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TRAUMA OF THE HEART*

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Trauma of the heart may result (1) from wounds and direct violence and (2) from strain of effort. Wounds of the heart are not common in this country, and will be excluded from this discussion. Direct violence to the chest wall may rupture the heart muscle, causing death, or may cause death without obvious heart injury; or it may give rise to the following clinical conditions: (1) pericarditis; (2) angina of effort; (3) a disorder of rhythm; (4) lesion of a valve; (5) contusion of the heart muscle. Strain may result in: (a) a disorder of rhythm; (b) lesion of a valve; (c) primary cardiac overstrain.

Spontaneous rupture of the heart muscle is, of course, the result of coronary disease. Goodall and Weir (1927) record eighteen cases, but in only four was there evidence of emotion or strain. Such an event can hardly be included in trauma of the heart.

Genuine cases of heart disease the result of injury or sudden strain are not very common. There is nothing distinctive about the physical signs. For a diagnosis we depend upon an accurate history and, if obtainable, previous personal medical knowledge of the patient. Many doctors can quote one or two cases in which they were convinced that trauma produced disease of the heart, or that it so aggravated the condition of a heart already diseased that the whole life-history was changed. A traumatic heart lesion shown at a clinical meeting will provoke criticism, because the physical signs will be similar to those met with in disease due to natural causes, and it must be demonstrated that the condition before, at the time of, and immediately after the trauma has been correctly assessed. Nevertheless, I believe that at the present time more cases of heart disability the result of trauma are overlooked than are diagnosed by mistake.

During the last thirty years I have made brief notes of twenty cases in which trauma of the heart must be considered seriously. Twelve have been the result of direct violence and eight the result of strain, although in three of them both forms of trauma may have been acting together. One man with a traumatic lesion of the mitral valve was under observation for seventeen years until he died, when the trauma was confirmed at necropsy. In the twelve cases of direct violence it is safe to presume that the heart was normal before the injury. In three of the eight cases due to strain it is presumed that the heart was already diseased, but the strain altered the whole life-history. A reasonable definition of a normal heart would be: one that is efficient for the age and usual activities of an individual, and which would show no

abnormality on examination with all the methods at our disposal.

Recorded cases in medical literature are difficult to collect, although there are examples typical of many well-recognized possibilities. It is reasonable that standard medical textbooks should be very guarded in relation to injuries of the heart. These play a small part in the natural history of disease. Books written as a guide in compensation cases are helpful in classification and make valuable reading, but they aim at a finality of statement that is not always in keeping with such an individual problem as injury. Allbutt (1870) wrote chiefly from the point of view of the part played by overstrain in artisans in producing degeneration. He made a point that he gained wide experience in the industrial area of Leeds. In mentioning certain cases in which a sudden strain appears to have caused structural damage he suggests that these may be extreme instances of an agency always at work, in some degree, in those who do heavy manual labour. He concludes: "But when we come to refine upon labour and endeavour to separate it into two categories of sudden and continuous efforts we find very often that the distinction is almost impossible." There are, however, a number of cases where the distinction can be drawn between sudden and continuous effort; and a number of cases of trauma or alleged trauma of the heart in those not engaged in heavy work—that is to say, the result of an exceptional event in one unsuited to the effort. And disease of the heart may result from the direct violence of a blow on the chest wall.

The study of traumatic heart disease resolves itself into two problems: (1) whether the heart be diseased at all—which is perhaps the more difficult; and (2) whether a cardiac disability is due to trauma or to natural causes. For this latter consideration the physical signs are almost negligible. A detailed and accurate history, in the widest sense, is all-important. The patient must be studied as a witness, and also his relatives. Not less important is a reliable family doctor who knows his artisan patients personally and makes a note when first called in. It is my association with a number of these who attend clinical meetings regularly which encourages me in the attempt to assess some of the evidence about trauma of the heart.

