

ON ACUTE ENDO- AND PERICARDITIS.

By MICHAEL F. COX., M.K.Q.C.P.,

Physician to St. Vincent's Hospital.

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I do not think it can be necessary to apologise to the Academy for introducing the subject of acute endo- and pericarditis; the frequency of the occurrence, and the gravity, of these affections, should be ample excuse. And yet it may be said, or thought, that the number of those—and they of such eminence—who have, in Dublin, treated of these diseases, has been so great, both in the past and in recent times, that it is little, if at all, short of presumption on my part to bring forward a subject with which the names of Graves, and Bellingham, of Stokes, and Hayden, not to mention the living, have been so honourably identified. And yet as Science advances, and as we all think, or *think* we think, independently, even so, with all diffidence, I venture to present my thoughts and observations on the matter before the Academy. I shall not presume, and do not pretend, to deal exhaustively, or perhaps systematically, with the subject; but merely propose to offer, for the consideration of the Academy, the facts which I have observed, and the ideas which have occurred to me, in connection with these facts.

I shall divide my remarks into three heads—first, as to the frequency of acute endo- and acute pericarditis; secondly, as to the causation of these affections; and, thirdly, as to their treatment.

In the first place, I have been greatly impressed by the enormous frequency of these affections, in private as well as in hospital

practice. I cannot suppose that my experience in this respect differs from that of other Members or Fellows of the Academy, but certain it is that I do not meet with any other class of acute affections more frequently than I meet with acute endocarditis. I do not know, as I have said, whether or not the experience of others coincides with mine; but, if the experience be common, we must, I think, conclude that it points to a change in the type, or in the prevalence of a particular type, of disease—such a change as has occurred, for instance, in the case of typhoid and typhus fevers. Certainly, with regard to acute endocarditis, I meet with it much more frequently than it seems to have occurred in the practice of Graves, or Stokes, or Hayden, or than recent authorities record it. The varieties of acute endocarditis and acute pericarditis with which I shall deal are the rheumatic and the idiopathic, as well as with what I prefer to call the septic variety of endocarditis. And here I must confess myself at a loss with regard to the differentiation between so-called idiopathic and rheumatic endocarditis. *Does* such a thing as idiopathic endocarditis exist? I cannot definitely undertake to answer; but certain I am that I have met with a considerable number of cases of acute endocarditis, of a mild type for the most part, and of brief duration and favourable termination, in which neither joint-trouble nor perspiration, nor, if I may so call it, *rheumatic*^a tongue, was present. I have records of nearly a dozen such cases, as well as of two cases of so-called *ulcerative* or *septic* endocarditis, and of one case of acute idiopathic pericarditis.

Authorities differ widely as to the prevalence of acute rheumatic endo- and pericarditis; the mean estimate being that about 50 per cent. of all first attacks of rheumatic fever are attended by some form of cardiac complication. My experience coincides more closely with that of Dr. Latham, who places the percentage considerably higher. Perhaps the explanation which he offered would also apply to me—namely, “the assiduity of his clinical clerks in providing interesting cases for his wards.” As to the relative frequency of endo- and pericarditis, I also find myself more in

^a White, creamy, furred tongue.

accord with Latham and Sibson, than with Stokes or Hayden, Fuller or Bamberger. Latham found acute endocarditis present in three out of every four cases (75 per cent.) of rheumatic cardiac complications, and acute pericarditis in only one out of twelve; whilst the two affections co-existed in one out of every seven cases. Of Sibson's cases, according to Hayden, four-fifths were of endocarditis, one-tenth of pericarditis, and of the combination of endo- and pericarditis also one-tenth. No account is here taken of myocarditis. Fuller found endocarditis and endo- and pericarditis combined, collectively, in one out of 2·3 cases, and pericarditis alone in one out of every six cases.

Now Stokes and Hayden found acute pericarditis more frequently than acute endocarditis in rheumatic fever, although their experience differed as to the relative frequency of acute pericarditis occurring in combination with acute endocarditis—Stokes finding the dual combination more frequently than any other cardiac complication, and Hayden finding it less frequently than any other: except, perhaps, myocarditis, which did not enter into their calculations; neither having ever met with a case in which it existed independent of endo- or pericarditis. Whilst Stokes barely admitted the possibility of its existence or recognition, Hayden and other good authorities admit its existence. I myself have met with one case of its separate existence, one recently in which it existed with endocarditis, and perhaps one in which it existed in combination with pericarditis.

