about inside the limiting membrane of the parasite. In fact one can sometimes almost see that the agitation, which is so characteristic a feature of this stage of the evolution of the parasite, is confined to the central portion of the organism, and is not shared in, except in a passive way, by the cyst wall; and that it is entirely owing to the vigorous movements of the imprisoned flagella. From the extreme transparency of the flagella and the rapidity of their movements, one cannot be absolutely sure of this in the unstained living specimens, but in these stained preparations it is distinctly evident that this fancy is well founded. In the preparation from which Fig. 5 was drawn, and also in that of which Fig. E is a photograph, the flagella can be readily made out surrounding the central mass and lying inside the limiting membrane of the organism.

G.

Looking at these photographs and drawings, I do not see how it is possible any longer to contend, as so many Italian writers have done, that the flagellated phase of the malaria parasite is an involution or degeneration form; that the flagella are comparable to the protoplasmic projections sometimes seen in scorched blood corpuscles; or that they are anything other than a distinct phase in the evolution of the living malaria parasite.

H.

I have not attempted to make similar preparations of the flagellated bodies of the benign, tertian, or of the quartan parasites. I have no doubt, however, that by selecting the stage of fever in ordinary tertian or quartan attacks their flagellated forms could be equally easily demonstrated, and that probably some interesting details bearing on the question of species would be made out.

When working at these preparations I took the opportunity to repeat Marshall's and Ross's experiments on the influence of water, and of exposure to the air, in inducing exflagellation. I can confirm their statements. I found that breathing once on the slip before taking up the droplet of blood was a better way of supplying moisture than Marshall's plan of placing a droplet of water on the coverglass; if the amount of moisture be too great, as it is apt to be by Marshall's method, the parasites are killed. I also repeated Ross's experiment of pricking the finger through vaseline, and not removing the grease film surrounding the droplet of blood till one hour thereafter.

I found that Ross's statement, that under these circumstances exflagellation did not take place, was perfectly correct; for when I spread the blood out, at the end of an hour, and at once dried and then fixed and stained it, only crescents, and

perhaps a few ovals were found. When, however, the blood, after breaking the vaseline film surrounding it, was exposed to the air for a minute or thereby, and then mounted on a slip and coverglass in the usual way for making fresh blood preparations, in many instances, after a short time, exflagellation was seen to occur.

THE CROONIAN LECTURES

ON

THE MEANS BY WHICH THE TEMPERATURE OF THE BODY IS MAINTAINED IN HEALTH AND DISEASE.

Delivered before the Royal College of Physicians of London,

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LECTURE IV.*

[ABSTRACT.]

CHEMICAL STUDY OF HEAT PRODUCTION.

THE breaking down of any chemically complex substance, such as a proteid or a fat, always leads to the liberation of energy. Various observers have, by burning these substances in a calorimeter, calculated the amount of heat set free by their destruction. In such a case the whole of the liberated energy appears as heat. Some of the most careful and recent results are Stohmann's.¹ His figures represent the amount of heat necessary to raise 1 gramme of water from 17° to 18° C. He found that the heat value, expressed in gramme-calories, of the substances enumerated was as follows: Most albumens, 5,298 to 5,941; meat, 5,662; albuminoids, 4,650 to 5,961; chondrin, 5,130; animal fat, 9,509; olive oil, 9,500; carbohydrates, 4,381 to 3,714; cane sugar, 3,955.

These figures are of great value, but we cannot, of course, calculate the heat production of an animal from its diet. In the first place we do not know what proportion of any meal is absorbed from the gastro-intestinal tract; secondly, not all the energy set free in the body is manifested as heat—some takes the form of mechanical work, and therefore in calculating the heat value of any particular diet we should have to deduct the energy which in the body takes the form of motion, but this is very difficult to estimate accurately, and therefore the deduction to be made from the total heat value of the food would be uncertain; and, thirdly, the food may be stored up in the body and not broken down for some time. The value of figures, like those of Stohmann's, which show the amount of heat produced by the breaking down of the albumins and fats of the body lies in the fact that we can,

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by observing the amount of nitrogen and carbon excreted, calculate what must have been the amount of proteid and fat which had to be broken down to produce these amounts of nitrogen and carbon, and then, by looking at Stohmann's figures, we find how many calories the breaking down of these proteids and fats gave rise to. But in these calculations we must remember that although the fats are completely broken down yet the proteids are not. Nearly all the proteid is, however, broken down to urea and uric acid; the excretion of these can be measured, and the number of calories resulting from their complete destruction estimated; if this done, and the calories of energy remaining in the urea and uric acid are deducted from those set free if a gramme of albumen is broken down as low as possible, we, of course, easily obtain the energy value to the body of a gramme of albumen, and this is found to be about 4,000 grammes-calories.

