An Address

ON

THE PHYSICIAN AND THE PATHOLOGIST ON HEART FAILURE.*

BY

SIR T. CLIFFORD ALLBUTT, K.C.B., M.D.CAMB.,
F.R.C.P., F.R.S.,

REGIUS PROFESSOR OF PHYSIC, UNIVERSITY OF CAMBRIDGE.

IN my youth it was a common jest to ask what N. or M. died of, the witty answer being "shortness of breath of course." The jest has vanished, but the platitude remains, with some alteration of the formula, which, instead of "shortness of breath," now runs "of heart failure." In the obituary notices of our current journals we read daily of these deaths by the half-dozen, that N. or M. died of "heart failure"; and the vogue of appendicitis is threatened. Now, do we owe this new fashion to the physician or to the pathologist? for it is often worth while to examine the straws floating in the wind; we may learn something from some of them. Is this a parrot phrase put up as a kind of shorthand between the doctor and the registrar of deaths? or does it, like appendicitis, signify a fresh point in pathology, claiming its catchword? Does the phrase mean that a snap of the heart stopped the whole machine, otherwise capable of working on, as a snap of a connecting rod may stop the engines of the *Olympic*? or does it mean that the heart was the *ultimum moriens*, and that, so long as this central organ held to it, death was kept at bay? In this case to say that one died of "heart failure" is surely as empty a truism as to say that he died of "shortness of breath." On the other hand, as it is true that we have of late years learned more—much more—of the heart's work and of its diseases, both on the clinical side and on the side of pathology, so the new phrase may be a shadow thrown by some shift of position—whether clinical or pathological—in our view of the heart and its functions. Four or five years ago, when engaged on an article on the treatment of heart disease for Musser and Kelly's *System of Therapeutics*, I was brought closer to this problem, and found the answer so difficult that it has occupied much of my attention since; and to-night I ask you to help me towards some solution of it.

Now, if I appeal to a clinical physician, asking him what he knows about "heart failure," I shall get a ready answer; I shall hear of many a case of sudden and mortal stoppage of the heart; of heart failure in infections; of cardiac dilatation and defeat under high pressures, venous or arterial; of senile decay, so corroding the heart that it can work no more; of angina pectoris; of aortic regurgitation, and so forth—his data are in plenty; his opinions decisive. And likewise, when I betake myself to the pathologist, I find him no less ready for me: from him I hear of fatty degeneration; of "cardio-sclerosis"—whatsoever this may mean; of rheumatic, diphtheritic, and such-like myocarditis; of fibroid disease of the ventricular walls; of sapping of the *a-v* bundle, and so forth; data again in plenty, opinions decisive. But when I desire to link up the two classes of information; when I confront the physician with the pathologist and seek to correlate the times, periods, and modes of disease and death the one with the other, I find myself but little nearer to practical guidance than I was before. Of course, we all know that a "fatty" heart is not so good as a sound one; we all know that a senile heart will not sustain the attack of influenza like a young one; we all know now that damage of an *a-v* bundle may disconcert the rhythms; but when we ask the physician to draw some parallel in the living patient between the formidable modes of decay described by the pathologist; how and when these degradations are manifested; how, in the long course of cardiac decay, the imminence of sudden death is to be foreseen and provided against; or how, indeed, we are to know that any such process is at work at all; or, lastly, how in a case of known heart disease the degrees of its advancement and of the cardiac reserves are to be noted and tested—then the physician retires from the argument. And so with the

pathologist: when we show him a heart from a case of sudden death he will confidently demonstrate to us on the specimen why a heart so diseased could not have gone on; if beside this one we place another heart—still more diseased, it may be—and tell him that the possessor of this heart lived for long enough, and perhaps in the end died of something else; or if we show him another heart in which, by the poison of rheumatic fever, the tract of Tawara was eaten away, and yet, notwithstanding, there was during life no dissociation of auricle and ventricle, he in his turn retires from the argument, saying that he is not concerned with clinical conundrums. Thus we have the physician and the pathologist trotting each on his own side of the hedge, each intent upon his own scouting and his own bearings, and neither able as yet to reconcile his own observations with those of his comrade, both of them being perhaps a little too indifferent to the need of mutual counsel and comparison.

Whether it be, then, for lack of mutual co-operation or because of the perplexities of the subject, the interpretation of cardiac pathology in terms of clinical medicine so far from becoming clearer has darkened, and the intricacies of the path have become more baffling. Our fathers, knowing less of the entanglements of the subject, and newly provided with a collection of the blunter facts of morbid anatomy, took the matter more easily. That people died suddenly of heart disease was for them an old story; when, therefore, they were introduced to diseases of the valves of the organ, and to coarse lesions of its substance, their difficulties were almost solved. The patient is dead, here is the lesion which caused his death; what more does one want? But, as pathology became more and more curious, these simple solutions became less and less acceptable. Kirkes, Bence Jones, Wilks, Fagge, T. H. Green, and many others with them, began to demur. In the dead man they may have discovered lesions enough, and apparently more than enough, to cause his death, but the man had died suddenly, and the lesions under demonstration were of long standing, or in a series of such cases were very various in seat and nature. Again, in a series of such cases valves were diseased and not diseased; kidneys were diseased and not diseased; atheroma was considerable and inconsiderable; the ventricles were dilated or were not dilated; the myocardium was in extreme decay or virtually normal; and its lesions were now focal, now diffused. Thus gradually it dawned upon the pathologist that the problems of cardiac failure were not so simple as in the rise of morbid anatomy had been supposed; and upon the physician, that his clinical methods were fallible indeed, far more so than had been supposed during the rise of stethoscopy. By what system of compensations was it that the same heart could be at once fairly competent clinically and yet pathologically the seat of old and intimate decay? By what system of compensations could a heart go on fairly well, or indeed without suspicion, while its structure was being undermined? Surely there ought to have been some stage of incompetency or at least of falter before the sudden collapse?

