CHAPTER 1 - CARDIOVASCULAR PHYSIOLOGY

Assembly of Japanese bicycle require great peace of mind.

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CLINICAL PHYSIOLOGY AND CARDIAC MURMURS

Introduction:

As Sir Thomas Lewis said, of cardiac valvular abnormalities, diagnosing them is like "recognising a dog by its bark". That may be generally true, but if we wish to make a complete and accurate diagnoses in all four diagnostic categories, then we must build up those diagnoses in each patient, using all of the available clinical features. This chapter sets out to illustrate how this can be done within the cardiovascular system.

The history will give us a great deal of our functional diagnosis. Thus there may be symptoms of "forward" or "backward" heart failure (Ch. 3) or, in less severe cases, symptoms of impairment of cardiac function under load; also palpitations from any cardiac irregularity, pain etc. As usual, the history of the time-intensity relationships will give us information on the clinical pathology. Aetiology, as usual, should be sought from the background history.

Physical examination of the CVS gives us additional information, particularly about the Anatomical diagnosis and its Functional significance.

Clinical diagnostic problem solving in a patient with a cardiac murmur.

Here, you need a detailed history and a complete, accurate physical examination of the whole CVS .

(A) PHYSIOLOGY AND ANATOMICAL DIAGNOSIS OF MURMURS

Murmurs are produced by turbulent blood flow across valves (or heart defects) in three main circumstances.

1. High blood flow rates through normal or abnormal valves or vessels.

2. Forward flow through constricted or irregular valves into a dilated vessel or chamber.

3. Backward or regurgitant flow through an incompetent valve, septal defect, or arterio-venous communication.

Just as aggravating and relieving factors are important in making an anatomical diagnosis from the history (e.g. shortness of breath on lying flat suggests left heart failure), so too can factors that diminish or increase the intensity of murmurs be of value on examination in anatomically localising the valve involved. These various manoeuvres include position, hand-grip, valsalva, squatting, relationship to respiration etc. Thus, hand-grip (increases blood pressure or LV "afterload") exacerbates aortic regurgitation (AR), and the mitral regurgitation (MR) murmur of mitral valve prolapse; standing decreases venous return and cardiac filling and so increased the loudness of the murmur of sub-aortic valve stenosis from hypertrophic cardio-myopathy; inspiration increases right-sided heart murmurs and sounds; expiration typically increases left heart murmurs and sounds.

SYSTOLIC MURMURS

1.Pan-systolic murmurs.

Caused by blood flowing from a high to a low pressure chamber. Classical examples are mitral regurgitation (MR), tricuspid regurgitation (TR), and ventricular septal defect (VSD). In all three, the murmur begins with the first heart sound (because the relevant pressure differential begins even during the isometric contraction phase of the appropriate ventricle) and continues up to, and even through, aortic valve closure (pressure differential across a-v valve continues even during the isometric relaxation phase of the ventricle); in TR the murmur may go right through the pulmonary sound into early diastole for the same reason.Characteristically, MR is a high-pitched blowing pan-systolic murmur maximal heard at the cardiac apex and radiating to the axilla.TR is best heard at the left parasternal edge, may radiate to the apex, but not the axilla; as with most right heart murmurs it is increased during inspiration (because of the normally associated increase in venous return at that time). VSD is heard maximally at the lower left sternal edge; in milder cases it may not continue right through to the second heart sound due to late systolic functional closure of the defect from septal muscular contraction. Again, having made the diagnosis of the valve involved, ask yourself whether this is the primary anatomical diagnosis, or whether it is a secondary functional consequence of some other cardiac problem (cardiac dilatation and failure can produce dilatation of the AV rings and poor contraction of the papillary muscles to the chordae, and hence *secondary* MR/TR). If the murmur does seem the likely primary problem, then its functional severity should also be assessed.