Some Post-mortem Records of Heart Trauma

1. *Without External Bruising or Injury to the Chest Wall.*—Groom (1897) was called to a boy of 16 who had collapsed while walking, and found him dead. A month previously the shaft of a pony trap pressed him against some railings. He lay up for five days. There was no external bruising. Post

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mortem the left ventricle was seen to be ruptured posteriorly. It appeared to have developed from the endocardium outwards, with eventual haemorrhage into the pericardium. O'Neill (1914) described the case of a boy aged 9 who was "knocked down and jumped on." There were certain bodily injuries, so that he was kept quiet but not absolutely at rest. The heart was not specially examined. He died forty-seven days later, and a perforating slit was found at the auriculo-ventricular junction on the left side, with haemorrhage into the pericardium. Hamilton (1934) recorded the case of a child of 7 years brought in dead to hospital. The front wheel of a car had passed over the chest wall without causing external bruising or damage to the bones. Both ventricles were ruptured. Fraser (1929, quoting Vaquez-Laidlaw) describes a man who was struck by a carriage pole. He was admitted to hospital with dyspnoea and palpitation. There was an unusual sound at the base of the heart. The symptoms soon cleared up and he was discharged. He was readmitted several months later with congestive heart failure, from which he died. Post-mortem examination showed laceration of the heart muscle in the region of the conus arteriosus. Wilks (1865) recorded the case of a young man who fell from a height, striking his left side on a stone. He died two days later from peritonitis due to a ruptured bowel. Dyspnoea had been present, but a stethoscope was not used. No external bruising was seen at necropsy. The posterior cusp of the aortic valves was split transversely, but there was no sign of chronic disease of the valves. A small deposit of fibrin was found on the ragged edges of the valve. Gibson (1909) recorded a case, in the Edinburgh Royal Infirmary, of rupture of the mitral valve from the kick of a horse.

2. *Experimental*.—Kahn and Kahn (1928) state that it has proved possible in the dead subject to rupture a valve by striking the chest, without injuring the bones of the thorax. Allbutt (1909) gives the names of several distinguished pathologists who have succeeded in this.

3. *With a Fractured Sternum*.—At a coroner's post-mortem recently at the Derby City Hospital I was present when Dr. Osborn demonstrated disease of the aortic valves very suggestive of being the result of trauma. There was an old fracture of the sternum over the base of the heart. The man was wounded during the great war, when a front-line soldier, so that presumably his heart was normal. Whilst he was in a casualty clearing station the sternum was fractured by a fall of roof during an air raid, after which he was invalided out of the service. The history is so fragmentary that it is not possible to make a definite diagnosis of traumatic disease of the aortic valves, but it is the most probable explanation. The aorta itself was particularly healthy, as also was the mitral valve.

Clinical Conditions which may Result from either Direct Violence or Strain, or from Both Together

DISORDERS OF RHYTHM

1. *Auricular Fibrillation*.—Of this condition Price (1937) states: "Its onset can now and then be traced to bodily effort, especially in the middle-aged or elderly, perhaps to trauma." This is what we should expect from the natural history of the condition. The event is more probable if the disposing causes of age or mitral stenosis exist. White (1937) states that it may result from direct trauma, excitement, or exertion. In some accidents all these causes will be present, as in two of my cases recorded below. It is surprising to find the statement in a book on incapacity or disablement (Brockbank, 1926): "I have never met with a case in which auricular fibrillation could be attributed to sudden strain, and at present I should be very unwilling to believe that strain could cause it." Hay and Jones (1927) recorded five cases of auricular fibrillation, of which four were due to sudden physical exertion and one to electric shock. They state that four of the patients were apparently well before the exciting cause or accident. They claim that sudden strain frequently initiates auricular fibrillation in diseased hearts, and in addition that fibril-

lation may result from physical strain in hearts that are apparently normal, so far as can be determined by ordinary clinical methods. As a typical example of the result of strain on a diseased heart I have the following notes (1934).

W. H. P., aged 46, gave a history of rheumatic fever twenty years ago. He was refused for war service, but led an active life, in good health, working on an estate. His doctor knew that he had mitral stenosis. One day, while carrying a tree-trunk, his mate failed and W. H. P. had to bear the whole weight. Pain and discomfort developed in the chest, and he had difficulty in getting home on account of shortness of breath. He passed one or two restless nights, after which his doctor diagnosed auricular fibrillation. Seen four months later, the fibrillation was confirmed by electrocardiogram. The mitral stenosis was obvious. There was no congestive failure. The exercise tolerance was poor. A course of quinidine in hospital produced toxic symptoms, but failed to restore normal rhythm. The arrhythmia has persisted since.

