CHAPTER 5 - CHEST PAIN WITH SPECIAL REFERENCE TO MYOCARDIAL ISCHAEMIA

1. ANATOMICAL DIAGNOSIS

In the respiratory system, we saw that pain was an unusual feature, except when the parietal pleura was involved. But pain may arise from other somatic structures in the chest, particularly the rib cage including the rib joints, muscles and the nerve roots themselves (as in nerve root compression at the level of the inter-vertebral foraminae; and Herpes Zoster or shingles). The diaphragm is also a somatic structure, innervated by the phrenic nerve (C3, C4) so that it too may give rise to (referred) pain felt in those dermatome distributions (shoulder-tip), when inflammed or injured.

Diseases of the lung itself (or visceral pleura) do not generally give rise to pain. However structures in the mediastinum can. The upper oesophagus, pharynx, trachea, and larynx have afferent vagal fibres which can mediate pain, and the other mediastinal structures, the heart, great vessels, the (parietal) pericardium, and the lower oesophagus are also supplied by (sympathetic) afferent fibres. As a general rule, the position in the spinal cord to which visceral afferent fibres pass from each internal organ depends on the segment of the body from which that organ developed embryologically. For example, the heart originates in the neck and upper thorax so its visceral pain fibres enter the cord all the way from C2 to T5.

In any patient with chest pain, we use our hierarchic approach to diagnosis, and first try to answer the BROAD question of whether we are dealing with ANY cause of mediastinal chest pain, or whether it is non-mediastinal (e.g. nerve root, chest wall, parietal pleura etc). In this respect, most pains originating from mediastinal structures have a central retro-sternal component. But before we can further dissect mediastinal pains, we have to consider the nature and mechanism of referred pain.

1. Referred Pain

Afferent nerve stimulation from mediastinal structures does not produce pain localised to that area alone (the organ concerned does not normally need to signal its presence at all). What it does produce is pain referred to the corresponding dermatome nerve distribution of the skin. We don't know the precise mechanism of referred pain, but the following is probably important. The ganglia cells of both sympathetic and somatic afferent nerve fibres are located in the most posterior aspect of the dorsal root ganglion expansion. Their proximity, and the fact that at least some of them synapse with common neurones in the posterior spinal horn means that when visceral fibres are stimulated, pain signals are conducted centrally in part through neurons that conduct pain from the skin, so that the patient has the feeling that the sensation actually originated in the skin.
This explains the **distribution of referred pain from mediastinal structures**. Thus, the heart, being innervated by sympathetic afferent fibres from C2 to T5 (bilaterally) gives rise to pain that can be felt in any of these areas, differing in each individual due not only to an involvement of different areas of the heart, but to variation in sympathetic innervation in different patients. The characteristic pain distribution is central retrosternal radiating to the neck, the lower jaws, and down the arms, particularly the left arm, usually only as far as the wrist, but sometimes involving the fingers. **Upper oesophageal pain** is usually referred to the pharynx, lower neck or upper midline chest. **Lower oesophageal pain** is usually felt lower in the midline sternal region, but can radiate to the (left) upper chest, shoulder, and even the left upper arm. It rarely radiates further down the arm than this. **Parietal pericardial pain** is usually centred over the lower sternum and adjacent left chest but may also radiate to the left shoulder tip, probably because of the involvement of its diaphragmatic portion, and therefore involvement of C3, C4 via phrenic somatic afferent fibres. The **great vessels** may also produce pain, particularly the aorta. The latter gives rise to pain similar in distribution to cardiac pain, but tends to be more localized to the chest, and (being a posterior structure) is more inclined to radiate to the back. In **thoracic aortic dissection**, the classical history is of pain beginning high retrosternally, but travelling down towards the epigastric area as the dissection spreads distally.

The **stimuli for pain** is different in different mediastinal structures. Thus, we have already seen that the trachea will give rise to most discomfort when inflamed. This is true also of the pericardium, but the great vessels respond to stretch, as in aortic dissection (and in that instance, as the dissection travels distally, the pain may move from the upper to the lower chest). Cardiac pain is only evoked by myocardial ischaemia, i.e. when the demand for blood supply in any area outstrips supply (see further below). The oesophagus characteristically produces pain on swallowing, especially in relation to spasm, either that initiated at the site of an obstruction, or precipitated by hot or cold fluids. The quality of referred pain is, as elsewhere, very useful in determining its anatomical nature, because this tends to vary from structure to structure. However, there is no way of predicting precisely the sorts of pain that occur, so they have to be learned by experience. The characteristic pain of myocardial ischaemia/infarction is a "heavy", "gripping", or "vice-like", "crushing" central retro-sternal pain, radiating to the neck and left arm. The pain of oesophageal spasm is not unlike this (although without radiation). Pericardial pain is not usually described in such graphic terms, but tends to be of sharper or more "stabbing" quality. Lower oesophageal pain is usually of burning nature, particularly in reflux oesophagitis. Tracheal pain tends to be a vague discomfort, burning or 'raw' when associated with inflammation.

