CHAPTER 3 - HEART FAILURE

INTRODUCTORY COMMENT

The primary aim of this chapter is to discuss the pathophysiology of cardiac failure. I do this in the hope of giving a simple view of it, to facilitate understanding of why different signs and symptoms occur, and so be in a position to best use clinical information in building up clinical diagnoses and treating individual patients.

Some normal values

Cardiac output - normally approx. 5 l/min.

Stroke volume (SV) - approx. 70 ml.

Heart rate - approx. 70 beats per minute

End Diastolic Volume (EDV) - approx. 120 ml.

Systolic ejection fraction = SV/EDV = approx. 67%.

Mean JVP - 0-2 cms. above manubriosternal angle.

End diastolic pressure - up to 10 mmHg before "a" wave. No more than 15 mmHg at "a" wave peak

Know your "pressure volume loops", and the Frank-Starling curves.

DEFINITION

This is not as easy as it sounds. An elevated venous pressure may reflect right heart failure, but it may also reflect overfilling of the circulation as in over-transfusion, or in sodium and water retention from renal impairment. Therefore, the most generally accepted definition of heart failure is that state when the volume output of the heart fails to keep pace with the metabolic demands of the tissues. By this, heart failure may occur in absolute terms when the pump itself fails, or when there is a higher demand than it can meet, as in severe thyrotoxicosis.

TYPES OF CARDIAC FAILURE

The above definition gives us our first subdivision of heart failure into high output and low output types. The former are relatively rare, but include such conditions as thyrotoxicosis, high fever, beri-beri heart disease (Vitamin B1 deficiency), anaemia, pregnancy, arterio-venous fistulae and intra-cardiac left to right shunts, Paget's disease of bone, prolonged severe tachycardia of any cause (e.g. supraventricular tachycardia).

You will find many classifications of heart failure in the literature, e.g. high- and low- output failure; heart failure related to an elevation of "preload" versus "afterload"; "pressure" versus "volume" overload; "systolic" versus "diastolic" failure; and "backward" and "forward" failure. In Anatomical terms we also talk of right and left heart failure, and in Pathological terms acute, sub-acute, and chronic.

In simple terms, the heart consists of a pump with one-way valves, and to work efficiently it must fill normally and not be expected to pump against an unduly high pressure/resistance.

Therefore, in general, cardiac failure (Left or Right) can result from:

1. "Volume" overload:

a) associated with valvular incompetence/regirgitation (eg. AR or MR causing increased end-diastolic LV volume).

b) From severe salt and water retention (e.g. in chronic renal failure) which causes secondary cardiac dysfunction from muscle and valve dilatation.

2. "Pressure" overload:

a) outflow valve stenosis (e.g. aortic valve stenosis).

b) increased peripheral vascular resistance (as in hypertension).

3. Dysfunction of the myocardium:

a) "systolic" dysfunction

b) "diastolic" dysfunction (see below).

We will now consider 'forward faiure.'

'Backward failure' will also be discusssed, though we will see that it is really a misnomer

IMPAIRED CARDIAC FUNCTION

Cardiac failure is not an all-or-none phenomenon. It mostly occurs as a late complication, and before that there are lesser degrees where it is important to discern the *degree* of Functional cardiac impairment. In doing so, initially we may see evidence only when the cardiovascular system is put under load, the most obvious here being exercise. In aortic valvular stenoses, there must be approx. 50% narrowing of the aortic valve diameter before there is even a pressure drop across the valve itself, and as much as a 70% narrowing before there is any detectable increased resistance to flow - mostly because such narrowing lack length, which is an important contributor to resistance.
Moreover, where there is no primary cardiac muscle dysfunction, the heart can call into play:
Compensatory mechanisms to maintain the cardiac output near normal for long periods of time viz:
1. Cardiac output can be increased by sympathetically-mediated increases in cardiac muscle contractility,
2. Cardiac dilatation and secondary increases in stroke volume (Starling's law of the heart) can compensate for the volume overloads,
3. Pressure overload like AS, PS and hypertension can be compensated, for a time at least, by secondary hypertrophy of the relevant ventricle.
This all helps compensate for a falling level of cardiac output at any given level of tissue metabolic demand, and it is only when these compensatory mechanisms fail that overall performance of the heart as a pump is impaired to any noticeable clinical degree.