Kahn and Kahn (1928) record cases of auricular fibrillation following direct trauma of the chest wall. I have seen two such cases, after a man had been knocked down by a motor car. There has been no evident bruise on the chest wall or actual history of a blow over the heart, so that both direct trauma and sudden strain may have acted together. Both were elderly, and possibly liable to the disability, but in good health for their years.

T. C., aged 61, acting as a night watchman, was knocked down by a car on January 21, 1932. He was taken to the casualty department at the Derbyshire Royal Infirmary; about this he remembers little. After being detained for a few hours he was sent home by car to rest in bed. He noticed he was short of breath, and his doctor recognized an irregular pulse. The shortness of breath persisted, and although his external injuries were trivial it was obvious that his capacity for exertion was entirely changed. Seen by me in December, 1932, the fibrillation was confirmed by electrocardiogram. There were no other abnormal signs, or other abnormalities in the tracing. There was no congestive failure. Five years later his condition was unchanged.

Mr. W., aged 63, was knocked down by a car on December 22, 1936. He was taken to the Derbyshire Royal Infirmary, where an irregular pulse was noted. He was kept in a surgical ward for fourteen days' rest on account of a scalp wound. On returning home he was dizzy and easily became short of breath. His doctor noted the irregular heart. Seen on February 3, 1937, he was of excellent physique for a man of his years, with the history of an active life. Physical examination revealed nothing abnormal except the arrhythmia, which was confirmed as auricular fibrillation by electrocardiogram. I understood he was making no claim, because he considered himself entirely to blame for the accident. But there was no question that a heart previously efficient for his life was now causing incapacity.

Both these cases illustrate the need for more accurate notes after an accident. This history of scalp wound is of interest because Bramwell (1934) has recorded the case of a coal-miner in whom auricular fibrillation was present after a blow on the head. He is rather guarded about attributing the fibrillation to the trauma, although the evidence strongly suggests it.

2. *Auricular Flutter*.—The disposing causes of this condition are similar to those producing fibrillation, but I have seen no reference to trauma as an exciting cause.

One case of auricular flutter came under my own observation in 1935 after a fracture-dislocation of the cervical vertebrae. From the hospital records it would seem that flutter developed several days after the accident whilst in bed. It is difficult to find a reasonable explanation, but the fact remains that when the patient had recovered from his surgical injuries he was totally incapacitated by reason of the heart

condition. There was evidence of a hypertensive heart, but one cannot entirely ignore the injury as a possible exciting cause of the flutter.

C., aged 58, with good health, had not seen a doctor for ten years. A signboard fell on his head, and he was admitted to the Derbyshire Royal Infirmary with a fracture-dislocation of the cervical vertebrae. The pulse chart records the rate as between 80 and 90 until the tenth day, when it is 120, at which level it persisted. Seen in July, 1935, about three months after the accident, when he had returned home, he was getting about, but was short of breath on exertion. The pulse was regular, the rate being 120 to 130. The blood pressure was 160 mm. systolic and 100 mm. diastolic. The heart was enlarged. An electrocardiogram showed a tracing typical of auricular flutter, with a ventricular rate of 120 and an auricular rate of 240. One may conclude that there was a hypertensive heart before the accident, but that the auricular flutter developed about ten days later.

3. Extrasystolic Arrhythmia.—Kahn and Kahn (1928) record a case of extrasystolic arrhythmia the result of direct injury of the chest wall. But such irregularity is not a definite entity and is no disability in itself. One may only conclude that there is an irritable focus in the heart muscle. The case in question may have been a contusion of the heart, or it may be that the rhythm was disturbed from a neurosis. As an example of such arrhythmia following injury I have the following notes.

J. H., aged 46, was gored by a bull. He was unconscious for twenty-four hours and was detained in hospital three weeks. There was a large scalp wound. Three lower ribs were fractured on each side. It was noted that the pulse was irregular. Four months later the exercise tolerance was poor and the heart showed numerous ventricular extrasystoles. The irregularity persisted a year after the accident. Physical examination and an electrocardiogram showed no other changes. He was capable of taking a fair amount of exercise without discomfort, but had not regained his full capacity. This case is complicated by the fact that he had a primary syphilitic lesion during the war and the Wassermann reaction is still positive.

