but in others being seldom absent and always greatly increased at the periods. It originates in the ovarian region, and shoots down the thigh, often also down the leg and round to the back. There is also often present, especially on the accession of atrophy, the peculiar submammary pain of ovarian disease, generally felt in the left side only. Headache, nausea, or even sickness and great general discomfort, are always present more or less.

In the milder cases, treatment is generally successful in mitigating the sufferings, and often the ovary may be made, even in some very well marked cases of arrested development, to fulfil its functions completely. First of all therapeutic remedies there stands iron, which will be found in such cases to be of great use, even though there should be no general indications for its employment. There can be no doubt that many forms of this remedy have a specific power over the sexual organs, male and female; for, in a case of chronic metritis or subinvolution, smart hæmorrhage may be induced by large doses of iron. In ovarian dysmenorrhœa, it is best given during the intermenstrual periods in small doses, one to five drops of the liquor ferri perchloridi, well diluted, and increased suddenly to fifteen or twenty for a day or two previous to and during the menstrual flow; or better still is the substitution for this large dose of an iron and aloes pill, there being few better combinations in the Pharmacopœia than that old-fashioned remedy. Hot hip-baths and leeches to the perinæum at the period are often useful additions, with an occasional blister on the sacrum. To such as this, the treatment of delayed or difficult menstruation at puberty, due to inefficient ovarian development, must be confined; for the other means are only allowable in very obstinate cases, after the patient has been married, or when there are indications of premature ovarian atrophy. Marriage is, perhaps, the most efficient remedy, and one we ought seldom to hesitate to recommend; for, even if the patients may not have children, they will have better health, and they may even become pregnant if they marry early enough and are not mismanaged.

The last and most powerful aid is mechanical irritation of the uterus; but, as it is not free from risk, and therefore requires careful use, it is not always to be recommended. It is, besides, in the class of cases where the uterus is most at fault that it is least risky and most serviceable. The method of irritation I generally employ, as the most convenient and least troublesome, is the insertion of Simpson's galvanic pessary. This instrument has by some writers been very much decried, but I think by those only who seem to have used it indiscriminately and without reference to a proper selection of cases.

The irritation set up by the presence of a galvanic stem in the uterus is communicated indirectly to the ovaries in a manner that is not as yet explicable, but that it has an influence is beyond doubt, and, if it remain within bounds, it is in a large number of cases beneficial. A large experience has shown me that it is only in occasional instances that the stem cannot be borne, and that, if carefully watched during the first few weeks of its use, these cases are easily eliminated. In a case where I have been led to regard the use of the stem as advisable, I always begin with a small size, and, after this has been worn for two or three months, I change it for a larger one. For the first week after its introduction, it is not unusual for the galvanic stem to give rise to considerable discomfort and even positive pain, but this usually passes off if the patient keeps her bed for a few days, and there is no further trouble save from the leucorrhœal discharge, which is a part of the process. The action of the stem is not purely mechanical, as has been stated; for, very soon after its insertion, the zinc becomes coated with an albuminous deposit, from which the copper is free, and the zinc becomes corroded. It is certain, therefore, that there is a galvanic action set up, and the stimulating effects are due partly to this, and partly to the interior of the uterus being constantly bathed in a weak solution of chloride of zinc. However produced, it is certain that the uterus rapidly enlarges under the action, and there is every reason to believe that the ovaries take part in the increased activity. If once the uterus become accustomed to the presence of the galvanic stem, it may be worn for many months, and the longer it is retained the more permanent will be the benefit; but, if after a trial of a few months, say four or five, there is no apparent alteration for the better, the attempt may be given up and the case considered as hopeless.

In a very large number of cases of incompletely developed ovaries, another remnant of infantile life is met with in an exaggeration of the normal curve of the uterus, amounting sometimes to complete ante-flexion, and in this class of cases the galvanic stem is especially serviceable.

