THE CAVENDISH LECTURE ON ACUTE CARDIAC FAILURE.

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AFTER a few preliminary remarks, the lecturer said: Acute heart failure is, I admit, a misleading phrase, but it is scarcely necessary to explain to an assembly such as this that it implies not any separate disease of the organ, but the culmination, in temporary or permanent arrest of the cardiac function, of a variety of conditions damaging to texture and exhaustive to nerve power or muscular energy.

We cannot perceive aright the heart and its mechanism, nor appreciate duly its modes of failure, unless we bear steadily in mind the fact that it is but a part, differentiated for special reasons, of a complete cardio-vascular tubal system containing the blood. The tubes are not passive tubes, sluiced by a central force pump; on the arterial side the powerful rhythmic pulsations of the heart are so modified and restrained by the elastic and muscular action of the vessels as to ensure a safe and adequate stream of blood throughout the body; whilst on the venous side the position of valves, the action of adjacent and often ensheathing muscles, the impetus of lymph and chyliferous tributary currents, and the constant but rhythmically-increased thoracic aspiration help to maintain the blood movement.

I need only hint at the large influence of the nervous system, which has in the course of evolution become so specialised with regard to the heart and vessels. The cerebro-spinal nerves, the sympathetic system, and a department of the nervous system—the vasomotor nerves—in touch with the preceding and yet under guidance from a separate centre, all take part in controlling the heart's doings, and in regulating the blood supply to each organ according to its functional requirements, which vary from time to time. Finally, there is held in the tightening and relaxing grasp of this complex tubal apparatus the blood itself, a living tissue, whose constant variations, both chemical and physical, in health and disease, affect the facility of the capillary flow, and influence the tonicity and nutrition of the circulatory apparatus. Can we look into this complex mechanism, which I have but enumerated, in which physical, chemical, and vital agencies are so intimately mingled, without a feeling of wonder that it should hold good unceasingly to the allotted term of life, and that so few lives should be brought to a premature close through heart failure? A chapter on compensatory adaptations has yet to be added to our textbooks on medicine for our better understanding of many such problems.

With such an infinite number of contributory factors acute cardiac failure cannot be a simple question; and if I, bound by the stern limits of time and the still more compelling limitations of my ability, approach it from the more concrete, clinical, and practical side, it is from no want of appreciation of the manifold intricacies of the subject; and it is with a very humble sense of our indebtedness as practical men to those whose patient labour in the laboratory have deepened for us our insight and interest in our clinical work.

RUPTURE OF AORTIC VALVE.

A most obvious cause of heart failure is direct injury—excluding, of course, external wounds—as, for instance, the very rare occurrence in which a healthy man during some sudden effort ruptures one of his aortic cusps. The whole cardiac mechanism is suddenly put out of gear; severe precordial pain, disorderly action, and faintness ensue, to be followed by other signs of perturbed circulation. In the only case of this injury that I personally remember seeing almost immediately there was smart hæmoptysis, no doubt from the over-distended left ventricle obstructing the return from the lungs. Under complete rest the heart, at first surprised and only very imperfectly able to deal with the altered mechanism consequent

upon the spoiled condition of its main valve, gradually steadied down. The left ventricle enlarged and increased in power; and the small, rapid, and irregular pulse was gradually altered to that having the characteristic features of aortic regurgitation. This alteration of pulse marked the adaptation of the heart to its new conditions in a manner precisely similar to what we observe in a case of aortic regurgitant disease, in which dilatation of heart and mitral inadequacy having arisen, the irregular mitral characters of the pulse gradually return under rest and treatment to those proper and normal to a well-compensated aortic regurgitation.

EMBOLISM.

The displacement of a clot from a systemic vein is a common cause of acute anginal heart failure, and is attended with urgent dyspnoea, an irregular, fluttering pulse, and very variable heart signs. The condition is, indeed, one of asphyxia of the heart, and it either terminates rapidly in death or the clot is further dislodged and propelled into the lung, when the cardiac symptoms quiet down, and those of another order commence with hæmoptysis significant of pulmonary infarction. Here, again, we can only recognise what is happening and relieve suffering and ensure quietude by subcutaneous morphine and like remedies. These cases are, however, by no means necessarily fatal; if the clot passes on to the lungs recovery ensues in a fair proportion, and the prognosis in each case must be considered on its separate merits.