In no organ is this lack of concord between the signals of life and death so disconcerting as in the heart. In all living structures there is a factor of safety, that potential which in the heart we call reserve, but in none, perhaps, is it so capacious as in the heart. Yet of this reserve, of what the heart can do in case of need, we have no scientific measure; the microscopist cannot detect it, the clinician has no valid tests for it. At one time it maintains a defective heart; at another time it betrays a heart which, to all appearance, if not impeccable was at any rate not plainly corrupt. So more thoughtful observers began to put the matter more cautiously, and to say that "when a patient succumbs to heart failure certain degenerative changes are usually found in the myocardium and vessels, and these are supposed to account for the heart failure." Evidently even yet some more refined investigation into the phases and intricacies of cardiac qualities must be instituted if these apparent caprices, clinically speaking, of the heart of man are to be explained, computed, and foreseen. "Heart failure" is evidently a very composite notion—one which must be submitted to a no less penetrating analysis. We begin to learn that in the heart, as we learn in less striking ways in other forms of organized matter, that complexity of structure and conditions mean

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not less but more stability; that equilibrium endangered in one direction, or in more than one, may be supported by readaptations of other factors of the concert; so that the organ, if it may not be restored to its pristine capacities, may be steadied into some tolerable adequacy such as, under favourable circumstances and within limits not too wide, to preserve it from default. The heart of an active undergraduate slightly and temporarily impaired by a sharp catarrh may by the stresses of rowing become gravely harmed and give rise to unmistakable clinical symptoms, while a senile heart far more radically vitiated may amble comfortably along the serener paths of tranquil old age. *Non intelligitur quando obrepit senectus.*

To pass from these general reflections into more concrete observations, we may begin either with the clinical or the pathological side: as the clinical side is the field of practice, let us begin there; remembering how Goethe taught us that the general is always to be found in the particular. In the clinical field we shall first be impressed by the part of the heart in almost all diseases, and under conditions such as anaesthesia. In infections we know that the heart is concerned in three ways: in some of them it is directly attacked; in others it seems to suffer rather from a general depreciation in common with other parts and tissues, an impairment due apparently to pyrexia or to some more diffuse effect of the specific poison; in others, again, from a dissociation from its peripheral complement, from a loss of tone in the "peripheral heart," so that it beats the air in vain. Now, for these different conditions have we any crucial symptoms by which to distinguish them? Have we in a particular case any criterion by which, for instance, in an infectious disease, we can discriminate myocardial lesion, myocardial enfeeblement of a more general and temporary kind, and slackening of the peripheral circulation? Moreover, these several factors overlap each other, cross each other, and supplement each other inconstantly, and in degrees of which appreciation seems very difficult. Nay, we are well aware that in infections—as in diphtheria, scarlet fever, rheumatic fever, pneumonia, etc.—a diagnosis of direct attack upon the myocardium is usually, perhaps, beyond our methods; if in rheumatic fever dilatation is not an infrequent accompaniment, in other infections, as in diphtheria, it is at least frequently absent. And in chronic myocardial deteriorations is it too much to say that in the majority of cases of sudden or rapid death the underlying condition is not detected till once for all on the *post-mortem* table?

From and since my student days attention to cardiac disease has taken various directions, dependent on the various improvements of our means of diagnosis. As a student I witnessed the developments of auditory diagnosis—methods which certainly served to detect many cardiac diseases, such as pericarditis and mitral stenosis, not decisively demonstrable before. Then, by the efforts of Sansom, Ewart, Lees, and many others, especial emphasis was laid on percussion as a means of measuring the dimensions of this chamber or of that—a method by later disciples pushed to extravagant lengths or used with indiscriminate assurance. The sphygmograph came next, and from it more was expected than it could give; though twenty years later it was restored to practice by James Mackenzie with largely developed powers; still even thus improved the instrument could tell us only certain things, chiefly facts about rhythm—facts important enough, but, excepting occasionally in respect of pulsus alternans or dissociatus, throwing little light upon myocardial values. Meanwhile pressure gauges were to open out the way for us. Inventors made and improved these instruments, and much was attained in measurement of maximal and minimal pressures and of pulse amplitudes; but after a while we found that we were, perhaps, as far as ever from appreciating the states of cardiac reserve; as much as ever in cardiac disease we were taken by surprise by sudden deaths. Again, the *x-ray* method, and at this moment the method of the electrocardiogram, are, or were, to place in our hands tests of myocardial dynamics; but hitherto we have had to be content with facts which, however interesting, do not give us the intimate criterions which we desire; for cardiac potential we are still without any gauge.

From these introductory remarks, gentlemen, you will infer, and infer correctly, that I do not intend to dwell upon what I may call broadly Valvular diseases of the

heart. In the valvular diseases we can often, generally perhaps, form some approximate forecast; the conditions are more frankly mechanical: in some of these cases the myocardium may count for much, but generally speaking it is the static structures rather than the dynamical potentials which determine the issue. If in a few instances, as in aortic regurgitation, the heart may drop out without warning, yet as a rule valvular diseases of the heart do not end suddenly; as a rule we may rely on it that a comparatively intact muscle will fight with fairly good reserve forces till defeated by accumulating venous stresses, and by those regional falls in velocity often known by the rather inept term of "backward pressure." And of these shortcomings we have for the most part no insufficient indications in symptoms and signs.

From the bearing of my essay it will appear that in the class of cases to be discussed I can say little about symptoms; for my point is that, even in the gravest cases of myocardial lesion, symptoms may be none; still, there are a few remarks of a critical kind to be made concerning alleged or occasional symptoms. Moreover, the problem of sudden death—that is, of absence of symptoms during the main run of a myocardial lesion, even to an extreme period—is not materially altered, if we include certain other cases in which, indeed, some symptoms did precede death, but did not put in an appearance till the lesion was far advanced. Dyspnoea, for instance, or falterings in the pulse, or, again, syncopic phenomena, if they appear at all, may not, and commonly do not, appear till the malady has entered upon its final stage; so that the issue is removed by but one step from a sudden death.

But, before considering these admittedly late and almost irremediable symptoms, let us dwell for a few minutes upon any symptoms or signs from which more timely warning may be obtainable, such as dislocation or readaptation of the chambers; murmurs, unless as superfluous signs of demolition, we may disregard. Hypertrophy of the ventricles is so alien to intrinsic degeneration of the myocardium that we may pass it over; but in passing I would say that the alleged intrinsic instability of hypertrophy—say of the left ventricle—apart from the general instability of the diseased individual so affected, seems to me to lack any serious proof. Apart from the causes and conditions of the hypertrophy in the particular case, I know of no evidence that the mere hypertrophy depends upon any encroachment on the normal reserves of the heart. So far as evidence yet goes, a hypertrophied myocardium is as good as a normal muscle; if the need for it be temporary, the part will recede to its normal dimensions, as if no such change had ever taken place; nor does a persistently hypertrophied myocardium stand at any intrinsic disadvantage. Whatever essayists may argue, we do not know that it is more easily fatigued, or more liable to degenerative disease; it stands at the disadvantages in which all parts of an embarrassed organ are involved, and no more.