Rubner² has in animals, by careful analysis of the expired air, the urine, and the fæces, observed the amount of nitrogen and carbon excreted, from which he calculates the quantity of proteids and fats that corresponds to such an amount of nitrogen and carbon. Then he compares the heat given off by the animal in an animal calorimeter with the heat that he would, from his knowledge of the number of calories set free by the breaking down of these quantities of proteids (after allowance for the urea and uric acid), and fats, expect to be produced by the animal during the time it was in the calorimeter. The following table shows how the calories reckoned by each method correspond. It gives in kilogramme calories the results of experiments on a small dog which was observed for five days starving, five days on a fat diet, eight on a diet of meat and fat, and six on meat; and on a big dog observed two days starving, twelve days no meat and fat, and seven on meat. Each twenty-four hours was observed separately, and the table gives the total results for each dog for each diet.

Diet.	Number of Days.	Total of Calculated Heat.	Total of Heat Estimated by Calorimeter.
Starving	5	1296.3	1305.2
...	2	1091.2	1056.6
Fat	5	1510.1	1495.5
...	8	2492.4	2488.0
Meat and fat... ..	12	3985.4	3958.4
...	6	2249.8	2276.9
Meat	7	4780.8	4769.3

Rubner's experiments are very carefully performed, and therefore we must look forward with great interest to their confirmation. If they are confirmed they will prove that he has successfully overcome many great difficulties, and the agreement between his chemical and calorimetrical results will be remarkable, for we have seen that animal calorimeters may be very inaccurate instruments, and unless very carefully constructed will give rise to all sorts of errors. Secondly, the chemical method gives the heat produced, but the calorimeter only indicates that lost, and we cannot infer quite accurately from that the amount produced by the animal, for we do not know whether, during the experiment, it varies its rate of loss; and, further, we cannot tell, unless we know the variations of its temperature during the twenty-four hours an experiment lasted, how much of its heat production has been expended in raising its temperature, and if any has been so used it would not be shown by the calorimeter. In these experiments the dissipation of energy as mechanical motion does not interfere with the results, for the animals keep quiet in a cage which just fits them; and further, such mechanical work would tell equally in both sets of figures, as also would the work done by the heart and respiratory muscles, most of which work becomes heat as it has to overcome friction.

NITROGEN AND CARBON EXCRETED IN FEVER.

We now pass on to a consideration of the experiments in which an attempt has been made to calculate the heat production of starving animals suffering from fever by observing the amount of nitrogen and carbon excreted. It is always

necessary to use starving animals, for it is well known that the ingestion of a meal increases the production of heat, as, indeed, the table just quoted from Rubner shows. This method can give some idea of the chemical processes involved in fever, but in applying the results to man we must remember that, as human patients are given food during a febrile illness, and as man possesses in his sweating and hairless skin, far better means than the lower animals of varying his loss of heat, the processes may be somewhat different in man from the processes in a starving animal. By far the best experiments are those of May,³ who comes to the conclusion that the balance of evidence supports the view that in febrile pyrexia the respiratory quotient is unaltered, that the excretion of CO₂ is not much increased, and that the brunt of the destruction falls on the proteids. He used rabbits in whom he induced swine erysipelas. For some days before the production of this fever they were starved so as to get their excretion of urea steady, and during the fever May most carefully observed the output of nitrogen and carbon both in the urine and the breath for more than twenty hours at a time, and he calculated what must have been the original amount of proteid and fat tissue which would, when broken down, correspond to the nitrogen and carbon he found excreted. Using Stohmann's heat values of proteid and fat, he was able to estimate the number of calories which would be set free by the destruction of these amounts of proteids and fat. He found that in a starving rabbit suffering from swine erysipelas there is an increased heat production, due entirely to an increased destruction of proteid.

THE EFFECT OF SUGAR IN SAVING ALBUMEN AND FAT.