OVER-TAXATION.

Next to such injuries to the heart, of which I have given a right-sided and left-sided example, we must glance at cardiac failure from over-taxation. There are two factors always at work, although in different proportions in different cases, in producing cardiac failure from great or prolonged exertion, namely, direct fatigue of the nervo-muscular tissue of the heart and a poisoning of the blood of auto-metabolic source.

Speaking of young people and healthy hearts, acute cardiac distress is, of course, very common in these days of athletic competitions, strenuous school exercises, hard bicycling, etc. It is surprising, indeed, that actual heart failure so rarely occurs; but few of us have an opportunity of witnessing such an attack. Fatal cases are rare, but have been recorded. For the most part we can only observe the after-effects of incomplete heart failure. Amongst the concomitants of heart distress or failure during violent exercise, as running, vomiting is one of the most common; a boy will tell you that at the end of a long race which overtaxed him, he cast himself on the ground and vomited. Dr. Clement Dukes speaking of boys who become overdone from running, from various causes, says:

Sometimes a boy will finish merely *tired*; or he will faint or vomit on the way and require to lie down on the road; or, he will complete his run but vomit all the evening after reaching home; or, again, if still more overdone, he may become absolutely insensible while running, and this state may last for hours; or he may die whilst in the act of running.¹

And amongst the after-effects one of the most constant is *anæmia*, and both of these phenomena—vomiting and *anæmia*—may be regarded as bearing witness to a changed condition of the blood consequent upon the accumulation of products of metabolism, which is now recognised as taking a not unimportant part in the mechanism of heart failure. I would remind you that gastro-intestinal attacks, vomiting, or diarrhoea are not uncommon occurrences in those who, habitually leading a sedentary life, suddenly take to exhausting exercise. There are other more certain proofs of altered blood in those who have died from excessive exercise, and in hunted animals, of which increased liquidness of the blood, absence of *post-mortem* clotting, and darkened colour are amongst the more obvious. I am not aware of any observations that have been made upon the blood in healthy people after a period of training followed by severe and exhausting effort, and, still more interesting, after prolonged exertion in those who are not in training. We are all of us familiar with cases of neurasthenia and *anæmia* distinctly traceable to some severe exertion in a person with no previous training.

In estimating the consequences of over-taxation of the heart from violent or prolonged exertion in young people especially, there are one or two points of practical moment to be observed. A glance at Beneke's² tables will show that the heart of the child is somewhat precociously developed. According to this

authority³ whilst the height doubles in the first seven years of life the heart volume increases nearly fivefold. In the second seven years things go more quietly, the height increasing by one-third and the heart volume by one-fourth, but the influence of this early developmental activity must be reckoned with throughout childhood.

This cardiac precocity observed by Beneke is in accordance with what I believe to be a fact which I have long noticed and pointed out in clinical teaching—namely, that the heart of the child—say between 6 and 12 years—is relatively hypertrophied—that is, the apex beat is somewhat to the left of the normal and the impulse relatively strong. This is the case especially with boys, and in my experience holds good with a considerable proportion of them. I have regarded it as due to the ceaseless activity of children and to a developmental *precedence* of heart to chest and lung capacity. The practical point is that I do not regard a slight extension of the apex beat within the nipple line, unaccompanied by confirmatory signs, as necessarily a morbid condition in a child. I look for the evidence of cardiac overstrain rather in an extension of the upper margin of dullness in the mid-sterno nipple line, an unduly perceptible impulse at the ensiform base, and a hesitating action of the heart, that is, an irregularity of rhythm and force. These signs, in association with languor, anemia, breathlessness on exertion, and sometimes a sharp pain at the heart which I have heard described as a "beasty pain, catching the wind," caused by a slight effort are sufficient for diagnosis. You may get others, such as murmurs; you often do get a hæmic murmur over the pulmonary area, but murmurs are by no means essential or to be expected.