Dilatation we cannot dismiss so readily. It is true that there is nothing very stealthy about a dilatation which is an accommodation for an excessive output or residuum; and, indeed, we need not take alarm at a dilatation significant of a loss of tone, if we have reason to believe that it depends on this only, and not upon a textural dissolution. If, however, it be a result of myocardial disintegration—as, for instance, in a heart of fatty infiltration in an obese person—a state which is nearly always associated with an unknown amount, often considerable, of fatty degeneration also, then we may well be anxious. Unfortunately, in these persons, with their brawny chests, an accurate delineation is not often practicable. But here we have to halt, or even to return upon our steps; the wall of an enfeebled heart, so the unwary would say, must yield, and yielding signify to the careful observer an increasing dilatation. So it would seem, no doubt; but so it is not—not as a rule. It is a strange thing, but one too generally acknowledged to need arguing here, that more often than not a degenerate ventricle does not dilate; not rarely it diminishes in bulk. There is no direct relation between dilatation and tissue change.

I am disposed to mistrust too ready a resort to logical explanations by severance of one cardiac factor from another; still it would seem as if the endowment of tonicity might maintain in position even a decayed—an extremely decayed—ventricle. Of the explanation there is much

doubt; of the absence of dilatation in such cases, even in the majority of them, there is no doubt. And it is still more certain that dilatation is no gauge of the histological condition of the myocardium; in some cases, as in acute rheumatism for instance, an impaired ventricle may be attended with dilatation; in others, as for instance in diphtheria, no such correlation may be found, usually is not. The difference may lie in a contrast between focal lesions and diffuse. A wider percussion area after effort is a test full of fallacies. We cannot admit, then, with the Leipzig school, that dilatation is any measure of the quality of the myocardium.

If, then, the delineations of the heart's dimensions which we obtain by percussion, or even by *x* rays, give us no constant or trustworthy criterion, what of auscultation? Well, we are but little better off. The textbooks complacently repeat that feebleness of the apex beat, falling off in the tone of the first sound, and prevalence in the same area of a thin acute second sound, are signs of myocardial degeneration. So they may be; but they are neither obligatory nor, when present, conclusive. In cases of atherosclerosis, with substantial myocardial deterioration, the impulse may be increased, usually it is not reduced; the sounds also may be good enough, or a snappy second may signify no more than a stiffened aortic valve, a condition of no great relative importance. Yet such patients often die suddenly, and at the necropsy the left ventricle may be stuffed with callosities, and the muscular largely supplanted by connective fibre. The contrast of small pulse and full apex beat need mean no more than a large area of vaso-constriction. And, as to defect of impulse and defect of sound quality, let any experienced auscultator ask himself how often he has found himself misled by ill definition of an apex beat, by a poor first sound and a thin second—severally or even together—in patients who were little or none the worse for the peculiarity. Among scores of such instances I recall two, both in men approaching threescore and ten. One of them had been reduced in health by business mishaps and by a gall-stone abscess; the other after many years of keen and arduous work was out of heart and anaemic. Neither was obviously emphysematous; yet in both the cardiac impulse was but feebly perceptible, if at all; the first sound was poor, the second thin and extensive. In both cases anxious consultations were held with "cardiac experts," and in both a diagnosis of cardiac degeneration was pronounced with little hesitation. The one lived thirteen years afterwards, and died at the age of 83 of some incidental illness; the other ten years later, at the age nearly of fourscore, is still giving his distinguished services to his country. These out of a many such, prove that a fagged or anaemic patient may without any grave affection of the heart nevertheless present for a while the defects of impulse and the quality of sounds which in another might truly signify such a flaw. These equivocal physical signs, then, are not criterions.

To turn from signs to symptoms. Syncopic or vertiginous phenomena often accompany cardiac default; but, whether from dyspepsia, slight labyrinthine disturbance, vasomotor atony, or other obscure cause, we are too familiar with this kind of symptom to rely upon it as crucial in the diagnosis of failing heart. If it be added to other signs and symptoms of heart disease, it may indeed enter, and enter gravely, into a diagnosis as cumulative evidence; but taken alone, or indeed without strong corroboration, it is a very equivocal item.

But surely by the pulse we ought to be able to make a fair inference concerning the state of the heart. Yet even here again we are baffled. It has, indeed, been asserted again and again that the profounder changes in the myocardium are betrayed by an irregular pulse; but as our experience of cardiac disease has widened we have learnt that in the rhythms we have rarely any criterion of cardiac values. Impairment of certain tracts should, so we think, be betrayed by altered rhythms, but we have no definite guide even here; we have learnt by autopsy that even the tracts of Tawara may be seriously undermined by an infectious disease without derangement of rhythm, so likewise may the nervous masses in the coronary sulcus. I think I may rely on a general agreement—an opinion definitely expressed by von Leyden, Bäumlér, Ebstein, and many others—that in the pulse we have no guide to myocardial damage, acute or chronic.

Positive variations of the pulse may mean little; and under the cover of normal rates, rhythms, and pressures decay may progress to the utmost. Notwithstanding there are two variations of the pulse which, when present, are ominous; these are a persistent rise of rate, and the pulsus alternans. On pulsus alternans I will not dwell, for Professor Wenckebach, Dr. Mackenzie, and Dr. Lewis have reinforced the lesson that this change is, if not invariably yet generally, a herald of myocardial defect. However, as the sign is no common feature of such defect, but a rare one—one far more often absent in failing myocardium than present—it is as a clinical criterion inconstant. Nevertheless, we shall remember Dr. Mackenzie's warning that, if imperceptible to the finger, it may be revealed in a sphygmographic tracing. The only exception to its significance as a note of disease is an occasional appearance of it under the action of some poisons. The researches of H. E. Hering¹ on the causes of pulsus alternans, if not strictly relevant, are so interesting that one is tempted to mention them here. The phenomenon depends, so it appears, on variable refractory periods of the fibres, some fibres being late in response, so that partial asystoles are summed up in rhythmical recurrence. Thus vagus stimulation, by slowing beats, may reduce pulsus alternans and substitute a continuous hyposystole, though occasionally it reinforces it. Such interferences suggest that between the myocardial fibres there are some obscure differences—at any rate, in certain hearts. The accelerans, on the other hand, as it quickens the rate, may increase the alternation or make it manifest by modulations of the refractory discords. Thus the cardiac nerves can not only influence the systolic volume directly, but, by modulating the refractory phases, also indirectly.