The results of my attempts to arrest premature atrophy of the ovary from any cause, when once begun, have been far from satisfactory; but this has been more especially the case when that atrophy has been due to a constitutional disease, such as tubercle. Sir James Simpson had a belief that the pretubercular amenorrhœa, so often seen in young

women, was a cause of the subsequent disease; and he, therefore, directed his attention to the restoration of the utero-ovarian function, as a means of treatment or prevention of the consumption. From the views previously expressed, it will easily be seen that I consider his theory to be based on error, though in some cases his treatment would seem to have been successful; but how much of his success was due to local, and how much to general, treatment cannot now be determined. It is not, however, a practice likely to meet with many followers.

A singular condition has been noticed recently by Dr. Priestley, of termenstrual pain occurring about midway between the periods, which is almost certainly due to an ovarian condition, though it is not clear of what kind. Since reading his paper, I have seen two cases, but have been unable to refer them to any category.

[To be continued.]

CLINICAL LECTURE

ON A

CASE OF FATAL HYPERPYREXIA OCCURRING IN ACUTE RHEUMATISM.

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ELIZABETH ELRIDGE, aged 23, a burler, was admitted an in-patient of the Leeds Infirmary November 17th, 1873. She was a well formed, healthy-looking young woman, of good family history, and had had good health till the present attack. About a week ago, she caught cold after being wet. She was feverish, had pains in the knees and ankles, but perceived no swelling. She took to bed; and, as she was getting worse, she came to the Infirmary.

On admission, her face was flushed; the body freely perspiring, and emitting the sour rheumatic odour; pulse frequent, rather hard, regular; much thirst; urine scanty. The auscultatory signs of the lungs and heart were quite normal. Temperature 104 deg.

℞ Misturæ salinæ effervescentis ʒi ss 4tis horis.

November 18th. There was much articular pain, aggravated at night. Evening temperature 104.5 deg.

℞ Liquoris morphinæ acetat. f. ʒss; aquæ f. ʒj; horâ somni.

November 19th. The morning temperature fell to 102.8 deg.

November 20th. The morning temperature had fallen to 102 deg.; evening, 104 deg.

November 21st. She did not seem so well; felt very hot, but was perspiring profusely. The pain had very much subsided, being felt only in the left shoulder. Temperature throughout the day about 104.5 deg.

November 22nd. This morning, she said she was free from all pain. She had profuse sour perspiration; the pulse had risen to 136; temperature 104.4 deg. The countenance was good. A gentle friction-sound was heard in a limited space over the middle of the heart.

℞ Potassæ bicarbon. gr. xv; aquæ f. ʒj; 4tis horis. Horâ somni sumat quinquæ sulph. ʒj.

During the evening of this day, she began to ramble a little; and towards midnight she became more excited, and attempted to get out of bed, and was noisy. The temperature rose to 106.3 deg. A scruple of chloral hydrate was administered, after which she became quieter; but she passed a restless night.

November 23rd. There seemed some improvement in the general symptoms. She was quite collected, and conversed rationally; but the temperature had reached the serious elevation of 106.75 deg. In the afternoon, the temperature rose to 110 deg., and she was passing into a comatose state. I was sent for, but before I arrived she was dead.

The *post mortem* examination was made twenty-three hours after death. The body was well nourished. *Rigor mortis* was imperfect. The blood was imperfectly coagulated. In the *Thorax*, the lungs were perfectly sound, free from adhesions, quite crepitant in all parts, rather full of dark blood. The heart was partially adherent to the pericardium anteriorly by recent lymph. There was not above an ounce of fluid in the pericardium. On the anterior surface of the heart, when removed, was seen a red patch two inches in diameter, roughened with recent lymph; and a similar patch posteriorly, of smaller size. The rest of the surface was smooth and normal. The substance of the heart was rather soft, not firmly contracted; the valves and internal structure were perfectly sound. In the *Abdomen*, all the organs were

quite sound. There was copious urine in the bladder. The *Brain* presented no appreciable deviation from a perfectly healthy condition. The capillary vascularity of the external pia mater was thought to be rather exaggerated.