There are, then, some propositions, or statements, of Hayden and of Stokes which my experience, so far, controverts. The first is that in rheumatic fever pericarditis is more frequent than endocarditis; and that when the latter exists, the former is likewise generally present. Secondly, that the liability to endocarditis is in direct proportion to the severity of the joint affection and to the number of joints engaged. Although Hayden admits that endocarditis *may* exist independent of joint-trouble in the rheumatic, he does *not* admit the existence of acute idiopathic endocarditis. Thirdly, that the liability to cardiac complications is in direct proportion to the severity and duration of the pyrexia.

Fourthly, that (according to Hayden) the murmur of acute rheumatic endocarditis is usually mitral in *origin*, and lasts for life, “usually coeval,” he says, “with the life of the patient”—differing herein from Sibson, Latham, and others; although he seems to assent to the statement of Lancereaux that in *ulcerative* endocarditis due to paludal miasma the aortic valves are usually the first engaged.

Now, as I have stated, my experience is directly opposed to that of Hayden and Stokes in these respects. In the first place, I have found—as Flint, Niemeyer, Bamberger, Fuller, Sibson, and Latham found—that endocarditis is much more frequent than pericarditis. Secondly, I have found the liability to endocarditis to be in the inverse, and not in the direct, ratio to the severity of the joint-symptoms.^a Thirdly, I have not found the liability to cardiac affections to be in direct, but rather in inverse, proportion to the severity and duration of the pyrexia. I have frequently noticed that the heart became engaged when, and as, the temperature fell—as a salt crystallises when fluid cools; and that endocarditis flourishes with a low, even with a subnormal temperature. Fourthly, I have found acute rheumatic endocarditis generally curable, agreeing herein with Latham and Sibson; Latham finding that nearly one-fourth left hospital apparently cured—free, at all events, from murmur; and Sibson finding as many as one-half of his cases recover; whilst Hayden lamented that he had not had the good fortune to meet with more than one case of complete recovery out of some hundreds.

As to the locality of origin of the endocardial mischief, I find the aortic outlet to be generally the first affected; but the mitral outlet, although engaged later, generally the more permanently affected.

In rheumatic endocarditis, I find that the pulse generally gives, by its altered character and tone, the first indication of the onset of the disease; whilst a softened systolic, or, in some cases, a post-

^a I have rather found the less the joint-trouble, the greater the danger of heart-engagement, and *vice versâ*.

systolic, or diastolic, aortic sound generally precedes, by some days, the invasion of the mitral area.^a

I submit some charts, which show not alone the possibility, but the actual occurrence, of endocarditis, apart from, and independent of, any elevation of temperature; and these charts are in no way specially selected, but rather types of what I generally find. This *apyrexia* has an important bearing on the question of treatment, for, manifestly, if there be no pyrexia, and no pain, there is no scope for drugs that have the faculty of easing pain or reducing temperature—on the contrary, they may do much harm, by lowering the vital power.

I submit here the charts of four cases of acute rheumatic endocarditis, which I have recently had under my care in St. Vincent's Hospital, in which the disease existed quite independently of any fever; in fact, as will be seen, for the greater part of the time the temperature was, in a marked manner, subnormal; and in one case improvement was coincident with a slight rise in the temperature. This last was the case of a delicate girl, a servant in a country house near Dundrum, which has proved a rather fertile nest for rheumatism, as I have had the wife and three children of the gardener, who live in the yard adjoining, suffering at various times from rheumatic endocarditis, two of them without any *arthritic* trouble. This girl had not much arthritis, but developed very bad endocarditis, which first manifested itself at the aortic outlet, but quickly migrated to the mitral area, and firmly established itself there for some weeks. Under the influence of treatment she gradually improved. She left hospital ten days ago; and I saw her yesterday, when the murmur had completely disappeared. Although she has *no* murmur, she is, as she always has been, very anæmic; and so, when leaving, I put her on full doses of the tinct. of the perchloride of iron, which she is to continue.