2.Ejection Systolic Murmurs

In the heart, ejection systolic murmurs relate to flow abnormalities (not necessarily organic) across the aortic and pulmonary valves. The classical murmurs are produced by aortic valvular stenosis (AS) and less commonly pulmonary stenosis (PS). These are mid-systolic murmurs because flow

begins over the narrowed valve only when the pressure in the ventricle concerned exceeds the aortic or pulmonary arterial pressure. This does not happen until beyond the first sound (AV valve closure) because it must await the completion of the isometric contraction phase of the ventricle. So the murmurs clearly begin after the first heart sound. Moreover, with increasing systolic flow across the valve they reach a maximum in mid-systole and fall away again as flow across the valve wanes late in systole with diminishing pressure differential between artery and ventricle. This gives a "diamond-shaped" murmur on phonocardiography, becoming inaudible before second sound valve closure. Again, ask whether this is an abnormal valve, or perhaps an increased flow across a quite-normal one (as in the high cardiac output states associated with thyrotoxicosis, anaemia, fever, etc.). AS and PS are heard as medium pitched, harsh, rough, mid-systolic 'ejection' murmurs maximally at the base of the heart, and conducted to the neck.

DIASTOLIC MURMURS

1.Diastolic Rumbles. Mitral Stenosis - MR. (Also TR)
These are due to increased flow across normal AV valves or normal flow across diseased, distorted, or stenotic ones. Since there are two phases of rapid diastolic ventricular filling, these murmurs are typically mid-diastolic, with presystolic accentuation. The mid-diastolic component arises because, after the semilunar valves have closed (second heart sounds), the pressure within the ventricles must drop to a level below that in the atrium before the AV valves can open (isometric ventricule relaxation phase); only then will diastolic ventricular filling begin. Then, a subsequent pre-systolic accentuation arises from atrial contraction late in diastole producing a boost or "kick" to blood flow across the valve. These murmurs, too, can be functional, e.g. increased diastolic AV valve flow in MR and TR (secondary to large volume back flow through the corresponding AV valves during systole); also functional flow murmurs in left to right shunts such as ASD, VSD, patent ductus, etc. In addition, an "Austin-Flint" mid-diastolic murmur may be heard in association with severe AR (the backward flow of blood from the aorta to ventricle pushes the anterior mitral valve leaflet half closed during diastole, thus causing a transient partial functional mitral valve narrowing).

2. Early Diastolic Murmurs. **Aortic regurgitation (AR), Pulmonary regurgitation (PR)**.

These both give an early diastolic murmur, coming right after the corresponding second heart sound, when their intensity is maximal (corresponding to maximal back flow from the corresponding artery to ventricle). This flow then diminishes rapidly so that the murmur is 'decrescendo', characteristically ending about mid-diastole. The murmurs are usually high-pitched, and best heard over the left third or fourth intercostal space; again various manoeuvres can intensify the murmurs to make the anatomical diagnosis clearer, in the case of AR by sitting the patient up and having him breathe out fully (to throw the aortic valve root closer to the sternum.

(B) DETERMINING THE FUNCTIONAL SIGNIFICANCE OF CARDIAC MURMURS.

1. Loudness of a Murmur and its Functional Significance

In general, with cardiac volume overload problems, the loudness of even organic murmurs is not well correlated with severity.This is because with MR and TR, the AV valve diameters are large in any case; also, with any regurgitation murmur, loudness is greater if there is any accompanying narrowing or stenosis of the valve. Loudness of AS and PS correlate a little better with the degree of stenosis.

2. Quality of a Murmur and its Functional Significance

Helps distinguish aortic stenosis from a haemodynamic mid-systolic aortic flow murmur, the former being of much more 'rough' quality.

3. Length of Murmurs and Functional Significance.

The longer a mitral mid-diastolic murmur (i.e. the earlier it occurs after the closing of the second heart sound), the more severe the MS. In addition the longer the murmurs of AS and PS extend into late systole, the more their functional significance.

4.Heart Sounds and Functional Significance of Murmurs

(a) First heart sound (S1)

Loudness of S1 determined by three factors:

(i) The position of mitral leaflets at the onset of systole.

(ii) The structure of the leaflets themselves (whether abnormal or thickened).

(iii) The rate of rise of pressure and tension in the (left) ventricle.