A foreknowledge of the contributory and direct causes of these preventable occurrences is, of course, of much importance, and, thanks very greatly to the labours of Dr. Clement Dukes and to the careful supervision of the able medical officers attached to most of our public schools of recent years, and to the care with which boys are watched, weighed, measured, and their sports organised at most of our preparatory schools, all such risks are reduced to a minimum, whilst, on the other hand, the moral and physical advantages of athletics are fully maintained. A routine examination and registration of the health of every boy on entering school life would, however, be an advantage, supplemented perhaps by some brief form of health report from the home medical adviser.

A cardinal point—and the only one I shall touch upon—to be borne in mind with regard to young boys and girls is their special aptitude for short spells of active exercise, but their complete unfitness for prolonged, fatiguing, monotonous exertion. This is almost the reverse condition applicable to older people, and it is one often forgotten, not perhaps so much at schools as at home. Athletic fathers, robust elder brothers, proud of the sporting alertness and eagerness of young boys, will often keep them out on long bicycle, golf, or shooting expeditions, and may thus cause grave damage to the young and rapidly developing heart.

There is certainly a lack of supervision of young men training for athletics at the universities, although it is true that they come already seasoned and tested from the public schools. There ought to be some medical "don" to supervise university athletics, who should, however, be himself thoroughly in sympathy with the athletic spirit.

TREATMENT OF OVER-TAXATION.

The treatment of acute failure of heart from overstrain involves a period of a few weeks' complete rest and often many months of careful supervision. Young people make a rapid and generally a complete recovery, provided no actual lesion has been produced. The younger they are the more ready and complete the recovery. In youths and young men a degree of irritability of heart is often to be observed for many months, even for some years, the patient suffering from attacks of palpitation and cardiac pain on slight fatigue or exertion, and often at night. Quiet exercise must be resumed after a time, and it is in my opinion better to allow interesting exercises within measure, such as croquet, level cycling, golf, and easy tennis, rather than more formal courses of training. But each case must be considered on its merits. In some cases of cardiac overstrain in young people it will be found that a feeble lung capacity has led in-

directly by imperfect pulmonary circulation to the heart distress. In such cases well-ordered respiratory exercises, by developing the lungs, will facilitate the work of the right heart, and help the general circulation by increasing thoracic aspiration.

Well-planned sea voyages or sailing trips from the coast are peculiarly advisable for adults, provided strict injunctions against smoking and all strong alcoholic drinks are given.

HEART-FATIGUE IN ACUTE DISEASE.

Another form of fatigue heart failure is that frequently met with in acute disease. It is too long a story for me to go fully into now. I have dealt with it with some minuteness elsewhere⁴ as met with under one of the most common of circumstances, namely, in acute pneumonia. It is met with also in enteric fever, in acute bronchitis in old people, during a severe asthma paroxysm and in other conditions, such as functional tachycardia. The heart failure in these conditions may come about with a gradual quickening day by day of the pulse until it becomes fluttering, confused, running, representing little more than peristaltic movements of the heart. In other cases, and this more commonly in plethoric, fleshy, large-framed people with a pulse at first not unduly quick, with considerable tension and accentuation of the second sound, the failure occurs quite suddenly, attended with rapid breathing, pallor and cyanosis, sweating, collecting mucus rattles in the chest, and almost imperceptible running pulse. Under strong stimulation and the subcutaneous use of strychnine the patient may rally and all the conditions improve, but relapse often, and in elderly people almost always, takes place. An attack such as I speak of occurring in an old person with bronchitis or pneumonia is almost of fatal augury; the senile heart cannot recover the fatigue, and hence when the pulse mounts up to 120 in old people recovery is rare, unless the rapidity be attributable to functional disturbance from some temporary cause. With a sound heart in young persons no condition is too desperate for possible recovery.