Sequence of events during the course of slowly-developing heart failure. We need first analyse the consequences of impairment in the "forward" function of the heart, or the progression to what we call "forward" failure. This concept is based on fairly firm grounds as we shall see.
"Backward" failure (e.g. elevated JVP and oedema in right heart failure) is really a misnomer. Part of the explanation of what is called "backward failure" lies in understanding the mechanism of "forward" or systemic failure, which can arise from impaired right ventricular function - after all, the left can again only pump what the right delivers to it.

'FOWARD' HEART FAILURE

Arterial pressure is maintained as a balance between cardiac output and peripheral resistance, so if the former falls the latter rises. This rise in resistance is brought about primarily by a sympathetic nervous discharge which shuts down renal, splanchnic and skin blood flow, to have the effect of maintaining not only blood pressure, but blood flow to vital organs, such as the brain and heart. Release of renin and the consequent angiotensin production, as well as vasopressin release helps to compensate to maintain B.P., at least for a time. This maintenance of blood pressure, and with it blood flow to the brain and heart, may mean that there are no symptoms of cardiac dysfunction at all, at least at rest. However, on exercise or when the system is put under other load, arterial blood pressure may not be so well maintained.

So early on, there is reduced cardiac output, and maintained blood pressure and perfusion to the vital organs, albeit at the expense of perfusion of the kidneys, skin and splanchnic beds. Eventually, the reduction of renal perfusion from arteriolar constriction leads to a significant fall in GFR, and this leads to the next phase of compensation, viz. sodium and water retention. This will expand the intravascular volume, increase cardiac venous filling pressure/JVP, and so help maintain B.P. (Starlings Law of the heart). However, it will also increase total extracellular fluid volume to cause tissue oedema, especially in dependent areas like the legs.

This oedema and increased JVP is therefore not related to "backward" right heart failure at all, but is a consequence of 'forward failure'.

INCREASED VENOUS PRESSURE IN HEART FAILURE

First consider the heart as a single chamber. Then, any increased venous pressure as above will lead to cardiac dilatation. Up to a point this will be helpful, because by increasing initial cardiac fibre length, it will increase the force of ventricular contraction on the basis of Starling's Law of the heart. Thus, provided dilatation is only moderate, the fluid retention and increase in blood volume will work in a compensatory way to bring cardiac output back now somewhere towards normal. (This is why we don't aim to bring the JVP right back to normal in treating right heart failure). But, beyond a certain point, further sodium retention and cardiac dilatation may become self-defeating for several reasons.

<u>Factors impairing cardiac function in worsening faliure</u>
1. When dilatation becomes too great, we get on to a plateau of the Starling curve where further dilatation cannot further improve the strength of cardiac contraction, and may even worsen it.
2. Cardiac dilatation also brings about a problem due to the law of LaPlace, because more work is required to generate a given force when the heart is dilated (the tension that has to be overcome by the contracting ventricle is directly proportional to both the pressure within its lumen and lumen diameter).
3. Cardiac dilatation may lead to incompetence of the AV valves (mitral and tricuspid), from a combination of
a). Stretch causing AV ring dilatation and
b). Papillary muscle dysfunction as part of the ventricular muscle failure.
4. A high venous pressure also means a high left ventricular end-diastolic pressure, and this reduces effective coronary perfusion. The reduction in coronary blood flow will impair myocardial function still further.

LEFT HEART FAILURE

So far, we have accounted for the "forward" aspects of heart failure, but we need to think about why we see this more in left ventricular impairment than right. There are probably two reasons for this.

1. Most impairment of (the stronger) left heart function is only manifest under load (e.g. exercise) and not at rest until very late. Therefore, any "decompensation" only occurs intermittently, with time in between for circulatory readjustment to the status quo - any tendency for "forward" failure to occur during exercise will pass off after the exercise period is over. Only late, when all compensastory mechanisms fail and resting left ventricular function is impaired will there be any persistent forward failure to produce lasting sodium retention.
2. Even then, provided the right heart is functioning normally, there may be very little peripheral systemic oedema and JVP elevation, because even a modest increase in right atrial pressure from the initial Na+ retention will increase right ventricular end-diastolic pressure, and hence maintain the output of the normal right ventricle.
The corollary is that whilst the right heart is functioning normally, any blood volume expansion brought about by L.V. forward dysfunction will tend pumped normally by the right heart into the pulmonary circulation. And whilst this may be very effective in limiting the JVP and general venous pressure entering the right heart, it has the consequence of increasing the volume of blood in the lungs. This will increase pulmonary venous pressure to the left heart, but because left ventricular reserve is already compromised, it cannot sufficiently increase its output in response. In effect, there is a temporary imbalance in the output of the two ventricles, with the right heart output initially being greater than the left. But eventually a new equilibrium will be achieved, because the impaired L.V. will still have some ability to increase its output through the increased end-diastolic ventricular volume causing increased left ventricular output via Starling's Law of the heart.

