Some Thoughts on the Clinical Diagnosis of Heart Disease

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INTRODUCTION:

In spite of the many advances that have been made in the past two decades in technical procedures designed to aid in the diagnosis of cardiovascular disease, the clinical examination remains the single most reliable method for determining the etiologic, anatomic and hemodynamic diagnosis. It is safe to say that more mistakes in diagnosis are made as a result of inadequate clinical examination than from any other single reason. This method of examination is available to all physicians and the only essential instrument is a stethoscope, although the ophthalmoscope and the sphigmomanometer are sometimes useful. In this age of right and left heart catheters, phonocardiography and vectocardiography, image intensification and cine-angiocardiography, the pure cardiac physiologists will probably agree that the plain PA chest X-ray and the ordinary 12 lead electrocardiogram may be considered as part of the clinical examination.

SYMPTOMS:

Most of the symptoms of cardiac disease may be explained on the basis of a relatively few pathophysiological abnormalities. These include ischaemia of the myocardium, elevation of the pulmonary venous pressure, decrease in the cardiac output, abnormal action of the heart and mixing of the right- and left-sided circulations. Another group of symptoms may be referred to as associated with cardiac disease.

Cardiac ischaemic pain occurs in any situation that results in a relative deficiency of oxygen supply to the myocardial fibres. Although the vast majority of patients with this symptom have disease of the coronary arteries, it must be remembered that it can be produced by any severe obstructive lesion in the circulation of blood in the heart or lungs. Most of the characteristics of this pain are well known and need not be repeated. However, some points may require extra emphasis. The short, recurrent attacks (angina pectoris) should be considered to be related to transient increases in myocardial work, extra exertion of the heart rather than extra exertion by the patient. Thus one is not surprised by the occurrence of pain during an exciting television program, or when the cardiac output is increased by an incidental infection. Peculiar locations of the discomfort are often stressed, but it is more important to remember that in almost every instance at least some component of the discomfort is located retrosternally. Too much emphasis can also be placed on the character of the pain as the patient may find it almost impossible to describe. More important is the fact that it rarely, if ever, begins very acutely but rather builds up over a period of seconds or minutes to a peak, remains at this peak for a period of minutes with ordinary angina pectoris or much longer with myocardial infarction, and then relatively gradually subsides.

The symptoms associated with elevation of the pulmonary venous pressure are the various types of dyspnoea that occur with lesions of the left side of the heart. Thus with left ventricular disease, dyspnoea on exertion occurs when the pulmonary venous pressure is normal at rest but becomes elevated with exertion, orthopnoea and paroxysmal nocturnal dyspnoea are apparently associated with elevations of the pulmon-

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ary venous pressure associated with recumbency, and pulmonary oedema with more prolonged pulmonary venous hypertension. This is an obvious oversimplification of the dyspnoea of left-sided cardiac disease; for example, in mitral stenosis which is associated with long standing pulmonary venous hypertension, structural changes occur in the lungs which have an important bearing on the symptom in this disease. However, the concept is useful in correlating the symptoms with the signs associated with left ventricular disease.

Decrease in the cardiac output may occur suddenly or gradually. Sudden decrease may result in syncope as occurs in Stokes-Adams syndrome. This symptom also occurs with exertion when the output is fixed, as in the exertional syncope of left ventricular outflow tract obstruction. The symptom most closely correlated to chronic fixed cardiac output, or low output states, is fatigue. This occurs particularly following exertion and thus is not present in the early morning but becomes increasingly more troublesome as the day progresses, and the patient often finds it necessary to have a rest period in the afternoon or early evening. Fatigue will not be the dominant symptom in low output states in which exertion results in elevation of the pulmonary venous pressure however, as the dyspnoea resulting from the latter is much more distressing. In a patient who complains of severe exertional fatigue, therefore, one should suspect the lesion proximal to the pulmonary vascular bed. It must also be remembered that chronic decrease in cardiac output, either relative or absolute, is the basic defect underlying the syndrome of congestive cardiac failure, with all the symptoms which result therefrom.

The symptom resulting from abnormal action of the heart, palpitation, is one of the most difficult to evaluate. It occurs as a result of one of four mechanisms: rapid heart action, irregular heart action, hyperkinetic ventricle, or from an increased awareness of normal heart action. Paroxysmal tachycardia is an example of rapid
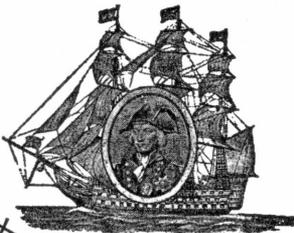
heart action and the diagnosis of this entity often must be made entirely from historical evidence. Irregular heart action occurs in atrial fibrillation and in ectopic beats, and again the nature of the arrhythmia can often be determined by careful evaluation of the history. Palpitation is often the only symptom of pathological states characterized by hyperkinetic ventricles, that is ventricles carrying an extra volume load. This characteristically occurs with exertion or excitement, but is very difficult to distinguish from the normal awareness of heart action in these situations. Finally, many normal people are occasionally aware of physiological cardiac contraction, although this type of palpitation is much more common in patients with emotional problems.