May's next experiments are of the greatest interest to clinical physicians. He gave a starving rabbit a known quantity of grape sugar, and made—both before and after the administration of the grape sugar—observations similar to those I have just described. After the observations the animal was immediately killed, and the glycogen in the body and the grape sugar in the urine, alimentary canal, and fæces were estimated, and from the figures obtained May was able to learn how much had been used in the body. He found that the administration of sugar saves the destruction of albumen and fat which goes on in starvation and in fever.

Experimental medicine here shows strikingly why it is, as clinical medicine has found by experience, that milk and farinaceous diet is best for fever, and further, as all May's experiments show conclusively that in starving rabbits suffering from swine erysipelas the heat production is increased, and that this increased production is due entirely to a greatly increased destruction of proteid, we are probably correct in concluding that in most fevers the greater part of any increased production of heat is due to an increased destruction of proteid rather than to an increased destruction of fats. It is true that May has not been able to allow for any energy which, resulting from the breaking down of tissue, manifested itself as mechanical work; but the increase of proteid destruction he found was so great that even if the fevered starving rabbit did a little more mechanical work than it did before the fever was induced the increased energy taking this form can have been only a small proportion of the whole energy resulting from the increased destruction of proteids.

THE EXCRETION OF CO₂ IN FEVER.

It is almost impossible to make any such observations on man, but some attempts have been made to find out whether in man the amount of CO₂ exhaled is increased during fever. Many more observations will have to be made on different fevers before the results can be accepted as final, but as far as they go they show that in man the amount of CO₂ excreted during fever is not increased.

Mr. Hopkins and I investigated the question of whether the output of CO₂ and H₂O from the lungs was increased when pyrexia was induced by cerebral lesions. We used the differential calorimeter I have already described. The animal was shut in one chamber, no hydrogen was lit in the other chamber, but it was closed and an equal amount of air was drawn through each chamber. The outgoing air from each was passed through a pumice and sulphuric acid tube to absorb the water, then through a U tube containing pumice soaked in caustic potash, and then through two U tubes, each

containing dry caustic potash. The sulphuric acid tube was weighed before an experiment, and each set of three caustic potash tubes was also weighed, and the tubes were then again weighed after an experiment, the duration of which was usually an hour. Of course the sulphuric acid tube and caustic potash tubes on the animal side gained more than those on the other side, for these only showed the H_2O and CO_2 in the air drawn through, while those on the animal side showed this, and in addition the H_2O and CO_2 exhaled by the animal in one hour and taken up in the air current passing through the chamber. By subtraction we found how much H_2O and CO_2 were produced by the animal.

In four experiments on two rabbits variations in both animals were so slight and so well within the normal variations that we may conclude that damage to the corpus striatum does not increase the output of CO_2 , and in this respect the pyrexia which follows it is similar to the pyrexia which accompanies those fevers in which the excretion of CO_2 has been investigated. Inspection of the figures also shows that the pyrexia caused no constant alteration in the amount of aqueous vapour exhaled. It was impossible to trace any relationship between the CO_2 and H_2O in the air and that exhaled by the animal. The position of the lesion in the brain was always confirmed by *post-mortem* examination.

HEAT PRODUCTION IN HYBERNATING DORMICE.

Dr. Pembrey and I have performed a number of experiments upon hibernating dormice.⁴ When these creatures are hibernating their temperature is very low, the rectal temperature usually being about 9° or $10^\circ C.$; if they are brought out of a cold into a warm atmosphere their temperature slowly mounts at about the same rate as that of an equal weight of water (for their specific heat is very little less than that of water) until it reaches about $16^\circ C.$, when it suddenly mounts many degrees in a few minutes to between 33° and $36^\circ C.$; at the same time the sleepy animal becomes active and runs about. Mere lowness of external temperature will not cause the animal to sleep nor its temperature to fall. This will only happen when lowness of external temperature causes the creature to become inactive.

Our observations brought out the interesting fact that this characteristic sudden rise of temperature which occurs when hibernating dormice become active is unlike the rise of temperature which occurs when the rabbit is afflicted with swine erysipelas, or has its temperature raised by damage to the corpus striatum, for it is accompanied by an enormous increase in the output of carbonic acid. This goes to show what is probably true, namely, that the metabolic processes of the pyrexia due to cerebral lesions and of that due to fever are quite different from those of health, and not merely an exaggeration of these.