So we can now see how so-called "backward" failure may be spurious. Older explanations were that failure of the left ventricle leads directly to an increase in left ventricular end-diastolic pressure, and therefore a "back-pressure" on the pulmonary veins, capillaries etc. But even when the heart stops beating, venous return also stops, and mean pulmonary "back pressure" does not rise much at all. By the present view, pulmonary congestion and oedema arise from fluid retention secondary to L.V. forward failure increasing right ventriclar output and driving the retained fluid into the pulmonary bed.

We are now in a position to explain some of the symptoms of "forward failure" resulting from left rather than right ventricular dysfunction.

1. Shortness of breath on exertion is a early symptom of left heart dysfunction, even before there is much in the way of overall fluid retention. According to the above, this arises because the left ventricle is unable to keep pace with the increased demand of exercise whereas that of the right heart can. This causes an imbalance between the outputs of the two ventricles during exercise, and leads to pulmonary congestion, so explaining the dyspnoea. This may well explain why there is more feeling of dyspnoea on exercise with a given degree of left compared with right heart failure.

Orthopnoea and PND are explicable on a similar basis. These symptoms occur with moderate left heart failure, when there is already an increase in circulating blood volume. This increased volume will be distributed throughout the whole vascular compartment, both systemic and pulmonary, but whilst the patient remains upright the rise in pulmonary venous pressure will be kept to a minimum by

the gravity-dependent pooling of blood in the legs. But once the patient lies flat, the situation will be altered, and where right heart function remains normal, the sequence of events is: Elevated JVP -> elevated atrial pressure -> increased R.V. end diastolic volume -> increased right heart output -> shift blood to the pulmonary capillary bed. Again, by dilating, the left heart will compensate for this by pumping better, but only at the expense of a much elevated pulmonary venous pressure, causing initial discomfort on lying flat, then shortness of breath, and finally pulmonary oedema.

This suggested mechanism of pulmonary congestion in left heart failure is important. The point is that really 'backward' failure, though an easy and convenient way of thinking, is a consequence of forward failure.

There is one sense in which backward failure does occur, viz. severe mitral valve stenosis. Here, very high left atrial pressure causes a rise in pulmonary venous pressure, with secondary hypoxia from interstitial lung congestion/oedema resulting in pulmonary arterial hypertension; and, since the right ventricle is just not built to withstand pressure overload, right heart failure ensues.

Finally we should talk not of left or right heart failure, but of degrees of dysfunction. In the same way we should not fall into the trap of using such phrases as 'left ventricular failure', unless we are sure the left ventricle is the anatomical site of the problem. Where we are not, we should use the term left heart impairment and state its degree.

PRIMARY RIGHT HEART FAILURE

Here, we will again see the evidence of forward failure, because the left ventricle cannot pump more than the right delivers to it. But, this time, the right ventricle is the one which will not be able to deal with the increase in fluid retention, so that there will be a disproportionate rise in jugular venous pressure, peripheral systemic venous pressure and peripheral oedema, compared with primary left heart failure. As R.V. function diminishes, the diastolic pressure gradient between the right atrium and the left can become the major driving force for pulmonary perfusion. In these circumstances, it is important to ensure that the pre-load on the R. ventricle (JVP) is not reduced too much (e.g. by overuse of diuretics).

ACUTE HEART FAILURE

In this situation, there is not time for sodium retention, which requires some days, and yet we often see a sudden elevation of left or right venous pressure when the corresponding side of the heart fails. The is probably related to sympathetic peripheral *veno-constriction* driving blood from a peripheral to a more central location. And, where there is left heart impairment more than right, this blood will again be driven to the pulmonary vascular bed. Eventually, this will cause left ventricular end-diastolic pressure to rise and increase left ventricular output, so that a new balance of the two ventricular

outputs ensues; but at such equilibrium the left atrial and pulmonary venous pressure will exceed the threshold for pulmonary oedema (Starling's law of capillary forces).