**Aggravating and Relieving Factors** are of much value. Thus, pain of myocardial ischaemia is typically related to effort and relieved (rapidly) by rest. Tracheal pain is aggravated by coughing. Oesophageal spasm pain is characteristically aggravated or precipitated by swallowing fluids such as ones which are either too hot or too cold. Pain associated with reflux oesophagitis (due to incompetence of the cardiac sphincter) is typically aggravated by posture, particularly lying flat and bending down. Pericardial pain due to pericarditis is also aggravated by different postures, no doubt because the associated pericardial effusion is shifted by such posturing to allow the two inflamed surfaces to grate together more, or less, over particular parts of the cardiac surface. Characteristically, pericardial pain is worse on lying flat and eased by sitting forward.
Associated phenomena - helpful in localizing chest pain to particular mediastinal situations. Visceral pain in general has a definite quality of "nastiness", and is often associated with nausea, vomiting, sweating, tachycardia, i.e. reflex efferent autonomic phenomena, perhaps not surprisingly in view of the stimulation of autonomic afferent pathways. Myocardial ischaemia is especially associated with these features, and some patients even have a feeling of "impending doom" as well.

In the abdominal viscera there are other aspects of referred pain, including (spinal reflex) guarding of associated abdominal muscles.

The above should suffice as a basis for the hierarchic dissection of the anatomical basis of referred mediastinal pain. You will see why we need to know all about the site, distribution, nature, radiation, aggravating and relieving factors etc. to make the anatomical diagnosis, because there is much overlap between pain arising from these structures, especially in distribution and radiation. In this respect the functional aspects, i.e. the aggravating and relieving factors, are often paramount in allowing us to pinpoint the underlying anatomical area or organ involved.

So far, we have mainly discussed history findings, but examination findings can be useful as well. Thus, during episodes of myocardial ischaemia or infarction there may be tachycardia and an added fourth heart sound (because the ischaemic area of myocardium becomes stiff). Pericardial friction rubs may be heard in patients with pericarditis. In suspected aortic dissection, feel all pulses and look closely for (left-sided) pleural "fluid" (blood) because the dissecting aorta often leaks as it traverses the thorax (the descending thoracic aorta lies to the left of the midline). Diagnosis can therefore be added to very greatly by pleural fluid aspiration. In any acute situation blood-stained pleural fluid means either trauma, pulmonary embolism, or dissecting aneurysm; (in the more chronic situation it would raise the suspicion of neoplasia). Cardiovascular system examination in chronic constrictive pericarditis may also often reveal variable intensity of the first heart sound, an elevated jugular venous pressure with a rapid "y" descent, and an early third heart sound or "pericardial knock"; also Kussmaul's sign in the neck (increase rather than decrease in JVP on inspiration), together with pulsus paradoxus. These latter signs also occur in acute pericardial tamponade.

2. CLINICAL PATHOLOGICAL DIAGNOSIS.

What is the general nature of the lesion?

Very sudden onset conditions are either related to mechanical events, or rupture or obstruction of hollow tubes. An abrupt yet reversible event suggests obstruction more than rupture, while a rapid onset yet persistent event could be due to either continuing obstruction (as in acute coronary thrombosis precipitating myocardial infarction) or rupture (as in aortic dissection).
As usual fever suggests an inflammatory condition, which might be confirmed in the case of the respiratory system by examination of the sputum. Any pleural fluid should be examined macroscopically, microscopically and by culture to aid to diagnosis, not only in inflammation, but trauma (red cells), rupture of blood vessels (red cells), neoplasia (malignant cells), and pulmonary infarction.

3. FUNCTIONAL DIAGNOSIS

The functional diagnosis should always include the degree of impairment of the primary organ function, and also any sequela and complications which may follow - for example, in the case of acute myocardial infarction, atrial fibrillation, ventricular dysrhythmias, etc.

The functional effects of a process on the patient are important in localizing its anatomical site. Thus, in making an anatomical diagnosis in a patient with obscure chest pain, the presence of left heart failure &/or atrial fibrillation would lend support to a cardiac anatomical site for the problem.

4. AETIOLOGICAL DIAGNOSIS.

Look for underlying "risk factors" (predisposing factors) in any patient with myocardial ischaemia or infarction (hypertension, cigarette smoking, diabetes, oral contraceptive therapy, obesity, stress, high LDL cholesterol, etc.). But this "risk factor" concept should be thought of in all patients when the primary clinical pathology, anatomy etc. are established i.e. we should always try to establish the underlying Aetiological diagnosis. Usually, we do this from the background diagnosis of family history, drug history, past history, social history, personal history, etc. In a patient with an acute myocardial ischaemic syndrome, look out not just for (long-term) risk factors for atherosclerosis, but more short-term factors which may have precipitated the attack. When all else fails, ask the patient!!

