

offered to build a new set of laboratories for him in Strasburg if he would stop and to raise his salary. The new pathological laboratory will cost £50,000. On the other hand the Government of England, 200 years ago, handed over the colleges and the Universities of Oxford and Cambridge to the clergy of the Established Church, and the consequence has been that these institutions have ceased to be universities in anything but name. Only the theological faculty is rich and flourishing; law and medicine have been stifled. At the same time, the few professors of law and medicine were encouraged to convert their offices into sinecures, and then, when the students had been kept out and the professors had ceased to lecture, the endowments of medicine were handed over to other purposes. There is not at the present day a single student of medicine at Oxford. The Royal Commissioners of 1854 endeavoured to start a school of scientific studies preliminary to medicine, but failed because they still left the control of the professors in the hands of the clerical majority, and did not properly constitute an independent medical faculty. The Commissioners now sitting have definitely refused to resuscitate the medical faculty of Oxford, although a petition signed by a large number of the professors and tutors residing in Oxford and urging them to do so had been presented to them. The list of endowments belonging to the suppressed medical faculty of Oxford is very considerable, but medicine is entitled besides to a full share of the ancient college endowments which have been quite wantonly usurped by the clerical profession. The Regius Professorship of Medicine founded by Henry VIII. still exists; it is now worth £500 a year. Its present holder, Dr. Acland, gives no lectures or other teaching. Lord Lichfield's Professorship of Clinical Medicine is also held by the same gentleman; this office is worth £200 a year, but no lectures are given by him in that capacity. In the last century Mr. Matthew Lee entrusted Christchurch with a sum of money to pay a reader in human anatomy and to buy subjects. The reader is no longer required to carry out the founder's intentions. The same is true of the Aldrichian Professorship of Medicine. The most ancient medical endowment in Oxford is that of Linacre, the founder of the College of Physicians. The Fellows of Merton College had the care of this, and for some 300 years spent the money on themselves. In 1854 it was made by the Royal Commission the basis of a professorship of physiology, but physiology is not taught by the present Linacre professor, who, in the recent agitation concerning vivisection, took a part hostile to the interests of those engaged in those experiments on animals which are the foundation of medical practice. The noblest medical benefactor of Oxford was Dr. John Radcliffe. He himself never studied medicine except in Oxford. After graduating M.D. he was persecuted and turned out of his lodgings by the clerical head of his college (Lincoln). He died in 1714, and left a large property to trustees for the purpose of improving the opportunities of medical study in Oxford. The Radcliffe Infirmary was thus built and a splendid medical library formed, while stipends were assigned to travelling Fellows who should learn on the Continent of Europe what Oxford failed to teach. Happily, Radcliffe, whose name we should mention with deep gratitude, left his property not to a college nor to the university, but to independent trustees. Oxford was not considered in former days too small a city to supply cases for the study of a medical faculty; at present it is more populous than several German towns which possess celebrated and active medical faculties, such as Hiedelberg, Bonn, Göttingen, and Jena. Cambridge had never sunk so low as Oxford now has done, and at the present time is likely to be brought into the right path by the strenuous efforts of Professors Humphry and Foster. But it is not only in depriving medicine of the endowments which belong to her that our universities have done wrong, it is owing to their neglect of medical sciences that it was possible for the Legislature to make those provisions against physiological experiments which were demanded by ignorant and weak-minded people, and have made us necessarily dependent on the physiologists of Germany and France for the knowledge necessary for the progress of medicine. A noble bequest to medicine had existed in London, but had been made away with in the most shameless and fraudulent manner. This was Sir Thomas Gresham's bequest, dating from 1596. The site of the house and garden which he left for his six professors was now worth three millions of money. The trustees, who were the Corporation of London and the Mercers' Company, had fraudulently disposed of

this site in the last century to the Crown, and were protected by Act of Parliament from the consequences of their outrage. Gresham, whose college might have become one of the noblest universities in Europe, had been cruelly betrayed by his fellow-citizens. Gresham's professors included one of medicine. Consequently, medicine has a claim on the city of London for £500,000. The trustees have the effrontery at the present day to continue to nominate the Gresham professors and to pay them each the magnificent sum of £100 annually, thus making a hollow pretence of carrying out the designs of the founder. Whether money for the purpose be obtained from such a source as this or from some other, such as a Government grant, there seems to be great need at the present time for the union of all the eleven medical schools of London under one central, well-supported, amply endowed medical faculty. The independent teaching of clinical medicine and surgery in the wards of the hospitals might continue, but in order that research might be carried out and students taught by men engaged in research, an endowed staff of professors was necessary. Such a staff might be connected with the University of London, which would then become a true university, and might carry on its work in London as does the University of Edinburgh in the northern capital.

## CONCERNING THE INFLUENCE OF PER- SPIRATION ON THE FEBRILE TEMPERATURE.

BY SYDNEY RINGER, M.D.,  
PROFESSOR OF MEDICINE AT UNIVERSITY COLLEGE.