In another way the pulse may signify decay of the myocardium—namely, by a persistent change of rate, whether of acceleration or of retardation; though here, again, we have not a constant but an inconstant criterion. We have seen, or shall see, that the tract of Tawara may be profoundly marred without perceptible effect on the pulse; and it is common experience that a normal rate of pulsation is consistent with profound deprivation of the left myocardium at large. Notwithstanding it has been strongly impressed upon me that a permanent change of rate is ominous of failing heart. On bradycardia, or again on tachycardia, I need not insist—these conditions are better and better recognized every day; I will insist rather on the less emphasized sign of simple acceleration. Let the pulse of a certain man be known as habitually 70, at rest 65-70; and let this pulse-rate be found to have become habitually ten more—that is, 85-90, on slight movements rising to 100 and over; and let this change show no substantial alteration under prolonged rest, let it fall, that is, under whatsoever rules of tranquillity of mind and body, never below 80-85; then heart failure is in the offing. If, moreover, with this acceleration there be attacks of faintness, or of slight dyspnoea on moderate effort, the failure of the heart's muscle is at hand; and but little amendment can be promised. Of course, before a final opinion is given, any possible nervous influence will be estimated; and the influence of coffee, tea, tobacco, and the like be eliminated, for in later life such agents often become less well tolerated. Two or three cases of this significance have impressed themselves on my memory: one of them in a patient of Dr. George Johnston, of Ambleside, a country gentleman of advanced years, but for his years of great activity, both of mind and body. In climbing over a stone wall into his own park he was aware of some little faintness, which might well have been a transient affair of little importance. However, he was persuaded to send for Dr. Johnston, who found his pulse at a continuing rate of 100-110. It seemed to the patient and his friends that to keep a vigorous man with all the appearance of health permanently at rest, or only to allow him to drive out in his carriage, was a serious decision. I saw him with Dr. Johnston twice at an interval of twelve months; and in spite of the demurs of his friends, who hinted at hypochondriasis or neurasthenia—neurasthenia does not come on in the later life of persons previously healthy—I could not but support Dr. Johnston's injunctions; for if during complete rest the pulse-rate were reduced under 100, or might fall even to 80, on exertion it returned to the higher numbers. Gradually, in the course of the next eighteen months, syncopic sensations at stool or under any more consider-

able effort, increased upon him, further and more ordinary symptoms of cardiac failure set in, and he succumbed without any abiding amendment. But here again, as with pulsus alternans, we have no criterion; acceleration of rate in myocardial breakdown is an inconstant phenomenon, and in certain peripheral relaxations may appear without any myocardial disease.

Of the many other and more familiar symptoms of heart failure, such as dyspnoea, slight oedema, slight albuminuria, cyanosis of ears and lips, fullness and tenderness of the liver, pulmonary crepitations, arrhythmia perpetua, etc., I need not speak; more or less sudden heart failure is frequent without any of these features; and in any case they usually signify late phases of the malady and speak only too plainly for themselves. Of the many symptoms to be discounted, I will refer only to arrhythmias pertaining to the extrasystolic kind, which in some cases are, to the inexperienced observer, terrific. One of the worst of these clumsy, bustling hearts I examined some time ago with Dr. Davies, of Histon, in an elderly gentleman, who, after a little careful treatment, was safely permitted to go about his business; and now I often see him engaged in public or social concerns, apparently without disablement. Another case, almost as troublesome, I witnessed about ten years ago in a gentleman, now living, then some 78 years of age; at Homburg he made a good recovery from some gouty conditions, and his heart practically recovered its steadiness. Almost as I write, he has, on the borders of fourscore and ten, successfully weathered an attack of genuine influenza. In these and such cases the heart may never recover its normal rhythm, but no harm may come of the disorder. The symptom group of every such case must, of course, be sifted carefully, for, on the other hand, in pneumonia or diphtheria a dropped beat may be the first presage of heart failure.

Sphygmomanometrical observations have proved, so far, to be of no use in the detection of myocardial disease. That under prolonged high arterial pressures the heart may be defeated is true enough; but this is not a myocardial decay. A test of blood velocity, which is probably always reduced, would be more trustworthy.

From the electrocardiogram we may in time get more definite hints. In general as age advances the ordinary height of the F(T) wave diminishes in relation to J(R), and in the dying heart it disappears altogether. It is premature, however, to say that from this variation a criterion of the state of the myocardium can be obtained; still a waning of this wave, and still more a negative oscillation, may prove to have a definite pathological significance, especially if the pauses are protracted. On the other hand, a persistence or recurrence of the juvenile type in adult life signifies a nervous factor. Fibrillation of the auricle no doubt signifies decay of the part; fibrillation of the ventricle may be one of the modes of sudden death, but is hardly consistent with survival.

I must not occupy your time with a discussion of the various devices for testing cardiac reserve proposed by Katzenstein, Graupner, and others; suffice it to say that, while some of them are inconvenient, none is effectual or even approximately indicative of the available capacity of the heart under observation.

Thus baldly I have tried to show from the point of view of the physician how indefinite, how inconstant, how obscure, are the symptoms and signs of myocardial insufficiency; let us see, then, what the pathologist has to say—if he with his scalpel and probe can offer us more definite measures of cardiac capacity. We shall say to him, surely you ought to discover why the patient died? why his heart or that man's heart broke down without warning? or in any case what was the critical point at which its resources could no more avail? But too often in his turn the pathologist gives the same uncertain sound. In this heart he found too little to account for death, in that too much—the patient had no business to have lived so long. On grounds as insecure as those of the physician, he finds one heart which, with comparatively little or even no evidence of decay (Cabot), surrendered at the first summons, or during life was obviously insufficient; and, conversely, another which by the very extent and chronicity of decay testified to the long period during which it had managed with silent tenacity to wage an unequal fight. From the side of morbid anatomy, then, the want of parallel,

of apparent correlation, between the physician and the pathologist lacks nothing of its emphasis.