The second case which I submit is that of a young constable in the Dublin Metropolitan Police—a body of men who, from constant exposure to wet and cold, are especially subject to rheumatic

^a The metastatic character of rheumatism is sometimes shown, no less by its jumping, as it were, from one joint to another, than by its *metastasis* from one cardiac area to another.

attacks. This man had recently left another hospital, after having recovered from an attack of rheumatism, and had resumed duty in very severe weather. Although on duty, he was feeling very ill; he had no joint-pains, but complained of malaise and sick stomach. On examining him, I found his pulse quickened and irregular, being occasionally intermittent. His temperature was nearly 101° F. The first sound was softened at the aortic outlet, and the heart's action was laboured. At my suggestion, he was permitted by Dr. Nedley to come into St. Vincent's Hospital. For three weeks I had him confined to bed. The temperature throughout was *subnormal*; the cardiac intermittency lasted for more than a week. At first, as I have said, there was softening of the first sound at the outlet of the aorta; then, in about a week, a murmur became developed at the mitral orifice, and remained for about ten days, when it cleared off; and now there is no trace of it, whilst a very slight alteration of the first sound at the aortic outlet remains. In this case myocarditis existed with endocarditis.

The third case is that of a boy, aged sixteen years, who came into the hospital on the 13th of last month (March), suffering from rheumatic fever. The joints only were engaged; the heart was unaffected. His temperature on admission was 100° F.; after some days it sank to the normal; and *then* endocarditis manifested itself, first revealing itself at the aortic, and then at the mitral outlet; thenceforth the temperature was, for the greater part, *subnormal*.

The fourth of these cases was also that of a constable in the Metropolitan Police, and, like the former case, he had recently left hospital after an attack of rheumatic fever. On admission he was suffering very much joint-pain, especially in his knees, shoulders, and elbows, and perspiring copiously. He had no heart affection. His temperature was 101.5° F. Under the influence of thirty-grain doses of salicylate of soda three times a day, and of blistering, the temperature steadily and quickly fell, and pain was relieved. *Then* endocarditis presented itself in softened cardiac sounds; and although the salicylate of soda was discontinued, the temperature thenceforth remained normal or subnormal.

The next case which I shall present to you was of a very different character. It was the case of a young fellow named Keegan, aged twenty-nine years, an intelligent and sober shop-assistant, who was admitted to hospital on the 10th of October last (1888), with symptoms which resembled typhoid. He looked chilly and pale; had a whitish-brown tongue, and a temperature of 102° F. He had got a wetting a fortnight previously, and felt unwell in the interval, but had no joint-pains. Being struck by the character of his pulse, which was rapid, soft, and yielding, I examined his heart, and found endocarditis already developed. On careful examination in bed, I found a loud-toned, rather musical, presystolic mitral murmur present. He had no joint-trouble. The temperature, as you may see, oscillated violently for some days, rising as high as 103.4° F., and falling as low as 97° F. On the sixth day after admission it suddenly sprang from 97° F. in the morning to nearly 106° F. in the evening, and in a few hours fell again (quickly, almost as mercury) to 97.5° F., subsiding a degree further before next morning, to 96.4° F. The rise in temperature was attended by anxiety and distress, but unattended by cerebral disturbance. The fall was marked by considerable prostration. The Resident Pupil had given, on observing the rise in temperature, twenty or thirty grs. of salicylate of soda, and attributed the fall of temperature to the action of the drug; but I thought it quite inadequate to effect such a profound change; and attributed the rise to profound central nervous disturbance; and the fall to reaction. The variation of temperature, amounting between the rise and the fall to between 18° and 19° F., was most remarkable, and seemed to me of bad omen. The case went on with fluctuating temperature, varying between 97° and 102° F., until it had sunk as low as 96° F., when it began again to climb up by rapid steps until it reached 104° F. on the evening of the 9th November. By next morning, just a month after admission, it had fallen to 97.4° F., a fall of nearly 7 degrees. As I have already mentioned, on admission the patient presented a loud, well-marked presystolic mitral murmur. This murmur now suddenly became altered in character; it got much

lower in tone and less distinct. This apparent improvement did not, I explained, augur well. Taken in conjunction with the rapid rise and fall in temperature, which favoured hyperinosis of the blood, I diagnosed cardiac thrombosis; and prognosed the danger of embolism. For some days the case progressed quietly; when suddenly one night the thrombus became dislodged from its moorings and was wafted into the brain, lodging in the right middle cerebral artery; and next morning I found my patient with left hemiplegia. It happened to be my class morning, and pointing out the case to my pupils—who had carefully watched its progress—I stated what had, in my opinion, occurred—namely, that the thrombus, the formation of which we had recognised, had become dislodged from the mitral orifice; and passing from the heart had been arrested in the right middle cerebral artery; and, if that were so, then I said the recently-altered presystolic mitral murmur would have probably regained its pristine tone. And so we found it. It needed little prophetic power to tell what the result should be—embolism, softening, and death. In a brief space the whole cycle of rheumatic endocarditis was traversed before our eyes. Rapid oscillation of temperature continued, a rise of over 5 degrees being quickly followed by a fall of 6, and then succeeded by rapid zig-zag rise and fall—like lighting flashes—until finally it sprang upwards and was lost in *ante-mortem* hyperpyrexia.