Cases of rather sudden death occurring in the course of acute rheumatism, not previously presenting any symptoms of unusual severity, have been recorded from time to time; and as, in several of such cases, cerebral symptoms, such as pain in the head and delirium, have preceded death, they have been attributed to rheumatic inflammation of the membranes of the brain; and since, also, some of these rapidly fatal cases have been marked by a sudden subsidence of articular pains preceding the development of cerebral symptoms, it has been customary to speak of such as examples of a *metastasis* of rheumatism from the joints to the membranes of the brain. But *post mortem* examinations have not produced any uniform result of distinct inflammatory appearances in the brain, such as are met with, for instance, in the pericardium when it becomes inflamed in the course of acute rheumatism; neither is the subsidence of previously severe articular inflammation an invariable incident in these cases.

A considerable proportion of patients who have died with these symptoms have been young, previously healthy, and experiencing an attack of rheumatism for the first time. Such was the case with each of the three cases recorded by Dr. Ringer in the *Medical Times* of 1867; such is the fact with regard to my own case. Dr. Wilson Fox's two cases of hyperpyrexia in acute rheumatism were both first attacks of rheumatism, but were aged 49 and 36; these cases recovered under the treatment he has invented and described in his book *On the Treatment of Hyperpyrexia*.

The symptoms of fatal hyperpyrexia occurring in acute rheumatism, as presented in most cases, are, subsidence of any existing rheumatic pain; increased frequency of pulse; rise of temperature; the occurrence of pericarditis in many cases, but this is not invariable, nor essential; pain in the head and delirium. When the temperature reaches to 110 deg., the natural tendency is to a speedily fatal result.

These cases are not to be regarded merely as aggravations of the rheumatic complaint. Some new disorder, having a pathology distinct from that of rheumatism, is superadded, giving rise to the special symptoms, and occasioning the fatal result. In some instances, the patients have been convalescent from the rheumatism, when hyperpyrexia has occurred, speedily terminating in death.

The one circumstance common to all these fatal cases is the extreme temperature of the body attained contemporaneously with the development of the serious symptoms, or shortly before death. Acute rheumatism not unfrequently gives rise to a high temperature. The evening temperature may reach 104 deg., or even 105 deg., especially if there be any pericarditic complication; but any higher temperature suggests serious danger, and few cases have long survived when the temperature of 109 or 110 deg. has been reached, the temperature continuing to rise until the patient is *in articulo mortis*, and even rising for a time after the heart has ceased to beat in some cases.

The simple treatment of the case reported left its spontaneous course practically uninterfered with, the only exception being the administration of a scruple dose of quinine, the effect of which (according to the report of the committee thereon, given in the third volume of the *Clinical Society's Transactions*) is markedly to reduce the temperature in pyrexia, but which in this instance had no very perceptible effect. The case may, therefore, be taken as a contribution to the natural history of hyperpyrexia.

The exceptions to the general result of death when the temperature reaches 110 deg. are afforded in the two cases recorded by Dr. Wilson Fox of University College, in which the extreme temperature was reduced by immersion of the patient into water of about the natural warmth of the body, of which the temperature was then gradually reduced, followed by the application of ice-bags and the free administration of stimulants. This treatment seems rather an attack upon the effect, or the symptom, than an attempt to correct or remove the cause; but it had the highest merit of saving the life of the patient in each case.

When I last saw my patient alive, on the day prior to her death, neither the general symptoms nor the rise of temperature were suggestive of immediate or very serious danger. When the danger was imminent, I was sent for, and attended without delay; but the patient's death just before my arrival prevented any attempt to follow out the plan indicated by Dr. Fox.

To the question, What is the cause of the greatly elevated temperature in these fatal cases? I fear we have at present no satisfactory definite answer. In the healthy body, some regulating powers maintain an uniform temperature, varying only within narrow limits. There must be calorific agencies continually at work, and the undue activity

of these is restrained by opposite counteractive influences. What is the nature, respectively, of these calorific and restraining agencies? and is hyperpyrexia due to exaggeration of the former, or to failure of the latter?

