Table A (*continued*).			M.	F.	Total.
Group 3, col. 1 . .	8. Midlothian	F. resident	114.		
col. 2 . .	4. Perth	should be	6·5	4·4	5·5
col. 3 . .	„	„	0·6	0·4	0·5
Group 4, Note †, 1898, not 1888.					
Table A_1, col. 2 . .	4. Ireland	„	6·8	7·4	7·1
Table B_1, Div. 1, hours outside,	English Counties		6·1.		
		Total	6·3.		
2, „	„		5·9.		

(1) Phthisical insanity is, of course, but a small component in these figures.

Clinical and Experimental Observations on Katatonia.

By LEWIS C. BRUCE, M.D., Physician Superintendent, Perth District Asylum, Murthly; and A. M. S. PEEBLES, M.B., Assistant Physician, Perth District Asylum, Murthly.

THE following observations were made by my assistant (Dr. Peebles) and myself with the object of observing the physical symptoms of katatonia and hebephrenia. As the result of these observations we were led to make some experiments in the way of treatment, and we combined with this work some experimental observations on rabbits.

We have had under observation twelve cases of katatonia—ten women and two men,—but we have been able to observe only three cases of hebephrenia. We are therefore only in a position to place before you to-day our work on katatonia.

Physical Symptoms of Katatonia.

The history of the disease in our cases was quite in line with the classical descriptions of Kahlbaum and Kraepelin. Hereditary predisposition was present in six out of the twelve cases. In some the habits were vicious and drunken, in others the habits of life were good, and as the course of the disease ran typically in both classes, it is hard to believe that the defective habits did more than lower the resistive power of the individual, and were therefore only a predisposing cause. Three of the patients had suffered from previous mental attacks; in one of these cases at least the previous attack was one of katatonia, which was apparently completely recovered from. In every case the origin of the illness was gradual and insidious.

Loss of energy, listlessness, and nutritive failure were invariably present. Then hallucinations of hearing of a distressing nature appeared, leading to impulsive actions, or delusions, or to paroxysms of fear with complete loss of self-control, which necessitated hospital treatment.

Out of the twelve cases, nine women and one man were adolescents. Of the other two cases, one, a woman, was over thirty years of age, and the other, a man, was over forty years of age; and yet the disease was absolutely typical in both.

On admission all the adolescents were poorly developed and poorly nourished. The two older cases were both well developed, but their body-weight was below par.

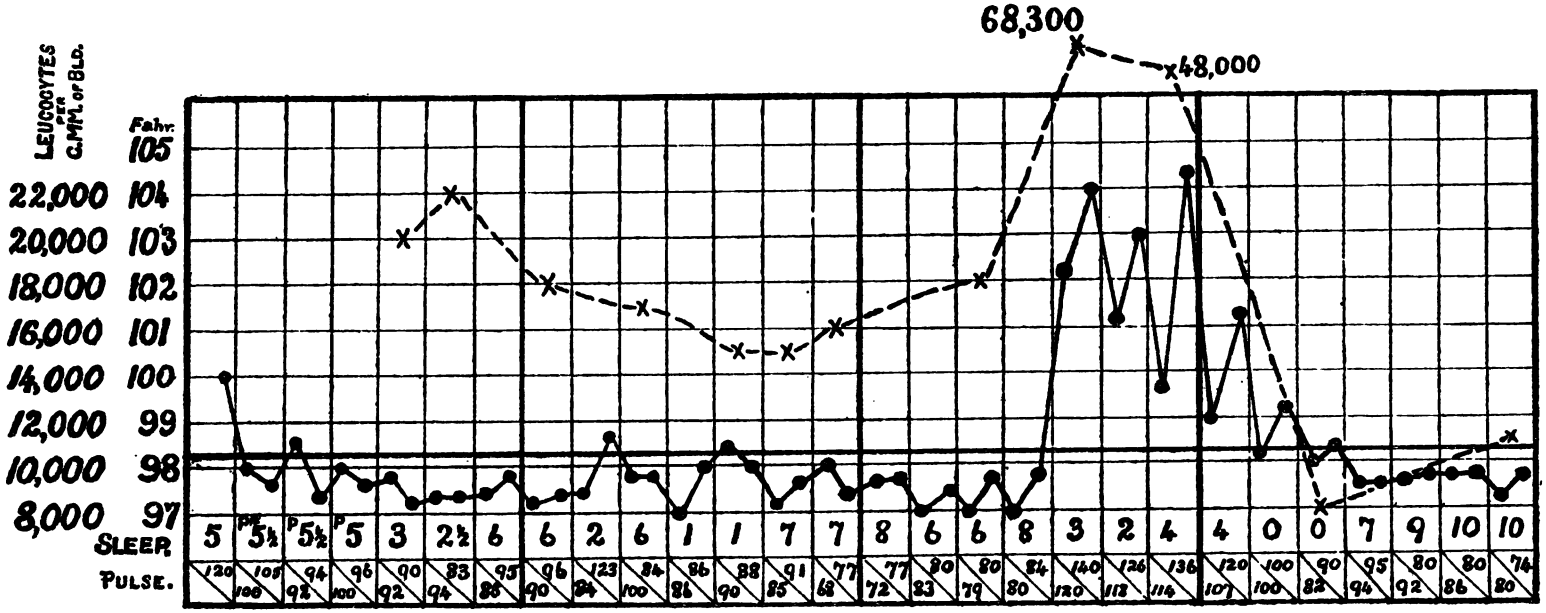
For convenience of description we have divided the disease into two stages: (1) the stage of acute onset; (2) the stage of stupor terminating in recovery or complete or partial dementia.