Mixing of blood from the two circulations within the heart occurs only in congenital heart disease and is associated with the symptoms of cyanosis and exertional dyspnoea. The cyanosis occurs when the saturation of the arteriole blood falls below 85%, and may only be seen with exertion. The dyspnoea is apparently related to the effect of arterial hypoxia on the central nervous system.

A wide variety of symptoms may be associated with heart disease, and although not directly related to cardiac function, may be of considerable aid in diagnosis. Included here would be the systemic emboli associated with mitral stenosis and atrial fibrillation, intermittent claudication indicating the presence of occlusive vascular disease in the legs, and the early morning earaches associated with severe systemic hypertension.

SIGNS:

The clinical signs related to cardiac disease must be divided into two groups: those related to changes in left ventricular output, and those which are due to specific anatomical lesions. It must be remembered that both factors may influence the same finding. An example of this is the small volume, slow rising pulse of aortic valve stenosis, in which case the small volume is related to the diminished left ventricular stroke volume and slow rise is related to the obstruction at the aortic valve. It must also be remembered that the left ventricular output as measured clinically refers to the fraction of blood ejected from the left ventricle into the aorta and is not necessarily the same as the right ventricular output, the total left ventricular output, or the effective systemic blood flow. In atrial septal defect with a large left to right shunt, the right ventricle pumps much more blood than the left, while in most types of cyanotic congenital heart disease, the right ventricle pumps less blood than the left. In mitral insufficiency and ventricular septal defect, only a fraction of the total left ventricular output enters the aorta during systole with the remainder passing into the left atrium in the former and into the right ventricle in the latter instance. Finally, in all lesions where there is a run-off from the aorta, such as aortic insufficiency or patent ductus, the total left ventricular output is far more than the effective systemic flow because of the leaking of blood back into the left ventricle in the former and into the pulmonary artery in the latter instance. For simplicity, left ventricular output will henceforth be considered to be synonomous with the amount of blood ejected from the left ventricle into the aorta.

Changes in the left ventricular output are generally accurately reflected in the clinical findings, at least to the degree that one may say with some degree of confidence that the output is normal, elevated or decreased. Severe depression of left ventricular output will result in cold, often peripherally cyanosed and perspiring extremities, constricted peripheral veins, especially in the back of the hands, and markedly diminished pulse volume. Other findings which may be helpful include absence of retinal venous pulsation, of the left ventricular impulse and of the physiological third heart

sound. In contrast, marked increase in left ventricular output is characterized by warm, throbbing extremities, full peripheral veins, increased pulse volume with a collapse of the downstroke, commonly slight elevation of the jugular venous pressure, marked pulsation of the retinal veins, hyperkinetic left ventricular impulse, a relatively loud third heart sound, and an ejection systolic murmur. Discrepancies arise when a factor other than output is acting on any of these findings. The least reliable signs are those directly related to cardiac action such as the cardiac impulse and auscultatory findings, as these are so frequently affected by the anatomical abnormalities. However, discrepancies may occur with the pulse and peripheral findings as well. For example, the pulse volume is affected by change in both peripheral resistance (systemic hypertension with elevation of the mean blood pressure) and the so-called "central" resistance (systolic hypertension associated with a rigid aorta). An example of discrepant peripheral findings occurs when a low output is associated with liver necrosis as the latter may result in vasodilation, even in the presence of a low output state.

The anatomical abnormalities which result in cardiac disease may involve primarily the left ventricle, the right ventricle, the pericardium, or the atria. The clinical pictures which result from the various types of pericardial disease are well known and will not be repeated here. Likewise, atrial disease, which, in its pure form, is almost always associated with abnormalities of the atrial-ventricular valves, and which is seen clinically as mitral or tricuspid stenosis, need not be further emphasized.

Abnormalities that affect the left ventricle may be divided into four groups. These include pressure loads, volume loads, primary disease of the myocardium itself (cardiomyopathy), and combinations of these.