CONCLUSIONS AS TO PRODUCTION AND LOSS OF HEAT IN PYREXIA.

I think now we have come to the end of our discussion of the problem of the variations in the production and loss of heat in pyrexia, and the three great main conclusions to which we seem to have come are:

1. In man at least pyrexia is not produced by the same method in all fevers; sometimes the rise of temperature is due to an increased production of heat, and sometimes to a diminution of the loss.
2. In the present state of our knowledge animal calorimetry is very difficult, and the results obtained from calorimeters must be received very guardedly, especially when they are applied to man.
3. In some forms of pyrexia in which the production of heat is increased, the metabolism leading to this takes place in the proteid tissues of the body, and probably the metabolic processes concerned in a pathological rise of temperature are different from those of health.

THE USES OF PYREXIA.

A very difficult problem which has been much occupying the minds of the profession lately has been that of why almost all micro-organisms which are harmful to the body raise its temperature, and the suggestion has been made that the rise of temperature is a defensive mechanism, or, in other words, pyrexia is, like phagocytosis or chemiotaxis, in

some way harmful to the fever-producing micro-organisms or their toxins. Of course, it does not follow from this view that the higher the temperature of the body the better the prognosis, for the higher temperature might be taken to indicate that the dose of infection was very severe, and that therefore the body did all it could to resist the invasion, nor, on the other hand, would it follow that if the temperature did not rise much the dose of infection was slight, for it might be that the body was feeble and had but little power of raising its temperature and therefore defending itself. Clinical observation can hardly settle the point, but in passing I may mention that I have seen a severe case of typhoid fever in which during the second week of the illness the temperature was very irregular; the patient, however, always felt better and had a better pulse when her temperature was high than when it was low.

ANTIPYRETIC DRUGS.

A few years ago much was hoped from the antipyretic drugs—antipyrin, acetanilide, and phenacetin; and if it could have been shown that they distinctly improved the condition of the fevered patient it would have been a strong argument against the view that pyrexia is a defensive mechanism. The reverse, however, is the case, for they are not now often employed as antipyretics, and in a discussion on pyrexia and its treatment which took place at the Bristol meeting of the British Medical Association held in 1894, speaker after speaker got up and said that, as a general rule, it did more harm than good to treat the pyrexia of fever.

Some few fevers are treated by agents which lower the temperature, but these very fevers are instances of the exception proving the rule. Ague is treated by quinine, the temperature falls and the patient is better. This, however, is not because the quinine is an antipyretic, but because it is a poison to the plasmodium malarie; the patient's temperature falls because the pyrexial agent is destroyed. The same is true of syphilis. If a patient is suffering from syphilitic fever his temperature falls when he takes mercury, not because mercury is antipyretic, but because the cause of the pyrexia is destroyed. Again, it is almost certain that any benefit that follows in rheumatic fever from the administration of salicylates is not due to their antipyretic action, but because they are specifics for rheumatic fever, for other antipyretics are not equally efficacious, and salicylates benefit other symptoms than the temperature, namely the pain.

MODE OF ACTION OF COLD BATHS IN FEVER.

It is generally believed, and in all probability correctly, that many cases of typhoid fever are benefited by cold sponging or by a cool bath. Many have hastily concluded that the bath does good because it lowers the temperature. But this is probably incorrect. In the first place we must remember that the cold sponging or bath does more than lower the temperature; it diminishes the delirium, the tremor, and the prostration. In any of these ways it would do good. But, further, Roque and Weil claim to have shown that "in typhoid fever left to itself the toxic products manufactured by the bacillus and organism are eliminated in part during the illness. The urotoxic coefficient is double the normal, but this elimination is incomplete, and is only completed during convalescence, for the hypertoxicity continues for four or five weeks after the cessation of the fever. In typhoid fever treated by cold baths the elimination of toxic products is enormous during the illness. The urotoxic coefficient is five or six times the normal. The hypertoxicity diminishes as the general symptoms mend and as the temperature falls, so that when the period of pyrexia and convalescence sets in the elimination of toxins has ceased." So we learn that it is by no means certain that in typhoid fever the benefit of cold baths is due to their antipyretic influence, and we see that clinical medicine affords no evidence that antipyretics are useful in fever.