The physical symptoms of the stage of acute onset were as follows:—The alimentary system was disordered in every case. There was no desire for food, often no thirst. Vomiting after food was common. The heart's action was frequently rapid, irritable, irregular, and intermittent. The arterial pressure gradually rose until the acute stage terminated. The skin during mental paroxysms poured with perspiration; blotchy and pustular rashes were present in 50 per cent. of the cases. No deficiency of urine or urea was noted in any case. Each of the women patients menstruated once during the period of acute onset, and then ceased to menstruate until recovery or dementia terminated the disease. Dulling of sensibility to touch, heat, and pain was very common. The pupils were always dilated and sluggish in their reaction to light. The special senses of sight and hearing were not affected so far as outward impressions were concerned, but taste and smell were often completely disorganised: two patients mistook strychnine for sugar, and at least five of the cases were unaffected by strong ammonia. In every case the organic reflexes of micturition and defæcation were not under the control of the patient; these cases always tend to be wet and dirty. The skin and tendon reflexes were exaggerated. At uncertain intervals the voluntary muscles passed into a state of katatonic spasm, which lasted variably for a few minutes or hours.

The mental state was essentially one of confusion. Vivid auditory hallucinations, always of a distressing nature, were

present in every case. There was an appearance of preoccupation and fixed attention in these cases as they sat up in bed listening intently. Or they would suddenly run to windows or doors in response to imaginary voices. Very frequently these hallucinations led to paroxysms of terror, when the patient shouted and struggled and perhaps tried to jump through a window, run out of the door, or hide under the bed. In the intervals between paroxysms the patients might lie for hours with eyes closed, apparently oblivious to all around. In other cases, again, there were brief periods of sanity, but the patient had always a confused appearance, and was soon exhausted if spoken to. The power of continuous attention was gone. There was no memory of what occurred during the acute stage. Sleep during this period was deficient. The temperature was irregular, sometimes slightly febrile in the evening, sometimes paradoxical. In 50 *per cent.* of the cases the acute period terminated in a distinct febrile attack. Leucocyte counts during this stage showed a moderate persistent leucocytosis, the increase being chiefly in the polymorphonuclear and large mononuclear elements. Coincidently with the febrile attack, or if the febrile attack was wanting, the stuporose or second stage was ushered in by a high leucocytosis, the increase being in the polymorphonuclear cells. Chart No. 1, illustrating the first stage of the disease, shows the temperature, pulse, and leucocytosis per c.mm. of blood. It will be noticed that the acute stage terminated in a sharp fever, that coincidently the leucocytosis rose to 68,000 per c.mm. of blood. At the termination of the acute stage the patient may pass into a typhoid state; only one out of our twelve cases presented this symptom. Bacterial examination of the blood was made in eight cases. The method adopted was to run 3 to 4 c.c. of blood into 200 c.c. sterile broth by means of an exploring needle passed into any prominent vein in the forearm. Five of the flasks were sterile. Three contained organisms. Two of these were apparently accidental contaminations, but the third, obtained from the case which had passed into a typhoid state, presented a pure culture of a short streptococcus. The patient recovered from the typhoid state and passed into stupor. On testing the agglutinative power of her blood upon this streptococcus, we found that in a dilution of 1 in 30 with a broth culture of the organism, agglutination was complete in

CHART 1.