To illustrate the point, one ml per heart beat extra blood pumped out by the healthy right heart (say, total 70 mls/stroke) in comparison with the left (say, 69 mls/beat or a reduction of about 1.5%) would add up to 60 mls extra blood delivered to the left heart every minute; and if unlimited over an hour this would result in an extra 3.6 litres of blood within lung, with an impossibly gross increase in venous pressure! Thus even this small transient discrepancy below the detection limit of measuring cardiac output, could lead to gross changes. The other point is that because there will be an increasing left ventricular end-diastolic pressure associated with the above imbalance, the left heart will dilate, and with that stroke volume will increase (Starling's Law of the heart), so that the output of the two ventricles will soon return to equality. At that point we would not expect to see any discrepancy in output of the left heart versus the right.

"SYSTOLIC" and "DIASTOLIC" HEART FAILURE

This is an increasingly important classification, born of the pressure-volume loops which have proved so useful in studying experimental heart failure. To understand these, you must grasp the point that the curves do not relate pressure or volume changes over time, but pressure vs. volume changes per se.

Systolic failure: This is fairly easy to understand. Such failure arises when the cardiac muscle becomes poorly contractile such as in alcoholic or viral cardiomyopathy. In this model, the initial response is one of cardiac dilatation, which for a time at least can be compensatory (Starling's law of the heart), but eventually failure supervenes as discussed.

Diastolic failure: This is particularly important in the left ventricle and arises when the ventricular wall is very stiff, be it due to left ventricular hypertrophy (e.g. in systemic hypertension, aortic valve stenosis), or to myocardial ischaemia/infarction/fibrosis, infiltration of the heart (amyloidosis, haemochromatosis, sarcoidosis), constrictive pericarditis or other "restrictive cardiomyopathies". Here, because of ventricular wall stiffness, diastolic filling is limited. And since the left ventricle can only pump out what comes in, cardiac output is impaired, despite good systolic contraction. Up to a point, even this form of impaired cardiac function can be compensated, by and increase in heart rate, by ejection of a greater quantity of end ventricular diastolic blood volume (normal ejection fraction only 70%), and by atrial hypertrophy increasing ventricular filling late in diastole through more forcible atrial contraction. However, as the ventricular wall stiffens further, these compensatory mechanisms will become insufficient, and heart failure will again eventually supervene; this can occur very rapidly in these circumstances if atrial fibrillation suddenly complicates the clinical course at any time.

RATIONAL TREATMENT OF HEART FAILURE

All of the above are important if we are to treat heart failure rationally. Positive inotropic agents, such as digoxin, are used to increase the strength of cardiac muscle contraction.

Treatment of Volume overload in heart failure.
As we have seen, many of the changes such as sodium retention and increased venous pressure are, up to a certain limit, useful in compensating for the reduction in cardiac output. So the first pont is that is that we should not attempt to dry a patient out too much with diuretics to return his blood volume and venous pressure right back to normal. This is particularly true in right heart failure, because the output of the left ventricle is totally dependent on what it receives from the right, and so in this situation is highly dependent on an increased right venous filling pressure for its continued function.

On the other hand, pulmonary oedema from acute left heart failure can be life-threatening.
IV peripheral venodilators such as isosorbide mononitrate or glyceryl trinitrate can also be useful, particularly in acute left heart failure, by increasing peripheral venous capacitance and thereby reducing central venous pressure.

Treating Increased Afterload in Heart Failure. We have just seen that where cardiac "pre-load" is high,venodilators can be very effective therapeutically. In the same way, where the primary problem underlying the heart failure is an increased ventricular pressure afterload as in systemic hypertension, then systemic arteriolar dilators can be equally effective.These act mainly by reducing the pressure against which the heart has to pump, in this way reducing cardiac work. They may act more directly in aortic valve incompetence, where reduction of blood pressure lessens the functional degree of aortic regurgitation.