But, at the outset of this side of the matter, I would with all due deference suggest that in many a report from the *post-mortem* chamber to the wards the histological changes described are consistent with, or even declare rather the effects of the decomposition of death than of living disease. I have had to discard many a note recording blurred striation, nuclear alterations, "hyaline" change, etc., when without any record of date after death, or of the state of the atmosphere. Indeed, so regular is the tendency in the viscera to a focal distribution of their lesions that one may wisely—I will not say reject, but regard with caution, all reports of diffuse and uniform myocardial disintegration, as suggestive of *post-mortem* decomposition.

Of the kind of detriment called "fatty degeneration" opinions of late have undergone some fluctuation; but the term, for a while discarded, seems to be returning into use, though with some difference of interpretation. The distinction between fatty degeneration and fatty infiltration still holds its ground, though the extremes are rare, and between them lies every grade of transition. If in the obese heart much of the fat is to be traced to an inter-fibrous penetration, yet on the other hand in very few such hearts is this penetration unaccompanied with definite deterioration of the muscular fibres themselves. And if in "fatty degeneration" the droplets of fat may be but particles of unassimilated nutriment rather than a disintegration of muscle cell, or a metamorphosis of albuminous structures into fat, and if we cannot say that fatty degeneration alone is a myocardial regression, yet this delay of absorption and assimilation seems to signify some alteration of chemical change, if in rate only, and if so to be not without some modification of muscular energy. Aschoff does indeed go so far as to think this fatty manifestation may signify an excess of physiological activity. It is certain at any rate that, in the midst of it, the fibres and their striations may still be well defined (Aschoff, Lubarsch, and many others). In diphtheria beside the fatty deposits the muscle fibres are also obviously diseased. In old hearts with coronary sclerosis Kent's bundle itself may be thoroughly "fatty," yet with no apparent heart failure. On the other hand, in some toxic cases it would seem that the "fatty" state may be established in twenty-four to thirty-six hours. However, what I have to emphasize is the agreement that even well-marked fatty deposits, severe and diffused, may take place without any clinical evidence of heart failure. In this context, then, pathology can provide us with no criterion. That, as I have recently seen stated, the "fatty heart" is to be known by dilatation or by irregularity of rhythm cannot be maintained; these coincidences are quite inconstant.

In no circumstances is our lack of any criterion of cardiac capacity and of cardiac stability more painfully manifest than in the acute infections. Again and again, in such diseases as diphtheria, young and promising lives are cut short by heart failure without any more definite warning than the vague forebodings cast by the shadows of such cases gone before. The young man or young woman may be going on well, so far as clinical observation can tell us, and yet be within a minute of eternity. Sometimes a persistent increase or retardation of pulse-rate may hint at the danger; but more often there is no such sign of peril. Now, a heart to shut up in this way must have been gravely affected for some little time before. Again, after an infection as mild as a "common cold," more probably after an influenza, the patient has no warning that his convalescence is incomplete until he engages in some effort, such as a game of football, and then finds himself invalidated for many a month to come. Without any clinical manifestation his heart had been the seat of occult disease. Here, again, our only guide is crude experience; we have no criterion. Tanaka² points out how remarkable it is that a "heart often functions well up to the moment of sudden death," yet after death the myocardium reveals disease both extensive and profound. The ventricle may or may not be dilated. In rheumatic myocarditis (Romberg, Cowan, Carey Coombs) sudden heart failure is less common, and usually is so combined with valvular lesions and dilatation that clinical symptoms are not wanting. Interesting as it is from a histological point of view, I must not in this context dwell longer upon it. Nor do

the distinctions, definite or transitional, demonstrated by Dr. Coombs and others, between acute interstitial and parenchymatous myocarditis much concern us here. We will pass on to chronic myocarditis.

In so-called "chronic myocarditis," again, probably not strictly an inflammation but partaking rather of an atrophic or degenerative nature, we find no closer correlation between the clinical and the pathological series. Cabot, in a remarkably able report³—one in which he compared clinical and pathological notes taken independently—says that he has quite given up attempting to diagnose chronic myocarditis; "in its diagnosis is a mere matter of luck. It has no clinical symptoms, no characteristic physical signs." So this point needs no farther labour.

Let us proceed, then, to fibrosis of the myocardium, an alteration which an eminent student of heart disease but a few weeks ago assured me was of great clinical importance. I demurred to this, for not only am I of opinion that this fibrous invasion may be wholly without clinical correlations, but, indeed, that we have not to spend much regret upon this indifference. Many years' experience has convinced me of the truth which Cabot⁴ has lately reiterated—namely, that "not infrequently gross fibrosis of the myocardium is found in hearts which had been perfectly efficient during life." Dr. Hale White⁵ also says of a fibroid heart that it is practically undetectable. This paradox is true of both forms of cardiac fibrosis—of the diffuse form described by Dehio⁶ in 1899, and the focal form, in which the wall of the heart, especially of the left ventricle, is beset with callosities. These callous nodules or lumps were at one time attributed only to local ischaemia due to a closure of a coronary branch, by infarct or by sclerosis. Histologically, however, they do not essentially differ from myocarditic effects of other origin; moreover, recent research has demonstrated many a callus, towards which its particular coronary branch was open.⁷ But these nodules, often old foci of toxic myocarditis, are not always to be found simply by slicing the ventricular wall with a knife; sections must be made and stained. Conversely, Kanthack and I, in investigating the effects of coronary disease on the heart, demonstrated on more than one heart that these arteries might both be blocked by atherosclerosis and yet the myocardium be normal enough for the age of the patient. Diffuse or dystrophic myocardial fibrosis Dehio attributed to venous stagnation, comparing such hearts with "cardiac kidneys" and the like; and it is true that fibroid ventricles are often dilated. Still, I have examined many hearts presenting this fibrosis in which evidence of venous congestion was not obvious. However, I must not occupy time on these points of pathology; suffice it to say that cardiac fibrosis, so far from being a cause of cardiac failure, seems not infrequently, as in hypertrophy with dilatation, to have some conservative value. Connective fibre is a less resilient tissue, and its elastic limits are narrower; but within them it is tougher. Syphilitic fibrosis is, of course, another matter; any effect this variety may have on the cardiac function depends on its site. "Cardio-sclerosis" may seem to be germane to this part of the argument; but I never succeeded in ascertaining from Huchard, or from his disciples, what the term signified. It seems to have been adopted rather for logical symmetry than for the complexities and elusiveness of Nature; and if it ever had any meaning at all it was as a cover for several different conditions. With fibrosed hearts, then, patients may, as Dr. Hale White says, die suddenly; but we have no criterion of its presence, nor can the pathologist tell us how or when it causes death, if indeed it does cause it; which is doubtful.