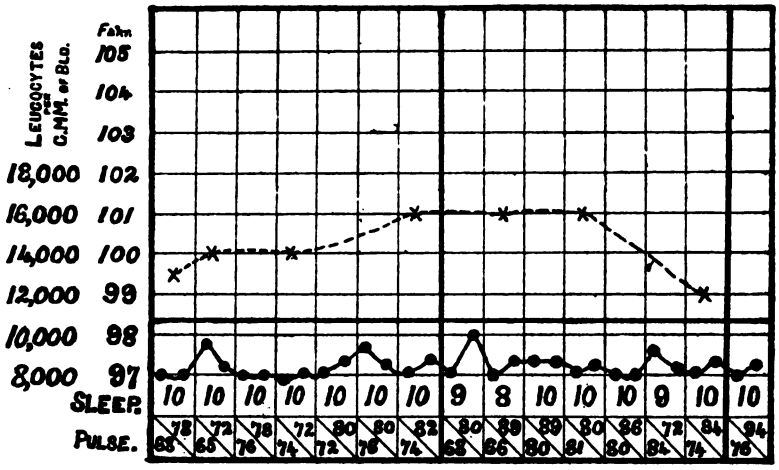

68,300
48,000
LEUCOCYTES PER C.MM. OF BLD.
Fahr.
22,000 104
20,000 103
18,000 102
16,000 101
14,000 100
12,000 99
10,000 98
8,000 97
105
SLEEP
5 P.M. 5½ P 5½ P 5 3 2½ 6 6 2 6 1 1 7 7 8 6 6 8 3 2 4 4 0 0 7 9 10 10
PULSE.
120 105/100 94/92 96/100 90/92 83/94 95/88 96/90 123/84 84/100 86/86 88/90 91/85 77/68 77/72 80/83 80/79 84/80 140/120 126/112 136/114 120/107 100/100 90/82 95/94 80/92 80/86 74/80

two hours. Control normal bloods gave no reaction at the end of twenty-four hours. The blood of the same patient failed to agglutinate *Bacillus coli communis*, nor did her blood agglutinate a very small coccus isolated from the blood of a case of acute mania. We have tested the agglutinative power of the blood of all our acute and three demented cases to this streptococcus in dilutions of 1 in 20 and 1 in 30. Eight gave definite complete clumping, three gave partial reaction, four gave no reaction. No control ever gave a reaction. The agglutinative reaction was slow, but was generally complete in six hours. No control ever reacted in twenty-four hours. We have tested the agglutinative power of the blood of fifty other patients, not cases of katatonia, to this streptococcus, and only five gave the agglutinative reaction. It is probable, therefore, that the agglutinin frequently present in the blood of patients suffering from katatonia is a specific agglutinin.

In no case under observation did the acute stage last longer than four weeks. This of course only includes the period of acute symptoms, not the prodromal period. The second or stuporose stage of this disease came on immediately after the febrile attack where such a symptom was present, or in default of the febrile attack a high leucocytosis heralded the onset of this stage. The physical symptoms of this stage are so well known that I need not do more than mention them. The alimentary tract was still disordered. The heart's action was weak and slow, the extremities were blue and cold, and the feet and hands became œdematous. The arterial tension fell. The lungs were liable to tubercular infection. The temperature was uniformly subnormal. The skin sometimes desquamated in small branny scales, sometimes was very greasy, and a condition of "varnished" skin was noted. Amenorrhœa was a constant symptom in women. It was impossible to test the sensory functions, but the special senses were quite active, as these patients knew what was passing around them. There was a tendency to retention of urine and fæces, the patient resisting these organic reflexes. The skin reflexes continued increased, but the tendon reflexes often could not be elicited on account of muscular resistance. The voluntary muscles were thrown into resistance by any attempt at passive movements. The mental state was one of stupor, often complicated by delusions. Impulsive actions, curious

attitudes, mutism, rhythmical movements, sudden outbursts of apparently maniacal excitement as sudden in termination as onset, all the innumerable physical and mental oddities to be seen in this disease, were well illustrated by the cases under observation. Sleep returned and was, as a rule, excessive. The condition of the leucocytes during this second stage was interesting. Immediately upon the onset of the stupor the leucocytes might fall to below 8000 per c.mm. of blood, but soon they rose again, running on an average between 12,000 and 16,000 per c.mm. The percentage of polymorphonuclear cells fell to about 60, the lymphocytes

CHART 2.



increased, and a transient eosinophilia occurred in every case. Three out of the twelve cases have recovered, and in them it was noted that the polymorphonuclear cells never fell below 60 *per cent.* As recovery progressed the leucocytosis did not necessarily rise, but the percentage of polymorphonuclear cells increased. When recovery was complete the percentage of polymorphonuclear cells fell again to about 60. The leucocytosis never fell, however, lower than 12,000 per c.mm. Three cases which have become demented, and a fourth which has every appearance of becoming so, presented the following peculiarities:—Early in the stuporose state their leucocytosis fell frequently to 8000 and 10,000 per c.mm., and the per-

centage of polymorphonuclear cells was below 50. In one of the cases the polymorphonuclear percentage fell sometimes below 30. Some indication as to prognosis can therefore be obtained by examining the blood of these cases.

Experimental Observations on Rabbits.

The object of the experiment was to ascertain if the streptococcus isolated from the blood of the case of acute katatonia produced any form of disease in rabbits. Rabbit No. 1 was injected intra-venously with 1 c.c. doses of broth culture of the organism. Rabbit No. 2 was injected intra-venously with 1 c.c. doses of a filtered broth culture of the organism.