Now, to discuss the treatment of heart failure under these varying conditions would be to discuss the whole management of the illnesses in question. There are a few factors, however, in special concern with heart failure in acute disease which should be looked for as indications for treatment—namely, (1) maloxxygenated and otherwise contaminated blood supply to the heart muscle and nerve; (2) excessive weight of blood burdening the heart; (3) exhausted innervation from sleeplessness and physical cardiac fatigue; (4) positive obstruction to the flow of blood through the lungs; and (5) changes in the texture of the heart muscle incidental to the disease, and especially to the pyrexia. Now the first two indications are undoubtedly met by depleting the blood volume from the venous side by attention to secretions, the occasional use of mercurials, careful limitation of the food taken in place of the over-feeding often to be observed, and in some cases a small blood-letting. The timely employment of oxygen inhalations at intervals through the day is a remedy of great value which ensures an improvement in the aëration of the blood sent to the left ventricle, and so direct to the coronary vessels. It is by no means necessary to employ oxygen inhalations in the majority of cases of pneumonia, of which disease I am now especially speaking, but in all severe cases the cylinder should be at hand to anticipate the threatened heart failure; (3) and (4), exhausted innervation and pulmonary obstruction are met with in pneumonia, advanced typhoid, and asthma especially. Strychnine is the most powerful cardiac tonic we know, and, subcutaneously used, is the best remedy for heart failure. In these cases, however, a frequent concomitant symptom is ballooning of the abdomen, and to give strychnine by the stomach in such cases is both dangerous and inefficient for the purpose required. In asthma, pneumonia, and in enteric fever it is often possible, in combination with the free use of oxygen and the occasional injection of strychnine, to give a sufficient subcutaneous dose of morphine to procure some much-needed sleep. When the heart threatens to fail in asthma the paroxysm must be thus shortened. In bronchitis, on the other hand, under no circumstances can morphine be so given. I have often observed cardiac exhaustion threatened in asthma through the want of small administrations of con-

centrated and assimilable food essences at intervals to support the patient. Alcoholic stimulants are, of course, necessary, the dose being adapted to suit the individual case. (5) The changes incidental chiefly to pyrexia in the heart muscle as a cause of failure are mostly to be considered in the prolonged fevers, and will be best warded off by a judicious mitigation of the pyrexia. I would only observe on this head that in cases where there is labouring action of the heart, and a tendency to cyanosis and stagnation of surface blood, I have seen much better results from hot water than from cold water sponging or cold applications. The surface circulation is more facilitated, heat is rapidly lost, and the skin functions encouraged, all tending to lighten the burden of the heart. In the after-treatment of pyrexial diseases, in which there has been prolonged rapidity of heart or any sign of heart failure, a chief regard has to be paid to restoration from cardiac fatigue. Cardiac fatigue is the disablement left behind by acute illness. Thus our level sea and country resorts are preferable to hilly places, and exercise must only gradually and tentatively be resumed. Digitalis and strychnine are useful as cardiac tonics.

I can only glance at failure of heart from the specific poisons in diphtheria, phosphorus poisoning, malignant anæmia, and the like, in which the cardiac nerves are directly poisoned, and in most cases a rapid fatty change in the heart fibres ensues. These cases are mostly fatal, with the exception of the influenzal cardiac affections, amongst which symptoms of slowing and increased rapidity of heart are met with, also some most intense forms of angina, usually vasomotor, and attacks of dangerous syncope may occur, yet the prognosis is, even in severe cases, so far as my experience goes, as a rule, favourable.

ANGINA.

Of anginal heart failure I have no time to speak at length. I would only urge that, as regards treatment, as well as from a scientific point of view, we must regard angina pectoris as in the largest class of cases a functional disease—a cardiovascular neurosis. In many cases it is dependent upon disorderly action of the vasomotor nerves, and is associated with a sound heart (angina pectoris vasomotoria). In many others we have a similar mechanism, but with, in the background, an unsound heart—aortic regurgitation, aneurysm, senile fibro-fatty hypertrophy, uræmic heart, coronary disease, and the like (angina pectoris gravior, or secondary cardiac angina).