A heart which is irregular on account of extrasystoles may be the only abnormal finding after some sudden strain. It is an important problem. The prognosis and treatment depend upon the underlying cause of the premature beats. Some cases are probably due to a strained heart and others to a neurosis. We may discuss the question under the heading of "primary cardiac overstrain," although it is understood that not all cases of strain or alleged strain exhibit extrasystoles.

VALVULAR LESIONS

Rupture of a valve from strain or direct violence illustrates the importance of a correct history, because it is upon this that the diagnosis is based clinically; and even after death the history is required to make the diagnosis certain. There will be immediate and urgent distress, with pain which is obvious in lesions due to effort, and, unless masked by external injuries, equally obvious in direct violence.

Rupture of an aortic valve from strain was described by Peacock (1865a). It is an accident that is not very uncommon. The valve is almost certainly diseased before the effort, although functioning reasonably well. For an insurance company I obtained the following history from the late Dr. Bruce of Melbourne, Derbyshire:

A farmer, aged 56, pushed a cart out of a ditch. There was immediate substernal pain with distress and palpitation. He managed to walk very slowly to the doctor, and said he could hear a noise in his chest since the effort. There was a loud to-and-fro aortic bruit. He died of congestive failure a few weeks later.

Traumatic lesions of the mitral valve are rare. Allbutt (1870 and 1873) recorded three cases of rupture of a mitral valve from strain. Gordon (1922) described a case of lesion of the mitral valve from strain while running.

It was in a young woman of 24, who afterwards could hear a noise in her chest on exertion. He considered that the lesion was slight. He also describes a man in whom the aortic valves were ruptured from a blow, and who died soon afterwards.

Peacock (1865b) described the case of a girl of 19 with valvular heart disease in which the symptoms were not serious.

She had a severe attack of vomiting, followed by immediate distress and death in a few hours. At necropsy there was chronic rheumatic endocarditis of the mitral valve; the chordae tendineae were ruptured, so that one cusp of the valve was entirely loose. Horton-Smith (1902) described rupture of chordae tendineae in a workman aged 33, due to a strain. There was immediate pain with shortness of breath, followed by passive congestion. He died in three months; at the post-mortem examination the valves showed no evidence of previous disease.

I have recorded (with Osborn, 1937) full details of a case of mitral stenosis the result of trauma.

A man 32 years of age joined up at the beginning of the war in 1914 with a previous history of good health and athletic activity. He served a year in the front line. In 1915 he was blown up and buried in the debris. A day or two later he recovered consciousness to find his heart very distressed. There was no external injury. He was five years in one hospital and another. From 1919 till his death from pneumonia in 1937 he was under my own observation. It was obvious in 1919 that the mitral valve was diseased, there being a systolic bruit and a localized mid-diastolic sound. Five years after the injury he could sit out of bed, and gradually he improved. By 1930 (fifteen years after the injury) the signs were indistinguishable from those of mitral stenosis of rheumatic origin. There was a presystolic thrill and localized presystolic bruit. His condition remained stationary. When he died from pneumonia twenty-two years after the injury the stenosis was very obvious and there were features which confirmed beyond doubt the diagnosis of trauma.

The evidence is conclusive that the mitral valve was normal before the trauma. The same applies to Wilks's case of ruptured aortic valve from a fall. It is this feature which makes direct trauma of a valve particularly interesting, because a blow might produce a less serious lesion, whereby a slight tear or contusion of a valve would progress towards chronic valvular disease when healing. Allbutt (1873) described two cases of mitral stenosis in young men—in one case due to the kick of a horse and in the other to a blow with a cricket ball. The immediate distress at the time of the injury was alarming, but the heart's function was not distressed soon afterwards to a degree that would indicate rupture of a valve. Allbutt saw each patient about a year after the blow and found definite evidence of mitral stenosis, which he attributed to trauma. These cases were recorded a long time ago, but the diagnosis can only be based on observation and assessment of the history and clinical evidence. No modern facilities would help to prove or disprove the diagnosis of a traumatic valvular lesion.

