CARDIAC EMERGENCIES (Part II).

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Cardiac Dyspnœa.

So far we have dealt exclusively with those forms of cardiac emergency which arise through *disturbances of rhythm*; now we have to turn to other forms, often much more distressing to the patient, and in which characteristically the *heart's rhythm does not show any departure from normal*.

The first group of cases of this type have as their main feature an attack of dyspnœa. Not uncommonly this comes as a bolt from the blue in a subject quite unaware of any disability connected with the heart. Nearly always it is in a middle aged or elderly subject, more often a man. The attack wakens him from sleep in the early morning hours, and may last for a few minutes to an hour or more. When severe the distress is very great. Respiration becomes more and more difficult, cyanosis deepens within minutes to a livid hue, the lungs fill with sibilant rhonchi and bubbling râles, cough with a frothy blood stained sputum begins to develop and perhaps increases to a frank œdema of the lungs, a rapid, but regular, pulse and a raised blood pressure begin to flag, and the unfortunate subject, sweating and exhausted, lapses into a semi-conscious state. Asphyxia from the degree of œdema of the lungs may be quickly fatal in such a patient, occasionally within a matter of minutes.

This is the so called "cardiac asthma" or "paroxysmal cardiac dyspnœa" and it is the severity only that distinguishes it from acute œdema of the lungs, to which it so often leads. Where the one ends and the other begins is an arbitrary matter gauged only by the relative dryness or otherwise of the lungs; in the minor attacks there is mostly wheezing, and in the severe ones mostly œdema. The cause of both is now thought to be essentially the same, an acute engorgement of the lungs brought about by a rapid kind of cardiac failure in which the left ventricle is more especially concerned. These attacks are far more common in those conditions where the left ventricle is exposed to strain or where its muscle is disproportionately diseased. At least 75 per cent. of patients with such attacks have hypertension as the underlying cause, and in the remainder there is either severe myocardial disease from coronary atheroma, perhaps with a bundle branch block, or disease of the aortic valve, syphilitic, atheromatous, or occasionally rheumatic. Every now and then a coronary thrombosis begins in this way, and may do so without much pain. Rarely such attacks as these occur in a younger patient with mitral stenosis, or in even rarer cases a paroxysmal tachycardia or some other abnormal rhythm initiates the severe dyspnœa.

Acute breathlessness of this type, a true "cardiac asthma," has to be distinguished from two other types that are of cardiac origin and also from others that have nothing to do with the heart. As regards the former there is that form of breathlessness that sometimes wakens any patient who already has congestive cardiac failure and orthopnœa if, during sleep, he slips downwards in bed to a recumbent position. Here the attack is like cardiac asthma of a moderate grade except that râles are less widely found in the lungs and the attack subsides fairly quickly when the patient sits upright, and can be prevented entirely by proper support during sleep. In the other form of sudden cardiac dyspnœa a patient with Cheyne-Stokes respiration becomes wakened at night by long and

often violent periods of hyperpnoea; this can easily be recognized for the lungs are dry, the colour remains unaffected, and the periodic quality of respiration is well in evidence.

Among the causes of an acute dyspnoea, not immediately related to the heart, that may lead to difficulty in diagnosis, are true bronchial asthma, spontaneous pneumothorax, pulmonary embolism, and the attacks of coughing with wheezing that may occur in persons who have cardiac disease secondary to chronic pulmonary disorders such as chronic bronchitis and emphysema. I think we must all admit the difficulty that there can be in separating true bronchial asthma from the cardiac form in an elderly subject, for wheezing rhonchi of bronchial spasm occur in both forms. A recent onset of the attacks, the presence of enlargement of the heart and of hypertension or aortic valvular disease, and of evidence of pulmonary oedema would be much in favour of the cardiac form; a long history of similar attacks in a younger patient, with a marked tendency to emphysema would favour true bronchial asthma. The attacks of dyspnoea that occur in those with a cardiac affection—*cor pulmonale*—secondary to chronic lung disease are similar in origin to true bronchial asthma or are brought on by cough due to bronchitis. Such attacks in these patients are never followed by oedema of the lungs, and there is no cardiac factor responsible, for if the heart becomes involved to the extent of failure it shows from the start as a congestive failure distending the neck veins, enlarging the liver, and leading to oedema of the extremities. The diagnosis of this cardiac affection secondary to chronic lung disease would rest upon the presence of clubbed fingers, the marked and persistent cyanosis, constant wheezing throughout the day, perhaps a history of allergic phenomena, the story of chronic cough over many years, the characteristic form of the cardiac enlargement as revealed by X-ray screening, and perhaps right ventricular preponderance in the electrocardiogram. Spontaneous pneumothorax has its unilateral signs in the chest with a displaced mediastinum as the guide to diagnosis; pulmonary embolism leads to acute dyspnoea only when a main vessel is obstructed, and there is then pain, early hæmoptysis, and of course the association of some cause for the embolus such as mitral stenosis, phlebitis, or a recent abdominal operation.

