Clinical lecture on the treatment of acute rheumatism, pericarditis, and pneumonia, by the eliminative method / by B. Wills Richardson.

With B Wills' Compts

CLINICAL LECTURE

II.

ON THE

TREATMENT OF ACUTE RHEUMATISM,

PERICARDITIS, AND PNEUMONIA,

BY THE

ELIMINATIVE METHOD.

BY

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ON THE TREATMENT OF

ACUTE RHEUMATISM, PERICARDITIS,

ETC.

THE case that I wish to bring under your notice this morning illustrates in a remarkable and instructive manner the restorative power of Nature when judiciously assisted by Art.

You are aware that there are several ways of treating the disease under which this patient laboured, but as these are to be found in almost every elementary work on medicine, I shall not re-produce them here, and will at once proceed to remind you of the mode by which our patient's case was conducted to a most favourable issue.

The treatment of acute rheumatism by elimination has had many advocates, and one of the ablest and most eminent of these was the late Dr. Robert Bentley Todd, whose practice showed such great success in carrying it out, that it is but fair to connect his name most intimately with this method of treatment.

Whenever a new or peculiar mode of treating disease is advocated, and by such men as Dr. Todd, it is the duty of the clinical teacher to bring it to the test of practice, provided he is convinced that the patient will incur no risk thereby. I cannot shut my eyes to the assertion of Dr. Todd, who tells us, that since he adopted the treatment by elimination, he had much more rarely "met with those

accidents of the disease, pneumonia, pericarditis, delirium," &c., in the same severe form in which he used to meet with them under a more depleting treatment. Again, he found "that under this treatment the duration of the disease does not exceed from ten days to three or four weeks, and that relapses, which were very frequent under the treatment by bleeding, are of rare occurrence under this plan."* In consequence of such strong testimony as this in favour of the practice, I determined, as the opportunity recently presented itself, that you should see it carried out as nearly as possible in accordance with the directions of Dr. Todd. The case is moreover of particular interest, because not only was our patient affected with the severe articular symptoms and the characteristic fever of acute rheumatism, but she also suffered from some of the most formidable accidents of the disease—namely, a very severe attack of pericarditis, and subsequently double pneumonia, from all of which she was at the end of the fourth week convalescent, and this without having taken any mercury except as a purgative, and without either general or local bleeding.

Case.—Harriet Bellis, æt. 18, domestic servant, and a native of Wales, was admitted on Tuesday, 8th January, 1861, into the Adelaide Hospital, labouring under acute rheumatism, which she attributed to having slept in a damp room detached from the family residence.

On the previous Saturday evening, the 5th inst., a fire had been lighted in that room for the first time since she inhabited it, and she says that vapour "was afterwards given off by the damp walls."

The next morning both her ankle-joints were tender,

* Clinical Lectures, edited by Dr. Beale, 1861.

painful, and swollen. On Monday, the 7th, the knees were similarly affected. About this time she had a rigor, followed by heat, and was quite incapacitated from moving the limbs.

Tuesday morning she was much worse, and was consequently brought to the hospital.

9th, third day of the seizure: Excessively restless; face flushed and anxious; skin hot and covered with a copious perspiration, having a strongly acid reaction; pulse 130, unsteady and irregular; respirations 34; tongue brownish; bowels confined; ankles and knee-joints swollen, exquisitely painful and tender; light pink blush over the affected joints; urine acid and loaded with urates.

On applying the ear to the cardiac region, no morbid sound was heard, but the heart's action was most irregular, which, I may observe, is a very suspicious symptom when occurring in the course of this disease, and too frequently is a forewarning of subsequent cardiac mischief.

She was first put upon the opium treatment, which was so much relied upon by Brugnatelli in this painful disease. One grain of the watery extract of opium was ordered every third hour, and its effects were carefully watched; for although there is in such cases a remarkable tolerance of this drug, we occasionally meet persons who will not bear such frequently repeated doses. Wine, three ounces, arrowroot, &c. The affected joints to be swathed in hot damp flannel rollers impregnated with opium.

4th day: She passed a restless night. The wrists and shoulders are now affected, the joints affected yesterday being a little easier. She complains of great uneasiness in the region of the heart, where the stethoscope revealed a remarkably well-developed friction sound accompanying

the beats of that organ. This was audible over the whole cardiac region, and was also felt by the hand placed upon the sternum, giving a sensation somewhat similar to that occasionally perceived in some forms of enlarged bursæ. An endocardial murmur could not be detected. No alteration in the other symptoms. Pulse 136. She was this morning put upon the treatment recommended by Dr. Todd. A large blister was applied over the heart, and she was given a grain of opium, a grain of hippo, and five of nit. potash every third hour, and an alkaline mixture containing half a drachm of bicarbonate of potash in each ounce of fluid, one ounce of which was taken night and morning. Her wine was increased to five ounces, and she was to have strong beef-tea during the day.