The first heart sound is loud in the mitral area in MS because the prior sustained pressure gradient through the stenosed mitral valve ensures that the leaflets are held wide open until sudden ventricular contraction snaps them shut with a bang like a wide-open door! Loud first heart sounds can also be produced by increased ventricular contractility, or by decreased PR interval on ECG - if PR interval long, then valves have a chance to ebb towards the closed position before the onset of systole, and hence close softly. There is usually only one element to S1 at the apex (tricuspid closure not being heard there). The corollary is that if you hear what appears to be a split apical S1, ask yourself whether you are dealing with an added sound such as a 4th (low pitch) or a systolic ejection click (higher pitch).The first heart sound is often diminished in functionally significant MR (because the valve just does not close). Soft S1 also occurs in AR because of a very short isometric left ventricular contraction phase.

(**b) The second heart sound (S2)**

Four factors determine the frequency and intensity of semi-lunar valve vibrations, viz: the rate of development of pressure, the pliability or stiffness of the valve leaflets, valve radius, and blood

viscosity. Normally, only one (aortic) component of the second sound is heard at the cardiac apex, but over the base we hear two components, the first being the aortic (A2 closes earlier because of the higher systemic arterial pressure) and the second pulmonary valve, closure (P2 also softer). The split is normally narrow but varies with respiration, being clearly split during inspiration and closing right up during expiration. Persistent splitting in expiration suggests right bundle branch block (RBBB) - because of delayed right ventricular relaxation. Fixed splitting suggests atrial septal defect (ASD), because the left-to-right atrial shunt determines that there is very little of the usual % increase in venous return during inspiration). Narrowed or even reverse splitting of S2 can occur in aortic valve disease and hypertension, but most commonly in left bundle branch block (LBBB).

(c) Added Sounds and Functional Significance of Murmurs

Third and fourth heart sounds will be discussed below in relation to the various forms of cardiac "overload" brought about by valvular abnormalities. Other abnormal added sounds may arise from the opening of abnormal valves, usually with clicks. These are important in two respects. First, opening valve sounds are *not* usually heard in functional murmurs. Second, an opening click or snap heralding a murmur implies that the valve concerned is non-calcified and mobile, and this is important in MS. Opening clicks are also frequently heard heralding the onset of the systolic MR murmur associated with floppy mitral valve leaflet syndrome. Here, the mitral valve tends to balloon like a parachute, so the leaflet(s) do not become significantly incompetent until late in systole. What we therefore hear is a mid-systolic ejection click followed immediately by a late systolic murmur, rather than a pan-systolic murmur. Moreover, if the posterior leaflet is the one more involved, the blood jet created by the mitral incompetence is directed over the dome of the contracting anterior leaflet towards the atrial septum, causing radiation of the murmur towards the left sternal edge and base rather than the axilla. With anterior leaflet prolapse the radiation is more to the back because the incompetent jet is posteriorly directed (over the posterior leaflet dome). Note that papillary muscle dysfunction in ischaemic heart disease can produce similar late systolic murmurs.

5.Haemodynamic Signs and the Assessment of Functional Significance of Murmurs

(a) Cardiac Signs: To understand these we have to appreciate that there are two general forms of "overload" that valvular abnormalities may place on the heart. These are "volume" and "pressure" overloads.

(i) Volume Overload

This occurs with many of the pansystolic murmurs (MR, TR, VSD), and semilunar valve incompetence (e.g. AR) murmurs. Also with high output states and shunts including ASD, patent ductus, A-V fistulae; also anaemia, beri beri, fever, thyrotoxicosis, Paget's disease, pregnancy etc. The commonest situation is a leaking valve allowing backflow and there, to maintain a normal cardiac output, stroke volume per beat must be correspondingly increased (e.g. even with the mildest haemodynamically significant MR, 30% of each stroke volume of the left ventricle is pumped backwards through the incompetent mitral valve, so that to get a normal 70 ml output per beat through the aortic valve into the systemic circulation, the ventricle must pump a total of 100 ml each

time. Because of this, volume overload forms of cardiac impairment are characterised by cardiac dilatation, and the degree of cardiac dilatation clinically corresponds with the degree of overload, i.e. with the functional significance of the valvular lesion. Not that cardiac dilatation necessarily signifies cardiac failure, because the dilatation increases cardiac muscle fibre length at the beginning of systole, so improving myocardial contraction; also, over the longer term, the heart hypertrophies, and this helps maintain a compensated state. Functional murmurs may be heard in association with various forms of volume overload, for example the functional mid-diastolic flow murmur of severe organic MR, TR; the aortic ejection systolic flow murmur of severe aortic incompetence; and the (tricuspid) mid-diastolic murmur in atrial septal defects with left to right shunts, merely reflecting the increased diastolic flow across the tricuspid valve.