IS PYREXIA A PROTECTIVE MECHANISM?

The best collection of the literature of this subject is given by Max Müller,⁵ and many of the following references are quoted from him. Heydenreich⁶ found that the spirillum of relapsing fever lost its mobility quicker at $40^\circ C.$ than at $37^\circ C.$, while at from 15° to $22^\circ C.$ it retained it a long time. Then in 1880 Pasteur⁷ made the observation that fowls which

were ordinarily refractive against anthrax bacilli succumbed to them if their temperature had been artificially lowered by their being cooled, and in the same publication next year he said he had found that the virulence of anthrax bacilli is diminished by a temperature of from 42° to 45° C. Wagner⁷ infected fowls with anthrax bacilli and spores, and kept down the temperature of some of them by partially immersing their bodies in water; these succumbed, but others left at the ordinary temperature did not. He also infected a series of eleven in whom the temperature was kept down by antipyrin; of these eleven, six were taken ill; and of these six, five died. This, he thought, was due to a lowering of the temperature by antipyrin. Rovighi⁸ gives an account of how he infected three series of rabbits with saliva; one lot were placed in an incubator, the temperature of which varied from 37° to 42° ; another lot were left at the ordinary temperature, and the third lot were artificially cooled. He found the mortality to be highest among those artificially cooled and least among those artificially warmed; and in the same periodical for the year 1891 it will be found to be stated that Walther observed that after infecting rabbits with the pneumococcus those placed in a warm oven lived longer than those placed at the ordinary temperature of the room. Koch found that if tubercle bacilli were kept at a temperature of 42° C. for three weeks their development was much hindered, while the best temperature for their development was from 37° to 38° C. We thus see that this bacillus, the scourge of the human race, flourishes best at the internal temperature of man, and it is quite possible that if a phthisical patient's temperature could be safely kept at 108° F. for a month he might be cured. Fraenkel¹⁰ finds that the pneumococcus named after him loses its influence on rabbits in twenty-four hours if they are kept at a temperature of 42° C. Both Bumm and Finger have observed that the temperature at which the gonococcus flourishes best is between 33° and 37° C., and that higher temperatures stop its development. Max Müller came to the conclusion that the higher the temperature the less rapidly typhus bacilli multiplied. Welender and Berlien¹¹ have been treating soft sores by means of a thermostat, an instrument which keeps the sore at a temperature of 41° C., and they claim excellent results for this treatment. Filehne, at a meeting of the Physiological Society held at Oxford on August 11th, 1894, gave an account of some experiments in which he infected rabbits with erysipelas and studied the changes at the seat of infection, the ear. In animals kept in a warm chamber the reaction in the ear was slight, and attained its height in two days, and the animal was soon well. In animals kept at the ordinary temperature the reaction was much more severe, and lasted ten or eleven days. In animals kept in a cool chamber nothing happened so long as they remained there, but as soon as they were taken out and left at the temperature of the room they had a very severe attack of erysipelas.

Löwy and Richter¹² caused a rise of temperature in rabbits by puncture of their corpora striata; they then infected them with the bacilli of diphtheria, fowl cholera, swine erysipelas, and pneumonia. They came to the following conclusions: (1) That with very large fatal doses the animals whose corpus striatum was punctured lived longer than the control animals; (2) if the animals whose corpus striatum was punctured had a dose two or three times as large as that necessary to kill an ordinary animal they recovered; and (3) when a local injection of swine erysipelas was made into a rabbit's ear the local reaction was most marked in those animals in whom the corpus striatum had been punctured, but they invariably got quite well.

I quite admit that all these observations do not prove that pyrexia is a protective mechanism, but they certainly suggest that it is, and many who have thought about the subject have given their adhesion to this view; such, for instance, are Roy,¹³ Cantani,¹⁴ and Sanarelli.¹⁵

PYRETIC DRUGS.

Dr. Hale White then pointed out that there were many drugs which caused a rise of temperature. In belladonna poisoning the temperature might rise to 104° F. In animals large doses of cocaine would cause a rise of temperature, and strychnine and caffeine both raised the temperature a little.

The drug which had the most remarkable effect, however, was β -tetrahydronaphthylamine. Stern¹⁶ had found that it could produce a rise of 4.5° C. in an hour, causing the death of the animal; a smaller dose caused a considerable rise in the temperature without killing the animal.