In a group of cases numerically comparatively small, but very important, the anginal failure is the consequence of cardiac degeneration secondary to coronary disease, sometimes to coronary thrombosis or syphilitic disease (syncopal angina or primary cardiac angina). It is obvious that all the causes of excitement, direct and reflected, of the sympathetic and vasomotor nerves, must be looked into in dealing with the larger proportion of these cases, whilst on the other hand any special form of cardiac lesion must be appropriately dealt with.

In accordance with what has been above stated the immediate remedies for the cardiac attacks are, in the majority of cases, of the antispasmodic and sedative class—amyl, trinitrine, opium, directed against the arterial spasm which is so important a factor in the attack. In cases of coronary lesion, the employment of regulated exercises, combined resistance and respiratory exercises, and in old people especially, the regulated use of oxygen inhalations will help to maintain cardiac nutrition and to ward off attacks for at least a time longer. Whilst gentle exercise calculated to raise the blood pressure within the coronary circuit is thus to be advised, caution is necessary that no cardiac fatigue be allowed; the periods of exercise should be of short duration.

I well remember a medical friend whom I had seen previously for anginal heart disease calling upon me a year later, having gone through a course of special exercises. I could not examine him at the time, but he assured me he felt a new man, and could walk miles. A fortnight later he died suddenly.

In old people with atheromatous vessels, cramp in the legs is readily induced by any fatiguing exercise in consequence of the imperfect nutritional change allowed by the narrowed arteries, and similarly we may get cramp in the heart muscle, which is very apt to prove fatal. This was pointed out by Allan Burns nearly a century ago, and further dwelt upon by

Sir Benjamin Brodie. It is to be noted also that the cramp of fatigued muscles does not necessarily come on at once, but frequently is postponed until the night. Many a fatal attack of angina pectoris or syncopal failure has been prematurely induced by some over-fatigue or excitement during the day.

CARDIAC FAILURE IN PLETHORA.

There are two conditions leading up to heart failure which I have not touched upon, and yet which, from the remedial point of view, are of immense importance, but they have been fully handled by my colleague, Dr. Mitchell Bruce, in his recent Lettsomian Lectures. One is typified in the middle-aged, plethoric man, who, working hard, lives too well and takes little exercise. This man gradually acquires a fatigued and over-burdened heart, over-weighted with the heavy volume of blood it has to propel. He may get a spring or summer cleansing once a year at Homburg, Harrogate, or Carlsbad, but with no intermediate precautions he drifts along often into irremediable disease of heart or kidneys or both. His first introduction to the physician is commonly after an attack of breathlessness and cardiac excitement or failure occurring on some unwonted exertion, after which he remains perceptibly short-breathed. His cardiac area is found to be extended, partly from a slight yielding but mainly from precordial and interstitial fat. The first sound is weakened often to extinction, the second sound labouring; there may be slight albumen or a little sugar in the urine. If taken in time and placed under a depletory treatment, a restricted diet, with limitation of fluids taken chiefly between meals, a great curtailment of alcohol and smoking and well-arranged exercise, with insistence on deep, full inspirations from time to time whilst walking in the fresh air, such a man will be restored to health, and practically rescued from some early catastrophe in the form of heart failure in acute illness, or from drifting into chronic cardio-vascular disease. A few weeks' change away whilst getting injured to the treatment is of great value, and such cases will derive especial benefit from the Nauheim or modified Oertel treatment.

TOBACCO AND EPILEPSY.

The other class of cases I would refer to in association with two causative factors—namely, nicotine and epilepsy. I may bring my remark to a point and a conclusion by a brief sketch of a case in illustration.