Clinical Conditions which may Result from Direct Violence but not from Strain

1. Pericarditis.—Traumatic pericarditis is a well-recognized possibility as the result of a blow on the chest wall. In itself it is not serious; and it may not have been searched for sufficiently often in the surgical wards. A

pericardial friction sound may have been overlooked in some patients with severe chest injuries, in whom the possibility of a contusion of the heart must be considered subsequently.

2. *Angina Pectoris*.—Fraser (1929) gives evidence of angina of effort resulting from direct injury to the chest wall. There are also references in the German literature.

I have recently attended a man 57 years of age. He was in charge of a cart when the horse bolted and the shaft struck him over the sternum. He was laid out on the ground for a few minutes, and then slowly, with distress, walked a short distance to a farm. He was sent to hospital next day, where fracture of sternum and ribs was excluded. He was short of breath on slight effort, with pain and aching in the chest. When he began to get about, in three weeks' time, the symptoms of angina of effort developed. The shortness of breath has continued; and his case may be included under the heading of a possible contusion of the heart.

White (1937) states that fatigue of nervous type may dispose towards angina. In the foregoing patient there is this type of nervous problem to consider. I believe that the angina of effort in his case is the result of trauma. Lewis (1937) writes: "Angina pectoris and cardiac failure never can find equivalents in the terms of structure."

As an example of angina of effort the result of direct injury to the chest wall the following case is of significance because there is no question of compensation. The diagnosis is based on what the man himself and his doctor can tell us.

W. H., aged 54, has always had good health. On June 14, 1937, he tried to jump on to the rear of a lorry, which moved; he fell on his left side rather heavily, sustaining a cut head and bruised elbow and hip, and "his ribs were sore." There was no immediate discomfort, apart from his bruising; but a fortnight later, when recovered from this, he noticed that when walking briskly for a bus he got a gripping pain in the chest, radiating down the left arm, and he had to stop. This discomfort became more frequent and occurred on very slight exertion, such as mounting stairs; so that after doing a fortnight's work he had to give up. The heart was recorded as irregular and the rate unduly increased on exertion. In August the attacks were less severe, but they persisted. The pulse was regular. He has improved, but in December would still get angina with moderate exertion. On December 7, seen by myself, the heart showed no abnormal signs and an electrocardiogram was normal. The exercise tolerance was rather poor, but he was proposing to go back to his duties, which are clerical. He did not feel capable of working in his garden.

3. *Heart-block*.—This form of arrhythmia indicates a lesion deep-seated in the myocardium. It may possibly arise from direct violence; that it may result from effort is very improbable. Walker (1933) states that heart-block has been recorded after a blow on the chest wall. I have no personal knowledge of such a case, but some of the post-mortem lesions described above, which have been the effects of direct violence without any external injuries, will illustrate the fact that such an exceptional event as injury to the auriculo-ventricular bundle, with survival of the patient, is possible. White (1937) states that it may result from direct trauma. Coffen (1930) described complete heart-block, persisting seven years, after a child 3 years of age had fallen four or five feet on to his chest. There was no external bruising. "Just before the accident he scored 98 per cent. in a baby clinic examination." The slow pulse, which persisted, was recognized within a few hours of the accident.

4. *Contusion of the Heart*.—It is of course well known that a severe blow on the chest may cause death without anything definite being found in the heart post mortem. Some such examples have been the result of diving.

Certain of the ruptured hearts described above in which the accident was survived for a time may be regarded as severe contusions which have eventually ruptured into the pericardium. To diagnose a contusion of the heart muscle is a difficult problem. White (1937) states that contusion or even partial rupture of the heart wall is probably much more common than has been thought in the past, because of the usual recovery. He suggests that an electrocardiogram taken early might show changes in the T waves, which would be transient. It might be of interest to search in the surgical wards for examples of contusion of the heart.