The next case which I will very briefly refer to is that of P. D., formerly a coal porter, and subsequently hospital porter at St. Vincent's; who has had no less than a dozen different attacks of rheumatic fever—characterised by excessive arthritic pain—but never by any cardiac complication—except possibly once, by myocarditis. In one attack his life seemed in imminent danger from hyperpyrexia, attended by restless and violent delirium, both of which were arrested and his life probably saved by severe blistering to the nape of the neck, and a large dose of quinine. I mention this case because of its bearing on the causation of hyperpyrexia, and its treatment.

The next case which I shall record occurred in private practice, and deeply interested me. It was a case of what must be regarded as

septic, typhoid, or ulcerative endocarditis. Its course extended over five weeks and terminated fatally. I saw the case in the beginning once, then twice, frequently three times, and occasionally as often as four times a day. It was seen on consultation repeatedly with me by Dr. Redmond, than whom no one could desire a more loyal or helpful colleague, and was also once seen in consultation with us by Dr. Nixon. It was the case of a young lady, aged twenty-four years, whose father had died, at a comparatively early age, of cerebral embolism, or hæmorrhage; and whose mother had died of broncho-pneumonia degenerating into phthisis. Her brothers and sisters were not robust; one brother was threatened with phthisis, and had taken a voyage to Australia, where he had been advised to remain at least for some years, because of pulmonary delicacy. The girl herself was slight but hardy, and was very fond of exercise. She had spent her holidays yachting around the Scotch coast, but did not seem to derive the benefit which was expected to follow from such a healthful and enjoyable holiday. On the contrary, she felt readily fatigued, and, before I was called in, had been to see a leading member of our profession here in Dublin. She was complaining at the time, in addition to want of strength, of want of appetite and disordered stomach. Feeling so weakly, she or her sister had suggested to the physician whom she consulted, to give iron. I only mention this to show that she merely thought herself out of sorts. However, the doctor, more wisely, finding her stomach disordered, prescribed for her a stomachic mixture; bicarbonate of soda in a bitter infusion. Evidently she had no cardiac affection at that time. Some days afterwards feeling very ill, and seeming feverish, a friend, a senior and well-informed medical student, induced her to take 10 grs. of sulphate of quinine. A few days afterwards, on the 11th of December, 1888, I saw her for the first time. Her temperature then was 103.4° F., her pulse 120. She presented a typhoid aspect; had a coated tongue but no diarrhœa; no eruption and no cough or lung trouble; she had not then, nor had she had previously, nor had she subsequently, any joint pain whatever. I inquired into the condition of the closets and drains but found nothing wrong. The house is in a healthy, elevated,

airy locality, in a southern suburb—a rather new but dry house—on good soil. On examining the heart I found well marked endocarditis present, marked by a prolongation and softening of the first sound at the aortic orifice; the mitral and pulmonary areas were then unaffected. By next day the temperature had fallen to 99° F., but the day following it rose to 104° F.; and later on that day to 105° F.; subsequently falling that same evening to 102.8° F.; next morning it had risen to 103.3° F.; later on it sank to 101.2° F.; at night rose to 104° F.; next morning sank to nearly 100° F. (100.2° F.); the next day it sank as low as 99.8° F., when menstruation occurred; but remained for only a short time. During the next week the case pursued a varying course, the temperature varying between 100° and 103.8° F. By the 25th of December (Christmas Day), the temperature was normal. For a week all went well. The endocarditis which had originally declared itself at the aortic orifice had shifted, first to the pulmonary, and then to the mitral outlet, where, for a time, it established itself. But the condition was steadily improving and promised a favourable termination—coincident with the fall in temperature, and the general improvement—till the first of January, when a fresh Alpine ascent was, so to speak, begun; and it became evident that a fresh battle had to be fought. By the 3rd of January the temperature had reached 104.4° F., the pulse 120. The respiration was never quickened. Gastro-enteritis of a very acute and severe character now set in, attended by vomiting and diarrhoea. At twelve o'clock, p.m., on the 4th of January, the temperature reached 105° F., but fell next morning to 100.8° F. Then for three days it varied from 102° F. to 104° F.; rising the day following (the 9th), to 104.8° F.; then it fell to 101° F. By this time the patient had become greatly exhausted; she grew weaker and weaker; was first restless, then delirious, then became unconscious. Diarrhoea was continuous, the odour of the most offensive, acrid, and almost intolerable, character. For some days a catheter had to be passed, urine being secreted freely; coma and profound collapse set in; she became algid and seemed moribund. As a last resource I injected a few drachms of sulphuric ether hypodermically. A slight rally took