CORONARY BLOOD FLOW

Understanding cardiac conditions first requires a knowledge of coronary blood flow regulation. Normally this is closely auto-regulated, and determined by the amount of cardiac work. But there are sympathetic nerve fibres, particularly to the larger coronary arteries which can come into play to modulate and even over-ride this auto-regulation from time to time. Coronary blood flow is mostly diastolic (85%) because of the high intra-myocardial pressure during systole. It is therefore inversely proportional to heart rate (systole occupies a fairly fixed time). The sub-endocardium normally operates more towards the limit of coronary flow reserve and is therefore first to suffer when there is any general fall in coronary blood flow. Coronary flow reserve and the associated high capacity for autoregulation of flow is not completely understood. One factor is that
not all of the very dense capillary network (controlled by pre-capillary sphincters) are open at any one time, but may become so through local reduction of pO2 or accumulation of local metabolites in the arteriolar/pre-capillary sphincter area; release of endothelium-derived releasing factor (Nitric oxide, NO) may also be play a role. Because most coronary blood flow is diastolic, another important factor in perfusion is the pressure within the ventricles during diastole. This will rise with heart failure (increased end-diastolic pressure) with the result that effective perfusion pressure for any given level of arterial blood pressure will tend to fall. Perfusion is also limited by increased venous (coronary sinus) back pressure, and both factors may contribute to the vicious circle of accelerating cardiac failure in its terminal stages.

The heart (300 gram weight) has a high coronary flow (250 ml/min), yet does not normally have a large reserve of fuel, and approximately 75% of oxygen is extracted in a single passage of blood through it (arterial blood has 20 ml/100 ml; coronary sinus blood, with pO2 approx. 20 mm Hg, has only 5-6 ml 02 per 100 ml blood). This is much more than the usual 25% extraction of most tissues; cardiac tissue thus has a high metabolic demand, and often must work aerobically (it can use lactate, glucose and fatty acids). Moreover, the exercising heart demands a great increase in coronary blood flow, and to achieve this in the face of a reduced diastolic perfusion time (increased heart rate) requires as much as a 30-fold decrease in coronary vascular resistance. This blood flow increase is made possible by the extremely dense microvascular network in the heart.

UNDERSTANDING ANGINA

Angina arises when there is either an increase in demand for oxygen and fuel because of increased cardiac work, or a reduction in blood supply.

1. "Demand" Angina. This is where the angina is related to effort (and other types of increased heart work), in a fairly fixed relationship. It is said most commonly to be due to local point(s) of fixed atherosclerotic coronary artery narrowing causing blood flow to be unable to keep pace with the increasing demand of the exercising heart muscle, so that ischaemia here is relative. (Occasional patients with 'variant' angina have clear coronary spasm as the cause of their exercised-induced angina - see below). This type of angina is also seen in patients with severe aortic valve stenosis, even without coronary atherosclerosis, because coronary blood pressure and flow are critically low in this situation, even at rest.

2. "Supply" Angina. Suspect this where there is no fixed relationship of angina to effort, where emotion induces it, and particularly where it comes entirely out of the blue, even when the patient is at rest (at least physically, if not mentally). Hence the importance of taking a detailed history in all patients to determine whether or not angina occurs regularly in a consistent relation of effort (e.g. walking up a particular hill), or perhaps otherwise comes on at times of psychological stress.
Characteristically, this less common 'reduced supply' form of chronic angina occurs in younger patients, particularly females, and is due to coronary artery spasm, often focal. This may well be at least part of the explanation of angina occurring in relation to emotion (although many believe that increasing cardiac work associated with the more rapid heart rate etc. is more important there). It is also thought to be the explanation of much variable angina, the classic form of which is so-called "variant" or "Prinzmetal" angina.

Such coronary artery spasm is probably related to sympathetic stimulation, although pharmacological investigation of this has been confused by two factors, firstly the difficulty of finding good experimental animal models, and secondly the rather non-selective pharmacological properties of some of the agents used to induce spasm, for example ergonovine (an alpha-agonist but also a serotonin agonist).

Some patients with variant angina have angina of effort, yet a detailed history reveals that this does not regularly occur, suggesting that coronary spasm may even occur during exercise in this situation. This may seem peculiar but, as we have seen already, the coronary arteries are richly supplied by sympathetic fibres, and during exercise there is reflex sympathetic discharge to many of the viscera, particularly the skin, splanchnic and renal beds to shut down their blood flow and divert it in response to the high demands of the exercising skeletal muscle. This does not typically include the brain, but sympathetic constriction of coronary arteries can limit the potential increase in coronary blood flow by as much as 30% in severe exercise, even in normal individuals (Stone 1983, ref 1), and presumably more so in those patients with variant angina prone to develop angina during exercise.