(ii) Pressure Overload

In contradistinction to volume overload, pressure overload on the heart, as in AS, PS, and hypertension, gives rise not to dilatation but to ventricular hypertrophy and usually concentric hypertrophy, so there is no dilatation at all, but a thrusting or heaving over the corresponding area (apical thrust with LV hypertrophy, left parasternal heave with RV hypertrophy). In addition, on auscultation there is characteristically an added fourth heart sound (S4 - soft late diastolic and low-pitched). This arises because the late increase in atrial flow produced by atrial contraction (the atrial kick") runs abruptly up against the hypertrophied, non-compliant ventricle. S4 also heard in myocardiali infarction and other conditions associated with increased ventricular "stiffness". Right-sided fourth heart sounds are usually best heard in inspiration at the left sternal edge, whereas left sided S4 best heard at the cardiac apex. Note that we refer here to the compensated state where cardiac output remains normal, at least during everyday activities.

3. (b) Peripheral haemo-dynamic signs indicating degree of significance of cardiac murmurs.

(i) Compensated State. Haemodynamic signs on the arterial side of the circulation are especially important in the diagnosis of the functional significance of aortic valvular lesions. Thus, the slow carotid upstroke and prolonged pulse of aortic stenosis; the 'collapsing' (rapid upstroke and rapid fall) wide-pressure pulse of aortic regurgitation. With MR, MS and other cardiac lesions, the pulse volume often remains relatively normal, at least in the compensated state at rest. With right sided valve lesions, we may also see peripheral signs that help us determine functional significance, particularly in the jugular venous pressure (JVP). Thus, the height of the JVP "a" wave correlates with severity of tricuspid stenosis; also the giant systolic "v" wave in severe TR.

(ii) Signs of cardiac decompensation and eventual cardiac failure.

The heart has several mechanisms to compensate for haemodynamic abnormalities arising from valvular disease, so that cardiac output may be well maintained and the patient remain relatively asymptomatic. And when it occurs, cardiac de-compensation is not an all or nothing phenomenon. Initial symptoms and signs will be present only under load (e.g. exercise), and it is only at a relatively

late stage that cardiac output will fall at rest. Hence the importance of your history in determining the presence of any early decompensation.
In general, we talk simplistically about **two sorts of cardiac impairment or failure**, namely**"forward" and "backward"** failure. In the history, "forward" failure or decompensation is characterised by lethargy and tiredness (initially on exertion), and if severe (as in AS) syncope on effort and even angina of effort in the absence of coronary disease (due to a drop in coronary diastolic arterial perfusion pressure under exercise load). The signs of "forward" failure can be just as prominent in right heart failure as in left, because the left ventricle can only pump on what it is delivered by the right. "Backward" failure is really a misnomer (see Ch 3), but let us accept the concept for the moment. In left sided cardiac impairment, this is suggested by shortness of breath on exertion (pulmonary congestion during exercise from increased pulmonary venous "back pressure"). Later, as the left ventricular output becomes more impaired (with remaining normal right ventricular function) resting left ventricular end-diastolic volume, and with it pressure, become elevated. This first comes to the notice of the patient when (s)he lies flat (orthopnoea, paroxysmal nocturnal dyspnoea or PND), because this causes blood normally pooled in the legs to be shifted to the lungs, increasing pulmonary venous pressure and pulmonary congestion still further, thus reducing the normal compliance of respiratory ventilation. Here patients have 'decompensation' under load but are not yet in end-stage cardiac failure. In this respect, it is often useful to examine patients under load. e.g. even the act of undressing for physical examination can bring out signs of cardiac decompensation not obvious at rest.