But, as time presses, let us now pass on from the histological nature to the sites of lesion. Is it less the nature than the position of a breach of continuity which determines the mortality? Here again I fear the answer of the pathologist is still equivocal, though it is true that closer methods of observation may prove more enlightening. The lesion, or the critical lesion, may be overlooked; for instance, Falconer and Duncan,⁸ beside valvular lesions in a certain heart, found diffuse lesions in the right auricle (less in the left), while, save for a slight perivascular lymphocytosis, the ventricular muscle was normal.

In respect of site our thoughts turn promptly to the tract of Tawara, and perhaps to Wenckebach's area about

the superior vena cava, for we must not be content to say that lesions in this area set up disorders of rhythm only. We readily suspect, in such a concert as the function of the circulation, that rhythmical disorder may abut upon and issue in defective work. At first sight it seems that if scattered focal lesions—such as those described by Carey Coombs, Poynton, and others—might come short of gravely disturbing conduction, yet that such a check must follow a more diffuse and general invasion of these structures, for ventricular automaty, detached from the cardiac system, is certainly less effectual. Syphilis, by its proclivity for the septum, especially menaces Tawara's tract. Now some observers are of opinion that this tract, whether by its looser texture or by its peculiar blood supply by a twig from the right coronary artery, has in its disease some independence, be it more or less, of the rest of the heart.⁹ Spots of haemorrhagic and leukaemic exudation, aortic rheumatic foci and the like are found not infrequently in this tract, especially in the left limb of it, when absent in other quarters of the heart. Tanaka insists, as I have done, on the curious maintenances of heart function and of medical confidence in not a few cases in which the myocardium or conductive tract proved to be in extremes of fatty degeneration, until in a moment death brought all to an end. This, he says, as Dr. Hugh Anderson has illustrated in the cat, is especially characteristic of old hearts, as contrasted with young ones.

But we must not, I think, make too much of this alleged independence; decay proceeds as often from the myocardium to the bundle and knot, and often, as Dr. Coombs has shown, both tracts and myocardium, if unequally affected, are attacked simultaneously. Saigo¹⁰ examined 100 hearts from this point of view, and concluded that rheumatic nodules do often arise especially in the bundle; that regressive processes also—such as fatty vacuoles—come early in it; but that in brown atrophy and fibrotic hypertrophy the bundle may be involved, or may be free or nearly so. Lubarsch,¹¹ again, investigated 500 hearts on this complicated problem; in many he found changes, such as acute rheumatic nodules, predominant in and about the tract, especially the left limb of it. Brown atrophy he found to occur in both, if in unequal degrees. Lubarsch's observations may be summed up thus: In moderate focal fatty myocardium the limb of Tawara's tract may be quite free; or in extreme and widespread focal fatty myocardium the *a-v* bundle may be very slightly affected; or fatty change may be quite isolated in it, even the papillary muscles being free (but this is rare); or fatty change may be marked and extreme in the *a-v* tract, with very slight myocardial affection, say slightly in the papillary muscles only; or fatty change, moderate or much, may be practically alike and equal in both myocardium and bundle; and finally, Lubarsch thinks these variations are dependent upon vascular conditions, such as local retardations of blood velocity.

In Tanaka's cases of diphtheria the bundle was five times more, three times less, and four times equally affected with the rest of the musculature. "Only once was an almost isolated bundle-affection found." But in the bundle the changes are more diffused than in the rest of the myocardium. "But, he concludes, a sudden failure of the heart can also come about without essential lesion of the bundle; indeed, occasionally arrhythmia appears without any marked degeneration of the bundle."

I make no excuse for dwelling at this length on the pathological relations of the bundle and the myocardium, for in these might lie some explanation of the obscurity of the causes of continued cardiac function on the one hand and of sudden cessation of it on the other; but the interpretation is not yet. In some cases, it is true, the proper symptoms of dissociation are present, but in some they are absent; and as yet we have no histological elucidation of this inconstancy. In one of Lubarsch's cases, in which dissociation was complete, after death, while in the myocardium were found large focal callosities, in the bundle were only small ones, with some fresher spots of exudation and minute haemorrhagic points, none of them extensive enough to rupture continuity. On the other hand, many cases are published (by Reinecke and others¹²) in which, under what seemed distinctly to be complete demolition of the bundle, there was during life no dissociation. In one case¹² the whole bundle was calcified;

in others in which the tract seemed broken up dissociation was often incomplete; and sometimes there was only simple retardation. If one limb is affected there may be incomplete dissociation. In a few cases tachycardia has been observed. Dr. Coombs's researches, again, make it probable that acute rheumatic or septic toxins may far invade the tract without any characteristic symptoms during life. I will consider presently how far intrinsic and extrinsic causes (bundle, vagus, bulb) must co-operate; or, again, how far chemical perversions may be concerned in arrests of the heart.