place—next morning the patient was semi-conscious, then became more conscious, then somewhat hysterical. Next day, January 13th, the temperature stood at 99° F.; she was more conscious in the morning; in the evening was quite conscious, and menstruation set in. Next day menstruation ceased, but the tongue was clean and she was quite bright, although the temperature rose to 101·2° F. That night she was quiet but sleepless, and sleepless during the following day, the temperature falling to 99·8° F. Still no sleep; then the mind began to wander; delirium set in, followed by exhaustion; the pulse grew weak; then complete unconsciousness ensued, and the temperature began to rise—creeping up higher and higher, steadily, until 107° F. was reached—and soon after the end came.^a

My next, and, I am sure, you will be glad to hear, my last, two cases are of pericarditis, the one with, the other without effusion. With these I will be brief. I shall deal first with the case of pericardial effusion. The patient, a schoolboy, aged fourteen years, was admitted to St. Vincent's Hospital on the 12th of February, 1889. He was a delicate, strumous-looking boy. His temperature on admission was 100·5° F., but it rose that evening to 102·5° F., and afterwards ranged between 99° F. and 102° F. His family history was fairly good. He himself had had scarlatina and typhoid fever, but they left no apparent ill effect. The history of the present illness was, that about a month before admission to hospital he had been thrown and hurt at football. A week afterwards he felt pain in the right side and epigastrium, and consulted a medical man who ordered him a plaster, which relieved the pain for a time. Subsequently another doctor, who was called in, seems to have diagnosed enlargement of the liver; for which he ordered a blister. On admission to hospital the mark of the recent blister was quite fresh. The liver was found pushed downwards, and above the blistered surface was red and puffy as if pus had formed there; but the swelling seemed due to the irritation of the blister. The right side was

^a I have since had another case of acute ulcerative, septic, or malignant endocarditis, which terminated fatally, in six days, by embolism, hemiplegia, and hyperpyrexia.

found greatly widened and flattened, being a few inches more in girth than the left, and was quite dull on percussion. The breathing was very hurried, and the pulse small, wiry, and rapid. Pleural effusion, displacing the liver downwards, was diagnosed, and verified by aspiration, which brought away nearly a quart of serum. A slight friction sound was audible at the summit of the dulness, but was barely perceptible before operation; however, a loud friction sound developed after the removal of the fluid, and thenceforward the pleuritis quickly cleared off without any recurrence of the fluid.

At the same time, however, as the pleural effusion was diagnosed, it was found that there was also pericardial effusion. The heart sounds were scarcely audible, and dulness was found to exist from the top of the sternum to the ensiform cartilage, and from the right of the sternum for some inches transversely. Dulness also extended from below the left nipple to about the third rib on the left side. I had almost determined to have the pericardium tapped; but I resolved to give a chance, poor though it seemed, for absorption. Accordingly I ordered the patient 1 gr. of grey powder with 5 grs. of saccharated carbonate of iron three times a day, and had the præcordial region painted with lin. iodi. After some days I was gratified to find an apparent shrinkage in the extent of the effusion, and then, after a few days more, a friction sound appeared toward the apex of the heart, which gradually crept upwards until it was audible all over the cardiac region, then it began to fade from below upwards until finally and quickly it completely disappeared—and with it the dulness vanished; the boy gradually improved, lost his dyspnœa, and gained strength; syrup of iodide of iron with cod liver oil was substituted for the hyd. c̄ creta and fer. carb. sacch., and the patient steadily and satisfactorily progressed towards apparently complete recovery. He has now left hospital with no effusion, and, so far as one can judge, with no adhesions.