"Afterload" or blood pressure reduction makes good sense in circumstances where the heart failure is secondary to systemic hypertension, but in cardiac impairment from other causes, e.g. primary left ventricular dysfunction, its rationale is not so obvious, especially because at the extreme it will tend to overcome the very peripheral systemic arteriolar constriction we regard as vital for maintaining blood pressure. Nonetheless it may help. Traditionally, it is thought that blood pressure reduction in this situation helps by reducing the "pressure work" the heart has to do, so allowing it to do more volume (i.e. cardiac output) work. But if we take this view, what we are saying is that the reflex and compensatory mechanism the body sets in train following a reduction in cardiac output are not necessarily beneficial, at least when carried too far. But there is another possibility. We know that the coronary arteries and arterioles are well supplied by sympathetic nerves, and if the sympathetic discharge associated with any heart failure ever spread to cause an increased coronary vascular tone, then this would reduce coronary arterial perfusion, and we would certainly have gone beyond the realms of ventricular compensation. Because of this, it is possible that some of the improvement

with arterial vasodilators is via a reduction of coronary resistance to increase coronary bloodflow, particularly in situations of underlying myocardial ischaemia.

The angiotensin converting enzyme inhibitor drugs and angiotensin receptor blockers are particularly effective agents in reducing blood preeure/LV afterload, because the renin-angiotensin system is known to be activated in heart failure.

Of course, where afterload is increased, compensatory ventricular hypertrophy can maintain L.V. function relatively normal for long periods of time. But this has its limits. First, the hypertrophied fibres are not absolutely normal in function, and also have a rather precarious capillary surface area to myocyte volume ratio. Second, even with the hypertrophied heart, there comes a time and degree of severity when it just cannot continue to maintain normal cardiac output, even at rest, so that cardiac failure occurs. Third, in aortic valve stenosis, narrowing does not offer much resistance to flow intil about 70% reduction in valve area. But then, stenosis may not have to progress much in absolute terms to give a great increase in resistance to flow and therefore demand for cardiac work. Fourth, any hypertrophied left ventricle is stiff, so causing problems of diastolic filling, and this can become a special problem if atrial fibrillation suddenly supervenes. The'atrial kick' in then ineffective.

In the final analysis, whether we are dealing primarily with progressive pressure or volume overloads, systolic or diastolic types of ventricular dysfunction, myocardial ischaemia or otherwise, there will come a time when the heart will fail and become grossly dilated - and at that stage high end-diastolic pressures will reduce coronary (diastolic) perfusion to aggravate the situation still further.

MCQs: MECHANISMS IN DISEASE

A. MCQs on Mechanisms in Disease:

A patient presents with left heart dysfunction secondary to aortic valve incompetence. Which of the following would be characteristic?

1. A wider pulse pressure than usual at any given level of heart failure.

2. A third heart sound.

3. Concentric ventricular hypertrophy.

4. A loud first element to the second heart sound at the base.

5. A raised systemic arterial diastolic blood pressure.

6. A low pulmonary venous pressure.

7. A 10 cm increased jugular venous pressure as an early sign.

8. Systemic arteriolar dilators should be of particular benefit in improving cardiac output.

9. An early diastolic murmur at the left sternal edge.

10. The intensity of the murmur associated with aortic incompetence is a good guide to its severity.

Answers at end of Chapter

CLINICAL PROBLEM SOLVING

An interactive computerized online tutorial is available @;
www.ictscd.com (no prior http://)
A 72 year old man (1) presents with increasing shortness of breath on exertion (2) over a period of 12 months (3), a three months history of discomfort and shortness of breath on lying flat (4), and a two week history of awakening at night short of breath (5). He has noticed no palpitation (6) but for about 4 months has had occasional "tight" retrosternal chest pain on severe exertion (7), and on one or two occasions a faintness on exertion (8) - indeed on one occasion he "blacked out" completely (9). No cough, sputum or wheeze (10). No shivers or sweats (11). No weight loss or anorexia (12). Past History: No other symptoms of relevance. Social History: Married. No stresses. Nonsmoker. Occasional alcohol (13).