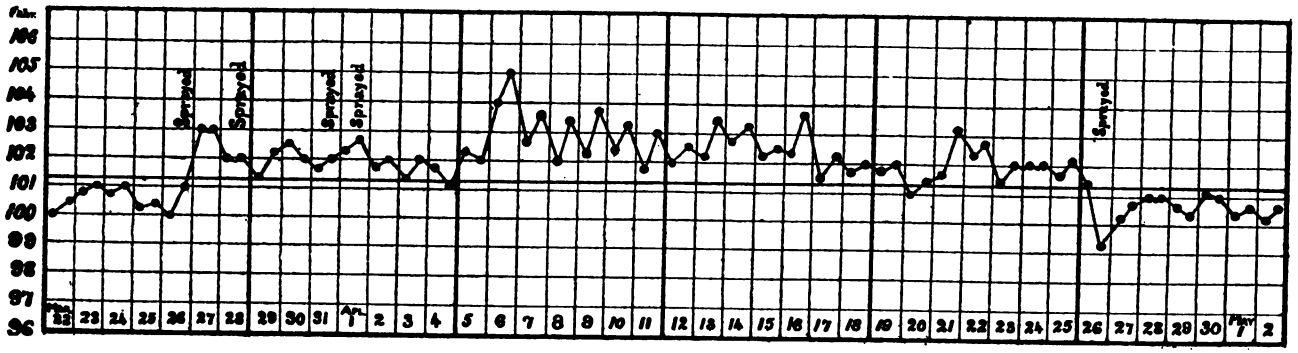
Rabbit No. 1 during a month received in all 7 c.c. After each injection the temperature rose one or two degrees, and latterly the temperature was irregularly febrile independently of injections. At the end of the month the animal became listless, dull, and lethargic, and the cutaneous reflexes were exaggerated. The animal always took food. In the middle of the sixth week we injected intra-venously 2 c.c. of an intracellular extract of the streptococcus. The temperature immediately fell to subnormal, and continued subnormal for two days. The rabbit at the same time wakened up out of its lethargy. Since then the animal appears to have become immune to the organism.

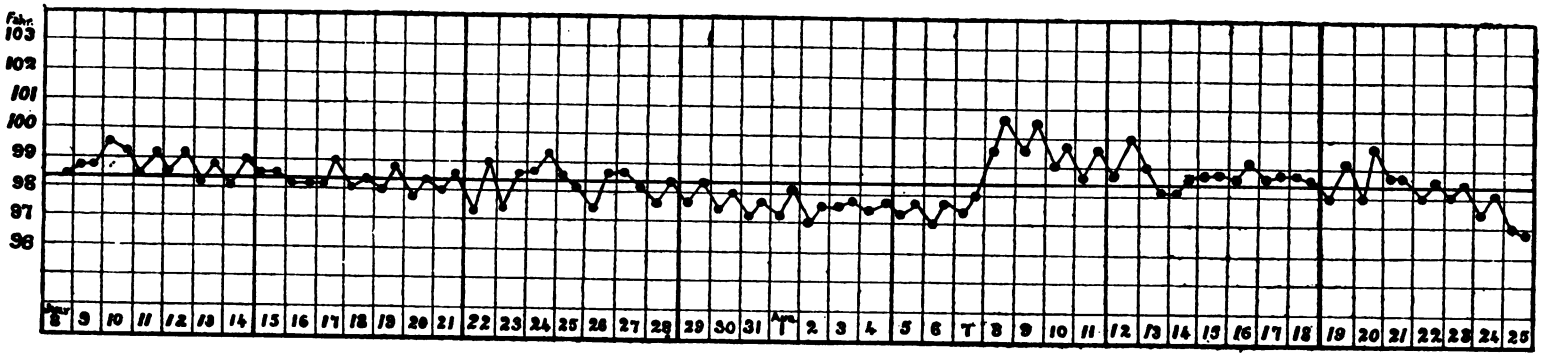
Rabbit No. 2 showed no reaction to the intra-venous injections of the filtered culture, which points to the fact that in broth cultures the toxin of this streptococcus is purely intracellular.

Rabbit No. 3 was inoculated subcutaneously with living broth cultures of the streptococcus. Rabbit No. 4 was inoculated subcutaneously with an intra-cellular extract of the organism. Both animals gave a slight febrile reaction to the injections, but no other symptoms were noted.

Rabbits Nos. 5 and 6 were sprayed with living broth cultures of the streptococcus, and by licking themselves were therefore infected by the alimentary tract. Both animals gave definite results. One or two days after infection their temperatures rose and continued irregularly febrile, independently of subsequent infections. In Rabbit No. 5 a definite febrile attack was noted ten days after the first infection with the

CHART 3.





organism. This febrile attack lasted irregularly for three weeks, when the temperature fell to normal, *i. e.*, 101·4 in a rabbit. Both animals took food, but they looked unhealthy, and nutrition was imperfectly performed. They both suffered from transient attacks of lethargy, and in both the superficial reflexes were increased. Both animals became immune to the organism about six weeks from the date of the first infection, and any further infection not only failed to raise the temperature, but actually lowered it to subnormal for one or two days.