Prognosis in an attack of cardiac asthma is a difficult matter, for the worst attacks are fatal within a few minutes and the least pass off within the same time without any serious distress. Others last for an hour or two, gradually worsening or recovering. The danger signal is the occurrence of signs of pulmonary oedema, for if this does not occur the outlook is good, death being almost always due to asphyxia from such oedema. The later prognosis should always be most guarded; Palmer and White found that 170 out of 250 cases which they studied had an average duration of life of only 1.4 years from the time of the first attack. The coincidence of gallop rhythm or of *pulsus alternans* indicated, as a rule, a very short life.

In the immediate treatment of the acute attack the patient should be sat bolt upright, given morphine, $\frac{1}{4}$ -gr. repeated if necessary in 15 to 20 minutes, and venesected to the extent of a pint or so. Morphine has a remarkable effect in these patients, and I know of no contra-indication to its use except the late semi-conscious state of respiratory paralysis that terminates a long attack, when venesection and artificial respiration should be tried. Atropine has no effect in reducing the risk of pulmonary oedema, but as it serves to maintain a free airway by inhibiting bronchial spasm I often use it for these patients. Alcohol, nitrites, and reflex irritants like camphor are useless, and adrenalin dangerous. Even strophanthin, which might be expected to be of considerable value, has not been

shown to be of much use. Oxygen, given through a funnel closely applied, or in a tent if available can give relief, and if there is respiratory failure should then be combined with five to ten per cent. of CO₂. The later care of these patients consists of absolute rest in bed for some weeks with restricted activity afterwards, and also full digitalisation. Though cardiotonics do not seem to help in the individual attacks they are of the greatest value in preventing recurrences. A low calorie diet, after the Karel type, with fluid restriction as well is particularly valuable in obese subjects, and recently the newer mercurial diuretics have been shown to have a place in continuous treatment, even though there may not be any congestive cardiac failure. In a suitable patient an injection once or twice weekly, or the suppository, may be tried. These patients should always sleep well propped up in bed, with support under the knees to prevent them from becoming recumbent during the night.

Cardiac Pain.

The last form of cardiac emergency is that in which pain is the chief feature. Pain of cardiac origin is almost always due to *acute pericarditis* in a young subject, or to *coronary ischæmia* in an older one. Such pain has to be distinguished from that of functional origin, as in *neuro-circulatory asthenia*, and that found with *thoracic aneurysms* and other sources of *mediastinal pressure*, from root pains occurring in *spinal osteo-arthritis* and *tabes dorsalis*, from pain arising in *pulmonary disease*, whether pleuritic or due to mediastinal drag, from *pain of œsophageal origin* due to growth, or to the *rare peptic ulcer* or *paracœsophageal hernia*, from pain due to *neuralgic or fibrositic causes local to the chest wall*, and from pain due to some *upper abdominal emergency*. I put the latter last, for though it is usually given such prominence in the books as a source of difficulty, I think that in practice we rarely should have real difficulty, despite the confusion that has been supposed to exist between pain of cardiac ischæmia and that from gall stones.

There is no need here to detail the various points in the separation of pain of true cardiac origin from that due to these various other causes, but there are one or two worth a brief mention. Severe pain in acute pericarditis is rare, and when it occurs is due to involvement of the pleural and outer border of the parietal diaphragmatic pericardium, frequently with complicating pleurisy. The pain thus has characteristics like those of pleural pain with in addition the fact that it is often intensified by pressure over the sternum or lateral to it. The rub, the associated cardiac murmurs in a young subject with fever and perhaps joint pains complete the clinical picture. Pain from cardiac ischæmia can almost always be recognized on the history alone; it is essentially sternal, and if it radiates it does so across the chest at or above the level of the nipples, or through to the back, upwards to the neck, or outwards to the shoulders and arms. Any pain exclusively submammary, or radiating into the abdomen is unlikely to be cardiac. Ischæmic cardiac pain is usually gripping, almost never stabbing, and compels rest. The duration and degree of relief from rest depend upon the cause; the pain of coronary occlusion often comes at rest but is quite unrelieved by it, and may last for hours; that of angina comes first during effort, usually lasts for a few minutes, rarely more than an hour, and is relieved by complete rest. It is most important in dealing with a suspected anginal attack that came on during rest to find out whether the first attacks came on only during exertion, for this is the crucial test. In a severe case the attacks, as is well known, may then come on at rest, especially at night. Quickly recurring short attacks of angina at rest strongly savour of coronary thrombosis in one of its atypical forms, and the distinction

may not be easy without the support of electrocardiographic evidence. In others the separation is easy as a rule; the duration of the pain and the absence of relief from rest and nitrites, and after that the oncoming shock with its rising pulse rate and falling blood pressure, and later still the pyrexia, signs of congestive cardiac failure, and perhaps a pericardial friction rub leave no doubt of a coronary occlusion. It is characteristic of angina of effort that when the attack is over the patient is as well as before; in doubtful and difficult cases disability and slight signs may often reveal a coronary thrombosis that would otherwise pass unsuspected unless neglect of rest happened to be fatal.