3. "Mixed Demand" and "Supply" angina

It is remarkable how it sometimes takes a clinical scientific observation to alter clinical approach to history taking. We all tend to see disease in a preconceived way, particularly if we are "pattern-recognition-ers." Hence our view of angina mechanisms determines the sort of history we obtain. It was because of this that the term "angina of effort" was equated with "stable angina". But now that that there is awareness of possible variation in coronary artery tone, and with it, coronary blood flow, Maseri in particular has pointed out that even so-called "stable angina" is often not nearly as stable as a superficial history would suggest. Indeed in the relatively early stages of much angina, even angina of effort, there is usually some variable component, as evidenced by so-called "walk-through" angina, and the observation that a preliminary warm-up period can improve "angina" distance. Close history taking will also often reveal that angina varies during the day, sometimes being worse early in the morning, and sometimes in the evening, particularly after meals; and sometimes also very dependent on the patient's state of mind.

Particularly interesting is that, on coronary angiography, coronary artery constriction at sites of atherosclerotic coronary narrowing is not infrequently observed during exercise even when patients have "stable" chronic angina. This may explain the above variability, i.e. that there is a fixed element of angina due to an atherosclerotic plaque limiting blood supply in the face of increased myocardial
demand, but also a variable element due to a variably super-imposed arterial tone at the atherosclerotic site. From clinical experience, the variable aspect of (exercise or other) chronic angina is more prominent early on during its course, and there tends to be less variability later on. However, always be on the look out for it, because treatment is quite different, with "demand" angina being improved more by reducing heart work (e.g. with beta blockade), and "supply" or "vasospastic" angina being best treated by agents such as the nitrates and calcium antagonists which reduce coronary artery muscle tone.

The above can be taken a step further, as I have suggested elsewhere, as follows: Because any focal coronary artery spasm is usually seen at the site of atherosclerosis, most think that the atherosclerotic plaque predisposes that site to spasm (possibly through a diminished local endothelial production of vasodilators). On the other hand, because we tend to see variant forms in the relatively young, and more fixed forms in older patients, I take the view that coronary artery constriction, frequently very focal along arterial length, may actually be the precursor to atheroma itself. By producing an episodic short focal narrowing, a segmental coronary constriction that reduces the lumen to, say, half its normal diameter, may not offer much increase in resistance to blood flow. This is because resistance depends on length and because any tendency to reduce coronary flow from local arterial constriction will be overcome more distally by the normal arteriolar auto-regulation of blood flow. In that event, volume flow through the area of coronary constriction would remain approximately constant, and so Poiseuille's law (which only holds for very long uniform narrowings) would not apply. Indeed, quite the reverse, with Bernouilli's law being more applicable, whereby there would be a (square power) increase in the velocity of blood flow through any such narrowing, and one now not inversely rather than directly proportional to reduced diameter.

Thus a 50% reduction in diameter from focal coronary "spasm" would produce a four-fold increase in focal blood flow velocity through the constricted point. And, since local wall shearing stress at the point of constriction is dependent on velocity (in some formulae, on velocity squared), enormous shear stress forces could be placed upon the endothelium at that point, perhaps enough to cause endothelial denudation (see Gertz et al. 1981). And we know that chronic repetitive endothelial injury is a precursor for human atherosclerosis. Just how this comes about is debated, particularly to cause focal atheroma. Normal mechanical forces acting in the circulation have been looked at very carefully as one obvious potential candidate, but such haemodynamic forces just do not seem sufficient to cause endothelial damage. However, the situation might be very different indeed in the case of the (focal) coronary arterial constrictions outlined above.

In this way, repetitive, even subclinical, focal coronary constriction might gradually produce endothelial damage and allow atheroma to build up slowly, so that we might not be surprised to see the whole spectrum of myocardial ischaemia going through an initial phase of asymptomatic atheroma and variable angina, right through to fixed angina at the end-stage.

