trophy that the subject may live for many years, as was illustrated by a case in which acute rheumatism at the age of 14, in 1868, left behind regurgitation, attended with a maximum amount of dilatation and hypertrophy, together with secondary mitral regurgitation and exaggerated signs of every kind, which the patient survived for fourteen years, earning his livelihood as a greengrocer. He died suddenly in May 1882, during convalescence from slight sub-acute rheumatism, when the aortic valves were found to be practically non-existent, and the heart weighed 42½ ounces.

When, on the other hand, aortic regurgitation comes on late in life, partly from the fact that it will in most cases be progressive, and partly from the diminished aptitude of the heart to undergo compensatory changes, the prognosis is most grave. An example was related, in which a fatal termination followed the establishment of the disease in a gentleman aged 34, at an interval of only sixteen months. In these cases, the presence or absence of the aortic second sound in the neck is of extreme importance. The unfavourable prognosis dates from its disappearance, which in some cases is delayed for a considerable time.

We have still to trace the symptoms which lead up to a fatal termination in the form of valvular disease under consideration. They fall into two series, according as the ultimate effect on the circulation is arterial stagnation, from lack of *vis à tergo*, so-called asystole, or venous obstruction. In the former class, there occur syncopal and anginoid attacks, paroxysms of dyspnoea, a sense of oppression in the chest, pain in the cardiac region, irregularity and intermission of the pulse and heart-beat, which replace the regularity which prevails up to the moment of incipient failure of the heart. Dropsy is significantly absent, and there is no habitual dyspnoea. The *pulsus bis feriens* is felt (this is a peculiar double beat of the pulse felt on moderate pressure; it is produced by a double systolic effort of the heart). The characteristic irregularity of the pulse begins with the occasional occurrence of a short and rapid beat, which breaks the rhythm. It is difficult to decide whether it is an abortive systole or an attempt to supplement a systole which has been more or less inadequate; it succeeds the previous beat very quickly, and has a longer than normal pause after it. It is when the predominant tendency is to arterial stagnation, that sudden death is liable to occur; and, as said before, aortic incompetence is the one valvular affection in which this danger exists.

Dr. Walsh's statement that sudden death is more liable to occur in simple cases—that is, in cases not complicated by dilatation and hypertrophy—is not borne out by the statistics of St. Mary's Hospital, or by personal experience outside the hospital; but in a certain sense it is true—viz., as applying to aortic regurgitation occurring late in life.

In the other series, in which the final symptoms are due to pulmonary and venous obstruction, the last stage does not differ materially from that of mitral disease. The mode of causation of the symptoms is, in fact, almost identical, since insufficiency of the mitral valve is an intermediate stage in their production. There must, however, be imperfect filling of the system from the arterial side, as well as obstruction in the veins, which must be an aggravation, and must contribute to the hastening of the final and fatal stasis of the vital fluid. The supervention of mitral regurgitation has been said to be an element of safety, by preventing paralysis of the heart from over-distension. Sudden death, however, occurs in spite of it; and the danger in aortic regurgitation arises, not from resistance to the systole, in which case alone mitral incompetence would afford relief, but from undue filling in diastole, which it would indirectly tend to aggravate. Serious inadequacy of the mitral valve being once produced, its effects will gradually develop themselves in backward pressure upon the pulmonary veins, with consequent congestion of the lungs, cough, shortness of breath; next, hypertrophy and dilatation of the right ventricle, and, finally, dropsy.

THE BOOKS OF THE YEAR.—We learn from *The Publisher's Circular* that 4,732 books were published during the year 1883. Theology heads the list with 794 newly issued volumes; 556 educational and philosophical works were published, and 349 works of fiction, while history and biography accounted for 414. Voyages and books of travel numbered 210; and books on political and social economy, and on trade and commerce, 187; 163 medical and surgical treatises, and 130 works on law and jurisprudence, also appeared during the year. Thus, while theology is responsible for one-sixth of the total number of books; education and philosophy, for one-eighth; and fiction for one-thirteenth; medicine and surgery have only contributed one-twenty-second part.

BRITISH MEDICAL ASSOCIATION.

FIFTY-FIRST ANNUAL MEETING.

PROCEEDINGS OF SECTIONS.

ON THE PRODUCTION OF HEAT IN FEVER.

Read in the Section of Medicine.

By W. WITHERS MOORE, M.D., F.R.C.P.,

Physician to the Sussex County Hospital, Brighton.