Examination: looks reasonably fit for age (14). Abnormalities are confined largely to his cardiovascular system (15). Pulse rate 72/min and regular (16). The pulse at the wrist has a normal downstroke (17), but the upstroke, assessed at the carotid pulse, is slow and prolonged (18). BP 120/80 mm Hg (18a). The JVP is at a vertically height of 8 cm above the manubriosternal angle (19). Both "a" and "v" waves are present (20). Trachea mid-line. Heart: a slowly thrusting apex beat (21) in the fifth left intercostal space 2 cm lateral to the midclavicular line (22). Auscultation findings: A normal first heart sound at the apex (23), with a soft second heart sound (24); the first component of the second heart sound over the base is very soft (25), and there is only a very narrow split to the second sound on inspiration (26). Added fourth heart sound at the cardiac apex (27). The murmurs heard are as follows: a loud, harsh, rough systolic murmur maximal over the base of the heart and conducted to the neck, more to the right than left (28). This murmur clearly begins after the first heart sound, becomes maximal in mid-systole (29), and runs through almost up to the first component of the second heart sound (30). On sitting the patient forward there is also a soft early diastolic

decrescendo murmur immediately following the first element of the second heart sound (31). Whilst the patient is sitting up, you note on chest examination fine, late inspiratory crepitations at both lung bases (32) as well as mild sacral oedema (33). Limbs: moderate bilateral ankle oedema (34). Temp. 37deg. C (35).

Investigations: Chest X-ray shows borderline cardiac enlargement with moderate pulmonary venous congestion (36). Blood urea and electrolytes normal (37). Haemoglobin, white cell count and platelets normal (38). Plasma albumin levels normal (39). Plasma creatinine slightly raised, and creatinine clearance at lower limit of normal for age (40).

Solving the Problem. Draw up four columns as previously outlined and solve the problem accordingly.

Graphic Solution: Available in next section as a jpeg. When viewing, centre the picture so that all 4 columns are able to be seen at the same time. The solution is available in two parts, in the sections: Problem Solution-1 and Problem Solution-2.

Diagnostic Dissertation. A Diagnostic Dissertation about the case follows. Make one yourself for comparison before viewing .

MCQs. Think about the questions following the case before turning to the graphic solution. Answers available in the final section of the chapter.

Which of the following statements is/are correct?

1. This patient shows evidence of impairment of left heart function.

2. The elevated JVP and peripheral oedema can be taken to indicate primary impairment of right ventricular function.

3. The added fourth heart sound is due to the cardiac dilatation.

4. The primary cause of this patient's problem is a "pressure" rather than a "volume" overload.

5. The diastolic murmur is classical of mitral valve incompetence.

6. He has evidence of haemodynamically significant aortic stenosis.

7. The thrust over the cardiac apex suggests right ventricular hypertrophy.

8. The oedema is most likely due to hypoproteinaema.

9. The narrow splitting of the second heart sound is most likely related to a right bundle branch cardiac conduction block.

10. Syncope with exercise is mpst likely related to a blood pressure fall on effort.

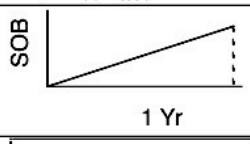
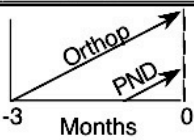
11. The length of this particular systolic murmur is of value in assessing the haemodynamic significance of the underlying valvular lesion.

12. Arteriolar vasodilators would be very useful in treating this particular patient's major haemodynamic problem.

13. This patient would likely benefit from replacement of the mitral valve.

PROBLEM SOLUTION-1

Heart Failure Problem

Where ?	What?	How ?	Why ?
I		(2) SOB on exertion	(1) Male 72 yrs.
? System	(3) Chronic progressive	SOB on effort	? Cause
II		SOB - as above (4) Orthop. 3/12 (5) P.N.D. Recent	?
Prob. Left Heart	Sub - acute on chronic	Impaired Function	? Reason for sub acute worsening
III	4 /12= Sub acute (11) No shivers / sweats. Prob. non-inflamm. (12) No wt. loss/ anorexia. Prob. non-neoplastic.	(6) No palpitation → (7 Chest tightness on severe effort ? Effort Angina ⇑ ? Structural coronary disease or functional probs. with coronary perfusion. (8) Faintness on effort ? 2° to BP ↓ ? ↓ C.O. on effort. (9) Effort syncope. (10) No cough, sputum, wheeze. Thus, prob. not resp. SOB.	⇑ Dysrhythmia unlikely cause. (13) Non smoker. Non drinker. No stresses.
Prob. not respiratory ←			
Cardio-Vasc. Sys	Sub - acute on chronic	BP ↓ 2° to ↓ C.O.on effort could explain all of above (III).	No aetiol clues yet.
(IV) Exam.		(16) No dysrhythmia. (17) Pulse downstroke normal. (? no significant A.I.)† (18) ↓ Carotid ↑ - suggests A.S.** (18a) BP Low - Consistent with A.S. ? Aortic valve stenosis (19) JVP ↑ - ? 2° to RHF (20) Normal "a" wave - No AF. (21) Apical thrust - ? LVH 2° to AS.** (22) Mild cardiac dilat^{n}.† (23) S1 normal. (24) Soft S2 - ? 2° to AS.* (25) Soft A2 - ? 2° to AS.* (26) Delayed A2 - ?2° to AS.** (27) 4th HS - No AF. - Prob. 2° to LVH.* (28) Aortic outflow murmur. (29) Aortic outflow murmur. (30) Long mid - sys murmur** ** /*= significant A.S.	
Aortic Valve. (Stenosis) ←			