The immediate prognosis in the attack of angina of effort is very good as a rule, for death is the exception if it ever occurs in pure angina. In coronary occlusion the outlook is much more difficult to judge; a long duration of pain and severe signs of shock or of cardiac failure early on are of grave import, but if these are absent or minimal the immediate outlook is usually quite good. The chief risks are sudden death, especially during some effort such as straining at stool, congestive cardiac failure, embolism, and later cardiac aneurysm.

In the acute emergency of the pain of coronary ischæmia, whether due to angina of effort or to coronary occlusion from thrombus or embolus, the immediate treatment is rest and a trial of nitrite. Rapid relief from nitrite almost excludes a coronary occlusion, just as a failure to get relief almost excludes angina of effort provided that the nitrite has been given in the proper way. If nitrite fails morphine, $\frac{1}{4}$ -gr. should be given at once and repeated in 15 to 20 minutes or later as required. In the later care of coronary thrombosis complete rest with adequate nursing cannot be too strongly insisted upon; the patient should be turned, washed, fed, but otherwise left alone for some days, and all sudden or active movements prohibited. Both the nurse and the relatives should be told that an action of the bowels is neither expected nor desired for several days, for nothing could be calculated to incur more risk than the purposeless insistence upon the bed pan during the early days. Nutrition matters little also during this phase, and it is best to starve judiciously except for small milk feeds and glucose drinks. Morphine can be stopped as soon as the pain ceases and other sedatives substituted; I prefer chloral and bromide, 15 to 20 grains of each in a draught as required. The view that chloral hydrate is harmful to cardiac patients lacks valid support and the drug has been used since its inception for these very cases and with success. The question of digitalis is still under discussion, but in general it is wiser not to use it unless signs of congestive cardiac failure supervene; then it should be given in full doses. If the danger is quickly increasing strophanthin $\frac{1}{200}$ to $\frac{1}{100}$ -gr. six hourly by a vein is suitable. In a gravely ill, cyanosed patient with congestive cardiac failure, an oxygen tent can work wonders, and even in those less severely ill it is a great help in securing proper rest and sleep.

To turn now to the immediate treatment of angina of effort, no remedy is so effective as nitroglycerine given in tablet form; the tablet should be chewed thoroughly, or one used with a lactose base for an edentulous patient. The important thing is to get the tablet dissolved as quickly as possible in the mouth, for absorption is much more rapid from here than from the oesophagus or stomach. Used in this way the action comes on within a minute, and only 2 per cent. of patients with angina of effort fail to get rapid relief from attacks. Occasionally another tablet may be needed, or even a third. Amyl nitrite, though it acts rather more quickly, has a more violent action which leads to pronounced side effects distressing to the patient and to onlookers, is more difficult to administer by the patient himself during the emergency of an attack,

and is less certain to relieve. For all these reasons it should be reserved for exceptional patients who fail to respond quickly enough to nitroglycerine.

For the prevention of subsequent attacks, beyond the usual general measures, patients should be taught the value of nitroglycerine tablets when used in relation to effort. The tablets should always be carried, and just before beginning to do some exertion known to produce or be likely to produce an attack of pain patients should chew a tablet thoroughly. In this way pain can be successfully prevented, and this process can be repeated as often as required for the moderate exertions of a reasonable life. I have had patients using 20 to 30 tablets a day for months or even years on end, and throughout this time able to do in comfort far more than would otherwise have been possible. With these large amounts there may be headache, but with smaller amounts, up to a dozen tablets daily, it is surprising how rarely this is at all troublesome. Used in this discriminate way in relation to immediate effort, nitroglycerine can keep many intelligent patients almost free from pain and yet allow them to lead lives far from that of cardiac invalids.