THE regulation of the temperature of the body, in health and disease, has occupied the attention of some of the best observers during the last quarter of a century; and yet it must honestly be confessed that the question is still a moot one. According to Senator, "the temperature of the body is a direct consequence of the chemical processes of life," or, in other words, of the oxidation of hydrocarbons and nitrogenous matters, and is regulated by the skin, which is the great cooling agent, the superfluous heat being got rid of by radiation, conduction, and evaporation. But as, under normal conditions, the temperature of the body is constant, while the loss of heat from the skin is very variable, and depends much on external circumstances, it seems necessary to suppose that some centre exists which specially presides over the production or discharge of heat. Such a controlling centre has been hypothetically placed by Virchow, somewhere in the intracranial nervous system; and this idea has to a certain extent been adopted by Dr. Burdon Sanderson, who writes to me thus: "At all times in the normal body the available means of heat-production is in excess of the demand; but under normal conditions the temperature is constant, because, as soon as it rises above the normal, the production of more heat automatically ceases. This automatic check-action is maintained by the nervous system. If it be impaired, the temperature rises quite irrespectively of the absolute amount of heat produced per minute or per hour in the body." I must confess to not quite grasping the point in this last statement; for, unless the automatic check-action also control the "liberation of heat" from the body, it is difficult to see how the temperature can rise independently of the absolute amount of heat generated.

It is, however, to the production of heat in fever that I would specially desire to direct attention. There are two main theories by which it is attempted to explain the origin of heat in fever; viz., the neuro-paralytic, which ascribes it to a derangement of the nerves regulating the escape of heat from the body, and the biochemical, which explains fever by assuming an increased production of heat, owing to accelerated tissue-change. Hence the question to determine is, whether production or discharge is primarily disordered. The thermometer teaches us that the actual heat of the surface in fever is greatly above normal; and it is difficult to understand, even if all evaporation be arrested, how the body can, by any nerve-influence, be rendered independent of the physical laws of radiation and conduction. Nay, rather the contrary; for, according to Dr. Leyden's conclusions, "the discharge of heat is increased in fever, whether the temperature be constant, fall, or rise; consequently it is certain that the production of heat is increased." In rheumatic fever the temperature is often very high, though the body be drenched with perspiration; so that great heat must be lost by evaporation. In addition, Murri's experiments made on dogs, negative the idea of fever being due to a neuro-paralytic lesion; for in fever more heat is given off than in health; whereas, after section of the spinal cord, when the inhibitory power of the regulating centre is arrested, less heat is given off.

The theory then, that the liberation of heat at the surface of the body is controlled or restrained in fever through disorder of the nervous centre, so that by "retention" the temperature rises, must for the present be abandoned, until more reliable evidence can be adduced in its favour; and we are of necessity compelled to look upon fever as a disorder of protoplasm. We may take it as granted that the temperature of the body is the balance of the production and discharge of heat, and that probably the phenomenon of excess of heat in fever is due to a disproportionately large destruction of albumen, which leads to an increased discharge of urea, and also to an increased formation of carbonic acid. The urea, which is probably derived from the blood-corpuscles and tissues, is increased, in an early stage, to three times as much as would be excreted by the same person in health on a fever-diet; and it is notable that

TABLE I.—*L. K., aged 32, Idiopathic Anæmia.*

Date.	Pulse.		Temperature.	
	M.	E.	M.	E.
Nov. 10	120	—	100.6	—
20	120	—	101.6	—
21	130	—	100.2	—
22	124	—	101.2	—
23	112	—	101.2	—
24	120	—	100.8	—
25	124	—	99.6	—
26	120	—	100	—
28	116	—	100.4	—
30	116	—	99.8	—
Dec. 1	116	—	100.5	—
2	120	—	98.5	—
3	120	—	101	—
4	116	—	100.7	—
5	121	—	100.2	—
6	128	—	102	—
7	128	—	102.3	—
8	132	—	100.5	—
9	120	—	100.8	—
10	120	—	100.8	—
11	120	—	100.8	—
12	112	—	100.4	—
13	112	—	101.4	—
14	108	—	100.4	—
15	108	—	101	—
16	112	—	101	—
17	—	—	101.2	—
18	—	—	102	—
19	120	—	101.2	—
20	108	—	99.6	—
21	104	100.4	—	—
22	104	—	100.6	—
23	110	—	101.6	—
24	112	—	99.3	—
25	112	—	101	—
27	104	—	102.2	—
28	108	—	101	—
29	116	—	102	—
31	104	—	101	—
Jan. 1	108	—	100.2	—
3	108	—	101	—
4	108	—	100.4	—
5	108	—	100.4	—
6	112	—	100.1	—
7	120	—	102	—
8	112	—	101.2	—
9	108	98.4	100.4	—
10	108	—	99.8	—
11	112	—	100.8	—
12	108	—	101	—
14	108	—	101	—
15	124	—	102.4	—
16	104	—	99.2	—
17	96	—	99.4	—
18	116	—	100.4	—
19	104	—	99.6	—
20	112	—	99.4	—
21	104	—	99.8	—
22	128	—	101.4	—
23	120	—	100.8	—
24	116	—	100	—
25	104	—	99.3	—
26	104	—	99.5	—
28	—	—	100.2	—
29	—	—	101	—
30	120	—	103.8	—
31	110	—	101	—
Feb. 1	114	—	99.6	—
3	114	—	100	—