Rabbits Nos. 7 and 8, sprayed with broth cultures of the streptococcus killed by heat (60° C. for 30 minutes), presented no symptoms. Every rabbit, with the exception of Nos. 2, 7, and 8—No. 2 was intra-venously injected with filtered broth cultures, and Nos. 7 and 8 were sprayed with dead cultures,—developed a specific agglutinin to this streptococcus, complete agglutination taking place within the hour with dilutions of 1 in 50 and 1 in 100. We found, however, in testing the normal agglutinative power of rabbits' blood, that certain rabbits possess serum capable of agglutinating this streptococcus in dilutions of 1 in 20 and 1 in 30. The agglutination is often incomplete. We have never been able to pass the organism through an animal and obtain it again from the blood.

The treatment of katatonia is eminently unsatisfactory. All but one of our cases, treated by rest in bed, fluid diet, saline purgatives, ran through the various stages of the disease unchecked. Our first effort at experimental treatment was to immunise a goat to the streptococcus obtained from the acute case of katatonia. We used the serum of this goat to treat two cases in a condition of stupor in subcutaneous injections of 12 c.c. daily. Treatment in both cases had to be discontinued on account of erythema and general urticaria. In one acute case it produced the same complication and no beneficial effect. We next exhibited the serum in 10 c.c. doses by the mouth in two stuporose cases. In both cases the temperatures fell very low, and continued very low during the period of administration of the serum. The patients showed no signs of improvement. The serum was again tried by oral administration in huge doses, 80 to 140 c.c. in a day in the twelfth case of our series during the acute onset of the disease. Here it again lowered the temperature, but its curative effect was

practically *nil.* We then treated five stuporose cases with subcutaneous injections of broth cultures of the organism killed by heat (60° C. for 30 minutes). Three of these cases were patients whose serum had failed to agglutinate this streptococcus. Our object in this experiment was to rapidly raise the active immunity of the patients.

Case No. 1 received injections of 33 c.c. in fourteen days. The highest temperature recorded was 99·8° F., and at the end of three weeks the patient had gained 6 lbs. in weight. There was no mental improvement.

Case No. 2 received 146 c.c. in forty-two days. The highest temperature recorded was 101·8° F. We believe this temperature to have been due to some accidental cause, as this case was quite immune to large doses of the dead culture. The patient gained 3 lbs. in weight during treatment, but there was no mental improvement.

Case No. 3 received 82 c.c. during a period of twenty-four days. The temperature was never febrile. There was no gain in weight and no mental improvement.

Case No. 4 received 66 c.c. during a period of thirty-two days. This patient gained 8 lbs. in weight. The temperature rose once to 99·2° F. There appeared to be slight temporary improvement mentally.

Case No. 5 received 56 c.c. during a period of thirty-three days. There was no febrile temperature. The body-weight increased by 3 lbs., but there was no mental improvement.

Eleven days after the last injection in each case the agglutinative power of the serum was tested. In every case the serum possessed a high power of agglutination. After the failure of the goat's serum to arrest the acute onset of the disease in the twelfth case of our series, we commenced to actively immunise the patient by means of subcutaneous injections of broth cultures killed by heat. This case was a very acute one; each mental exacerbation was heralded by an attack of vomiting and a feeling of sinking in the epigastrium. Within twenty-four hours of these prodromal symptoms the patient passed into an attack characterised by vivid hallucinations, wild terror, impulsive actions (especially trying to jump through windows), noise, and sleeplessness. We anticipated each attack by injecting first 4 c.c. and later gradually increasing doses. On each occasion the attack was aborted. In a week

the pulse-rate had fallen, and each injection lowered the temperature, which was inclined to rise prior to an attack. In this case the treatment undoubtedly arrested the disease, but how the injections acted we cannot explain. It is not possible that an immune body was formed in the short period which elapsed between the injection of the dead culture and the improvement in the patient's condition, a matter of two or three hours at the very outside. Towards the later part of treatment this patient also received by the mouth 2-minim doses of Acidi Carbolici, highly diluted, thrice daily, but improvement was most marked before this treatment was added to the injections of dead cultures.

Our conclusions from these observations are that katatonia is an acute toxic disease with a definite onset and course, in which the symptoms vary according to the resistive power of the patient, but in which the following diagnostic symptoms are never absent:—A prodromal period of gradual onset, which leads into the period of acute onset, with aural hallucinations, mental confusion, paroxysms of excitement, impulsive actions, katatonic spasm of the muscles, a hyperleucocytosis which at the termination of the acute stage indicates a virulent toxæmia. In the second stage a condition of stupor with muscular resistiveness to passive movement.

2. That even at the onset of the disease there is in about 70 *per cent.* of the cases an agglutinin in the blood-serum which appears to be a specific agglutinin to a short streptococcus which was isolated from the blood of an acute case of katatonia.

