THE INVESTIGATION OF THE SIGNIFICANCE OF DISORDERS AND DISEASES OF THE HEART IN SOLDIERS.

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DR. LEWIS'S Report on the work done at Mount Vernon on Disorders of the Heart¹ as seen in the soldier is so careful and complete that there is little, at present, to add to it, and the comments I am requested to make upon it can be but few.

One main result of the investigation is to strengthen the opinion which, before these researches were established, was gaining ground—namely, that "heart strain," a phrase a few years ago in common use and application, is a rare event. In the article on Stress on the Heart in Allbutt and Rolleston's *System of Medicine*, an article which expressed the opinions at that time of Roy, Michell, and myself, we likewise had affirmed that "for one case of disability due to strain, or even of sharp overstrain, there are fifty of secondary and incidental derangement." And again: "As the animal mechanism attained many wider and more various powers of survival yet scarcely even a momentary independence of the heart, this organ must itself have attained to an enormous endurance, and resources almost illimitable." But in the same essay, under "Infections," I said that "stresses upon hearts under this detrimental result only too often in (heart) strain . . ." "experience inclines me more and more to refer cases of heart strain to this category"; and I quoted Dr. Arthur Lambert of Harrow who "regarded (in 1904) the specific fevers as the determinant of heart strain in schoolboys; anaemia, general debility and lack of training being subordinate causes. Up to 1903," he added, "I was able to collect a number of cases of 'heart strain,' and during those years influenza was always with us. After 1904 we saw very little of heart strain." With acute cardiac dilatation after such obvious infections as of scarlet fever and diphtheria we are all familiar, and in the article from which I am now quoting I stated that even a "bad cold" might have some such consequence, if during the brunt of it excessive exercise were undertaken.

It was in 1903, when de la Camp's paper² came into our hands—a paper in which the author reported, rather to our surprise, that the heart, far from expanding during muscular effort, diminished in mean diameter—that, infection apart, we began to take a different view of cardiac conditions under effort; that, instead of working under dilating stresses, the heart under exertion is normally smaller. Yet had we considered cardiac physics more intimately, we need not have been surprised. It is true that in exercise beyond the capacity of the myocardium the blood is heaped up in and near the right heart at the gates of the lungs; and this the more when the individual is imperfectly trained, or is becoming stale. But as rate and velocity increase, and the periphery expands, the output per beat must be less; and therewith the cardiac diameter. Furthermore, as the heart has its own blood supply, blood runs round the heart many times for once round the systemic area.

The heaping up of the blood in the right heart and veins is heavier, and tarries longer, in the untrained. Training is something far more than dietetics and muscular exercise; in the circulatory concert it means a development of the thorax, an unfolding of the lungs, and a swifter interplay of vascular, nervous and chemical responses.

In former papers I have described how in the first few minutes of increased effort—for example on starting to climb a steep hill—the arterial tree, as represented by its radial branch, opens out, not gradually but suddenly; and the better the training the sooner this expansive readaptation takes place. Under this change—this great reduction of friction—it is intelligible that the diameter of the left ventricle should diminish, or the chamber empty itself more completely. But in a man growing stale under training the vasomotor mechanism loses its tone, and its responses are enfeebled. There is no better test of the "fitness" of an athlete than to draw the finger-tip sharply down his cheek, when, if he be "stale," the red line will be strong and persistent. In many cases of "D.A.H." at

Mount Vernon this loss of vasomotor tone is conspicuous. The hand when hung downward turns a dusky blue, and its vessels empty when it is held above the head. If the finger be pressed upon such a hand and removed the white patch is long in recovering its colour.

But the researches of Dr. Lewis and Mr. Barcroft at Mount Vernon raise quite a new and a very interesting problem: setting aside for the while poisoning of the myocardium, may incidental infections produce any other effects in which the heart is concerned? The answer appears to be in the affirmative. These researches (p. 19) seem to discover a new disease, or rather to discriminate more exactly the features and nature of a disease less clearly apprehended already, by Da Costa and others, as "soldier's heart"; or by those of us who have written upon "neurasthenia" as "cardiac neurasthenia." The disease is one which is not uncommon in the civil population; but under the stress of the soldier's training, and amongst the aggregations of men in modern armies, it is more conspicuous and inconvenient. It is of the kind of diseases known to our fathers as "*morbi sine materia*"—a disease without a lesion. "Disease" is a clinical not a pathological term, and consists in a series of symptoms recurring with a fair uniformity. At Mount Vernon I have been much impressed by the uniformity of the series now chiefly under consideration. It consists in the main of the following terms or symptoms:—submammary pain, palpitation and quickened pulse, shortness of breath on exertion, tremor, exhaustion, "dizziness," and certain vasomotor phenomena. This group of symptoms is too uniform to be fictitious or fantastic.