The last case—positively the last—is that of a gentleman who happens to be a member of the Irish parliamentary party, and will not, therefore, I hope be debarred from obtaining your sympathy. Just a little more than two years ago, he became suddenly ill in

London. The weather at the time was intensely cold; the House of Commons intensely warm, stuffy and contaminated with sewer-gas. Sittings were late, and leaving the hot, if not *heated*, air of the House of Commons, and going out into the ice-cold, foggy night or morning air of London, with the vital power necessarily lowered by the vitiated air of the House—what wonder that one should, so to speak, catch cold? This gentleman, about thirty-five years of age, was very strong, had never previously, one might say, been ill—since when a child he had had scarlatina. One day he was suddenly seized with violent pain, apparently in the stomach. A doctor was called in, who seems to have prescribed a stomachic. The pain continued, so another doctor, a friend, was called in. He ascribed the pain to *angina pectoris*. During the week or ten days which followed, the patient suffered great pain of a stabbing character; was unable to walk without extreme distress, and had no appetite. He remained up till the Monday week after his attack, for nine days, and continued going about, although with difficulty. He made two short railway journeys on the Monday. Thenceforth he was in and out of bed for three or four days. At this time his medical attendant wrote to me—“I find a very distinct diastolic murmur at the base of the heart, immediately over the painful part, heard more distinctly towards the lower end of the sternum, with muffling of the second sound. I believe the aorta to be the seat of the trouble; but, of course, I cannot tell whether or not it is quite recent.” Evidently the case was regarded as one of aortic regurgitation. I crossed over to see the patient, and found him in the early morning on my arrival, with a temperature of 101° F., and suffering from well-marked acute pericarditis. The case was complicated by much gastric disturbance, a common phenomenon in such cases which often proves a source of error. The temperature ran up to 103° F. for the next few evenings. There was great exhaustion consequent on the previous exertions, as well as caused by the fever and sickness of stomach. Collapse was a few times threatened, and at night restless delirium was present. Rectal medication had to be resorted to. Suppositories with quinine and morphia being administered; as well as free

vesication, over the region of the pericardium affected, being resorted to. After a few nights a regular crisis set in, marked by fall in temperature, copious perspiration, and cessation of delirium; and thenceforth the patient made a rapid and complete recovery; and has since, now two years ago, been free from any cardiac trouble; presenting not the least trace of his dangerous malady, notwithstanding much exposure and, perhaps I may say, occasional hardship.

Now a few words briefly as to causation and treatment: To treat of endo- or pericarditis and leave rheumatism out of consideration, were to play Hamlet without the Prince of Denmark. If rheumatic fever cannot say, like Coriolanus, "Alone I did it"—it may at least say of the complications, "*Quarum pars magna fui.*" They are, in truth, well nigh, if not quite, inseparable. The causation and treatment of rheumatism are, therefore, largely the causation and treatment of endo- and pericarditis; but not wholly; for what might, for instance, benefit one aspect of rheumatism might injure another—what relieves the pain or reduces the temperature, may not equally benefit, nay, may interfere injuriously with the heart affection. And yet there is much truth in Watson's saying, that to all intents and purposes the heart must be regarded as a joint in rheumatic fever. Yes, and an important joint, nay, the most important of all, on which all the rest hinge. And yet, although rheumatic fever involves the heart as well as the joints, all endocarditis, and all pericarditis, is not rheumatic: no more than all arthritis is rheumatic. What is the origin of rheumatic endo- and pericarditis is involved in the still larger question—What is the cause of rheumatic fever? Is it a blood disease, or is it a neurosis? Where are its "*fons et origo?*" In the blood, or in the nervous system? I believe it to be a neurosis. All recent investigations, as well as a careful observation of the facts, point in that direction. The researches of the two Mitchells, father and son; of Gull, of Charcot, of Latham; the indications of pathology and of physiology, which is the elder sister of pathology, point alike to its nervous origin. In no other way, by no other theory, can we account for all the phenomena witnessed—for the sudden metastases, for the hyperpyrexia. Lactic acid we must regard as a *product* of the disease,

and not as the disease itself. In the nervous system, whether in the medulla, or in the floor of the fourth ventricle, must we look for the exciting and controlling power. In the dominant trophic centre and in the thermo-taxic, or heat-regulating, centre, lying side by side in the medulla, must we seek for an explanation of the development and, of the arrest, of the changes which take place in rheumatic fever. In them must we seek, and in them only can we find, an explanation of the remedial effects of treatment, be it counter-irritant, sedative, antipyretic, or revulsive. In the thermo-taxic centre, which some place in the medulla oblongata, some in the pons varolii, and some in the floor of the fourth ventricle, do we find a sufficient and rational explanation of the phenomenon of hyperpyrexia—for simple pyrexia stands on a different basis. And in its existence we can see an explanation of the apparent arrest of hyperpyrexia by the employment of blistering.