Pressure loads may be due to systemic hypertension or left ventricular outflow tract obstruction. They result in marked left ventricular hypertrophy, characteristically with little change in cardiac size until left ventricular failure occurs. Prolongation of left ventricular systole is an invariable accompaniment of any significant left ventricular outflow tract obstruction, and also occurs regularly when there is a sudden development of systemic hypertension. The cardiac output remains relatively normal until the left ventricle fails. The pulse has an increased volume and sustained peak in systemic hypertension and a slow rise in all types of rigid left ventricular outflow tract obstruction. In severe cases, a dominant "a" wave in the jugular venous pulse is common because of impedence to right ventricular filling by the enlarged interventricular septum. The most striking feature of the cardiac impulse is its "sustained" nature reflecting the prolongation of left ventricular systole, and, although this is most characteristic of the obstructive lesions, it also is quite commonly seen with either acute hypertension or prolonged hypertension with marked left ventricular hypertrophy. On auscultation, the decreased compliance of the left ventricular musculature will often be reflected in a palpable and audible atrial sound, whereas the prolongation of left ventricular systole, when present will result in delay in aortic closure, which, when marked, will produce reversed splitting of the second heart sound. The murmur due to the obstructive lesion is well known and requires no further comment here.

There are two types of left ventricular volume loads. In the one, the increased left ventricular output is simply associated with a total increase in cardiac output as occurs in hyperthyroidism, fever and the like, in which case the findings are those as indicated previously for high left ventricular output states in general. In the other group, the left ventricular output is in excess of the effective systemic blood flow. Characteristically, this type results in considerable enlargement of the heart prior to the onset of the left ventricular failure because of the high end-diastolic blood volume. The pulse has an increased volume and is of collapsing type when the entire

left ventricular output is ejected into the aorta, whereas when some leaves the ventricle by an abnormal pathway, as in mitral insufficiency, the pulse volume is decreased although it is still often collapsing in character, as left ventricular emptying occurs more quickly than normal. A dominant "a" wave in the jugular venous pulse is uncommon in contrast to the pressure load group, because the predominant pathological change in the left ventricle is one of dilation rather than hypertrophy. However, lesions which commonly have a pressure component as well, such as aortic insufficiency, are frequently associated in the later stages with this sign. The cardiac impulse is the most distinctive feature of these lesions. It is characterized by excessive movement, and the chest wall can often be seen to be actively moving on inspection. The auscultatory findings depend much more on the underlying anatomical defect than on the basic hemodynamic left ventricular abnormality.

Left ventricular disease due to "primary" involvement of the myocardium is extremely common, as ischaemic heart disease falls within this group. These diseases are characterized pathologically by diffuse, or focal, infiltration, or replacement, of the myocardium. The signs result from reduction of the cardiac output, decreased compliance of the left ventricle, prolongation of left ventricular systole, interference with right ventricular filling, and, eventually, left ventricular failure. It is obvious that the presence or absence of any of these abnormalities will be related to the severity of the left ventricular disease. Thus, in advanced stages of this type of lesion, the extremities will be cool and the forearm veins fine, pulse volume will be small with the contour normal (although sometimes collapsing if there is an element of sub-valvular outflow tract obstruction), a dominant "a" wave will be seen in the jugular venous pulse because of the impedence to right ventricular filling from the bulging interventricular septum, the cardiac impulse will be normal or sustained depending on the degree of prolongation of left ventricular systole, there will often be a palpable and audible atrial sound due to decreased left ventricular compliance, aortic closure may be delayed resulting in a single or reversed split second sound because of the prolongation of left ventricular systole, and there is often a left ventricular outflow tract murmur due to impingement by the hypertrophied muscle on the outflow tract during systole.

Left ventricular lesions associated with combinations of these basic abnormalities result in various combinations of the signs. Examples of this type of lesion would include mixed aortic valve disease with stenosis and insufficiency, systemic hypertensions associated with any of the volume loads, and the like.

Clinically important disease of the right ventricle results only from pressure or volume loads on this chamber or combinations of these. Isolated disease of right ventricular muscle is of little clinical significance. Regardless of the type of lesion, the general effect in severe cases will be reduction of cardiac output and thus will result in the findings associated with low output states. The basic differences between the signs produced by the two types of hemodynamic lesions are analogous to those produced by similar lesions on the left side. In general, however, the findings may be somewhat more difficult to sort out as mixed pressure and volume loads are very common on the right side.

SUMMARY:

Recent advances in hemodynamic investigative techniques have increased, rather than decreased, the importance of clinical examination, as they have added immeasurably to our understanding of the pathophysiology of symptoms and signs of heart disease. Some of the more important clinical features of heart disease particularly that involving the left ventricle, have been reviewed with emphasis on their importance as the cornerstone of modern diagnostic methods.