Clinical Anatomy Series – Cardiac Anatomy

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ABSTRACT

The necessity for an appreciation of human anatomy has been integral to medical teaching for hundreds of years. Today, it still remains a crucial component of the undergraduate curriculum, but with opinions being voiced over the adequacy of this teaching [1], and Royal Colleges noting a reduction in the anatomy knowledge base of applicants, this is clearly an area of concern. Indeed, there was a 7-fold increase in the number of medical claims made due to deficiencies in anatomy knowledge between 1995 and 2007. [2] Therefore, not only does a greater understanding of anatomical principles bring advantages to clinical practice, but also, it can make individuals stand out at interview. This series of articles sets out to explore key anatomical structures placed in their clinical context, starting with cardiac anatomy.

Mediastinum and Covering of the Heart

The heart is located in the middle mediastinum i.e. the area inferior to the sternal angle and bounded by the pericardial sac. The pericardium encapsulates the heart and base of the great vessels, and is composed of a fibrous and serous layer with the fibrous layer being outermost. The serous pericardium is further divided into the parietal and visceral (epicardium) layers; the latter is firmly attached to the surface of the heart and the potential space between the two creates the pericardial cavity. [3]

Clinical relevance

Somatic sensation to the pericardium is supplied by the phrenic nerves (C3-5) which pass in the fibrous pericardium to innervate the diaphragm. As a result, pericardial pain may be referred to the shoulder. [3]

Pericarditis

The aetiology of pericardial inflammation is multifactorial including infection (bacterial, viral, fungal and tuberculosis), post-myocardial infarction, malignancy and autoimmune. Clinical features include sharp central chest pain exacerbated by movement and lying down and relieved by sitting forward. Pain may be referred to the shoulder or neck. Auscultation may reveal a pericardial friction rub, typically at the left lower sternal edge on end expiration with the patient leaning forward. Features of the underlying cause such as pyrexia during infection may also be present. The key investigation is an ECG, which often shows saddle-shaped ST elevation in the early stages. Differentiating this from a myocardial infarction is clearly crucial. Importantly, abnormalities are not normally illustrated on a plain film chest radiograph. [4]

Pericardial effusion

The potential space between the parietal and visceral layers of the serous pericardium normally contains a small volume of fluid. Excess fluid is termed a pericardial effusion. If the volume is sufficiently large, this can reduce ventricular filling as a consequence of the lack of elasticity of the fibrous pericardium and this is termed cardiac tamponade. In severe cases, this can cause heart failure. Echocardiography is the key investigation for diagnosing this condition, and although most cases resolve spontaneously, a pericardiocentesis (drainage of the excess fluid through insertion of a needle) may be required to alleviate tamponade. [4]

The Heart Chambers

The heart is divided into two atria and two ventricles. Externally, the coronary sulcus separates the atria from the ventricles and contains several vessels including the right coronary artery and circumflex branch of the left coronary artery. Delineating the separation of the left and right ventricles are the anterior and posterior interventricular sulci, which also contain major vessels - anteriorly, the anterior interventricular artery and great cardiac vein and posteriorly, the posterior interventricular artery and middle cardiac vein. Internally, these chambers are separated by the interatrial and interventricular septum.

The right atrium forms the right anterolateral border of the heart. It receives deoxygenated blood from the superior and inferior venae cavae and the coronary sinus, which drains the myocardium. On its interatrial wall is the fossa ovalis – the embryological remnant of the foramen ovale that allowed oxygenated blood to enter the right atrium during foetal life. [3]

The right ventricle forms the majority of the anterior border of the heart. The inflow tract to this chamber is rough in appearance due to the presence of multiple trabeculae carneae, which are muscular strips. Three of these strips attach to the tricuspid valve – which separates the right atrium and ventricle – to prevent eversion of its cusps during ventricular contraction. These are the papillary muscles, and are connected to the tricuspid valve via the thin fibre-like chordea tendineae. The outflow tract, which passes to the pulmonary trunk, is smooth by comparison, and is termed the conus arteriosus. Preventing backflow of blood into the right ventricle is the pulmonary valve, which is also composed of three cusps.

The left atrium forms a large proportion of the posterior aspect of the heart. It receives oxygenated blood from the four pulmonary veins. Again, the fossa ovalis is present on the interatrial septum.

The left ventricle forms the left anterolateral and diaphragmatic surfaces of the heart. Similar to the right ventricle, the inflow tract is rough in comparison to the outflow due to the presence of the trabeculae carneae. In comparison to the right side, only two papillary muscles are present to prevent backflow of blood through the mitral valve; one for each of the valve cusps. The aortic valve is placed posterior to its pulmonary counterpart and serves the same function. The right and left coronary arteries originate from the left and right sinuses, which are the space

between the aorta and the aortic valve. This allows blood to enter the coronary arteries when the valve closes during diastole.