It is of interest to take this line of reasoning further. Once atherosclerotic plaques become established, they develop a rich vasa vasorum blood supply, almost entirely from the adventitia, so
that any severe episode of arterial spasm at that coronary point would result in this blood supply being 'strangled' by its media coat muscular contraction. Plaque ischaemia would result, and if the constriction lasted long enough, plaque necrosis would lead to endothelial denudation, sub-intimal haemorrhage, and plaque rupture and superimposed thrombosis. Such plaque damage is characteristic acute coronary syndromes. Hence what was initially a potentially reversible episode of myocardial ischaemia from coronary artery constriction could lead to less-reversible "unstable" angina, or even to irreversible myocardial infarction from coronary thrombosis. It must, of course, be said that the pathogenesis of atherosclerosis progression and acute myocardial ischaemic syndromes remains controversial, with acute coronary events being thought due to mechanical coronary artery plaque rupture. I find this line of thinking somewhat odd, and poorly supported

**MYOCARDIAL INFARCTION**

The anatomical diagnosis here is given by the same features as angina, and the real distinction clinically between an infarct and an episode of reversible myocardial ischaemia relates, as we would expect, to the time-intensity relationships of the pain, particularly its duration. In the case of the heart, 3-4 hours is usually taken as the cut-off time of pain separating reversible ischaemia from irreversible infarction. However, this is a very broad generalisation, partly determined by some patients having less complete coronary spasm and/or thrombosis, so allowing enough blood through to maintain muscle viability. Occasionally, even in this situation, some myocardial damage may occur (as manifest by a transient rise of troponin) and yet be reversible.

In terms of pathophysiology, we know that within an hour or two of the onset of pain, most patients with transmural infarction have coronary thrombosis, but the question we should ask is: 'What triggered this event?' In my view, it is likely that coronary spasm initiates plaque ischaemia and endothelial cell damage, and that this precipitates acute coronary thrombosis, as discussed above. Certainly this has been shown experimentally (Gertz et al. 1981), though again, it is a controversial concept in clinical myocardial infarction. The most widely held current view is that "spontaneous" fissuring or "cracking" of a susceptible (lipid-laden) plaque precipitates the series of events leading to plaque complications and superimposed thrombosis. Still, by our categories of diagnosis, we can and should still ask: 'Why did this occur when it did?'

**General points.**

We used to look upon myocardial infarction as an all or none phenomenon - an elevation of cardiac enzymes and ECG change being its hallmarks. Organs vary in the time they can tolerate total ischaemia. The neurones are exquisitely dependent on aerobic metabolism and extremely sensitive to oxygen and glucose lack. Correspondingly, cessation of blood supply is tolerated for only a few minutes. By contrast, in the skin, ischaemia may be tolerated for many hours. Some organs,
particularly those with a double blood supply like the lung and liver, are relatively protected against infarction. The heart, which functions only aerobically, tolerates ischaemia for up to 3-4 hours.

We now know that we should think of myocardial infarction as an evolving event, because:

(i) Though ECG and blood enzyme changes occur within an hour of coronary artery ligation in the experimental animal, subsequent release of the ligature with reperfusion of the area can result in salvage of a large area of what was considered to be totally infarcted. Also

(ii) There is evidence that some drugs may be able to limit myocardial infarct size. More importantly, there is now evidence that early (less than 4 hours) lysis of a coronary thrombus with IV tissue plasminogen activator can limit infarct size in man. Thus, we should regard heart attacks really as "myocardial infarctions in evolution." This is important for therapy, because it means that when a patient presents to hospital, they must be viewed as a medical emergency, i.e. a situation where quick action is needed to salvage as much myocardium from infarction as possible, even where markers like troponin are elevated.

(iii) Effect can sometimes aggravate cause in biology. Thus in evolving myocardial infarction, pain may reflexly activate the sympathetics and further aggravate coronary constriction; as may thromboxane A2, etc. liberated from the coronary thrombi.

PROBLEM SOLVING & MCQs

A. Mechanisms in Disease

A patient presents with a diagnosis of episodic vasospastic (variant) angina. Which of the following would be characteristic?

1. Sharp left-sided chest pain radiating to the back, aggravated by changes in posture.

2. The pain would characteristically be aggravated by lying flat.

3. Any relationship between the pain and effort would typically be constant during the day.

4. Angina occurring at rest.

5. Severe pain in reversible attacks could last for 1 hour.
6. Rationally, and in practice, we would expect to be able to reduce the number and severity of attacks more with calcium antagonists than beta blockers.

7. ECG ST-segment elevation may occur during pain if there is transmural ischaemia.

B. CLINICAL PROBLEM-SOLVING

A 61 year old (1) hypertensive (2) man (3) was admitted to hospital after sudden onset (4) of a severe (5) retrosternal chest pain (6) radiating through to the back (7). When the pain began it was high retrosternal (8) but by the time of admission it had extended to also involve the lower retrosternal area as well (9), even down as far as the epigastrium (10). There was no pain in the neck, jaws or arms (11). The pain was said to be "searing" in nature (12). There were no particular aggravating or relieving factors (13), but there was associated nausea, sweating and pallor (14). No palpitations or shortness of breath (15) and no other complaints on direct questioning (16).