5th day: Frottement very well marked; restless night; tongue brown and furred; bowels not moved; pulse 136; respirations 36. To continue the remedies, and also ordered five grains of blue pill, to be followed in the morning by a purgative draught containing sulphate and carbonate of magnesia.

7th day: The patient's general symptoms much better, the joints being considerably easier, although the skin is still hot and perspiring profusely; pulse 120. To continue the remedies.

8th day: Still further improvement; pulse has fallen to 112, and the cardiac friction sound is not so intense; could not detect any endocardial murmur; joints almost free from pain, and the heat of skin is lessened. Continue the remedies. Urine still acid.

9th day: Although there is an amendment in the general symptoms this morning, there is some increase of dulness over the heart, with diminution of the frottement, probably

in consequence of liquid effusion. Another blister was applied near the site of the first one, and the wine was increased to eight ounces. Pulse 112.

10th day: The cardiac dulness is lessened, and the frottement is more indistinct, and can now only be heard in isolated portions of the cardiac region. The joints have ceased to give any uneasiness, although they are still slightly swollen. Pulse 104. Same treatment.

13th day: Further improvement; the skin is slightly moist and cool, and the joints are perfectly free from swelling or pain; urine free from lithates and is neutral; pulse 102; the pericardial friction sound is now audible only at the base of the heart; no increase of dulness on percussion. Same internal treatment.

16th day: Pulse 98; bronchitic râles over the chest. Turpentine stupes to back of thorax.

17th day: Pulse 104; skin somewhat warmer; cough troublesome; the frottement is now altogether inaudible, and there is no endocardiac murmur to be heard. Repeat turpentine stupes.

18th day: Pulse 110; skin hot and dry; slight crepitus in lower and posterior part of left lung without perceptible dulness on percussion. Same remedies; turpentine stupes to back of chest.

19th day: Pulse 120; respirations 38; skin remarkably hot and dry; both lungs have become hepatized posteriorly since last report; sharp whiffy bronchial respiration with increased vocal resonance, supplanting the respiratory murmur as high up as the spine of each scapula, corresponding to which there is dulness on percussion, but not so decided as in pleuritic effusion. No fresh mischief in the cardiac region; urine neutral; the opium and alkalies

were now stopped, and their place was supplied by one ounce of aq. ammon. acetatis every second hour, and the turpentine stupes were directed to be applied to the back of the chest three times daily. Half an ounce of wine every second hour.

20th day: Pulse 126; respirations 38; skin hot and dry; face flushed; cough troublesome; sputa slightly rusty and viscid. Continue remedies.

21st day: Pulse 136; respirations 40. Same remedies, with the addition of a draught, at night, of tincture of hyoscyamus, chloric æther, and aromat. spt. ammoniæ.

22nd day: Pulse 160; respirations 44; skin very hot and dry; no increase of pneumonic consolidation, but the patient is very depressed, and has a flushed and anxious countenance. One ounce of wine was ordered every second hour, and also to be given during the night. The water of acetate of ammonia was replaced by three grains of sulphate of quinine every third hour, and the draught and turpentine stupes were continued.

23rd day: Much improved; the pulse has fallen to 132 and the respirations to 38; general perspiration; crepitus audible immediately below spine of right scapula, and the bronchial respiration is here not so decided as it had been. Same treatment.

24th day: Pulse 126; skin moist; respiratory murmur replacing bronchial respiration in right lung. Continue remedies.

25th day: Pulse from 106 to 116; respiratory murmur audible in all the site of the bronchial respiration in right lung, and also returning in the left lung.

28th day: Pulse 96; skin cool. Continue remedies, excepting turpentine stupes.

34th day: Both lungs free from morbid sounds. Instead of the sulphate of quinine she was now given three grains of citrate of iron and quinine three times daily. Wine reduced to eight ounces in the twenty-four hours.

36th day: Convalescent. No morbid sound in the posterior part of the chest; cardiac region carefully examined without the discovery of any indication of valvular lesions. The only symptom to lead to the inference that a morbid process had been going on in this locality being some slight dulness on percussion, probably the result of thickening of the pericardium by inflammatory products. As she has a great aversion to cod-liver oil, we ordered her an ounce of the following mixture three times daily:—

℞. Glycerinæ ℥j.
Cit. ferri et quininæ, gr. xxiv.
Aquæ ℥vii. Misce.