(iii) Cardiac Failure

Now, the patient with left ventricular failure will be short of breath at rest, and have severe orthopnoea, PND, and even pulmonary oedema at rest (late inspiratory crepitations over the lung bases posteriorly). There will also be signs of "forward" failure with decreasing pulse volume, increased pulse rate, poor peripheral perfusion, lethargy, tiredness and mental confusion, even at rest. When this is so, the body attempts to maintain normal arterial perfusion pressure to the brain, coronaries etc., through a reflex sympathetic shut-down of blood flow to the hands, splanchnic circulation and kidney. The resultant reduction of kidney function leads to salt and water retention, oedema and elevated JVP, signs we usually take as evidence of "right ventricular failure". Of course, once the left heart failure becomes severe, there eventually comes a time when the right heart actually does fail, because the high pulmonary venous pressure is transmitted to the pulmonary arteries; this may not seem important alone, but as pulmonary oedema follows, so pulmonary alveolar pO2 falls, and with it, local hypoxia with reflex pulmonary arteriolar constriction/pulmonary hypertension. This may ease pulmonary congestion, but leads to a pressure overload on the right ventricle, and because this ventricle is just not built to withstand such load, it rapidly fails.

Primary right heart failure is usually indicated in the history by symptoms of increasing oedema and elevated JVP.

When ventricular failure occurs, auscultation frequently reveals an added third heart sound. This is because the maximum (mid-diastolic) ventricular filling comes up against a flabby myocardium,

causing it to abruptly halt, just as the wind abruptly catches a sail. This gives rise to a low-pitched sound best heard over the appropriate ventricle.

Even when a cardiac "pressure-overload" problem becomes decompensated, the ventricle will dilate, and as it fails, a third heart sound may be added in just the same way. Therefore, if there was a fourth heart sound before (e.g. in aortic stenosis), you may now hear first, second, third and fourth sounds, called a "summation gallop" - like a galloping horse. In cardiac failure, you will often hear of the terms "preload" and "afterload", and how manipulation of one or both may improve cardiac output. We will deal with this later, but for the moment you may, as an approximation, equate "afterload" with the systolic pressure seen by the appropriate ventricle, i.e. pressure overload; and "preload" with end-diastolic ventricular pressure.

Cyanosis:
Cardiac failure can be associated with both peripheral and central cyanosis; peripheral because of the poor peripheral circulation and long transit time through the tissue capillaries; and central cyanosis when pulmonary interstitial and/or alveolar oedema from pulmonary congestion compromises arterial oxygen re-saturation in the pulmonary capillaries.

Building a Functional Diagnosis. In making a clinical diagnosis, build up your functional diagnosis hierarchically. Thus, in a patient with shortness of breath, we can't assume the condition to be cardiovascular, but must look for the appropriate signs that point more specifically to that (i.e. orthopnoea, PND etc.) In the same way, in patients who present with oedema, we can't assume that this is due to right heart failure - e.g. other causes include hypoproteinaemia and renal failure to excrete salt and water.

C. CLINICAL PATHOLOGY DIAGNOSIS

As usual, we judge the general clinical pathology from the time-intensity relationships of the symptoms, and look for weight loss and fever. For example, a patient with a prominent valvular murmur and fever would make one wonder strongly about an inflammatory valvulitis (bacterial endocarditis); indeed, so important is this that it would be the case until proven otherwise. Of course, a high fever itself may give rise to an increased cardiac output and a secondary flow murmur, so again we must learn to look at associations of symptoms and diagnostic categories both ways around to determine cause and effect.

D. AETIOLOGICAL DIAGNOSIS

Again, look in the background for what has preceded the condition to determine this; e.g. rheumatic fever as a prelude to later rheumatic carditis; syphilis predisposing to AR, Marfan syndrome associated with mitral valve incompetence (floppy mitral valve). Sometimes examination also gives a

clue, e.g. a click preceding a pansystolic MR murmur suggests floppy mitral valve leaflet syndrome rather than rheumatic carditis.