Past history included hypertension (17) for about 15 years (18), treated on and off, but not for the last two years. Also a family history of hypertension (19). Social History: the patient was a "moderately heavy" smoker of 20 cigarettes per day (20), but drank only "occasional" alcohol (21), and had no stresses or worries (22). Drugs on admission - nil.

Examination revealed a cold, pale, "shocked" man (23) in obvious distress with pain (24), anxious and sweating freely (25). Hands cold, pale and clammy (26). Radial pulse rate 130/minute (27), regular (28), but of "thready" character (29). Blood pressure 90/60 mm Hg (30) in the right arm, 70/40 mm Hg (31) in the left. JVP not raised (32). Carotid pulses relatively weak (33), especially the left (34). No carotid bruits (35). Heart not enlarged (36) but there was a heaving apical cardiac impulse (37). Second heart sound loud at the apex (38). First element of the second heart sound also loud at the base (39). Soft added apical fourth heart sound (40); no murmurs over the precordium (41), neck (42) or epigastrium (43). Femoral pulses equal but weak (44); slight delay of both femoral pulses compared with L. radial (45). No crepitations at the lung bases (46), but the left base very dull to percussion with absent breath sounds (47). Patient unable to pass urine for testing (had not passed urine for six hours) (48). No supra-pubic dullness to percussion (49), rectal examination NAD; in particular, prostate normal (50). Bladder catheterisation - no urine obtained (51). Optic fundi - some narrowing and irregularity of retinal arterioles with some arterio-venous "nipping" (52).

Investigations: ECG showed a deep S wave in leads V1 to V3 and a large R wave in V4 to V6 (53), with ST segment depression (54), particularly marked over the antero-lateral leads I, AVL, V4-6 (55). Cardiac enzymes normal 3, 12 and 24 hours after onset of pain (56).

The patient was transfused with blood (1 litre) over a period of 30 minutes, until the jugular venous pressure began to rise just above normal (3 cm vertical elevation above manubrio-sternal angle) (57).
He was also given the beta agonist isoprenaline to stimulate cardiac contraction with very little improvement in blood pressure as measured from either arm (58). One hour after admission to hospital he began to complain of blurring of vision in both visual fields (59).

Two hours after admission to hospital, he suddenly collapsed (60), when examination revealed an undetectable blood pressure (61), no detectable cardiac impulse, quiet heart sounds, and a 10 cm elevated jugular venous pressure, more marked during inspiration (62). Chest signs remained unchanged (63).

The patient was now in extremis.

Solving the Problem. Now draw up your usual four columns (widest for "How?" column), and work through to a solution of this problem linking inferences leading to like conclusions.

Points to note.

1. Connecting links should be made, as usual, between observations leading to like conclusions. Many of these have been deliberately left in the graphic solution in the next section(s). This far into the tutorial system, you should be able to do them yourself. Therefore, draw arrows making these connections in the usual way.

2. This is a comparatively long case, so you will need to make many interim conclusions. These will then form the basis of the overall final diagnosis.

Then make a final overall diagnosis before answering the MCQs below.

Graphic Solution: Available in next section(s) as a series of graphics, including interim conclusions. When viewing, centre the picture so that all 4 columns can be seen at the same time. You may wish to consult the appropriate graphic individually after solving each section.

Before looking at the final graphic conclusion, answer the following MCQs.

Answers at in final section of this chapter

WHICH OF THE FOLLOWING ABOUT THE PATIENT'S CONDITION ON ADMISSION TO HOSPITAL IS/ARE CORRECT?

1. The likely diagnosis is acute myocardial infarction.

2. The signs at the left base are characteristic of pulmonary consolidation.

3. There is normally a discrepancy of blood pressure between the right and left arms of this degree.
4. There is evidence consistent with an increased left ventricular systolic pressure.

5. There is evidence consistent with an elevated blood pressure in the proximal (ascending) aorta.

6. Left pleural aspiration is likely to be helpful in diagnosis.

7. The arterial pressure in the legs is likely to be less than that in the arms.

8. Bladder neck obstruction is the likely cause of this man's anuria.

9. He has ECG evidence suggesting left ventricular hypertrophy.

10. He has ECG evidence consistent with transmural anterior myocardial ischaemia.

11. The administration of isoprenaline was not a rational treatment.

12. There are signs that the hypertension has caused chronic damage to at least his small blood vessels.

Regarding the episode of visual disturbance occurring an hour after admission, which of the following is/are likely to be correct?