That purely spinal injuries can cause hyperpyrexia is well-known. Some years ago I saw a case of a labourer who had fallen off a high cartload of hay. A few hours after the injury he had a temperature of 107°–108° F., with spinal paralysis and priapism, which persisted till a speedy death released him from his sufferings.

“Is there,” asks Dr. Buzzard, “something which we may call, provisionally, a trophic centre for the osseous and articular system in the immediate neighbourhood of the vagi? As I have suggested,” he adds, “on a previous occasion, the discovery of such a centre would materially help us to explain the remarkable association of cardiac complications with the joint affection of acute rheumatism, as well as the sweating^a—characteristic of this disease—and the occasional hyperpyrexia which occurs in it. And it might also help to throw light upon the obscure pathology of arthritis deformans.”

In this connection, it is interesting to observe that Sir. Dyce Duckworth holds that gout is also a primary neurosis, having its seat in the medulla. Paget also believes in the nervous origin of gout—“There are reasons enough for thinking,” he writes, “that changes in the nervous centre determine the locality of each

^a Due to paralysis of the sympathetic.

gouty process ; while changes in the blood and tissues determine its methods and effects ; and that thus we may explain the symmetries of disease in gout—sometimes bilateral, sometimes antero-posterior—and thus its metastasis.”

It is now admitted that vaso-motor centres exist along the spine, each presiding over a particular vascular area : that each one may be excited reflexly ; but that all are controlled by a dominant head-centre, so to speak, in the medulla. This dominant vaso-motor head-centre it is which controls the nutrition of joints, and thus is explained why an injured joint is especially liable to rheumatic attacks—its centre having been excited, fatigued, and partially paralysed ; and hence it is that blistering a joint by constringing the vessels, lessens the blood supply and reflexly stimulates the jaded centre. These trophic centres are like so many roots—with the chief centre as a tap root—whose branches, as it were, spread far and wide throughout the body, to heart and joints ; and hence are so liable to be influenced by changes of temperature. If a person, weakened from any cause, be exposed to wet and cold—be, in fact, subjected to chill—glycocine, which is formed in excess, is transformed into uric acid ; which, acting on the vaso-motor centres, at first stimulates, and then exhausts them ; the superficial blood-vessels become dilated and partially paralysed ; destructive metabolism is promoted in the muscles, and so lactic acid is produced, which dilating the smaller vessels, stimulates the centres ; the paralysis of the sympathetic produced causes perspiration, which aids the kidneys in eliminating the diseased products—uric and lactic acid.

As to treatment I shall be very brief. We have seen how blisters may act beneficially ; that they do so I am thoroughly satisfied by experience. Now-a-days salicin, salicylic acid, and the salicylates have taken the place of the once much-lauded alkalies. Of these I prefer the salicylate of soda. That it relieves pain and lowers the temperature, in most cases, I am satisfied—but there its utility, I think, ends. Whilst it is said to prevent the development of cardiac complications, as was claimed for the alkalies, even Dr. Latham, who praises it highly—showing how it breaks up, and prevents the

formation of, uric acid—does not claim that it cures them. It sometimes causes distressing tinnitus, like quinia. Harm is done certainly, if not directly at least indirectly, by the speedy immunity from pain, which is taken to mean recovery; and so patients go forth gladly, to return sorrowfully, labouring under a relapse; it may be suffering from firmly-established endocarditis. In rest, long-continued and persistent, I believe as the sheet-anchor of acute endo- and pericarditis. In repeated blistering, and in the internal and persevering use of iodide of potassium and red iodide, or perchloride, of mercury, followed by the tincture of the perchloride of iron, or ferrum redactum; and occasionally in the employment of oil of turpentine, have I found the only means of mitigating or of curing—if we can cure—acute endo- and acute pericarditis.