A captain in an infantry regiment was invalided home from South Africa on account of attacks of heart failure. He was a married man, 37 years of age, had never had syphilis, but was an inveterate cigarette smoker and a little free with alcohol. In July, 1900, whilst mounting a hill, he felt distressed, and on return to camp fainted, and after this he was unable to exert himself without some distress. In September, having escorted some prisoners from Durban to Capetown, without sickness or fatigue or any hardship, he on arrival was about to get ready for dinner, and in the act of taking a glass of wine bitters, when he suddenly fell down unconscious and pulseless, so that a doctor who saw him immediately thought him dead. He remembered nothing until he awoke at 2 A.M., feeling very faint and ill. There was no preceding sense of faintness, and no convulsion. On a later occasion whilst feeling very well he suddenly fell unconscious for a quarter of an hour. He has had one similar attack since his return in November. He was subjected to a course of Nauheim treatment without benefit. His complaint was, besides these seizures, of almost daily attacks of cardiac pain, especially induced by any exertion. Dr. Walker, under whose care he came subsequent to the Nauheim course, by rigorously restricting the tobacco and regulating his diet and stimulants, materially relieved the symptoms, and when I saw the patient (with Dr. Walker) in April the cardiac dimensions, position, and sounds were normal, and the pulse, although feeble and rather quick, presented no other special feature. On inquiry into this gentleman's family history nothing cardiac was elicited, but a brother was a deaf-mute, and his second son was the subject of very frequent and severe attacks of *petit mal*.

This case was primarily, no doubt, one of anginal heart failure in association with excess in nicotine, and as such belonged to a class of frequent occurrence, but the instantaneous attacks of unconsciousness were suggestive of at least an associated neurosis of an epileptic character. I have met with one other very strikingly similar case in a younger man, also an excessive smoker, and I have a less full recollection of another case in connection with excess in cocaine.

I have from time to time been consulted about a lady who has long been the subject of night attacks of an epileptic character, and who has recently developed marked and rather severe attacks of angina with heart failure of an alarming degree, many of which attacks usher in a mild epileptic seizure.

In her case the pulse of late years has become extremely slow, and she has presented some symptoms of myxœdema. It is remarkable that in this case the amyl class of drugs are useless, whilst strychnine gives speedy relief both to epileptic and cardiac phenomena.

"CARDIAC HESITATION."

Cases of cardiac hesitation with pulse of uneven rhythm, not amounting to intermittency and presenting occasional attacks of fainting, are not very infrequent amongst children, young boys especially, between six and puberty. They are easily tired, and are generally anæmic. It is quite exceptional to find them addicted to any secret vice. There is nothing abnormal beyond altered rhythm and vacillating force to be found in the heart. The distinction between this form of heart failure and *petit mal* is often difficult, and I am strongly inclined to regard the two conditions as allied. Removal from all mental pressure, an outdoor life and arsenical tonics generally result in gradual recovery.

I have endeavoured in this address rather to suggest lines of thought than methods of treatment, feeling that you are as competent as I to give a practical therapeutic interpretation to whatever of my suggestions may meet with your acceptance.

REFERENCES.

¹ *Health at School*, 1894, p. 331. ² *Die Altersdisposition*, 1879, p. 94. ³ *Loc. cit.*, pp. 6, 7. ⁴ Lumléan Lectures, On Treatment in Diseases and Disorders of the Heart. Lecture III.

CLINICAL AND EXPERIMENTAL OBSERVATIONS UPON GENERAL PARALYSIS

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In this paper I cannot enter into full details descriptive of the disease known as general paralysis of the insane. It is necessary, however, to define general paralysis as a disease marked by progressive degeneration of all the bodily and mental functions, commencing first with those most highly developed and recently acquired, and ending with complete wreckage of mind and body. The course of the disease varies greatly according to individual resistance. Some patients show mental symptoms, becoming maniacal or melancholic, before motor symptoms become prominent, while others present motor symptoms sometimes for years before any mental peculiarities develop. The disease is regarded as incurable; but remissions, often of such completeness as to simulate recovery, are by no means uncommon. The typical disease is further peculiar in having several stages. The first stage, in which the patient is often acutely ill, in which febrile temperature, loss of body weight, restlessness, excitement, and sleeplessness are the outstanding symptoms; the second stage, in which the patient becomes fat, facile, lazy, and liable to congestive seizures; the third stage, when the patient again loses weight, becomes mentally demented and physically paralysed.

The causes of general paralysis are very variously stated, but the opinion that syphilis, if not the only cause, is certainly one of the chief causes is at present gaining ground both in this country and abroad.