3. That by infecting rabbits through the alimentary tract or blood-stream with this streptococcus a condition of malaise with irregular temperature, increased skin reflexes, and mental hebetude is induced. This disease tends to terminate naturally in healthy rabbits in about six weeks, and a condition of immunity is established to this organism.

4. That treatment by an antiserum obtained from a goat has given no beneficial results.

5. That active immunisation of patients in the stuporose state produced no curative effect.

6. That active immunisation in the acute onset of the disease, tried so far in one case only, produced undoubted benefit, but how this beneficial effect is brought about cannot be explained

by any theory at present held with regard to the production of immunity.

Discussion

At the Annual Meeting in London, July 17th, 1903.

The President.—We are much indebted to Dr. Bruce for this most excellent paper, and for the experimental work which he has done in regard to this interesting disease. I shall be glad if those present will give an account of similar cases which have occurred in their practice.

Dr. Robert Jones.—I am very unwilling to begin the discussion, because I know there are several members present who have not only had cases of the same kind under their care, but have written extensively on the subject. I congratulate Dr. Bruce upon the experimental work which he has been doing. I am incapable of discussing the paper from this standpoint, because I have not worked in that direction; but if his paper leads to anything which will modify what I consider to be the greatest scourge among our educated youths, it will do a great amount of good. I am astonished to see the number of stuporose cases which have come under my care recently, comparatively speaking; that is, within the last half of my experience—say ten or more years. My experience goes back nearly a quarter of a century, and it was quite uncommon to have cases of katatonia and dementia præcox many years ago, but now they have become comparatively common. At Claybury Hall, where we have only fifty private cases, we have as many instances of this stuporose form as in the main asylum with its 2400 cases! I was very much struck by our President's remarks—with whom, indeed, I have lately had an opportunity of discussing this form of insanity—respecting the prevalence of these cases, and I shall make use of my own experience in an address on a coming occasion. Dr. Bruce says the rise of temperature is more or less typical of a patient who is under treatment for some time. I should like to know how much of that is due to the patient's condition under treatment,—that is to say, how much is due to the difficulties that nurses and medical officers have in feeding these cases? I referred yesterday to a case of œsophagotomy at the London Hospital. Precisely the same chart is seen in this case. If food goes into the bronchial tubes or gets into the lungs, after a certain time it gives rise to the same temperature reaction, more especially if the food taken has been milk, and I have at the present time a case of this kind which takes nearly ten pints of milk in the twenty-four hours! I should like to know what Dr. Bruce's experience with regard to the difficulty of feeding these katatonic cases may be, and whether he connects the late rise of temperature with a sort of subcatarrhal pneumonia—a form of broncho-pneumonia which may eventually end in death, but which presents no symptoms in the way of cough or expectoration; and, indeed, very few symptoms on careful auscultation. The early temperature one can to a certain extent understand, for there is a very marked "apprehensiveness" in these cases. A case comes to my mind which used to be dressed surgically at St. Bartholomew's Hospital in my student days, and which was reported in the Hospital *Reports*. When the dresser went to dress a fractured tibia in the case of a child, the temperature sometimes rose to 100°, and after the dressing was over it went down again to normal. There is no doubt that fear or apprehensiveness may cause such constitutional disturbance as may involve a rise of temperature. I do not wish to take up the time of the Society any further, but we have had an extremely interesting paper from an accurate observer, and I congratulate Dr. Bruce on producing what I consider to be a distinct addition to our knowledge.

Dr. Andriezen.—I have for some time paid attention to this particular subject of dementia præcox, and recently I have published in the *Hospital* an article on the subject, dealing especially with the varieties of this disease. I am extremely pleased to have the opportunity of hearing Dr. Bruce's paper, because it is an example of the newer and better type of clinical work which is so necessary for the advancement of our knowledge of many of these obscure mental disorders. A large amount of evidence has been collected to show—and that is borne out by cases one has seen and studied—that toxæmic conditions occur in many varieties