I was asked to see a case of "bronchitis" in a surgical ward on September 16, 1937. A man aged 58 was admitted three days previously after a severe blow over the sternum from some timber. There was pain and dyspnoea. An x-ray examination excluded fracture, and the heart shadow and lungs were normal. The pulse rate was 80, and regular. The heart showed no abnormal signs, and an electrocardiogram was normal except for inversion or biphasic P wave in Lead III. There was no evidence of bronchitis and the temperature was normal, but the patient was expectorating moderate quantities of frothy serum. It was exactly comparable to the fluid coming up in acute pulmonary oedema but in much less amount. The physical signs in the lungs were quite normal, except for slightly deficient vesicular murmur, equal at both bases. I should conclude that the circulation through the lungs was inefficient on account of a contusion of the heart muscle. He was discharged after four weeks, having been about the ward for a few days. When he returned home he was short of breath on exertion. He began light work about two months after the accident, but could not carry anything up stairs or climb a ladder without distress. Examined on November 30 there were no abnormalities, except a poor exercise tolerance. The pulse rate was 84 at rest, was 100 after dressing, and rose to 144 after slowly climbing some stairs; he was short of breath. The electrocardiogram showed no change.

I have seen six other men in whom the heart appears to have been inefficient after a severe blow on the chest. There has been no record of the heart condition soon after the accident; it has only been considered weeks or months later, on account of shortness of breath on slight exertion. My first example (Barber, 1912) was lost sight of during the war, and the other five are alive. In two cases the sternum was fractured, and in two several ribs; the fifth and sixth cases are recorded above, with angina of effort. The others were aged 27, 46, 50, and 53 years, and they have never recovered fully. The symptoms have been shortness of breath on slight exertion, with dizziness. There has been left mammary pain to some degree, but true angina in only two cases. The heart showed no abnormal signs on examination, and the electrocardiogram was normal: in no case was it taken until several months after the injury. The only evidence of myocardial weakness has been inadequate response to effort. Under these circumstances it is easier to convince oneself than to produce evidence which can be recorded. White (1937) draws attention to the fact that neuro-circulatory asthenia and muscular flabbiness are more easily exposed by exercise tests than is true heart disease. Two of my patients have been colliers, whose muscles have been in reasonable training, and none of them has had the "irritable heart," unsteady under observation, which one tends to meet with in the effort syndrome. An exercise test which is reliable, even if it cannot be standardized, is to lead a patient along the hospital corridors to another department and set him talking by the way. If the proof of an inefficient heart be accepted, it is following the teachings of Lewis (1937) "not to make the attempt to diagnose in the terms of anatomy but in the more certain terms of

function." The condition has been more like the senile heart than any other condition due to natural causes. The following typical example may be cited:

H. B., aged 50, a collier. Five years ago a steel bar crushed his chest against the roof of a coal-mine, causing fracture of five ribs on the left side. He was three weeks in the Chesterfield Hospital and three months an out-patient. He returned to work, but gave up after three days because he was short of breath on exertion and dizzy, especially after stooping. He has worked for short periods since, but has always required extra help from his mates. He has remained short of breath on exertion, with some aching in the left side. His general condition and muscular tone were good. He was a steady and reliable man of the best type. The chest expansion was $2\frac{1}{2}$ inches. X-ray examination showed a heart of normal size, with the lungs clear and the ribs united. The arteries were healthy. The blood pressure was: systolic 115 mm., diastolic 75 mm. The heart showed no abnormal signs, and an electrocardiogram was normal. The pulse rate was 96 on arrival; it was 84 after a short rest; after slowly climbing eighteen stairs it was 140, and he was short of breath. While walking slowly he was incapable of talking without exhibiting shortness of breath.

Primary Cardiac Overstrain

In studying this difficult problem the type of strain causing the disability, or alleged to cause it, may be of three possible varieties, which must overlap to some extent, but of which the typical examples are quite distinct. These are: (1) the strain of athletic event, (2) the effort syndrome, and (3) the intense unexpected effort for which a man is not trained and during which the chest is probably fixed, with the glottis closed.

1. Strain in athletic event is almost confined to those taking part when suffering from some infection. Occasionally the untrained man may disturb his heart with a rowing or climbing effort.