Now it will be seen from the Report that in these cases—in this disease—although not rarely an infection takes some place in the history, the heart is mechanically unaltered. I am glad to add my testimony to the great care and precision with which these mechanical appreciations were carried out; the orthodiagraphic observations of Major Meakins and his colleagues being especially diligent and valuable.

The outcome so far then is that the fretful heart, known as the "soldier's heart," is not cardiac strain; and, whatever the explanation may be, for this I refer to the Report, there is this gain to nosology—that although, in the past, heart strain and what for temporary convenience I may call still "soldier's heart" have been confused, and even classed together, this disorder and that are now made separate and distinct. For "soldier's heart" we need a better name, and, if it is to be Greek, I have suggested ponopalmosis, a compound word which means palpitation on effort. Therefore, with a few chance exceptions, from the Mount Vernon category "heart strain" is dismissed.

Now by this selection I do not mean that heart strain does not occur; but that it is generally a result of some infection, is relatively infrequent, and does not constitute the bulk of the cases invalided from the army as "D.A.H." (see Report, p. 54 f.). With the gradual and inevitable deterioration of the myocardium after maturity—say in man from the age of 35 to 40 onwards—I have not now to deal. I have discussed this grave question in my *Diseases of Arteries*.³ At Mount Vernon nearly all the patients are under this period of life. This is one, and the first in importance, of the principles obtained under Dr. Lewis's research and direction. In the article referred to in the *System on Overstrain of the Heart* the two classes of cardiac disorder were more or less confused; the cases which Roy and I and Michell studied were, many of them, it is true, cases of actual heart strain; but effects for the most part, if not in all instances, brought about under the influence of some infection. Moreover, in them the heart, the right side of the heart usually, was definitely dilated and the valves for a while often incompetent. In "D.A.H." the form of the heart is unaltered and the dyspnoea seems not to depend directly upon the heart.

Here however I would venture on some demur to the disrespect with which certain cardiac murmurs have been treated by Sir James Mackenzie—to whose researches we are all so deeply indebted—and by his disciples. This is an extreme reaction against the obsequious regard for these signals which not unnaturally prevailed in the generations immediately after Laënnec. It is true that in concluding "that the presence or absence of murmurs (wherever they may be audible) is of no value in estimating the soldier's

capacity for work." Dr. Lewis premises that he concerns himself only with the capacity of a man for military service for a subsequent period of undefined duration. This I agree is true; under my care at this moment is a man, aged 32, suffering from mitral regurgitation, now to the degree of large dropsy and dyspnoea, and in a condition beyond substantial remedy. He had rheumatic fever at the age of 6, and was then told that his heart was affected. But thereafter nevertheless for some twenty years or more he had been at work as a labourer. Last year I had a similar case in a man who, after rheumatic fever, with damage to the mitral valve reported at that time, had notwithstanding been occupied since as a coal-heaver for sixteen years; and for the while with impunity. Of such cases I think we must admit that though for many years the mitral regurgitant murmur had not indicated immediate physical incapacity, yet it did indicate cardiac disease, and sooner or later cardiac failure. That out of such a man the State may nevertheless get a few years' work, is a decision which at Mount Vernon we may very properly make without cynicism; as Dr. Lewis says, at Mount Vernon our business is only with capacity for farther service; still, as he admits, the diagnosis, or prognosis, is "open to criticism on academic lines";—that is to say, from the patient's point of view? And not the diagnosis only but also the rule of life.