of dementia præcox. But I think we should not ignore the fact that has been insisted on by the French school of alienists, that the whole group of insanities which come under the heading of dementia præcox shows, almost from childhood, symptoms indicating some degree of what the French call degeneration of the brain. Sometimes it is allied to imbecility. Many subjects of dementia præcox who after adolescence become katatonic or demented, show in childhood extraordinary characteristics, and tendencies to obsessions and impulses of various kinds. Such conditions last practically throughout life—at least until dementia supervenes,—showing that throughout the whole period of growth, in later childhood at any rate, the brain is, as it were, evolving in a very abnormal, anomalous fashion. And this must be borne in mind as the chief factor, because it is in such subjects that toxæmic conditions will give rise to such extraordinary reactions as profound stupor, resistiveness, and silly vagaries of conduct which the mentally healthy individual afflicted with toxæmic conditions would not exhibit apart from mental confusion. It struck me very strongly in the course of Dr. Bruce's paper that his observations went far to show that we must drop to some extent the old psychological metaphysical views which we have held about mental diseases for so long. It is not so very long ago that, in Dr. Tuke's *Dictionary of Psychological Medicine*, the author of the article on katatonia, looking at it from the old standpoint only, said there was practically no such illness—that katatonia was really a melancholia which passed through a period of mania and went through stupor, and finally ended in a state of dementia. It would be extraordinary if a disease worthy of the name were a compound of four diseases. We know that that is not so, but that it is one disease which passes through four or five stages, which in their entirety constitute the disease. Katatonia and varieties of dementia præcox appear to us, from the most refined type of clinical research, to be undoubtedly diseases in the strict sense of the term, for they run their course through various stages, but these stages are not diseases by themselves, —in other words, that katatonia is not mania, or melancholia, or stupor, nor is it dementia; but that it is a disease which has characteristic stages through which it passes, and which have a natural sequence, although some of these stages may be slightly abbreviated or aborted. But the whole series of stages, taken together, comprise the disease. I am glad to hear Dr. Jones say he meets with more cases of katatonia than formerly. I meet with more cases of it than I used to. It is interesting to hear Dr. Bruce's observation that the toxæmia need not necessarily be febrile. It used to be widely believed in asylums that it was. In the very early stage of general paralysis, the temperature having been regularly taken, we looked for a rise of temperature but seldom found it, and then we doubted whether there was toxæmia at all. But at that time, which was ten or eleven years ago, we made no observations on the leucocytes, and we were not certain whether there was toxæmia or not. But the observations made during the last eight or nine years tend to show that a certain amount of leucocytosis above the normal occurs in the early stage of general paralysis, and where there is a slight febrile reaction leucocytosis is very much more marked, showing that we have here a new means for determining whether some serious degree of toxæmia is present or not. As regards katatonia my studies have been chiefly in the clinical direction, but the conclusions I have come to in this respect seem to show that it must be deemed worthy of inclusion in our system of classification; it has not yet been included in our psychological tables or statistics. Many of the cases of katatonia have been called "stupor," and other cases have been included as katatonia which were merely secondary stupor. If more papers of this character were read which contained clinical evidence of the sort which is necessary, it would do much to clear our ideas and make us drop a good deal of our old psychology, helping us to a better classification of the types of mental disease.

Dr. HAYES NEWINGTON.—I used to pay attention to this condition of stupor, and katatonia is certainly a new product since the time when I did pay attention to the matter. I have read one or two papers on katatonia, and I have noted what has been said about it here, and especially what fell from Dr. Andriezen. Katatonia is talked of as a disease. But if it is to be regarded as a separate disease we want a definition of it, and when we have got that we want it accepted generally. But in many of these questions of nomenclature—especially that dreadful word "confusion," which is becoming so prominent—one comes to see that

so many people have so many different opinions. We find that one man is talking of a group of symptoms under a name which perhaps does not quite cover the particular group of symptoms which another man might associate with the same term.

Dr. MOTT.—I should like to congratulate Dr. Bruce on this attempt to throw some light on an obscure disease. It is an effort in the right direction, it seems to me, to find out what the exciting cause in these cases is. And the toxic idea is the one which I think should be studied carefully. I would like to ask Dr. Bruce one or two questions. First of all, does he claim that there is a specific streptococcus in this disease? His experiments on rabbits rather led me to suppose that he did claim a specific organism which would produce in the rabbit, when the toxin was injected, a condition somewhat similar to that observed in the patients. Now, to prove that, it would be necessary, it seems to me, to take streptococci from other sources, or else perhaps you have only sick rabbit. I do not wish for a moment to throw any cold water on the very laudable attempt which Dr. Bruce has made, because I really think that this is a move in the right direction, and I think Dr. Bruce will fully admit any criticism that I offer is in the most friendly spirit. It is no good sitting down and looking at these patients any longer; the proper thing is to find out what is the cause of the toxic condition of the blood. To make his experiment more complete with regard to rabbits it would be better—and I speak from some experience in experimental medicine—if he would try some other animal. Rabbits are very fallacious animals; many mistakes have been made by using them. If he could use the streptococcus or the toxins from the blood of these cases on the dog, he would find that animal much more intelligent and satisfactory, because streptococci are very potent organisms, and produce profound effects. Another question I would ask Dr. Bruce is, are these streptococci generalised in the blood in such cases, or where do they exist? Where has he obtained his cultures from? I understand he has only got it in one case (Dr. Bruce: Yes). Of course it would be much more demonstratively proved if he got it in every case. And I think that the temperature chart which he shows is rather suggestive of a possible complication. If this disease were due to streptococcus one would have expected the temperature to be high early in the disease, when the leucocytosis is still active. I offer it as a suggestion, that this would have to be answered before we could accept the view that this was the cause of the condition. Dr. Jones pointed out that it is very easy to get a little broncho-pneumonia in these cases, and one which you cannot discover by physical signs. I have seen that so often in making *post-mortem* examinations, and no doubt Dr. Bruce has also seen it; and it would give rise to that temperature and to leucocytosis. But I do say that Dr. Bruce is to be heartily congratulated on a move in the right direction, and I wish him every success in this attempt, by clinical observation and experimental research, which is the only way, to solve some of these difficulties which we have to deal with.