In the commencement of my observations I told you that, as far as practicable, I was resolved to try the treatment by elimination in this case, but as some of my junior hearers may not yet have studied this mode of treating rheumatism, I must make it the subject of a few remarks. The object of this treatment is "to promote the elimination of morbid matter by the various emunctories," such as the skin, the kidneys, and the bowels. "To use antacid remedies, and to give large quantities of fluid for the free dilution of the *materies morbi*, and to supply the waste caused by the drainage from diaphoresis and diuresis."

Now, in the case before us, in order to promote the action of the skin we used the combination of opium, hippo, and saltpetre, recommended for this purpose by Drs. Todd and Copland, and which I may remind you is very similar to the original constitution of Dr. Dover's powder, and

is, in consequence of the substitution of nitrate for sulphate of potash, better adapted for producing diaphoresis than the Dover's powder of the Pharmacopœias. To act upon the kidneys we gave the bicarbonate of potash, and upon the bowels an occasional mercurial purgative, followed by sulphate and carbonate of magnesia.

What the *materies morbi* of acute rheumatism is, has not yet been satisfactorily ascertained, but there are very strong grounds for supposing that lactic acid plays a very important part in developing the symptoms. It is probable, however, as Dr. Copland suggests, that there are other causes in operation, and that they are also favoured by the increased fibrination of the blood and diminution of the red globules so usual in the disease.

Within the last few years Dr. Richardson of London, performed some very important experiments while endeavouring to solve this mystery. They are, moreover, peculiarly interesting from the fact that lactic acid was the substance he selected for the purpose. Indeed, he tells us he made choice of it in consequence of the theory advanced by Dr. Prout, and subsequently supported by the arguments of Drs. Todd, Williams, Fuller, Headland, and Mr. Spencer Wells, that it is the probable *materies morbi* of acute rheumatism. After having tried various modes of introducing the acid into the circulating fluid, he found that it was readily absorbed into the blood when thrown into the peritoneal sac. In the first successful operation the acid diluted with water was forced by a suitable apparatus into the peritoneum. The next day the animal was dead, and the post-mortem appearances were of the most important character. I cannot give all of them, but shall confine myself to those connected with the heart, which

chiefly interests us to-day:—The left auricle contained a small coloured coagulum. The left ventricle contained some fluid blood. The mitral valve was greatly thickened, red, and œdematous. On the surface of the valve there was a small fibrinous deposit, which adhered to its position by means of an under layer of a white glutinous fluid, the like of which he found afterwards, could be made to exude from the valve on puncturing it with a needle. The surface of the endocardium lining the left ventricle had an intensely vascular appearance, resembling in fact bright red velvet. In another experiment, where the diluted lactic acid was thrown into the peritoneum of a terrier dog, he found after death that the vessels of the pericardium were finely injected. The endocardium of the left ventricle was of a brilliant red colour, having a velvety appearance. The segments of the mitral valve were thickened, œdematous, and also of a bright red colour; while around their free margins were several beads, varying from the size of a pin's head to that of a millet seed. The aortic valves were of a deep red colour, with thickening and eversion of their free borders. In a subsequent experiment with the same acid on another dog, the pericardial vessels were also injected, and a small quantity of lymph was effused over the left ventricle. In addition to well-marked inflammatory appearances in the tricuspid valves, the segments of the mitral valve were of a pale red, and very much thickened, they were firm in consistence, and their free borders were studded with whitish, pearly beads. In both sets of valves the pale appearance was caused by the exudation of lymph beneath the lining membrane. Dr. Richardson performed other experiments for the same purpose and with the same substance; but, as the results

were almost similar, it is unnecessary to occupy your time with reading them. Although his experiments are not conclusive as to what is the *materies morbi* in rheumatic fever, still they show, as he remarks, that an excess of lactic acid in animal bodies produces symptoms similar to those of acute rheumatism. But this is all that can be safely inferred from them. Indeed, their author himself states that whether lactic acid is the veritable cause of the symptoms in every or any case of rheumatism, is only an inference, and it has yet to be learned by experiments what formic, acetic, lithic, and butyric acids will do under the same circumstances.

The pathological appearances observed after Dr. Richardson's experiments give some countenance to the supposition that the fibrinous effusions on the valves of the heart in acute rheumatism are the result of inflammation. Some pathologists believe that the lining membrane of the vascular system cannot take on this process. Mr. Simon, one of the most able advocates for such a view, asks: "Is the lining membrane of the arterial system susceptible of inflammation?" "What are the bloodvessels of the lining membrane of the arterial system?" And he also tells us that the *vasa vasorum* "do not penetrate to a sufficient depth to influence materially (if at all) the nutrition of the lining membrane," which he considers is derived from the blood with which it has contact. He therefore "has great difficulty in believing that the endocardial deposits could be of inflammatory origin." With regard to the anatomical question raised by Mr. Simon, I may mention, that although anatomists seem to agree that the nutrient vessels of arteries ramify principally in the outer coat, yet in the larger vessels they also extend into the middle coat, but