¹ On February 23rd, at 2 P.M., temperature 103; at 5 P.M., 106; at 9 P.M., 103.

² On February 27th, at 2.30 P.M., temperature 103.6.

³ On February 28th, at 2.30 P.M., temperature 104.

this excessive discharge of urea precedes the rise of temperature, and therefore is not the result of it. Leyden's observations show also a continued increase in the amount of carbonic acid discharged in fever; but, according to Senator, this increase is less than 30 or 40 per cent., and may possibly be partly due to the heightened temperature, expelling more rapidly the normal carbonic acid, and so the increase may be more apparent than real. Dr. Burdon Sanderson, in his able paper on the process of fever, says: "we are at liberty to adopt the tissue-origin of fever, as the basis on which we hope, eventually, to construct an explanation of the process." These reflections, on the source of heat in fever, were suggested by the consideration of the following case.

In November 1879, L. K., a married woman, aged 32, was admitted under me in the Sussex County Hospital, with idiopathic anæmia. It was a typical case, both as to the general appearance and symptoms, and the microscopic characters of the blood. All the usual remedies were tried, including arsenic, none of which, she said, she could or would take; and for some weeks she lay at death's door,

TABLE II.—*L. K., aged 32, Idiopathic Anæmia.*

Date.	Pulse.		Temperature.		Urine.	
	M.	E.	M.	E.	Spec. Grav.	Urea.
June 11	98	101.4	101.2	80	1007	0.6
12	80	102.6	101.4	74	1008	—
13	76	100.4	100.7	64	1008	—
14	82	101.4	101.8	68	1008	—
15	76	100.2	101	54	1008	0.6
16	120	100.4	101	72	1010	—
17	120	101.2	101	42	1007	—
18	112	100.8	101.2	62	1008	0.7
19	110	100.4	101	60	1008	—
20	110	100.6	101	58	1007	—
21	120	100.6	101	58	1009	—
22	116	100.3	101.8	53	1010	0.8
23	112	100	103.6	54	1009	—
24	118	100.2	102	60	1008	—
25	110	100	102	66	1009	0.7
26	116	101.2	102.4	40	1010	—
27	120	102.2	102	62	1010	—
28	136	100	101.6	68	1008	—
29	112	99.6	100	34	1012	1.2
30	112	99.4	101	44	1010	—
July 1	124	100.2	100.6	44	1011	—
2	120	99.8	101	44	1010	1.1
3	160	100	99.8	44	1011	—
4	100	99.6	100.4	46	1011	—
5	112	99.4	100	52	1012	—
6	108	99.4	100.4	42	1013	1.3 nearly
7	108	99.6	100.4	43	1012	—
8	116	99.6	99.1	43	1013	—
9	108	99.6	100.2	44	1012	1
10	116	100	100.8	42	1013	—
11	108	99.2	100.4	44	1013	—
12	104	99.2	100.6	45	1014	—
13	108	99.3	100.8	52	1010	1.1
14	100	99	100.5	51	1015	—
15	104	99	100.8	52	1013	—
16	120	99.2	100.8	45	1015	1.1
17	108	98.8	100	40	1010	—
18	120	98.8	100	51	1013	—
19	108	98.8	100.2	50	1012	—
20	96	99	100	51	1015	1.4
21	108	98.8	100.1	52	1015	—
22	102	98.6	100.2	49	1015	—
23	108	98	98.8	63	1013	1.3
24	108	98.8	99.6	53	1015	—
25	96	98.8	101.2	49	1015	—
26	120	98	100.2	53	1011	—
27	120	98	101.4	58	1015	1.4
28	96	99	104	58	1013	—
29	168	99	101.6	66	1012	1.3
30	120	98.8	102.6	59	1015	—
31	108	98.5	101	40	1016	1.3
Aug. 1	108	97.8	100.6	60	1015	—
2	108	98	100.8	38	1015	1.4
3	96	98.2	100.8	48	1014	—
4	96	98.4	100.4	58	1015	—
5	96	99	100	60	1016	—
6	108	99	100	56	1016	1.6
7	108	99	99.4	56	1015	—
8	108	98.6	99.4	60	1014	—
9	108	99	99.8	64	1014	—
10	96	98	100.2	63	1014	—
11	120	98.5	100	58	1015	—
12	108	99.2	100.2	54	1015	—
13	108	98.6	100.2	58	1016	1.6
14	108	98.4	99	62	1016	—
15	100	99	99.2	57	1017	—
16	108	98.6	100.2	66	1016	—
17	120	99	100.6	68	1017	1.8
18	120	99	99.2	68	1016	—
19	120	99.6	99.6	54	1015	—
20	108	99.2	100	48	1015	1.9 nearly