Dr. BRUCE.—I am much obliged to the various gentlemen who have spoken for their criticisms. Taking the first temperature chart, I quite admit that the temperature, on the face of it, looks exactly like that of a case where there has been a little accident in feeding. But that is not the only temperature chart I have. The majority of my cases I never touched, beyond observing them carefully at the bedside and working in the laboratory at the blood. I interfered in no way with the course of the disease. The cases were in charge of special nurses, and there was a temperature similar to this in other cases, without symptoms in the lung; and they were overhauled by both of us and by a clinical clerk, and we could detect nothing in the lung which would account for this. The blood was sent up to Burroughs and Wellcome's laboratory to be tested for typhoid fever, because the condition of the patient suggested that illness. Again, there are other cases where you get a rise of temperature to only 99° or 100° for one night, but a huge leucocytosis; and then two or three days afterwards the patient passes into a state of stupor. I am willing to admit that these temperature charts, of which I have at least ten good ones, might be construed as being due to pneumonia, and I have so frequently seen broncho-pneumonia in acute mania producing such charts that I was very suspicious. But it was not till I got a series of cases that I began to think, here is the termination of an acute attack; the temperature falls, the leucocytosis goes down, and the patient goes straight off into stupor. These cases were fatal, with one exception. I had to feed one case with the nasal

tube, but all the others were carefully fed with milk. They got three to four pints of milk during the day, and a pint at night, in addition to other fluid diet. The idea of filling these patients up with custards and hard food which requires much digestion is irrational and bad treatment, besides being cruel. I agree with Dr. Andriezen that in all these cases hereditary predisposition is the chief cause. There must be some very serious change in the resistive power of the patient from youth upwards. But I do not agree with Dr. Andriezen when he calls these cases dementia præcox. I do not know that there is such a thing. Why should we classify a disease in accordance with its termination? If we carried that out we should say all disease is death, because it ends so. And if you classify a case according to whether it ends in dementia or not, to be consistent you would have to classify all diseases under one heading. There is a great difference between katatonia and hebephrenia; you do not get the same agglutinin in the blood in hebephrenia that you do in katatonia. Dr. Hayes Newington says when you get a collection of symptoms you cannot always call it a new disease; and apparently he is not very much in favour of the new name katatonia. You have a distinct collection of symptoms, which are apparently a distinct disease. What shall you classify it as? Are you going to classify it as melancholia, or as mania? You get a collection of symptoms which are neither the one disease nor the other. What will you do? Shall we stick where we have been for the last twenty years because we are afraid of putting a new name to our collection of symptoms? Katatonia is not melancholia; compare them one with the other. Take the blood of a case of mania, and you will not get the blood agglutinated by streptococcus. There must be some specific condition; there must be some difference between katatonia and mania, and between that and melancholia; and we must have some name, otherwise we cannot classify such cases.

Dr. Hayes Newington.—The reason I raised the question at all was that we heard dementia præcox mentioned, which was also a new name, and they both seemed to be recognisable diseases in certain quarters.

Dr. Bruce.—I think every case except general paralysis would come under the term dementia præcox according to some works in America; and under some of the names they have brought out, such as "depressive insanity," even general paralysis could be included. Dr. Mott's criticism I value very highly, and I agree with him about the rabbits. A rabbit, I find, is a most unsatisfactory animal to work with; its temperature seems to go up very readily, and it is easily frightened. I do not know that I could go the length of saying that this streptococcus is the specific one which causes the disease, but apparently it must have something to do with the disease; it is either a primary or a secondary infection, because you get this agglutinin so constantly in the blood of these cases, and I am not certain whether the streptococcus is present in every case. In the only case in which I got it, it was in the blood; and in the few instances in which I have got organisms in the blood of the insane the patients have been in a desperate state—in a state of typhoid collapse,—and you may say the organisms were the terminal infection. They existed in the blood, and on examining the films which we took on the same day I got two typical examples of this organism, showing it must have been fairly numerous in the blood on that day. The girl was treated with frequent saline infusions, and she made a very good recovery indeed. I have examined the alimentary tract in all cases where there was vomiting, and we have made cultures and tried to isolate the organism from it, but have failed. We have not obtained that organism again from any source whatever in these cases of katatonia. I got it from a girl, but I never examined the vagina, as there was no likelihood of infection. If this sort of work will stimulate anyone to make similar observations, then I shall be very pleased, because I am absolutely certain that any future advance which we are to make in psychology, so as to bring our speciality abreast of other specialities and equal to the advances in general medicine, must be made by work at the bedside in association with work in the laboratory.

Clinical and Experimental Observations on Katatonia

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