2. The effort syndrome, strictly speaking, has little relation to strain, except in the differential diagnosis from overstrain. In its most characteristic form it first shows itself during some effort which a man has carried out before. During the war the first impression may have been that the symptoms developed as the result of physical over-exertion and mental emotion, acting on those unsuitable for the strain. Probably to some extent this was the explanation. In civilian life it occurs in women as well as in men. The modern view is that effort does not cause the condition, but calls attention to it. At the same time it is admitted that it sometimes develops after a severe effort, and also that in spite of the best of treatment some of the subjects must be warned that they are unsuited for strenuous callings.

3. The intense unexpected effort usually comes about, with the chest fixed and the glottis closed, during a lifting effort which is unexpectedly strenuous or prolonged. Perhaps the effort cannot be relaxed without some disaster occurring. Anxiety on this account may directly affect the heart capacity at the time. It is this type of effort that is most likely to damage the heart. The following brief notes will illustrate a clinical example.

J. B., aged 30, was carrying a bag of cement weighing two hundredweight across a stream. His foot slipped, and he made a very considerable effort to save the bag from falling into the stream, which would have ruined the cement. He had acute distress in the left side of his chest and collapsed on the ground. This was in the middle of the morning. He went on doing light work all day, suffering from palpitation and shortness of breath. He walked home a mile or two very slowly with his father, for whom he worked. He went to bed and passed a restless night. His doctor kept him in bed three or four days, after which he got about a little. The

palpitation and shortness of breath continued to some extent for two years. Physical examination at this time showed a pulse rate of about 100 when out of bed, with occasional ventricular extrasystoles after resting from exertion, to which there was poor response. He was of good athletic physique. Before the event in question he was in excellent health. He served four years in the war, during which time he won a three-mile race without discomfort.

For myself I should diagnose primary cardiac overstrain chiefly on the history. If one has understood the theory as usually taught of the reaction of the heart to effort I suppose the original conception would have been that the heart had suffered from acute dilatation. Allbutt diagnosed the condition on himself, although it led to no permanent ill effects in his case. The teaching followed that the heart did not dilate during effort. Wilson (1930) recorded exceptional circumstances which enabled him to note what he regarded as dilatation of a heart after effort, with quick return to normal. In the correspondence columns Wilkinson (1930) suggested that the dilatation in Wilson's case was only apparent, and that the diffuse impulse was due to forcible beating, adding at the same time that he did not believe that either dilatation of the heart or the strained heart existed. Those of us who believed that strain had sometimes injured a normal heart were prepared to consider whether forcible beating might injure it. Lewis (1932) recorded examination of the heart under the x-ray screen, with inspiration held and the glottis closed. He states that it is seen to dilate very greatly. There was abundant evidence that healthy men could perform this test with impunity. S. A. Smith (1931) wrote of the "erroneous diagnosis of acute dilatation of the heart." But if enough clinical evidence based on the judicious assessment of case-histories is forthcoming it is interesting to speculate that we may come back to the simple conception, which was accepted a generation ago, of acute dilatation from strain. It seems a reasonable possibility in the untrained average man undergoing an exceptional strain with the glottis closed. There may have been some defect in the pericardium or heart muscle, but examination would not have detected it, and disability would not have resulted had there been no unusual event.

It is not a common occurrence. I think I have seen four examples of hearts diseased from strain that were apparently healthy before an exceptional effort. Two have never recovered fully. The diagnosis from the effort syndrome is very difficult. Shirley Smith (1932) summarized the symptoms which simulate heart disease. There is no absolute proof of strain in any of my cases. The diagnosis has been based on the probabilities in relation to the man, the history of effort, and the report from his doctor, although a consistent non-varying degree of disability is taken to be suggestive of genuine heart disease.

S. A. Smith (1931) explains all the cases of what he calls "the erroneous diagnosis of acute dilatation" as due to a cardioneurosis, usually supervening on a minor superficial injury. I have seen a typical example of pain of muscular origin at the lower end of the sternum on the left side, from a lifting effort, for which the heart came under observation. The man could walk quickly without discomfort, and merely required reassurance. The pain passed off in a week or two. He was not a manual worker, but it is reasonable to suppose that if he had been compelled to go back to work that aggravated the pain he might have developed a cardiac neurosis. Pain which persists, however, is not a feature of primary cardiac overstrain.