I have alluded more than once to the part of the nerves in cardiac function, and so in the clinical surprises which we are discussing; but the nervous coefficients in these functions are very dark. Is it by means of nervous influence that a heart which is the seat of advanced disease may be carried along so far and so efficiently as again and again to deceive the clinical observer outright? We know that the heart is thickly beset with nervous fibres and ganglia, that the plexuses are motor in function, and that much, if not all, of the harmony of the circulation must depend, and intimately depend, on their integrity; but our new interest in Kent's and Tawara's tract has diverted research from these nervous governors. We may destroy aortic cusps without heart failure, we may poison the myocardium into extreme fatty change without immediate heart failure; but if with the soundest myocardium we cut the vagi, or even cut one only, the organ is promptly stopped—at any rate in dogs and cats.¹⁴ Kronecker's experimental determination of a nervous co-ordinating centre in the upper third of the interventricular septum is well known. A needle puncture here (in the dog) produces fatal delirium cordis, a result not to be obtained by punctures anywhere else. But on inquiry it seems that the morbid histology of the nervous cells and fibres of the heart is very little understood; even their normal anatomy is vague, and the pathological records are very dubious. Our methods may be inadequate, but hitherto the allegations of morbid changes in the nerves do not amount to proof. Vagus alterations have been reported, but on scanty evidence; yet if these nervous strands and centres deteriorate, and we cannot suppose them to be immune to the lot of all organized structures, their irritation or decay may account for a waning cardiac reserve, and for unexpected heart failure, even in cases in which muscular degeneration may be inconsiderable. Romberg, Krehl, Rosenbach, all turn for explanation of the paradox of endurance, and of deferred crisis in degenerate hearts, not exclusively to the "innate properties" of the organ, but also to its nervous endowments, and especially to its ganglionic integrity. Whatsoever our opinions on the rather academic controversy between the neurogenists and the myogenists, we shall not forget that the nervous system of an organ, such as the heart, is not only the conductor of its co-operative and harmonious concert, but also an instrument of its development. To Professor Langley we are chiefly indebted for such knowledge as we possess of the sympathetic system, and he tells us that although the ganglionic cells of the heart comport themselves differently from sympathetic cells, yet that such ganglia die rapidly if deprived of nutriment—as, for example, in animals bled to death. Oxygen may be more important to them than to nervo-muscular tissue, although they have a greater resistance to deprivations of oxygen than has the central nervous system.

And beside the intracardial nervous structures we have also the extracardial, the vagus and the bulb; not to mention those psychical influences which on the heart are supposed to have a peculiar potency. One would guess that a heart failure attributable to these influences is a cortical disturbance propagated through the vagus.

Again, the vasomotor system has degrees of reinforcement or default of which we have little notion. We do know that many a death attributed to the heart was due to a failure of the "peripheral heart," to a relaxation in large vasomotor areas. The "weak hearts" of women are often thus to be explained; their peripheral vessels are slack and labile. One is tempted, contrary however to present evidence, to dally with the hypothesis suggested by Henle, and ingeniously advocated by Hasebrock and Grützman,¹⁵ that the peripheral heart—the muscular arteries and capillaries—act not so much as a break on

the central propeller, but serve rather as forwarding machinery, a subsidiary means of propulsion.

And there is a side of vascular dynamics which is not fully realized—namely, the reciprocation of the several areas; or it is realized only between the splanchnic and the encephalic; yet these fluctuations are ubiquitous and incessant. If the warm hand be placed between the thighs, and the pulsations felt for a few minutes, the oscillations of vascular volumes, independent of respiration, will be clearly perceptible. And Müller¹⁶ and Weber have shown that areas are very independent, and often conflict, and such pressure interferences must have their echo in the heart; if in an area dilated for work velocity is not to fall, there must be an equivalent and harmonious constriction elsewhere, or the heart will have to make up the difference. Even to move a limb determines at once an afflux of blood thither.

Once more, we have to remember the chances of sudden death by fat embolism, nerve toxins, pulmonary embolisms, and so forth; or possibly, as suggested by Dr. Hawkins-Ambler, by increases of the specific gravity of the red corpuscles. There are probably many subtle agents of which we have no idea, but which enter, alone or accompanied, into these nice poises of life; and alterations in molecular constitution invisible even to the "ultramicroscope." An old rubber band may to the eye present no change, but on use its loss of elasticity will become quickly apparent, and it will die suddenly. The heart of Dr. Hugh Anderson's elderly cat could not withstand the vagus inhibitions to which the young cat reacted indeed plainly, but which it withstood with security. There is a world within the visible features—a world of surface tensions, osmoses, reaction potentials, viscosities, differential pressures, etc., of which we know little. If we apply cold to the sinus area, we retard the rate of the whole heart; if heat, we accelerate it, as in fever; yet to the eye there is no tissue change, and to other parts of the heart such applications of heat or cold are apparently indifferent.

Still, if sound and not too old, the heart, as Sir James Goodhart has insisted, will stand almost anything; no combination of adverse factors will kill it. The distinctions, therefore, urged by Merklen, Hoffmann, Rosenbach, and others between anatomical and functional cardiac integrity are pushed too far; there can be no function without correlative molecular motions, and if the function continues irregular, the corresponding molecules cannot have returned to their normal pattern; there must be some dislocation, and this may be permanent—a "new set." For instance, I am told that if a piece of steel be fatigued by repeated blows, even to the point of fracture, although to the microscope no molecular alteration may be visible, yet a permanent molecular dislocation may be detected by a change in its electric conductivity. Perhaps we also some day may be enabled to gauge an old, a fatigued, or a poisoned heart by the test of electric conductivity. Now, if its texture has got a permanently new set, the heart is no longer sound, whether the intimate alterations be visible or invisible.

Another curious point about cardiac reserve, or what we think to be such, is that it is trainable. A man in training seems to have many times the reserve, the cardiac reserve, of one out of training. In athletic training the heart probably grows larger with its skeletal kindred, and therewith probably also the tone of its motor ganglia and of peripheral vascular system, and the capacity of the lungs are also developed. In Cambridge an old boatman readily tests an oarsman for staleness by drawing a finger down his cheek and noting the persistence of the ruddy line. Moreover, training is not a mere matter of heart and lung endurance; the acquired economy of the skeletal muscular groups must count also for much. We cannot analyse all these manifold factors. In some elderly persons the skeletal muscles seem to have been relatively too good; the heart was not able quite to follow them.