when I determined to give her small doses (three minims) of Fowler's solution three times daily in her food. Under this treatment, she rapidly improved, and, in six weeks after its commencement, was able to walk out of the hospital. This case is remarkable, not so much for its recovery, as for the state of pyrexia which prevailed throughout its course, the thermometer going as high as 106° in the axilla. I may say, in passing, that scruple-doses of quinine always lowered the temperature.

On thinking over this case, I was much struck with the unvarying record that the urine was normal; that is, in fact, the clinical clerk never found the quantity excessive or the specific gravity above normal. This, coupled with the facts of the greatly reduced number and debased character of the oxygen-carriers, and the tranquil breathing save on the slightest exertion, made me marvel as to how the increased heat was produced; and I therefore determined that, should another case occur, I would look specially to the urine.

Singularly enough, in June 1881, L. K. came in again, in a very similar condition; the prostration, however, being scarcely so profound as before.

Her general health had been good until three months previously, when she had frequent bleeding from the nose, and which still continued, lasting three or four hours at a time. She complained of headache, and had not kept any food down for seven weeks. It is needless to detail her symptoms, as it is not pernicious anæmia, but rather the coincident fever, to which I would direct your attention. Suffice it to say that she again presented the typical characters of the disease, but the fever did not run so high as on the previous occasion. Profiting by former experience, the treatment by arsenic was at once commenced, and in the same manner and dose as before; the result was equally satisfactory, for she went out cured in ten weeks.

The same absence of all chest-trouble was as conspicuous as on the former occasion. The temperature did not rise beyond 104°. The quantity of urine represented the fluid taken—chiefly milk, soda-water, and lemonade—for at first no solid food was tolerated; but, towards the end of her stay, the appetite became very ravenous for animal-food.

The emaciation was considerable; but our late house-surgeon, Dr. Uthoff, saw L. K. in February 1883, looking plump and well coloured. The percentage of urea in the case was low, and exactly tallied with the specific gravity, as shown in Table II. The marked increase in the amount of urea towards the ends of the case was no doubt due to the large increase of store-albumen, from the greatly improved appetite and desire for animal-food.

In considering this case, we may, I think, take it for granted that the fever was not the cause of the anæmia, for it is not present in all cases, and it usually occurs late in the disease and precipitates a fatal issue.

Is the fever the direct result of the anæmia? Many considerations favour this view. In cases of chronic starvation, a rapid and decided rise of temperature often occurs in the last few days of life; a like symptom, too, is sometimes present in other forms of anæmia, *i.e.*, leukæmia, chlorosis, etc., and is equally variable in its character. M. Mollière made observations on eight young chlorotic women, who presented no other signs of disease beyond the anæmia for which they were under treatment. The temperature was taken every morning and evening in the rectum, over a period varying from two to fifty-one days, and was found to oscillate between 101.8° and 102.8°; and, what is more to our purpose, the amount of urea eliminated daily was estimated, and found to be normal.

Again, we know that a diminution of the oxygen-carrying hæmoglobin, as a general rule, lowers the temperature of the body; and such is the case usually in this disease; but it is quite conceivable that, in an extremely impoverished condition of the blood, as, for instance, when the red corpuscles are reduced to a fourth, or even a tenth, the nutrition of the nerve-centre, or centres (if such there be), which preside over the regulation of heat, may be so impaired that they no longer exercise effectually their inhibitory power; or, again, that the vital power in the tissues is so lowered that they are unable to resist the action of even the very limited supply of oxygen which is presented to them.

But all this presupposes that fever is due to the oxidation of nitrogenous tissues; and we ought, therefore, to find a corresponding increase in the solids of the urine, and not the reverse, as in the case under consideration. We are, therefore, driven to seek for some other explanation of the febrile state.