It may be difficult to decide whether a man has strained his heart or whether he is suffering from the effort

syndrome or, to use the transatlantic term, from neuro-circulatory asthenia. But from the all-important point of view of treatment the problems are not so essentially different. A short rest will be suitable for both; reassurance will help both, as also will suitable exercise and retraining. When it is admitted, as most people will, that in the effort syndrome group a man must sometimes be warned that he is not suited for intense or prolonged effort the practical considerations are identical. The divergence arises on account of compensation litigation on the one hand and of theoretical prejudgment as to whether the normal heart can be strained on the other.

The American teaching (White, 1937) seems to be that heart symptoms or signs that follow industrial strain or accidents are usually of the neuro-circulatory asthenia type, or are due to aggravation of heart trouble already existing. But Donahue (1927), in an article entitled "Accidents and Heart Disease from the Court's Point of View," writes: "We do have a great many cases of dilatation of the heart occurring at work while the employee is under some particular strain. He is lifting some unusually heavy load, or is in a rather awkward position doing heavy lifting, when he has dilatation and becomes sick, sometimes collapsing immediately." If we leave out the word "dilatation" the events are as described. Probably some are due to rupture of pectoral muscle fibres, with a cardiac neurosis superimposed. Probably some are due to the effort syndrome, to which the event has called attention. But the probability still remains that some are due to strain of the heart muscle or of the pulmonary or aortic ring. It would be in the best interests of medical science if such cases came before a medical board. We could study them with a mind more open. In those industries where there is a medical adviser to the firm he should include judicious management of the case in his decision.

We can remember that a generation ago sometimes the strained heart was diagnosed, quite mistakenly, on the evidence of a negligible systolic bruit in a growing boy. Invalidism on this account is a thing of the past. The profession has learned that the effort syndrome requires guidance and retraining, lest a selfconscious heart become permanent. Much semi-invalidism has been prevented by the accepted teaching that the heart is the part least likely to break down from exhaustion in some athletic event.

Our knowledge of physical signs and of their interpretation has advanced. There remain, however, some problems which cannot be solved by these means, and the last word must come from reliable clinical histories. By such histories, from doctors who have made notes, I believe enough evidence may be collected to prove that the healthy heart can be strained.

Summary

The forms of heart disease which have been recorded as the result of direct violence to the chest wall or as the result of strain are discussed. The conclusion is drawn that the physical signs on examination are of little assistance in assessing the diagnosis of trauma. Reliable clinical histories, in the widest sense of those terms, must be assessed with judicious care. This includes such evidence as the patient's own doctor can supply.

The clinical diagnosis of a contusion of the heart is discussed.

It is claimed that there is clinical evidence that the normal heart may become diseased as the direct result of overstrain from effort.

With regard to these two conditions, contusion of the heart and primary cardiac overstrain, it is difficult to obtain the proof that the symptoms in question are due to a genuine heart disability, but the probabilities in some cases are sufficient to justify the diagnosis.

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CHANGES IN THE LIVER OF MALE RATS AFTER CASTRATION AND INJECTIONS OF SEXUAL HORMONES

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Normal life of the organism is dependent to such a large extent upon the various functions of the liver that any changes observed in this organ need to be very carefully investigated. The manifold effects of castration on the different organs, including the liver, of male rats have been described in a previous paper (Korenchevsky and Dennison, 1934a). In most cases about seventy days or more after castration the actual weight of the liver was on the average 18 to 22 per cent. less than that of the control normal litter mates. When the weights were calculated per unit of body weight this decrease was not so great—namely, 11 to 18 per cent.—owing to the fact that castration also caused a decrease in the body weight of the rats. Histological investigation of paraffin sections did not show any considerable changes, although in some cases the cells of the "castration" liver were more vacuolated than those of the normal control (Korenchevsky and Dennison, 1935).

The injections of purified extracts of sexual hormones from male urine (Korenchevsky, Dennison, and Kohn-Speyer, 1933) into castrated rats were followed by an increase in the weight of the liver up to 32 per cent. (actual) and 25 per cent. (per unit of body weight). Histologically (Korenchevsky and Dennison, 1935) no difference