Then there is the world of chemical reactions in the heart; the ionic motions and substitutional interchanges of which we know little; but, from the behaviour of salt perfusions, they must be of the most intimate kind. The little we know we owe to such researches as those of Dr. Walter Fletcher and Mr. Barcroft. Dr. Gaskell showed years ago that to perfuse the heart with even a very weak solution of lactic acid is to stop it in diastole; a reaction due

to mere acidity, to the positive ion H. Stronger solutions drive it into rigor. Dr. Fletcher has shown that by a previous application of oxygen these effects can be prevented. The mammalian heart lying after death in an atmosphere of oxygen does not become acid, or but slightly so; and after perfusion with oxygenized Ringer's solution, even some hours after, no trace of lactic acid appears. Dr. Fletcher has shown me how many occult flaws there may possibly be in the balance of colloidal mechanisms in the cardiac muscle; and more than this—that the heart may have abundant fatty deposit in it, and yet the muscle cell on the motor side be intrinsically intact, and prove no more vulnerable than one histologically normal; and this he illustrated by the figure of an engine clogged with dirt and grease, which notwithstanding may do better work than another engine bright and clean but with tight bearings, or an unseen crack in its cylinder. When beyond unequal metabolisms we pass into the region of toxic influences we recognize again how near the edge our gyroscope may be revolving.

Finally we know that the heart's balance depends on that of many other parts also; the gaseous exchanges are not in the myocardium only, not even in the lungs only; they are burning in every muscular area, in every viscus. Thus, even if the haemoglobin be constant in quality and quantity, if velocity begins to fall we are in a vicious circle; velocity—which means oxygen exchange—slows down in every organ, especially in those which, beside the lungs, have a double capillary machinery, such as the liver and the kidneys; in these areas we very soon find evidences of stasis. And retardation in the lymph system also is not to be forgotten. Such defaults as these may for whiles miss or neutralize each other, but when at some unhappy moment a summation of adverse alternating conditions comes about—capillary retardations in the viscera, tissue calls, lymph pressures, vagus and vasomotor waves, psychic thrills—in such cases as these, cases not without their prior forebodings, we shall not be at a loss to comprehend at last the suddenness of death. But the riddle, which I have done so little to read, is the frequent suddenness of death in one who scarcely having known illness expires under no extraordinary effort; or in the peace of his own bed or elbow chair passes silently away.

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At the annual meeting of the board of governors of the Wolverhampton and Staffordshire General Hospital, held on March 12th, Dr. John Grout was elected a Vice-President in recognition of the valuable and memorable services rendered by him in connexion with the King Edward VII Memorial.

THE late Lord Wandsworth, a member of the firm of Stern Brothers, bankers, has bequeathed £10,000 to be used for medical research "as my old friend and doctor Sir William Bennett shall decide is best"; £5,000 to the Bolingbroke Hospital, Wandsworth Common; £1,000 each to the London Hospital and to St. George's Hospital, and £500 to the Hospital for Cancer, Fulham Road. After the payment of certain sums the residue of the estate, which it is estimated will exceed £1,000,000, has been left for the foundation of an orphanage to bear Lord Wandsworth's name.

THE third International Medical Congress on Accidents will be held at Düsseldorf in August (6th to 10th). Among the subjects proposed for discussion are the present state of the law as to compensation for accidents in different countries and the question of insurance against industrial diseases; the importance of early treatment directed to the restoration of functional disturbance after accidents; vascular disease and accidents; arthritis deformans and accidents; the influence of accidents on the origin and increase of tumours; methods of examination in cases of injury after accident. Communications relative to the Congress should be addressed to Professor Liniger, Düsseldorf.

A Post-Graduate Lecture ON THE ESTIMATION OF HEART POWER: AND THE TERMS BLOOD PRESSURE; HYPERTENSION; HYPERPIESIS; HYPERTONUS.

BY

WILLIAM RUSSELL, M.D., F.R.C.P. EDIN.,

PHYSICIAN AND LECTURER ON CLINICAL MEDICINE,
ROYAL INFIRMARY, EDINBURGH.

GENTLEMEN,—A request made by some of you that I should give a clinic on the present position of "blood-pressure" was communicated to me, and I of course at once expressed my willingness to speak to you on the subject.

At the outset permit me to say that, in the consideration of many clinical problems, the accuracy of our first step often determines the accuracy of our further steps, and of the conclusion we ultimately arrive at. As illustrating this proposition I would impress upon you that it is necessary to realize that, when we as clinicians feel the radial pulse, the initial and primary object in doing so is to obtain information as to the vigour or strength of the heart. The second object is to gain some knowledge of the condition of the channels through which the blood is propelled. As directly bearing upon our subject I recall to you various terms used in relation to it.

BLOOD PRESSURE.

This term means the pressure exercised by the blood inside the vessel, and the driving power of the heart is judged of by the estimate we form of this pressure of the blood upon the arterial wall. In much recent writing the term unfortunately is used to indicate, or as a mere synonym for, haemomanometer readings. Whatever the blood pressure be inside the artery it represents in some measure the power with which the blood is driven out of the left ventricle. This is the plain and fundamental fact, and this is the reason why the examination of the pulse occupies such an honoured place in our clinical methods. Other words are, however, in use in the study of this same problem of heart power.

HYPERTENSION.

The term "hypertension" is at present in common use, and has, I think, been popularized by the French school. In past years high and low tension pulses were spoken of. The word "tension" applied in this way was, however, long regarded by critical clinicians as unsatisfactory. The question always was whether it merely meant the amount of pressure exercised by the blood on the arterial wall or the taut state of the wall caused by the pressure, combined with changes in the wall itself. So far as I am able to see, the use of the term "hypertension" in no way answers the old question, although in many quarters it is treated and used as a "blessed word" and placed above the zone of criticism. Other clinicians steadily abstain from using the word "tension," in any of its possible meanings, as applied to the pulse, and with this attitude I am in complete accord. Without clear thinking and the use of suitable words the confusion which surrounds this subject will continue.

HYPERPIESIS.

The term "hyperpiesis" has been introduced by our veteran clinician, Sir T. Clifford Allbutt. Etymologically it means excessive pressure, and throws no light on the problem of how much is blood pressure and how much is arterial wall. If it carries with it the idea of fullness, the question at once arises of the relation of fullness to pressure. In measuring arterial pressure with the haemomanometer, the question remains, Are we measuring the pressure wave only, or are we not also measuring vessel wall?

HYPERTONUS.

The only other word I need refer to is "hypertonus." I am commonly regarded as having definitely applied it to an arterial condition. It means a contraction of the muscular