according to Mr. Simon not (as I have before mentioned) "to a sufficient depth to influence the nutrition of the lining membrane." He therefore attributes the endocardial deposits in acute rheumatism, not to the inflammatory process, but rather to a fibrinous precipitation from an overcharged solution, the valves encrusting themselves with fibrine, "just as a stick in certain streams coats itself with a calcareous envelope." It appears to me that there need not be any difficulty in believing in the existence of *endocarditis*. Indeed, Dr. Richardson's experiments are quite conclusive on the point. In addition, we have the analogy of inflammation in other structures, in which, as Mr. Goodsir has truly observed, the capillaries are all on one side of the inflamed membrane, and the inflammatory products on the other. Now, in Dr. Richardson's experiments, when the swollen valve was pricked with a needle, a clear lymph oozed from the puncture. If the congestion and œdema were very great there was *a transudation of this lymph through the membrane on its free or ventricular* surface, and when that occurred there was at once laid the basis of a fibrinous deposit from the blood. Instead, then, of supposing that the deposits in the cavities of the heart in acute rheumatism are merely precipitated from an overcharged blood, there can be little doubt that these are the result of an antecedent inflammatory process. Dr. Richardson's experiments, I should also mention, do not support the description given by Virchow of the endocarditic process, as, according to the latter gentleman, there is first a swelling of the diseased spot itself, but there is *no exudation*, the cellular elements take up a greater quantity of material, and the spot becomes uneven and rugged. In some of the experiments of Mr. Bowman, it was observed that the

earliest changes in injuries of the cornea consisted in the deposition of nuclei or cytoblasts, which are soon found choking the interstices of the tissues in the lips of the wound, and covering its surface so as to occupy the space left between its opposite sides, and bringing them into temporary union. A somewhat similar result Mr. Redfern produced by passing setons through the costal cartilages of dogs, a considerable generation of cells in the part next the silk being the consequence. Now, if we have such changes in the *early* stage of inflammation in non-vascular structures, I think there can be little difficulty in admitting endocarditic inflammation as an established fact. The pathological appearances may be explained, however, in another way. We all know that outward transudation of some of the constituents of the blood frequently takes place through the walls of the vessels. Such being the case, may not also some of the inflammatory products of the outer structures of the vascular system pass in a contrary direction, the coagulating lymph undergoing (as Hunter long since stated in his observations on phlebitis) some changes in ts passage through the inflamed vessels, which obliges it to coagulate more immediately or much sooner than it would otherwise.

In this separation from the blood, it is probable, as he also observed, that it must "have undergone some change arising from the action of the vessels; for if this lymph was no more than the coagulating lymph, with its common properties, it would in such cases only continue to throw in more coagulating lymph, in addition to what was circulating, and therefore probably it would be carried along with the blood to the heart as a part of the common mass." Should these views be correct they would account for the

coagulation of the blood in an inflamed vein, without the necessity of supposing that the *lining membrane* is actively engaged in the inflammatory process, its nutrition suffering secondarily from that process going on in the other coats. For although, as Cruveilhier observes, the internal surface of the vein does not present any trace of inflammation, we find in the injection of the capillary vessels of the external surface of the vein and of the surrounding areolar tissue in the infiltration, the density and the fragility of this areolar tissue and of the *external tunic* of the veins, unequivocal traces of a prior inflammation. Finally, gentlemen, if the opinion that the appearance of vessels carrying red blood is a secondary stage of inflammation; in other words, that there is "a *vis à fronte*, which, by its attractive power, assists and regulates the *vis à tergo* of the heart," we can readily understand that inflammatory processes may take place in a part, although anatomically we cannot demonstrate vessels in such a structure.

In drawing this lecture to a conclusion, I must observe, we should not dogmatically assert that the favourable result of the case, is to be solely and altogether attributed to the treatment pursued. Some would possibly say that very little was done for the patient, and that the *vis medicatrix naturæ* was the chief, if not the sole agent, in effecting the cure. We could not, however, adopt such an opinion, when we recollect that during the treatment free excretion and secretion were maintained; that alkalies were liberally given, which we know act upon fibrine; that irritability of the nervous system was, as much as possible, guarded against, and that free vesication was had recourse to. At all events, we have the satisfaction of knowing that the vital powers were not undermined by the *nimia diligentia medici*,

and the history of the case fully proves that a person can, in four weeks, recover from an attack of acute rheumatic fever, acute pericarditis, and double pneumonia, without mercurialization of the system, or the abstraction of red blood, generally or locally.