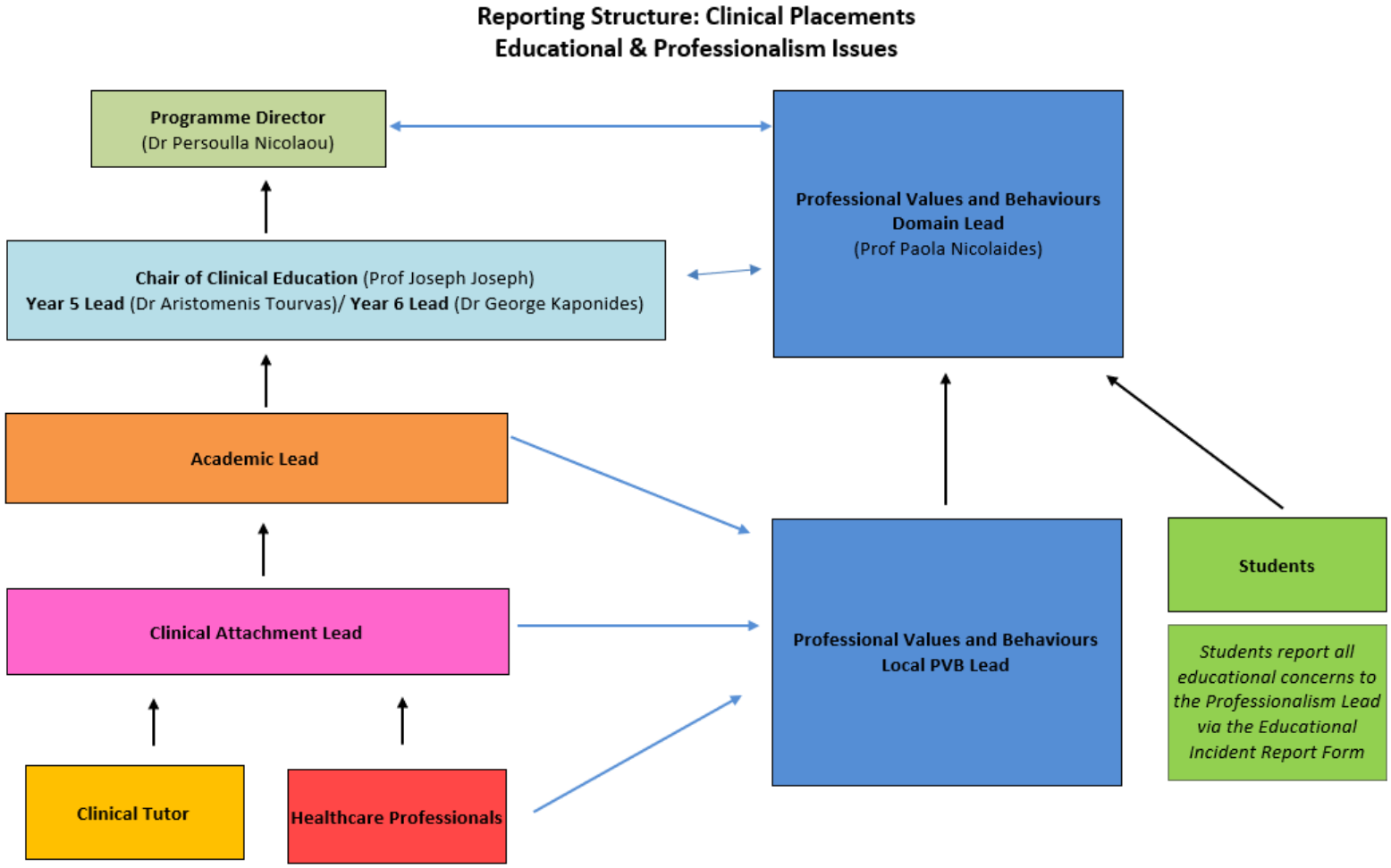
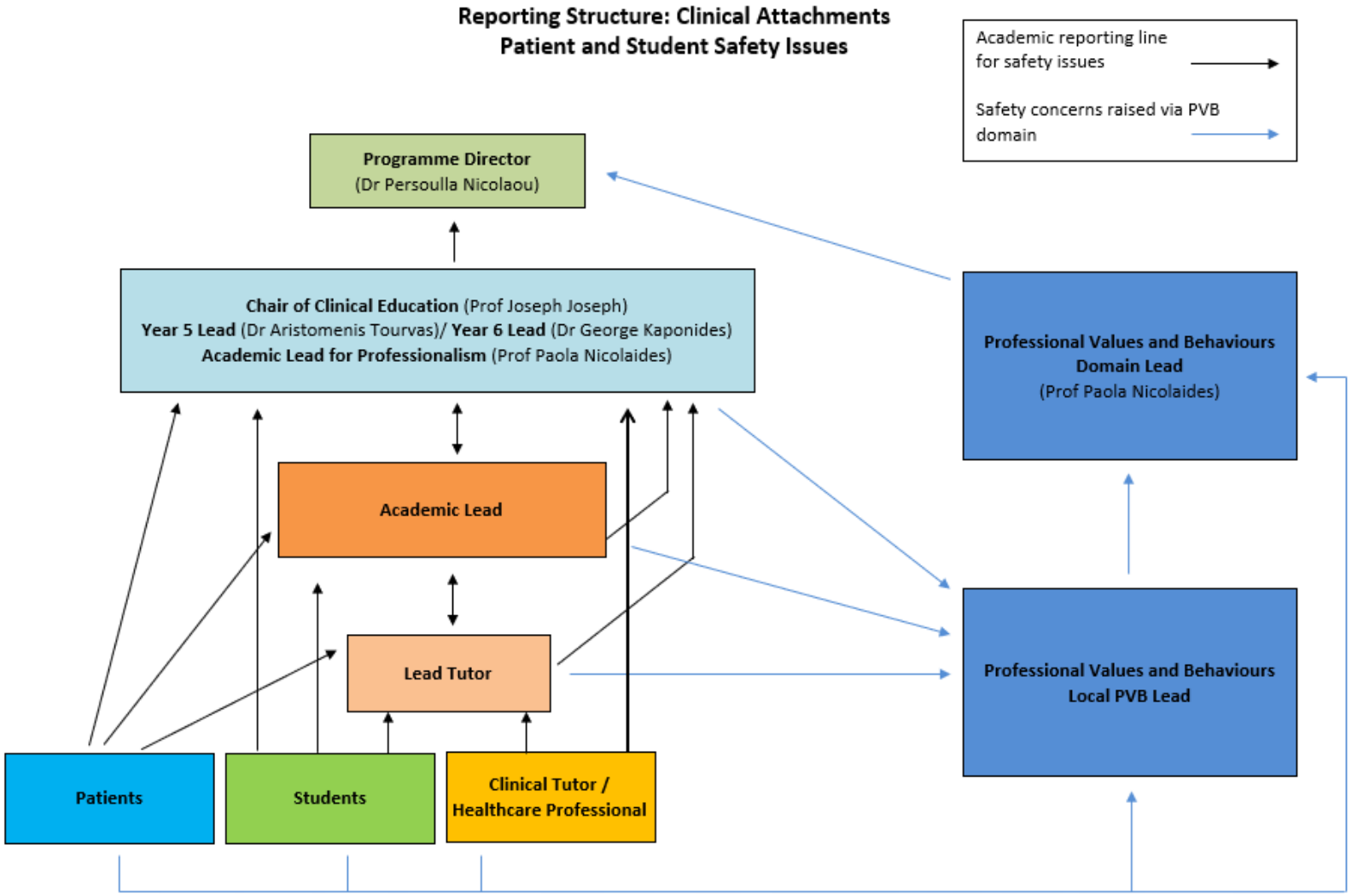
- Even if the patient is not in R-ventricular failure, they will have giant v waves +/- **pulsatile** hepatomegaly.

9. Reporting Structures




Reporting Structure: Clinical Attachments
Patient and Student Safety Issues
Academic reporting line for safety issues
Safety concerns raised via PVB domain
Programme Director (Dr Persoulla Nicolaou)
Chair of Clinical Education (Prof Joseph Joseph)
Year 5 Lead (Dr Aristomenis Tourvas)/ Year 6 Lead (Dr George Kaponides)
Academic Lead for Professionalism (Prof Paola Nicolaides)
Professional Values and Behaviours Domain Lead (Prof Paola Nicolaides)
Academic Lead
Lead Tutor
Professional Values and Behaviours Local PVB Lead
Patients
Students
Clinical Tutor / Healthcare Professional