Here may I remark that the phrase "back pressure," in common use even by expert writers on affections of the heart, is to me unintelligible. Surely by an increase of resistance in front the blood pressures must be raised in the whole area concerned; and equal in all directions, fore and back and lateral. To students at any rate, not to mention some of their seniors, this fallacious phrase suggests that the blood stream is reversed! In the generation of many a loud murmur the regurgitant wave, in the strict sense of the word, may be very small; possibly none, as a fluid vein and turbulent collisions of the particles of the blood may be caused by changes in the relative diameters of the chambers only. Or again, like a stream from a tap which when turned slightly on may be noisy, but when "full on" almost inaudible, so the slenderest thread of regurgitation may make a loud murmur. Constancy in the degree and quality of a murmur, as it suggests stability of structure and compensation is therefore, on the face of it, a good sign. In all cases of definite systolic murmur at the apex I believe there is regurgitation, whether the condition be temporary or permanent. I see no other explanation of the change of linear into turbulent motion.

In the class of cases we are considering, functional "murmurs"—that is murmurs dependent upon transitory causes—are very common; and I think we are bound not to ignore them but to do our best to analyse them and discriminate between them. This I think is not so difficult a task as it is often said to be. On the one hand in the "soldier's heart," as Dr. Levy pointed out to me, a systolic murmur having its chief seat along the left costal arch is very frequent; one would guess it to be but a whiff of air expelled at each jerky heart stroke from the lappet of lung. So again in the healthiest young man we may hear a systolic murmur over the area of the pulmonary artery; by this idle noise, although usually due merely to a slight impingement of the vessel upon the wall of the chest, and of no importance, many a lad has been kept out of the football field. Let us first eliminate all these insignificant noises from the list, and then on the other hand concentrate our more serious attention upon murmurs dependent on altered mechanical states of the heart; though, as even these are often transitory, a farther subdivision is necessary.

And for the present let us leave out of reckoning murmurs about the base of the heart, and direct our attention to those referable to the mitral and tricuspid areas. Even when these are transitory, as for example during convalescence from an infection, it seems certain that when systolic they signify valvular disorder; the valve, or more probably, in transitory cases, its bed, is awry; and the patient, so long as the murmur remains, should be treated on this assumption. For a murmur signifies the existence of an abnormal fluid vein, and an abnormal fluid vein means abnormal friction, and friction is a waste of energy. A young, vigorous, and normal heart, with some readjustment, may afford to waste this energy—for a while; but a perpetual waste, in an organ whose reserve is so precious, cannot go on without ultimate harm. It is a

handicap, and the efforts cast upon such a heart should not be immoderate.

In looking back upon many years of practice I recall very vividly, in respect of the present subject, the subsequent lives of many a patient in whom a mitral regurgitant murmur was for years the precursor of subsequent cardiac incapacity and ultimate failure. For the change from linear to vortex motion meant a fall in velocity, save in so far as the muscular structure of the heart at the part was buttressed. For many a year perhaps a young or comparatively young heart, out of its abundant stores, can meet excessive demands, and build itself up at threatened points; but if the interval is often a long one the event is none the less manifest. The murmur in crucial cases has been discovered accidentally; as for example in an examination for some temporary disorder, or for life insurance; though cardiac incapacity may not have ensued for years afterwards. After an uncertain period however the heart begins to enlarge, and the patient to feel a little transient dyspnoea on unusual effort; this symptom increases, and the heart begins to make itself felt; yet even thus the patient may live still for a few more years, and under due precautions do not a little work of a sedentary kind. It is then that effort tests may betray myocardial default, but the patient has then entered not upon his disease but upon the last stage of it. Then nocturnal dyspnoea will appear, the ankles puff up, and other symptoms of the final phase of the malady accumulate. Perhaps after this lapse of time I may refer to the first case of this kind of which I had any knowledge—that of my old master and friend Bence Jones. He bought a flexible stethoscope, then a novelty, and tested it first upon himself; unhappily a mitral regurgitant murmur made itself heard, and although this distinguished man lived on without suffering for a while, and in his impulsive way would still forget himself so far as to fly up the staircase at St. George's two steps at a time, yet gradually the increasing friction in the blood, and the consequent fall of velocity, made themselves felt, and brought about his premature death. Scores of such cases arise in my mind as I turn my eyes to the past; some of mitral regurgitation consequent upon an infection, some upon slow athero-sclerosis of the valve, and so forth. For, I repeat, an endocardial murmur means a fluid vein, an eddy; and an eddy means friction and a disturbance of linear motion; these, unless in some indifferent area, mean sooner or later a breakdown of the machine.