The following observations were made upon patients suffering from undoubted general paralysis. I would divide these observations into:

I. Those observations made upon patients in whom the disease was steadily progressive.

II. Those observations made upon patients in a state of remission.

III. Those concerned in an attempt to produce remission.

I.—PATIENTS IN WHOM THE DISEASE WAS PROGRESSIVE.

The system which I believe to be invariably affected throughout the whole course of general paralysis is the alimentary system. I have never yet seen a case of early general paralysis in which one did not meet with intestinal and gastric disorders—obstinate constipation, biliousness, capricious appetite, gastric crises, failure of digestive and assimilative power are the commonest symptoms. In the second stage bulæmia may be and generally is added to the symptoms of the first stage, while in the third and last stage constipation alternating with diarrhoea is the rule. These symptoms have

been commented upon by innumerable writers but their significance has not been appreciated, nor do I think any observer has ever realised that they run from beginning to end of the disease, as they are so frequently masked by mental symptoms. When you have heard from Dr. Ford Robertson the pathology of the alimentary tract in general paralysis you will agree that the alimentary symptoms as a group are very important symptoms.

Frequent observations have been made upon the temperatures of cases of general paralysis. Savage believes that there is nothing characteristic about the temperature in general paralysis. Clouston believes the febrile attacks so common in the course of the disease are maniacal temperatures. Mickle in an exhaustive treatise, draws many conclusions from the temperatures he observed, which for want of space I cannot repeat here. I have observed the temperatures of cases of general paralysis for several years, making continuous charts of four, five, and six months' duration. I admit there are some grounds for the diverse statements made regarding the temperatures in this disease. Some few cases, though steadily progressive, never have a temperature above 98.4° during the first and second stages of the disease; in others the temperature is an irregular one, febrile attacks occurring at long intervals. These peculiarities are explainable on the grounds that individual resistance to disease varies, and remissions in the course of the disease are very common. As the result of my observations I strongly endorse Macpherson's opinion that the most characteristic temperature in general paralysis is a recurrent febrile attack every one or two weeks. I have endeavoured to represent what I believe to be the characteristic temperature of the disease in the accompanying chart.

Upon admission there is probably mania with a febrile temperature of about a week's duration, then there is a remission for, say three weeks, and again a febrile attack, with or without mental symptoms, followed again by a period of remission which may be months in duration and take the patient well into the second stage. When the second stage is established the febrile attacks become more frequent and the remissions shorter, and this may continue for months. As the second stage gradually passes into the third stage the temperature tends to become subnormal, the febrile attacks of the second stage being represented by a gradual rise from subnormal to normal, but 99° F. is not often reached, the explanation being that the patient is becoming exhausted and does not react so vigorously to the disease as previously. When the third stage is fairly entered the temperature becomes very irregular—to-day slightly febrile, to-morrow subnormal, and so on, the outstanding feature being the irregularity. I have purposely omitted the febrile reactions to congestive seizures and apoplectic attacks, as these only complicate the main issue, and must be regarded as accidents in the course of the disease. Regarding such a temperature chart as a whole, the conviction is forced upon one that such recurrent febrile attacks can only be regarded as due to some recurrent cause, and that cause is a toxic one. Each febrile attack represents the resistive reaction of the body to some toxic substance, and each inter-febrile period an intermission when the resistive powers of the patient have subdued the action of the toxin. Kanthack considers it a rule "that previous to recovery from bacterial products an increase in the number of leucocytes takes place." It is hardly necessary to say that many workers have examined the blood in general paralysis; but such observations were single ones, made upon different patients, and gave the most irregular results. I therefore counted the leucocytes and took the temperatures daily, or every second day, of some 6 cases of general paralysis for three or four months continuously, and a glance at the dotted line on the chart will show that to every rise of the thermometer there was a corresponding rise of leucocytes. In the first stage the hyperleucocytosis was often very high, sometimes 20,000 to 30,000 per c.mm., and the higher and longer continued the leucocytosis the longer was the remission that followed the attack. In the second and early third stage the leucocytes still rose with each rise of temperature, but the reaction was not so vigorous, and between the attacks the leucocytes tended to fall lower than in the first stage. The remissions were also shorter. In the later period of the third