Clinical relevance

Auscultation

The heart sounds heard on auscultation of the precordium relate to valve closure. The first heart sound ("*lubb*") represents closure of the atrioventricular valves, the tricuspid and mitral, at the beginning of systole. The second sound ("*dubb*") signifies closure of the aortic and pulmonary valves at the end of systole and beginning of diastole. Cardiac arrhythmias and murmurs can be identified and diagnosed when placed in the context of the cardiac cycle.

Valvular heart disease

Valve disease can broadly be split into regurgitation (a backflow of blood secondary to inadequate closure) and stenosis (insufficient valvular opening causing obstruction to flow). Although these pathological features can arise in any valve, the mitral and aortic valves are most commonly affected.

Mitral valve

A combined pattern of stenosis and incompetence is often present, although one may be more prominent clinically. This leads to dysfunctional blood flow which can produce left ventricular hypertrophy, increased pulmonary pressure, pulmonary oedema and left atrial dilatation. Typically, mitral stenosis produces a mid-diastolic murmur, whereas a pansystolic murmur occurs in mitral regurgitation.

Aortic valve

Again, either stenosis or regurgitation can occur, and both can result in heart failure. Aortic stenosis characteristically produces an ejection systolic murmur heard in the aortic area with or without radiation to the neck. Notably, the volume of the murmur does not correlate to disease severity and indeed the murmur may become quieter as heart failure ensues. Symptoms of dizziness, syncope and angina are common, and a slow rising pulse may be apparent in aortic stenosis. Aortic regurgitation produces a diastolic murmur, often associated with a collapsing pulse.

Right sided valve disease

Tricuspid or pulmonary valve disease is often a result of infection, such as rheumatic fever and infective endocarditis (notably in IV drug users), or congenital malformations. [5]

The Coronary Circulation

The right and left coronary arteries arise from the aortic sinuses as discussed above and act to provide oxygenated blood to all of the cardiac tissues.

The right coronary artery passes inferiorly in the coronary sulcus between the right atrium and ventricle. Along its course, it branches to provide the atrial and sinu-atrial nodal branch and the right marginal branch. It then terminates as the posterior interventricular branch in the posterior interventricular sulcus. As such, it supplies the right atrium and ventricle, the sinu-atrial and atrioventricular nodes, and a proportion of the interatrial and interventricular septum.

The left coronary artery also enters the coronary sulcus, before terminating as the anterior interventricular and circumflex arteries. The former lies in the anterior interventricular sulcus and the latter in the coronary sulcus. This allows the left coronary to supply the left atrium and ventricle and a proportion of the interventricular septum.

Venous drainage of the cardiac tissue is achieved via the great, middle, small and posterior cardiac veins, which all drain into the coronary sinus. This in turn drains into the right atrium. It should be noted that there is a degree of anatomical variation found in the pattern of these vessels.

Cardiac Conduction System

Contraction of the cardiac muscle can occur independently as a result of the presence of an internal conduction system which sends electrical impulses to the myocardium. These signals begin at the sinu-atrial node, otherwise known as the pacemaker. This is located in the right atrium close to the entrance of the superior vena cava. Impulses from this node result in contraction of the atria. The electrical signal then passes to the atrioventricular node, which is located in the atrioventricular septum near the tricuspid valve. This acts as the starting point for signal transmission to the ventricles. Extending from the atrioventricular node is the atrioventricular bundle. This splits into the right and left bundle branch which both pass in their corresponding side along the interventricular septum, before terminating as the Purkinje fibres. This creates a coordinated spread of excitation along the ventricles with subsequent effective myocardial contraction.

Clinical relevance

Coronary artery disease can result in myocardial ischaemia and infarction. The site of artery occlusion determines the area of infarction, and damage to the conduction system in these areas can be assessed by ECG monitoring. Therefore, different ECG readings can point to the affected site. If the resulting ischaemia sufficiently affects the conducting system, fatal arrhythmias and heart failure can occur. The following ECG changes may be seen following a myocardial infarction (MI):

Anterior MI

- ST elevation in V1 – V3

Inferior MI

- ST elevation in II, III and AVF

Lateral MI

- ST elevation in I, AVL and V5/6

Posterior MI

- ST depression in V1 – V3
- Dominant R wave
- ST elevation in V5/6

Further Reading

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