On one other point I would spend a few words—namely, on *latent mitral stenosis*. In consultation the question often arises whether or no in a particular case there is a murmur at the apex. "Murmurish," says one; another that the first sound is prolonged; another that the first sound is reduplicated, but no murmur. No "murmur," it is true; the added sound may not be murmuring nor whispering, but an addition to, or a hitch on the ordinary first sound. The first sound may seem divided, with a notch as it were, in the middle of it. Now the addition may be before or after the true first sound; in the latter case, sooner or later it murmurs more definitely, and betrays a mitral regurgitation. With this I am not now concerned; in the former case it is a sign of a mitral stenosis, slight in degree and "compensated." Now in this case a thrill—short perhaps, and narrow in area, but unequivocal—may, if carefully sought for, be detected. But in the absence of cardiac deformation, of definite murmur, and of proper symptoms, this thrill may not be looked for. These latent cases turn up in practice, civil or military, and, if not readily picked out, may be reckoned among "our failures."

As an illustrative example of latent mitral stenosis I may take the case of Mr. B., a patient of Dr. Johnson of Cambridge whom I had seen some years before for neurasthenia with irritable heart, to which he was, and still is, liable. Dr. Johnson sent him to me, in respect of a call to military service, with a note of a faint and variable presystolic murmur. He had had rheumatic fever but with no record of cardiac affection. The first sound was short and snappy, but his heart had always been irritable, and on this visit I could not hear any murmur unless it were, for a beat or two after putting him through some exercises, a very fleeting presystolic sound—so fleeting as to be hard to define or record. The heart's dimensions were to percussion normal. While murmur hunting I forgot till the last moment to feel for a thrill; but then I found a short apex thrill clearly perceptible, and presystolic in time.

Some ten days later I examined him again, and on this occasion a presystolic murmur was audible, brief and soft but decisive. Oddly enough, now the thrill had vanished; again and again I tried to find it but in vain. So capricious is this disease in its physical signs, and so elusive are its signs, in respect of military service or of life insurance. I am not as yet ready to accept a definite distribution of stenosis murmurs into early, middle, and late. Again, a young friend of mine, very active both in mind and body, and anxious to serve in the army, has been rejected on two occasions for "soldier's heart." He has a slight addition to the short first sound and a short presystolic thrill. He has never had rheumatic fever nor other illness.

We have had a complicated illustration of this phase in the Mount Vernon Hospital. With Dr. Wilson and another colleague I examined a man with the obvious signs of aortic regurgitation. The question arose whether or no a divided first sound, audible at the apex only, signified a reduplication of the first sound. This it scarcely seemed to be; the taps were too close and the first one too light and brief; but I thought I recognized in it such a split in the first sound as I have described. The carotid beat being, as usual in such cases, only too definite, we were able to conclude that the first tap of the duplex sound was presystolic. Following this up we detected a presystolic thrill, short and light but quite distinct.

A thrill must be very carefully placed and timed, for in many of these irritable hearts a systolic thrill can be felt; perhaps in most of them if some effort be made just before examination. It is short but often very distinct to the touch. It is not felt about the base of the heart, and is therefore not due to a slack aorta. How the thrill is generated it is not easy to say. Very often in these cases a systolic murmur at the apex is recorded, an alteration of the first sound, more like a prolongation of it with some change of quality than like a definite murmur of mitral regurgitation. To suggest the cause of this thrill is but to guess; but, if I may guess, my suggestion would be that in these cases the papillary muscles and their chords may be a trifle slack, so that the mitral sheets shiver a little in the current, as a sail shivers when the boat gets near the wind. Such a quaver would be transmitted to the ventricular wall, and thence to the thoracic. In such a condition there might well be a minute, inconstant, and insignificant regurgitation also; though ordinarily in mitral regurgitation there is no thrill.

In conclusion, I hope it may not be unbecoming in me as a member of the staff, but rather an onlooker than of effectual service, to offer my tribute of praise to the author of this Report; a brief summary of infinite pains, of his own and of those of his colleagues who are and have been on resident duty at the hospital.

REFERENCES.

¹ Medical Research Committee's Report upon Soldiers returned as Cases of "Disordered Action of the Heart," or "Valvular Disease of the Heart." Spec. Rept. Ser., No. 8. ² *Zeit. f. klin. Med.* ³ See C. Allbutt's *Diseases of the Arteries*, ii, 475.

ON GUNSHOT INJURIES OF THE CHEST.