PROBLEM SOLUTION-2

Heart Failure Problem (Continued)

Where?	What?	How?	Why?
Interim Conclusion 1.			
Aortic Valve Stenosis	Chronic progressive	AS. Haemodynamically significant 2° LVH., angina & ↓ C.O. on effort.	?Cause.
	(35) T° = N. Prob. non-inflamm.	(31) A. I. murmur. (32) Basal creps. - ? LVF (33) Sacral oedema. - ? 2° RHF.	
Interim Conclusion 2.			
Aortic Valve (Incomp.)	No evidence of "valulitis"	1. Incompetence † = Prob. mild. 2. Evidence of 2° R & LHF.	?Cause.

FINAL DIAGNOSIS

Anatomic Diagnos	Pathological Diagnosis	Functional Diagnosis	Aetiological Diagnosis
1. AORTIC VALVE	No evidence of inflamm or neoplasia. CHRONIC ? DEGENERATIVE.	HAEMODYNAMICALLY SIGNIFICANT STENOSIS. With 2° ↓ cardiac output & BP. (Worse on effort). ⇓ Effort angina / syncope. 2° LVH → RHF	NO OBVIOUS BKGD AETIOL. ?DEGENERATIVE.
AORTIC VALVE	SUB-ACUTE DETERIORATION.	INCOMPETENCE -probably significant (normal pulse downstroke, but some cardiac dilat^{n} -? 2° to AI. (although could be 2° to decompensated LVH from AS).	?WHY.

DIAGNOSTIC DISSERTATION

1. Chronic background condition: This patient has a 12 month history of increasing SOB which subsequent events suggest to be secondary to aortic valve pathology, predominantly stenosis. Though the symptoms are of only 1 year's duration, we know that valvular stenosis can progress silently in clinical terms initially - e.g., up to 70% narrowing before any symptoms - so the actual pathological process could be very chronic.
2. Recent sub-acute deterioration: No obvious reason - no dysrhythmia/A.F. Not told about drug history, therefore ? beta blocker (negative inotrope) or sodium retaining medications (?NSAID for arthritis) added at that time. ? any alternative medications. Further history needed. No evidence of inflammation, no signs of bacterial endocarditis. but think about this as a possible reason for deterioration.

3. Aortic valve stenosis is haemodynamically significant:

a) Angina of effort - ? secondary to decreased (diastolic) BP on exercise causing decreased diastolic coronary perfusion.

b) Syncope of effort - ? secondary to marked decreased BP on effort causing inadequate brain perfusion.

c) Secondary LVH progressing to LVF.

d) RHF probably secondary to LVF via increased pulmonary cap. pressure -> pulmonary interstitial/ alveolar oedema> hypoxaemia -> pulmonary arteriolar constriction -> increased pulmonary arteriolar pressure -> RV pressure overload -> RHF .

4. Aortic valve incompetence - relatively mild clinically.

5. Background Aetiology - Unclear, apart fro increased 'wear and tear' on any valve with age, particularly one under high pressure and flow. Perhaps his systemic arterial pressure, now normal, was high earlier in life to increase shear stress across the valve.

Investigations: Not dealt with in the graphic solution to this case. They add little except to say that the sacral and ankle swelling is clearly not related to any impariment of glomerular filtration, nor to any fall in plasma albumin.

ANSWERS TO MCQs

Answers to MCQs:

Mechanisms in Disease: 1, 2, correct. 3-6 incorrect

Problem Solving case MCQs: 1, 4, 6, 10, 11 correct. All others incorrect.