CONCLUSIONS

The above has given you the tools to solve common clinical problem where a cardiac murmur is the predominant feature. But patients usually present with other symptoms and signs, and this will give you additional information about anatomical diagnosis (from the nature and quality of symptoms, their site and radiation if pain, and any aggravating or relieving factors). Similarly with Pathology you may get additional information on examination e.g. detection of an elevated temperature.

MCQs: MECHANISMS IN DISEASE

MCQs are of two types.

A. MECHANISMS IN DISEASE

This is where you are given a diagnosis, and have to work out whether or not the signs and symptoms are appropriate. This is not as unrealistic a clinical exercise as it may sound, because patients are often sent to you with a diagnosis, and your first task there is to see whether, on clinical grounds, that diagnosis is tenable.

Example:

1. A patient is said to have a haemodynamically significant stenosis of his aortic valve. Which of the following would be characteristic?

a) A thrusting cardiac apex.

b) A loud mid-systolic murmur and thrill over the upper sternum.

c) A slow upstroke to the carotid arterial pulse.

d) A loud second element of the second heart sound.

e) A third heart sound.

f) Dyspnoea and faintness on exertion.

(Answers at end of chapter.)

CLINICAL PROBLEM SOLVING & MCQs

The second type of question the building up of a diagnosis on the basis of a case study. To solve the case problem by our method, draw up a blank sheet of paper, headed in four columns, viz. 'Where?' (Anatomical diagnosis); 'What?' (Clinical Pathological diagnosis); 'How?' (Functional diagnosis); 'Why?' (Aetiological diagnosis). The Functional diagnosis should be by far the widest column, not only in its own right, but because much of your Anatomical diagnosis is derived from conclusions drawn from functional abnormalities. Having done this, you should then go through the clinical information phrase by phrase, assigning each (numbers in brackets) to one or other category/ column. Then, you should try to draw an conclusion/inference from it, no matter how tentative at the start. And so on in each column. This will greatly simplify your data-synthesis within and between columns, so facilitating increasingly hierarchic levels of diagnosis to an eventual diagnosis overall.

Continue thus below, or go to the Online Cardiovascular Tutorial.

Example

A 36 year old woman(1) presents with a six week(2) history of irregular palpitation(3) followed by increasing shortness of breath on exertion(4) recently waking her at night(5). She has not noticed any shivers or sweats(6), but has noticed increasing ankle oedema over the last three weeks(7). Past history: rheumatic fever at age 16(8), and two years ago, on routine examination, she was told that she had a "heart murmur"(9)

On examination, pulse rate 140/min and completely irregular(10), blood pressure 125/75 mm Hg(11). In the neck there is a 6 cm. elevated JVP(12); only one venous wave is detectable, and on timing with the opposite carotid pulse this turns out to be a systolic wave(13). Despite very full pulsation, pressure over the base of the neck obliterates this wave; it is also increased by abdominal pressure, and reduced by inspiration and by assumption of the upright posture(14). No diastolic venous wave is detectable in the neck(15). Inspection of the precordium: apex beat in the midclavicular line in the 5th left intercostal space(16). Palpation reveals a palpable first sound(17), and normal quality apex beat(18), but a prominent left parasternal heave(19). Heart not enlarged to palpation or percussion(20). Auscultation: A loud first heart sound(21); second heart sound normal at apex, but second component is increased in intensity over the second left intercostal space(22). There is an added diastolic heart sound of a clicking nature(23), heard very soon after the second component of the second heart sound(24). The murmurs heard are as follows: There is a soft high pitched blowing pansystolic murmur heard maximally at the apex and conducted to the axilla(25); also a long, low pitched, rumbling apical mid-diastolic murmur following immediately after the added clicking very

early diastolic heart sound, and continuing throughout most of diastole(26), although without any presystolic accentuation(27). No other murmurs.

Examination of the chest: fine late inspiratory crepitations at both lung bases(28). During examination, the patient also complains of discomfort and shortness of breath on lying flat(29). There is moderate ankle oedema bilaterally(30). On general examination there are no abnormalities. In particular, temperature is normal (31). There are no splinter haemorrhages, no finger clubbing, fundi normal, no splenomegaly, no painful finger nodes, Urine microscopy - No abnormality detected (NAD) (32).