13. There is evidence of dysfunction of the left fronto-parietal cortex.

14. The arterial supply relevant to the visual problem is the vertebro-basilar circulation.

Regarding the final episode, which of the following is/are likely to be correct:

15. The clinical findings are best explained by rupture of the cardiac interventricular septum.

16. Intravenous activated tissue plasminogen activator should be given immediately.
## PROBLEM SOLUTION: Pt.1.

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<td>4. Sudden onset, = Hyper-acute.</td>
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<td>quality or tendency to ‘travel’.</td>
<td>22. No apparent bkgd. stresses or medications.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>14. Assoc. nausea, sweating &amp; pallor. = ?2o to intensity of pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>?2o to visceral origin of pain,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>or ?both.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>15. No palpitations =</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no dysrhythmia No SOB. = no gross L heart failure, &amp;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>no gross resp. involvement.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>16. No other complaints =</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>No other clue to mediastinal structure involved at this stage, incl.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no dysphagia (oesophageal).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no hoarseness (rec. laryngeal nerve)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no aggravation of pain by position to suggest pericardial origin.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no wheeze or cough to suggest tracheal/bronchial involvement.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? no hiccoughs (phrenic nerve).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

### Interim Conclusion 1.

<table>
<thead>
<tr>
<th>Mediastinal.</th>
<th>Recent severe hyperacute episode.</th>
<th>Severe pain, extending down as far as epig. over time.</th>
<th>? Why.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spreading,</td>
<td>Nausea sweating and pallor prob. 2o to severe, visceral nature of pain.</td>
<td>Bkgd. long-term H/T, prob. poorly controlled.</td>
<td></td>
</tr>
</tbody>
</table>
**PROBLEM SOLUTION: Pt. 2.**

### Part 2: Exam. Findings

|--------|-------|------|------|
| Hyperacute | Cold, pale “shocked” = | **Peripheral circ. shutdown.**
- see above. | ? 2º to ↓ cardiac output.
- ? result of 1º ↓ in cardiac funct. ?
- ? 2º (internal) blood loss. |
| 24 | Distress with pain = | **Severe pain.** |
| 25 | Anxious & sweating. | — prob. 2º to severe pain. |
| 26 | Hands cold, pale = | **Periph. vasc. constrict.**
- sweaty hands =
- sympathetic hyperactivity (reflex). |
| 27 | Sinus tachycardia, 130/min. | **Low periph. pulse volume/pressure.** |
| 29 | ‘Thready” pulse = |  |
| 30 | R. arm BP = 90/60. |  |
| 31 | L. arm BP = 70/40., i.e., | Drop in BP. between
(R.) brachio-cephalic art. trunk, &L. common carotid artery |
| 32 | JVP not raised. = | **Partial obstruction of aortic arch.**
No R. Heart Failure,
No Blood Vol. overload
or ECF. fluid retention. |
| 33 | Both carotid pulses weak |  |
| 34 | L. carotid weaker than R. | This + 30 / 31. = |
| 35 | No carotid bruits = | **Cardiac pressure afterload.**
No notable carotid art. narrowing. |
| 36 | Heart not enlarged |  |
| 37 | “Heaving” cardiac apical impulse = | No evid. of cardiac vol. overload. |
| 38 | 2nd. heart sound loud at apex. = | **Loud aortic 2nd. sound.** |
| 39 | 1st. element of 2nd. heart sound loud at base, again = | **Loud aortic valve closure.** |
| 40 | Soft apical S4 = 22º to I.V. afterload. |  |
| 41 | No cardiac murmurs. |  |
| 42 | No neck bruits. (see also 35.) |  |

---

**Aortic arch.**

\[\Rightarrow \text{Partial obstruction} \Rightarrow\]

**Extending (?? spreading) from at least proximal to distal arch.**

\[\Rightarrow \text{Partial obstruction} \Rightarrow\]

**But —**

**Not involving prox. asc. aorta.**

\[\Rightarrow \text{Partial obstruction} \Rightarrow\]

**Aortic involvement begins high in asc. aorta.**
### INTERIM CONCLUSION. 2

<table>
<thead>
<tr>
<th>Aorta - from prox. to distal arch. arch.</th>
<th>Hyperacute Partial obstruction</th>
<th>Obstruction $\Rightarrow$ Increased aortic resistance to flow from prox. aortic arch onwards, including all major aortic arch trunks 2$^\text{nd}$ increase in LV. &amp; asc. aorta pressure &amp; decrease in distal systemic art. pressure</th>
<th>??Why. Bkgd. H/T.</th>
</tr>
</thead>
</table>