I AM induced to publish these experiments, as they throw some light on the cause of the morbid elevation of the temperature in fever. This preternatural heat of the body has been accounted for in different ways. Some ascribe it to the dry skin in fever, whereby less heat is lost by evaporation; whilst the production of heat is maintained, the excess accumulates in the body and raises its temperature to fever height. If the production of heat remains the same whilst less is lost by evaporation, heat of course must to some extent accumulate in the body, and so raise its temperature; but this accumulation of heat, as I shall shortly show, plays a very insignificant part in the generation of fever.

According to another, and, as I believe, the correct view, by far the greater part of the preternatural heat of fever is due to increased formation of heat by increased combustion of the tissues, especially the nitrogenous tissues. During fever the quantity of urea is largely increased, and this is generally considered to depend on general combustion of the nitrogenous tissues. Some have demurred to this conclusion, maintaining that the increase of urea is due to the retrograde metamorphosis of morbid products formed by the disease, as the exudation into the lungs of pneumonia. This explanation, however, fails altogether to explain the increase of urea in an attack of ague, where no morbid products are formed. Some years ago I showed that during a paroxysm of ague the urea is increased in proportion to the height and duration of the fever, so that given the height of the fever we can approximately calculate the increase in the urea; and, *vice versa*, given the increase of urea, we can ascertain the height of the fever. The increase of the urea begins directly the fever begins, and declines with its decline. This increase in the urea must be ascribed to increased combustion of nitrogenous tissue; and as the increase of urea is in proportion to the severity of the fever, it is fair to conclude that this increased combustion causes the fever.

A case of rheumatic fever with high temperature but with freely perspiring skin conclusively proves that fever is not simply due to accumulation of heat through loss of evaporation from a dry skin. In other febrile diseases, too, as in typhoid fever, especially where there is considerable exhaustion, the skin may be occasionally moist, and even soaked, whilst the temperature is very high.

I will now adduce additional evidence to show how little

share the dry skin plays in the production of the febrile temperature. Two patients were admitted under my care with ague. I determined to excite profuse perspiration before, or just before, the commencement of the febrile paroxysm, and to watch what effect this free perspiration might have on the high temperature.

The first patient suffered from quotidian ague, and his temperature rose in an untreated paroxysm to  $105^{\circ}$  and  $106^{\circ}$  F. Just before the onset of an attack, I gave him half a grain of pilocarpine, which in twenty minutes produced copious perspiration; yet, in spite of this, the temperature rose six degrees, to  $104.4^{\circ}$ , and the fit lasted as long as on previous days, the temperature falling short of the attacks on the previous days by about a degree. As in ague, the untreated fits often differ to a greater degree than this, it is doubtful if even this slight diminution was due to the jaborandi. I may mention that the sweating produced by the jaborandi had very little influence on the shivering, and blueness of the lips, nose, and extremities.

The next patient suffered from irregular tertian fever caught in Florida. In an untreated attack, on August 1st, his temperature rose to  $104.8^{\circ}$ . On Aug. 4th the rigor began at 3.20, his temperature at that time standing at  $101^{\circ}$  F., conforming to the rule with ague, that the temperature rises one or two degrees before the rigor begins. Five minutes after the beginning of the rigor, Mr. Neale, my resident assistant, administered hypodermically a quarter of a grain of pilocarpine. In a quarter of an hour perspiration began, the temperature standing at this time at  $102.6^{\circ}$ . The perspiration soon became profuse, and yet at 4.30, fifty minutes after the commencement of free perspiration, the temperature stood at  $105.5^{\circ}$ , and continued above  $105^{\circ}$  till 5.30 P.M., when the fever began to decline, and the temperature became normal between 1 and 3 A.M. on the following morning, the fit lasting more than ten hours. On Aug. 7th he had a fit which was untreated. On Aug. 10th he had another attack. At 3.20 his temperature was  $101.8^{\circ}$ . At 3.30 Mr. Neale gave him a hypodermic injection containing half a grain of pilocarpine. At 3.45 he was perspiring very freely, and his temperature marked  $102.1^{\circ}$ . At 5.30 the temperature was  $105^{\circ}$ , and subsequently rose to  $105.2^{\circ}$ . It remained at or above  $105^{\circ}$  till 8 P.M., and then fell, becoming normal at 4 the following morning; the fit, therefore, lasted over twelve hours. On August 13th he had another attack. At 5 P.M. his temperature was  $101^{\circ}$ . At 5.30 half a grain of pilocarpine was administered hypodermically. At 5.45 he was sweating, and his temperature then marked  $103^{\circ}$ . At 7.15 his temperature stood at  $105.4^{\circ}$ , and so remained till 8 P.M., and after this time it fell, becoming normal between 3 and 5 the following morning. In this case the attack lasted more than ten hours.