Solving the problem

The table in the separate online **'Cardiovascular tutorial'** will show how each phrase (&/or conclusion from it) can be assigned to a particular diagnostic category, and how conclusions can be drawn within each of those categories, so that a final overall diagnosis can be derived. Having done that, you will then be in a position to readily answer any question related to this patient without having to go back again and again to read the case findings each time.

The alternative is to solve this problem separately here yourself in four columns. Some practical hints should prove useful, particularly in the early stages of your approaches to this method. First, the Functional or "How?" column tends to contain most of the raw clinical information. This not only emphasizes the importance of physiology in clinical diagnosis, but tells you that you must draw up your four columns so that this particular one is widest. Next, as you go through the various clinical information try to draw inferences from each observation. Such inferences may of course be somewhat tentative initially [e.g. (3) = palpitation, ?? atrial fibrillation], but as more information unfolds, early tentative interpretations can be either confirmed (if so, join by arrows) or rejected. In this way firm <u>sub-conclusions</u> become possible, and should be highlighted as you go through your column work [e.g. (20) confirms atrial fibrillation]. This approach is particularly helpful in a long case with a lot of clinical information.

Sometimes your working sub-conclusions may involve other aspects than the particular column you are working in. For example, (26) suggests, from the functional abnormalities, involvement of the mitral valve and since this is an anatomical conclusion, this piece of information should be transferred across from the "How?" to the "Where?" column - good example of 'Diagnostic Category Overlap.' However, be careful not to transfer conclusions too early to the Anatomical column. Reserve this "Where" category as much as possible for the *primary* Anatomical region/system(s) involved. For example, (19) suggests right ventricular involvement (hypertrophy) but this is probably a secondary rather than the primary level of anatomical involvement and as such is best left in the "How?" column of Functional consequences. Similarly with (22) - pulmonary hypertension.

The commonest example of information transfer between columns is when the physiological/ Functional consequences of the process give a clear indication of the Anatomical organ system

involved. Information transfer between other columns is also possible at times. But if you are not sure where any piece of information should go, put it in the "How?" or Functional column, but with a big question mark beside it, and be on the lookout later for information which may combine with it and allow transfer to a more relevant column. (For example, if some patient has an enlarged liver (Liver - 'HOW?' column) associated with signs of liver dysfunction ('How?'), and you later find that the liver has a firm edge, be prepared to transfer the words 'Firm liver' from the Functional column to the Pathological ('What?') column - Chronic.

Write down not just the phrase from the problem itself, but your inferences from it - as in the accompanying Online Tutorial. Thus, under (22), an increased P2 is not of much value as a bald statement unless accompanied by the tentative inference that there may be pulmonary arterial hypertension. Of course, this process gets easier as more and more information becomes available, and in the early phases you may find it difficult to draw even tentative conclusions; it is for this reason (7) is put down merely as ankle oedema without any inference at this stage.

GRAPHIC SOLUTION TO CARDIOVASCULAR DIAGNOSTIC PROBLEM:

When you have finished your four columned solution, compare with that of the author, by reference to the **graphic solution in the next section: Problem Solution 1 & 2.**

Before consulting the graphic, you should be able to answer the following questions about this patient in any of the four diagnostic categories. Answers in last chapter section. Also, when referring to graphic solution, for best viewing centre it with the space bar below the graph.

Then **answer the following MCQs**.

WHICH OF THE FOLLOWING STATEMENTS IS/ARE CORRECT?

1. This patient has evidence the impairment of the right heart function.

2. The dominant lesion from the haemodynamic viewpoint here is mitral valve stenosis.

3. There is evidence that any impairment of left heart function is primarily related to systemic pressure overload.

4. There is evidence that impairment of right heart function is related to volume overload.

5. The pansystolic murmur heard could well be due to mitral ring dilatation in this patient.

6. Careful ascultation should typically reveal a fourth heart sound at the cardiac apex.

7. Wide splitting of the second heart sound would be expected in this patient.

8. The absent "a"-wave in the JVP reflects reduced diastolic ventricular filling.

9. There is evidence consistent with pulmonary hypertension and right ventricular hypertrophy.

10. The recent appearance of symptoms would be best explained by a rapid progression of the underlying valvular heart disease.