In high fever, such as occurred in L. K., the quantity of heat given off is nearly twice as much as the normal, which loss, according to Senator, occurs by radiation and conduction, and, during the crisis, by evaporation. Mollière suggests that the increased heat may be explained by the hypothesis of a combustion, the products of which are not so easily recognised as urea. It may be so, but he offers no proof of it.

We are told by chemists that heat is always set free when chemical compounds of a more stable kind are generated from such as are less stable, even when oxidation has no part in the process; and upon this fact Immermann has built up a theory to account for anæmic fever.

"There is nothing absurd," he says, "in supposing that the molecular structure of the cell is, from a chemical point of view, lax and unstable, requiring (at any rate in the more highly organised animals), a continual restorative activity on the part of the blood, for its maintenance. Should this restorative influence fail, or prove inadequate, owing to some defect in the composition of the nutrient fluid, the complex substance of which the cells consist will spontaneously decay and break up into a number of more stable products."

Thus we have a possible source of increased production of heat in those extreme forms of blood-disease of which pernicious anæmia is the chief type; but what becomes of the nitrogen? Dr. Burdon Sanderson, who kindly suggested the publication of the case, says, in a note to myself: "The pyrexia of your patient I take to be dependent on disorder of that part of the central nervous system which regulates most chemical processes by which heat is produced. The temperature is higher, not because oxidation is taking place at a greater rate, or heat produced at a greater rate, but simply because oxidation and heat-production go on, notwithstanding that the increased temperature of the body ought to have, so to speak, shut the safety-valves." But ought we not to have the *débris* of combustion manifest somewhere, if the fire have gone on burning in excess of the needs of the body from neglect of the regulator? Again, to what was the first rise of temperature due? If not to excessive production, it must be to lessened liberation of heat.

Dr. Anstie, to whose mind a similar difficulty seems to have occurred, offers the suggestion that, in fever, the force which ordinarily becomes latent in tissue-building is set free in the form of heat; thus accounting for its excess independently of the destruction of nitrogenous and fatty tissues. But Dr. Anstie furnishes no data on which he founds this hypothesis.

In the case related, we have two important facts; viz., an enormous decrease in the number of the oxygen-carriers, and an absence of the usual evidence of increased combustion in the urine, which seem wholly irreconcilable with the further fact of the state of decided pyrexia which prevailed in both of L. K.'s illnesses. Seeing, then, that the urine in the present case fails us as an emunctory of the *débris* of combustion, can we find another outlet for it?

The alvine evacuations at once suggest themselves; but for the first four weeks there was seldom a second daily action, and sometimes none at all. And, further, Dr. Parkes has pointed out that the diarrhoea of typhoid fever, even though it does favour the escape of chlorides, is not to any considerable extent the channel for the escape of nitrogen; so that we cannot look for any help in this quarter.

Failing the bowels, we turn to the skin; and here we are met with the fact that in this case all sensible perspiration was arrested; and we can scarcely take for granted that much solid matter passed away with the insensible perspiration, more especially when we recollect that, in the profuse sweating of rheumatic fever, the urine bears unmistakable evidence of its being the natural outlet of febrile *débris*.

The lungs, no doubt, excrete in fever an increased amount of carbonic acid, but, as already noticed, not to such an extent as has been assumed; and we have no evidence that nitrogen finds egress there.

Such are the reflections suggested to me by this interesting case, and I must confess my inability to carry the inquiry further; and shall, therefore, be grateful to any member who will offer any solution of my difficulty as to how the temperature came to rise in L. K.'s case.

ON THE USE OF SULPHIDE OF CALCIUM IN THE TREATMENT OF SCABIES.

Read in the Section of Medicine.

By THOMAS M. DOLAN, M.D., F.R.C.S.Ed.,
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In some of the most recent works on medicine, I find that sulphur-ointment alone is suggested as a remedy for scabies. Thus, in a work which has gained for itself a world-wide reputation, Quain's *Dictionary of Medicine*, the line of treatment is laid down as follows.

"In order to cure an ordinary case of scabies, it is simply necessary that the patient before going to bed should thoroughly apply, and gently rub in, sulphur-ointment to every part of the body, excepting the skin of the face and scalp; and in order to keep the ointment in contact with the skin, he should sleep in his underclothes, such as drawers, jersey, shirt, and gloves, and in the morning take a warm bath. The process should be repeated the next night, after which the ointment should be used every night for a week or ten days to those parts of the body only which are specially attacked."

All who have to deal with scabies—as medical officers of industrial schools and Poor-law medical officers—will agree with me that such a method of treatment is not only unnecessary, but tedious,