In these experiments, then, the temperature in an untreated attack rose to  $104.8^{\circ}$ . In the three fits treated with pilocarpine, which produced copious perspiration, the temperature reached respectively  $105.6^{\circ}$ ,  $105.2^{\circ}$ , and  $105.4^{\circ}$ , the fits lasting, respectively, ten, twelve, and ten hours. We may therefore fairly conclude that the free perspiration had a very insignificant influence on the febrile temperature, and the increased heat cannot be explained by its accumulation owing to a dry skin, but must be due to increased production of heat from increased combustion.

In his interesting and suggestive lectures on Cardiac Depressants, Dr. Fothergill explains the effect of aconite and tartar emetic on the febrile temperature by their changing the dry to a moist perspiring skin, and so increasing the loss of body heat by increasing radiation and evaporation. As I have already said, by making a dry skin moist we must of course abstract a certain amount of heat by evaporation, and to this extent cool the patient; but the experiments given in this paper show, I think, how insignificant a part the loss of heat, induced in this way, plays in causing that great fall of temperature so often produced by aconite or tartar emetic. Other reasons may be adduced in support of the same conclusion.

1. Whenever aconite promotes perspiration, a proportionate reduction of temperature ought to take place in all diseases; but whilst, in many cases, as in tonsillitis, &c., the fall of temperature is considerable, in other forms of fever, though the perspiration may be very free, yet scarcely any, or even no fall of the temperature takes place: for instance, in many cases of erysipelas, pneumonia, pleurisy, and especially in the specific fevers, the fever continues unchecked.

2. We, not uncommonly, find that aconite quickly reduces

temperature without promoting sweating, especially with children, in whom this drug in many instances fails to produce it.

3. Sometimes we see cases like the following: In typhoid or scarlet fever a patient with a hot dry skin, to whom we give aconite, becomes in a few hours freely bathed with perspiration, which continues several days, and then, in spite of the drug, the skin again becomes quite dry. Now in a case like this we find that the temperature undergoes no change. It remains as high during the sweating as before giving aconite, and does not rise on the cessation of the perspiration.

4. Some years ago, in conjunction with Mr. P. Gould, in order to test the influence of perspiration on the temperature, we three times performed the following experiment:—We placed a fever patient in a hot-air bath, with the exception of the head and face. When free perspiration came on the bath was removed and the patient covered lightly with clothes, and in this state he sweated for some hours afterwards. Whilst in the hot-air bath his temperature did not rise, nor did it fall after the bath, notwithstanding the free perspiration and light clothing. If it be objected that the clothing prevented evaporation, and the consequent reduction of temperature, I may reply that these are the identical conditions under which aconite in so many instances causes so marked a fall of temperature.

Cavendish-place, W.

## THE TREATMENT OF PSORIASIS BY ARSENIC IN LARGE DOSES.

By LEWIS SHAPTER, M.D. CANTAB.,  
PHYSICIAN TO THE DEVON AND EXETER HOSPITAL, CONSULTING PHYSICIAN TO THE WONFORD HOUSE HOSPITAL FOR THE INSANE.

CASE 1.—Clara B—, aged sixteen, had been the subject of “general” psoriasis for six months, and, notwithstanding treatment, the disease remained unchecked. On her admission into the Devon and Exeter Hospital the whole of the body, including the head, was covered with the disease, and in some places, such as about the ears, the derma had become infiltrated and thickened.

During the first two months, treatment consisted in the administration of Donovan’s solution in fifteen-minim doses thrice daily, and the local application of a lotion containing carbolic acid in large doses, and with liq. arsenicalis in five-minim doses, combined with citrate of iron and ammonia. The case still remained obstinate, and could only be regarded as having been relieved and rendered more tolerable.

I then ordered one-twentieth of a grain of arsenious acid to be taken in pill thrice daily, and the surface to be kept as clean as possible with vaseline and the occasional use of warm baths. At the end of a week the dose of arsenious acid was increased to one-fifteenth, then to one-tenth, and after the lapse of a fortnight it was again increased to one-fifth, and subsequently to one-fourth of a grain. When the dose of one-tenth of a grain was reached improvement commenced, but the slow and limited progress warranted a further increase in quantity, and the results showed that even larger doses were needed. The disease was checked, and each increased dose of arsenious acid appeared to do its work in limiting its extent; but after a time each successive stage of progress was stayed, and more of the drug was demanded to complete the work that had been begun. Two months after this system of treatment had been commenced the dose of arsenious acid was increased to one-third of a grain thrice daily; this was continued for another month, when some few isolated patches upon the legs were the only remaining indication of the disease. The dose of arsenious acid was again increased to half a grain; and at the end of another month the patient was discharged recovered, the arsenious acid having been omitted for a week before her final discharge from the hospital.

CASE 2.—George H—, aged eighteen, the subject of “general” psoriasis of two months’ duration. The disease