We attribute the maintenance of animal heat to a slow combustion going on throughout the body; the oxygen inhaled in respiration is given off, in combination with carbon and hydrogen, and these combinations evolve heat. This must be regarded as a destructive process, whether exercised partly on nutrient substances in the blood, whilst yet unassimilated (according to the view that certain hydro-carbons, taken as food, are employed directly for the generation of heat, and not for nutrition proper); or whether exercised on tissues undergoing destruction in the continual process of decay and reproduction which is essential to functional activity.

On the other hand, it is certain that the healthy living body is endowed with the power of restraining extreme temperature, whether derived from external media, or developed within itself. The experience of persons who have survived accidental confinement in the heated atmosphere of buildings on fire proves this, as does also that of workmen exposed to the heat of furnaces in some manufacturing processes, as in that of puddling in the manufacture of iron. Many years ago, a man attracted a good deal of public attention, under the name of "The Fire King", who, amongst other performances, would shut himself up in an oven, of which the internal temperature was raised to several hundred degrees, with a raw beef steak, which, after a time, he brought out cooked, himself being uninjured. Whatever jugglery there might be in this performance, no doubt the man was exposed to a very high temperature. Profuse perspiration is generally regarded as one of the means by which the body is cooled, when exposed to high external temperatures, or when heated by violent exertion. But arrest of perspiration is by no means an invariable condition in the progress of a case of fatal hyperpyrexia; and it is evident that there must be some essential and continually acting physiological agency at work in restraint of the continual generation of heat in the healthy body. It is believed that the particular action whereby heat is rendered latent, which is directly opposed to that destruction of tissue by which heat is generated within the body, consists in the opposite process of the re-formation of cells and the development of tissue, by which growth is effected, and the nutrition of the body maintained.

This cooling process is essentially vital, whereas the internal combustion, whereby heat is generated, may be regarded as physical. And it may seem to be in accordance with this view that, in many cases, the approach of death and the act of dying, when vital powers fail, are accompanied by a marked elevation of temperature. The process by which the heat of the body is maintained, is allied to the development of heat in a hot-bed, or in a damp hay-stack, or that by which accumulations of waste in a cloth-mill occasionally become ignited; but in the healthy living body this generation of heat is kept within due bounds by vital agency. And this restraining influence is exercised and regulated by the subtle influence of the nervous system.

The experiments and observations of Claude Bernard, Brown-Séquard, Schiff, and others, lead to the conclusions that the increased warmth sometimes found in paralysed parts is due to a freer current of hot blood flowing through vessels relaxed and dilated by vaso-motor paralysis, and that the temperature from this cause cannot reach a high degree; but that the general condition of hyperpyrexia, in which a temperature is reached considerably in excess of the natural heat of the blood, is due to profound disturbance of the functions of the great nervous centres.

In meningitis, tetanus, direct injuries to the brain and spinal cord, and in typhus fever, puerperal peritonitis, pyæmia, and other blood-poisonings, hyperpyrexia not unfrequently precedes death; so also the effect of some poisons, having a sedative effect on the nervous centres, is to raise the temperature of the body. Woosara has this action; and my colleague Mr. Oglesby has shown, by experiments on animals, that both morphia and atropine have a primary effect of lowering the temperature, which is followed by a considerable elevation. Farther experimental inquiries into the effects of poisons, and of injuries inflicted on the brain and spinal cord, might lead to very valuable results. It is desirable, also, to ascertain whether hyperpyrexia is accompanied with increased exhalation of carbonic acid and watery vapour, as from increased combustion of hydrocarbons. There is not, in this state, any invariable increase in the excretion of urea in the urine, as from rapid destruction of tissue; but the state of the blood should be investigated, to ascertain whether urea is there accumulated. If we adopt the view that hyperpyrexia is due to some depressing influence upon the nervous centres, the administration of agents which are believed to stimulate the functions of the brain and spinal cord, such especially as phosphorus and strychnia, would seem to be a theoretically appropriate treatment of this condition.