With Especial Reference to Haemothorax.

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WOUNDS of the chest do not as a rule reach the bases in France until the fourth day after the infliction of the wound, except in periods of heavy fighting, then they may arrive much earlier, that is, on the second day. The observations recorded in this paper deal more especially with cases observed from the third or fourth day up to about three weeks after the occurrence of the injury, and thus they deal with only a portion of the subject. Gunshot wounds of the chest are usually divided into those penetrating and involving the lungs or other thoracic viscera, and the non-penetrating, although it is not always possible to be certain during life that an apparent non-penetrating wound is really such. Non-penetrating wounds may, however, give rise to pleural and pulmonary lesions, and in a certain proportion of cases of simple clean perforating

bullet wounds of the chest the positions of the entry and exit wounds suggest that the bullet does not only pass through the chest but also through the lung, nevertheless there are no signs or symptoms indicating the presence of either air or fluid in the pleura, and such cases may recover rapidly and apparently completely. Pleurisy and empyema are the most common pleural lesions produced by wounds of the chest wall not involving the lung. The pleurisy is often at first dry, but not uncommonly an effusion follows, and since such lesions are often due to streptococcal infections spreading from the wound track in the chest wall to the pleura, an empyema of the usual type develops and requires the usual treatment.

Haemoptysis, haemorrhagic infiltration of the lungs, and pneumonia of a septic bronchopneumonic type are the pulmonary lesions that may follow on a wound limited to the chest wall. In addition to these, collapse of the lung of a massive type may occur on the side opposite to the injury even when the latter causes no pleural or pulmonary lesion on the side injured. The haemoptysis and the septic pneumonia are both directly associated with the bruising and consequent bleeding into the lung produced by the impact of the missile on the chest wall and the resulting injury to the chest wall and lung. The haemoptysis may not only be severe, but may also run a long course, and some of the most severe cases of haemoptysis seen by the writer in a very large series of chest cases were those in which the missile only bruised the chest and had not only not produced any wound, but where there was no evidence of any fractured ribs.

The bruised and blood infiltrated lung is very prone to become infected either from the lung or by the spread of infection from the wound track in the chest wall, and hence pneumonia of septic or of a bronchopneumonic type develops. In some cases wounds of the chest wall lay open the pleural cavity but do not involve the lung, a portion of the chest wall, more especially in its lower parts or at the back, is carried away and a gaping hole, often of considerable size, is left. In such cases the lung does not as a rule undergo complete collapse, although the pleural cavity is in free communication with the air. In fact, the volume of the lung seems to approximate to that seen after death when the pleura is open, and therefore the pulmonary collapse is markedly less than that seen in ordinary pneumothorax where, as is well known, very complete collapse occurs, yet in the latter cases the intrapleural pressure may not be above that of the atmosphere.

Penetrating wounds of the chest usually produce one or more of the following conditions: subcutaneous emphysema, haemothorax, pneumothorax, laceration of the lung. These conditions may not only arise where the wound is one obviously involving the chest, or where the wound of entry is near the chest—for example, the neck or the abdomen especially in its upper part; but wounds in other regions—for example, the face or arm—may also give rise to haemothorax or pneumothorax from the course of the bullet being peculiar owing to the position of the soldier at the moment he is struck. In such cases there is often only a wound of entry, and this may be small and inconspicuous, but yet a large haemothorax or even a pneumothorax may be present.

Further, in wounds of the chest the haemothorax or pneumothorax may be on the side opposite to the wound of entry; the missile taking an oblique course may enter on one side, more especially in wounds of the back, and follow such a course that it does not open the pleural cavity on the side of entry, although opening that on the opposite side without or with a wound of exit. Again, in some instances a bullet may enter and leave the pleural cavity on one side without causing any appreciable lesion on that side, and yet so wound the pleura and lung on the opposite side as to cause marked lesions.

Bilateral haemothorax is sometimes seen as a result of a single wound the track of which is transverse or oblique, and haemopericardium may occur in association with either unilateral or bilateral haemothorax.

Unilateral wounds, however, may cause bilateral effects owing to the development of contralateral collapse or contralateral pneumonia.

The subcutaneous or so-called surgical emphysema so often present in chest wounds is usually limited to the vicinity of the wound or to an area of the chest wall adjacent of variable size. In exceptional instances it