### PROBLEM SOLUTION: Pt. 3

**Part 3: Exam. findings (contd.)**

|--------|-------|------|------|
| ? Some obstruction of descend aorta. | 43. No epigastric bruits =  
No evi of renal artery stenosis as a cause for the bkgd. H/T. | | |
| L basal pleural cavity - fluid, but can we see this in any way as being $2^\circ$ to the aortic problem?? | 44. Femoral pulses weak & equal, but  
45. Radio-femoral delay. =  
(- ? coarctation as cause of H/T. )  
However, in present context,  
?? $2^\circ$ to ?? Further obtm. of the aorta distal to the aortic arch? | (Is there L. radio-femoral delay.?  
46. No basal lung creps. =  
No evi. of L.V.E.  
despite LV pressure overload.  
47. L. lung base ++ to dull to percussion + absent breath sounds, prob. =  
L. basal Pleural fluid. ??  
leftrightarrow  
48. Pt. unable to pass urine =  
? oliguria.  
49. No supra-pubic dullness to percussion =  
bladder not grossly enlarged. Thus, if oliguric,  
Prob. no post-renal outflow tract obstruction.  
50. Rectal exam NAD, incl. prostate.  
51. No urine in bladder on cath. =  
confirms oliguria, also confirms  
No post-renal outflow tract obstruction. |
| | Is oliguria $2^\circ$ to $\downarrow$ renal blood pressure  
(i) as part of the general $\downarrow$ in BP.?  
(Sweating & anxiety suggest sympathetic overactivity, which could override renal blood flow autoregulation).  
(ii) ? also further aortic obtm.,  
? as far down as the renal arts. | | |
| ? Desc. aorta | 56. ‘Cardiac’ enzymes normal. i.e.,  
If cardiac, (unlikely on other grounds in any case) then ischaemia rather than infarction. | | |
| | 52. Fundi - some narrowing & irreg. of retinal arterioles, with a-v nipping  
= ‘Grade I’ hypertensive changes, =  
$2^\circ$ effects of H/T. of long-standing H/T. on vasculature (arterioles). | | |
| | 53. Large ECG. voltages over LV. =  
LVH, prob. $2^\circ$ to H/T.  
54. ECG. S-T depression over L.V. Prob. =  
LV. ‘strain’ $2^\circ$ to LVH.  
(Can be $2^\circ$ to myocard. ischaemia,  
but no evi. for that here). | | |
INTERIM CONCLUSION-3

Interim conclusion 3.

| — from prox. to dist. arch. | Obstruction to flow commencing in prox. aortic arch, & extending down to: at least beyond the L. sub-clavian artery (radio-femoral delay), & probably even to the renal arteries — → pre-renal oliguric failure. | (Bkgd H/T). |
| 2. L. pleural space. |  |  |
|----------|------|--------|-------|------|
| 57. 1 litre blood transfusion over 30 min. \(\rightarrow\) JVP N. = at least central blood vol. returned back to normal. ( ? peripheral circ. status). |
| 58. -agonist should indeed stimulate cardiac contraction, but that hardly seems necessary, since L.V. is already contracting v. strongly. Moreover, the -agonist could cause unwanted peripheral vaso-dilatation - which could be v. dangerous in this situation where peripheral art. bp. has not been restored to normal. |
| 59. One hr after admission — “flashing lights in R. visual field(s).” \(\leftrightarrow\) ? Involvement of L. occipital cortex. \(\leftrightarrow\) ?Why |
| 59. If vascular: Post. Cerebral Artery \(\leftrightarrow\) territory, - a branch of the basilar artery or vertebrais. |
| 60. 2 hrs. after admission - sudden \(\leftrightarrow\) CIRCULATORY COLLAPSE. |
| 61. No detectable BP. |
| 62. No detectable apex beat, quiet heart sounds, JVP. 10cms., higher during inspiration (paradoxical.) ↓ |
| 60-62. = Cardiac tamponade. |
| 63. Chest signs unchanged = No more pleural fluid than before. ? also blood, given the circumstances. |

??Why

Bkgd. H.T. but why have events unfolded in this way ic., the nature of the basic underlying cause cause in still not apparent.
### PROBLEM SOLUTION: PT.5. FINAL DIAGNOSIS.

#### Part 5: Final Diagnosis

<table>
<thead>
<tr>
<th>Anatomic Diagnosis</th>
<th>Pathological Diagnosis</th>
<th>Functional Diagnosis</th>
<th>Actiol. Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. Post. Cerebral Artery territory.</td>
<td>Sudden onset. prob. Vascular.</td>
<td>Involvement of L occipital cortex.</td>
<td>? Extension of obstruction to vertebro-basilar arterial system. (But why only R. visual field involved.)</td>
</tr>
<tr>
<td>4. Peri-cardial bleed.</td>
<td>Sudden onset, this time Haemorrhage.</td>
<td>Circulatory collapse. due to Cardiac tamponade.</td>
<td>Is this due somehow to Leakage of blood from aorta into pericardial sac in the same way into the L. pleural space?</td>
</tr>
</tbody>
</table>
Answers to MCQs

A. Mechanisms in Disease: 4, 5, 6 & 7 correct. All others false.

B. Problem Solving: 4, 5, 6, 7, 9, 11, 12, 14 correct. All others false.