In experiments with the same drug on rabbits Dr. Hale White and Dr. J. Fawcett had found that recovery might take place even after a dose which caused a rise of 3.2° C. (5.8° F.) in three hours. In all the experiments, five in number, the rapidity of the pulse and respiration was increased enormously, and both were often so quick as to be uncountable. In one animal in which the rise of temperature was raised 5.5° C. (9.9° F.) death ensued.

Animals which, like rabbits, do not perspire much must lose most of the heat they part with by the lungs; the rise of temperature produced by β -tetrahydronaphthylamine therefore cannot have been due to a diminution of the loss of heat. Indeed, judging by the rapidity of respiration, this must have been much more rapid than usual, and probably the creature was by increasing its heat loss struggling against the rise of temperature produced by the drug. The animals for an hour or two after the drug was injected were usually rather restless, frequently shifting their position and turning round, but they never ran about nor made any prolonged severe muscular movement. This restlessness was always followed by marked loss of muscular power, but nevertheless the temperature continued to rise. The only animal that had convulsions was the one that died, and they did not appear until shortly before death. These facts seem to me to be strongly in favour of the view that the drug has in some way greatly increased the production of heat, and that this increased production is not due to muscular movement, for the rise is altogether too great and too rapid for it to be caused even in large part by the increased muscular movement due to the greater rapidity of the respiration and pulse.

I cannot help thinking, therefore, that we have in β -tetrahydronaphthylamine a drug which stimulates the thermogenetic centre without exciting the motor function of muscle, and that therefore we see—as we have already seen in the patients afflicted with hemiplegia—that the two functions are distinct. I have already pointed out in these lectures that damage to the corpus striatum produces a considerable rise of temperature. The rise produced by β -tetrahydronaphthylamine so resembles in extent and rapidity that caused by these injuries that it is at least likely that the drug acts on the muscles through the central nervous system.

I have not yet had time to see whether the administration of this drug increases the resistance to any febrile diseases, but it is clear that even if it does it will have to be given very cautiously to man on account of its marked effect on the heart.

REFERENCES.

- ¹ Ueber den Wärmewerth der Bestandtheile der Nahrungsmittel. *Zeit. f. Biol.*, Band xxxi, Heft 3, S. 364.
- ² Die Quelle der thierischen Wärme. *Zeit. f. Biol.*, 1893-94, Band xxx.
- ³ *Zeit. f. Biol.*, Band xxx, S. 1.
- ⁴ *Journal of Physiology*, vol. xix, No. 5 and 6.
- ⁵ Wirkung der Fiebertemperatur auf den Typhusbacillus. *Zeit. f. Hyg. u. Infect.*, 1895, S. 244.
- ⁶ *Centrab. f. d. med. Wissen.*, 1876, No. 28.
- ⁷ Charbon et Septicémie. *Comptes Rendus*, 1880.
- ⁸ *Centrab. f. Bakt. u. Parasiten.*, 1881, Band ix.
- ⁹ *Ibid.*, Band vii.
- ¹⁰ *Grundriss der Bakteriologie*, 1887.
- ¹¹ *Nord. med. Archiv.*, 1895, No. 12.
- ¹² Ueber ein Einfluss von Fieber und Leucocytose auf den Verlauf von Infektionskrankheiten. *Berl. klin. Woch.*, 1895, No. 15.
- ¹³ *BRITISH MEDICAL JOURNAL*, 1893, ii.
- ¹⁴ *Transactions of the International Medical Congress at Berlin*, 1890.
- ¹⁵ *Annales de l'Institut Pasteur*, April, 1894.
- ¹⁶ *Virchow's Archiv*, 1889, S. 14.

At the recent *conversazione* at University College, London, the apparatus to be used in a course of experimental psychology, which is to be started at University College in October under the direction of Professor James Sully and Dr. W. H. R. Rivers, was exhibited. The apparatus includes a Hipp's chronoscope, with control hammer for measuring the various forms of reaction time; Wundt's instruments for studying the estimation of time and the complication of sensations, an experimental organ, and apparatus for studying the sensations of active and passive movement. This and other apparatus forms the nucleus of a laboratory for experimental psychology, which will be the first of its kind in London, and, with the exception of that at Cambridge, in England, although there are now many such laboratories on the Continent and in America.