11. Development of symptoms of fever in this patient at any time in future would most likely be due to myocarditis, which is the most important thing to exclude.

12. There is good evidence of acute valvulitis ("endocarditis") in this patient.

13. The likely underlying aetiological cause of this patient's valvular condition is mitral valve prolapse due to lax chordae tendineae.

Answers in final section of the chapter.

PROBLEM SOLUTION-1

Cardiovascular Diagnostic Problem

Where?	What?	How?	Why?
	2. 6/52 = sub-acute	3. Irreg. Palpitation = ? A.F.	1. Young woman
		4. SOB.on effort ? L. heart dysfunction.	
		5. ? PND.	8,9. Prev.rheumatic fever, 2 yr. heart murmur = **? Bkgd. rheum. valve disease.**
	6. No shivers/sweats ? Non-inflamm.	7. Ankle oedema - ? R heart dysf^{n}.	
		10. V irreg. pulse = Probable A.F.	
		11. Normal B.P./"afterload".	
	9. Prev. murmur = ?chronic sub-clin. background	12. ↑JVP. = prob. R. heart dysf^{n}	
		13.) Large venous "v" wave, 14.) suggests possible T.I.	
		15. No "a" wave — confirms **A. F.**	
		16. Apex beat M.C.L. = no cardiac dilatation.	
		17.Palpable S 1.	
		18. Normal Apex beat =.No L.V.H.	
		19. L parasternal heave = R.V.H.	
		20. No cardiac dil^{n} . - confirms 16.	
		21. 1st heart sound ↑ - confirms 17.	
		22. Increased P 2 = ? pulmonary hypertension	
	23, 24. O. S . Thus, if M.S., then mitral valve is mobile i.e.non-calcified.	23. Diastolic click = opening snap of mitral valve.(O.S.).	
		24. Early O.S = **severe**	
Mitral Valve		**Mitral stenosis** (M.S.)	
		25. P.S.M. at apex → axilla = Mitral incompetence (M.I.)	
		26. Long mid diast.murmur. = M.S.	
		27. Absent pre-syst. accentuat^{n} of M.D.M.— due to A.F.	
		28. Basal lung creps. = L. heart failure.	
		29. Orthopnoea = L heart dysfunction.	
		30. Ankle oedema - confirms R.H.F.	
	31. Temp. normal = prob. non-inflamm.	32. No evidence of systemic emboli (e.g. from "valvulitis").	

PROBLEM SOLUTION-2

Cardiovascular Diagnostic Problem (contd).

CONCLUSIONS.

Anatomical Diagnosis	Pathological Diagnosis	Functional Diagnosis.	Aetiological Diagnosis
Organic **Mitral Valve** disease with MS and MI	**Sub-acute non-inflammatory**	**Mitral Stenosis** (17, 21, 23, 24, 26). — severe (21, 24, 26). **Mitral Incompetence.**(25). — mild.(16, 17, 20). **Atrial Fib**. (3, 10, 15, 27), prob. came before SOB, PND, (3. vs.4, 5.); therefore prob. ppt^{d}. it by sudden ↓ in diast. L. Vent. filling .→ **2° L.H. failure** (4, 5, 28, 29). = pul. venous congestion /oedema → local pulmon. hypoxia → pulmon. arteriolar. constrict^{n} → pulmon. hypertension (19, 22.) → **2° R. heart failure** (7, 12, 30) → **T.I.**(13, 14) - ? functional (i.e.? 2° to R.V.dilat^{n} — is there systolic liver pulsation, ascites? Go back and look for these).	**Underlying rheumatic heart disease**

MCQ ANSWERS

A. Mechanisms in Disease.

Answers to MCQ:
1. All a) - g) are true.

B. Problem solving.

Answers:
1, 2, 7, 9, true. All others false

Case Study Examples