There are of course two important questions that need an answer before using the drug in this way—the question of tolerance and that of risk. Tolerance even with large doses does not seem to develop in the human subject; the tablets remain just as effective over a period of years and the dose does not need to be raised. The question of risk is more important, for one might well ask whether the prevention of pain, by allowing unchecked exertion, would not prove to be a highly dangerous weapon for the anginal subject. It is a question that can be settled only by trial of such preventive treatment, and Evans and I in a large series of cases a few years ago found no single instance in which sudden death or in fact harm of any kind could be attributed to the free use of nitroglycerine in this way. It is far more effective than the routine use of this or any other nitrite three or four times daily given without reference to exertion, and it permits a much fuller life than is otherwise possible. Patients who are used to this preventive treatment for their attacks gain in confidence, get about often with ease, keep free from major attacks, and often can remain at work which would otherwise be impossible. They may be permitted all these things provided that no severe effort is undertaken, and provided that during whatever effort they undertake they remain free from pain; if pain comes in spite of using nitroglycerine they must stop and take further tablets. The explanation of the safety of this preventive use of nitrite is most probably related to the fact that it relieves anginal pain by dilating coronary arterioles and thus overcoming myocardial ischæmia. As pain is the index of this, when there is no pain there is no severe grade of ischæmia. The anginal attack comes with effort when the demand for oxygen by the heart muscle during exertion exceeds the supply available through the narrowed coronary vessels; nitrite produces a vascular dilatation with a much increased coronary blood flow, thus raising the oxygen supply to the heart above that required for the effort and abolishing the pain. If nitrite is given before the exertion begins, the only difference is that the coronary blood flow is already raised above that needed to supply oxygen for the effort, so that no pain comes on. Looked at in this way there is no greater danger in using nitroglycerine for prevention than for the relief of anginal pain, for the two conditions are parallel. The dangerous thing in either case is to allow the patient to continue to exercise when pain is present, just as it is dangerous to do so in coronary thrombosis. Sudden death in an attack of pure angina of effort is very rare, and when it happens there is pain. Death is not due to the pain but to the cardiac ischæmia which it tokens inducing ventricular fibrillation or arrest.

In this survey of the cardiac emergencies there are two points upon which I have not touched so far, the question of heart failure in such conditions as pneumonia and other fevers, and the use of modern remedies such as pyridine-B-carboxylic acid diethylamide.

Heart failure in the acute fevers, including pneumonia, is rare. When it occurs it is sometimes sudden with syncope and is then due to heart block or to toxæmic arrest or fibrillation of the ventricles; in the other group of cases it is congestive and is then due to some disorder of rhythm with tachycardia. Both these types of emergency have already been dealt with, and the principles of treatment discussed. But in the great majority of cases so called heart failure in these acute fevers is not primarily cardiac at all, but a form of toxæmic vascular shock. Here the heart is only involved in a secondary way.

The purest form of this kind of circulatory failure occurs in severe hæmorrhage, but it is also responsible for the similar kind of thing which eventually may arise in true traumatic or post operative shock, in severe vomiting or diarrhœa, in diabetic acidosis, in Addison's disease, and in burns. This is the condition, confused with cardiac failure, that also occurs in fevers, and far more commonly than true cardiac failure. The clinical picture is characteristic and very different from that of a cardiac failure; the patient has a greyish pallor, never deep cyanosis, respirations though rapid are not accompanied by difficulty in breathing or orthopnoea, the extremities are cold but not œdematous, the superficial veins are collapsed, not distended, the face is sunken and the intra ocular tension reduced. Engorgement of the liver is quite rarely detected clinically, and œdema of the ankles almost never occurs. Treatment in this condition of toxæmic vascular shock centres around the use of intravenous glucose and saline solutions, blood transfusions, bandaging of limbs and raising the foot of the bed, warmth, morphine and sometimes oxygen. Ephedrine is a drug worth trying, but adrenalin is destroyed so quickly in the body that it is useless in a protracted fall in blood pressure of the kind that occurs here. Caffeine, camphor and its allies, and pituitary extract are without effect, and digitalis is not indicated and never helps.

In regard to the second question concerning newer remedies, I do not think that any of them have as yet been shown to have any definite advantages in the management of cardiac emergencies over those which we have discussed. We know now as much about the pharmacology of many of them as we do about the well tried and efficacious drugs like adrenalin, digitalis and strophanthin, quinidine, morphine, and nitroglycerine; there have, for instance, been over 1,400 papers written on pyridine-B-carboxylic acid diethylamide alone. But in spite of this spate of research and publication, much of it of doubtful quality, none of the newer remedies in the treatment of cardiac emergencies can be said to have established an enduring reputation. Had they done so they would have been in universal use, and have received the universal support of pharmacologists. Pyridine-B-carboxylic acid diethylamide, for instance, is a powerful respiratory stimulant with a real value in conditions such as poisoning from carbon monoxide, hypnotics, and certain anæsthetics, or in cardiac failure that is purely secondary to asphyxia as here. But it has none of the value of adrenalin in overcoming the Adams-Stokes attack, nor of digitalis in slowing the ventricular rate in congestive heart failure, nor of quinidine in correcting auricular fibrillation, nor of nitroglycerine in relieving anginal pain, nor of morphine in overcoming the distress of cardiac asthma. It is by such tests that we must measure any remedy in a cardiac emergency, and by such tests these newer drugs have so far been found to be wanting.