THESIS.

ACUTE ENCEPHALITIS.

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C A S E I.

J.C. aged 32.

The above patient was admitted to the South: :land Hospital, New Zealand in May 1897. He was sent in from a Country District where he had been working as a farm labourer. There was no history of any accident or injury to the head. He had been working as usual about the farm, and had stated that he felt cold and was shivering a little, then felt unfit for work and went to bed. As he got no better he was sent into the Hospital. As far as could be ascer: :tained the family and personal history were quite good. On physical examination he was enclined to be somnolent, but on being spoken to opened his eyes and answered questions but could give us no definite answers, he did not remember where he had come from and could not tell where he was correctly, he gave us the name of a place five hundred miles away, nor could he remember in the least anything about himself, he did not complain of any uneasiness and was free from any pain whatever only he seemed to have lost his personal identity.

After a careful physical examination of his circulatory system, his heart and blood vessels all seemed to be perfectly normal, his chest and abdomen showed nothing abnormal nor could I find any trace of disease. His breathing was noted as twenty per minute, his pulse seventy and normal tension. Temperature on admission 101. No cough was present. On tapping the skull with the finger he evinced no tenderness or the least sign of pain, he had no retraction of the occiput and appeared perfectly con: :tented. The conjunctivae were injected, the pupils normal, reaction of iris was sluggish. The fundus oculi I was not sure about. Next morning he seemed a little irritable but this passed away and he still answered somewhat stupidly. What was most noticeable was the change in his moral character, his employer stated that he was always respectful and sober, but the first few days he was very filthy in his conver: :sation and in his remarks to the nurses. He was very cunning and only filthy when I was away, on speaking to him he expressed sorrow but did not stop his bad habits. After five days his temperature fell and he

was again polite and civil and a good patient. On the second day he had for about twenty four hours paralysis of the right external rectus but at the end of this time it completely disappeared. There was at no time any photophobia. The sphincters were normal. Urine free from albumen. The pulse at no time was very slow. No vomiting was present. His ideas did not form any logical sequence but he never raved and it was only on being addressed that he spoke much. He slept a good deal, but eat very little, all symptoms seemed to be cerebal in nature, on recovery he remembered very little. Two years after this he was sent to the asylum and remained there for a few months.

Respirations were never about twenty.

Temperature 101 on admission.

1.	M. 99	E. 101.5
2.	99.3	101
3.	99.6	101
4.	99	100
5.	98	99
6.	98.6	98.6

C A S E II.

C.H.

This was a case of a child aged 11 years who had previously been quite healthy. The family history was very good. She was running about quite well in the morning but in the afternoon her mother stated that she took a fit of shivering and seemed to be very ill and had vomited once and that was all, she com: :plained to her mother that she had pains all over her and was crying a good deal with the pains in her back, she gradually became worse and then was partially un: :conscious, when I arrived the child at first appeared to be asleep and seemed somewhat flushed, the pulse was 70 per minute, but the thermometer registered 105.5 the respirations numbered 25 per minute. While I was examining her she woke up and seemed to recognise me. I spoke to her and asked her if she had any pain any: :where, and she shook her head and seemed unable to articulate, she seemed to know perfectly well what both the parents and myself said to her and to under: :stand what we were saying. When asked to put her tongue out she did so at once but at no time could we

get her to speak, this aphasic condition lasted until death, when asked if thirsty she nodded and took the milk that was given her. The pupils were dilated and acted somewhat sluggishly. The conjunctivae were sensitive but deeply injected. There was no hyper: :aesthesiae about the head or neck, the head was not retracted nor any pain evidenced in the child's face, the chest was normal and free from cough or any rales, the circulatory system and the abdomnal were carefully examined but seemed to me quite normal. Urine normal. Nothing but cerebral symptoms were evident, she got gradually comatose and died about six hours after I saw her being dead when I arrived at the house.

Post mortem examination was performed next day. On opening the skull the duramater appeared to be perfectly normal, the pia mater and arachnoid were injected, the gray substance was distinctly hyper: :aemic. A piece was put in mudlers fluid and hardened. The sections cut showed distinct evidence of white corpuscles and what I took to be red corpuscles. Parts of the fresh brain also showed evidence of the aexudation of blood cells. I felt certain that that there were plenty of leucocytes. There was fluid in the sub-arachnoid space but no deposit.

C A S E III.

Mrs K.

This patient was about 32 years old of a good family history, she had two children and her personal history was fairly good having had no serious illness, she had been working hard and was consider: :ably exhausted, she went out for a ride on her bicycle and on returning to her home complained of some shivering and pain in her back, she went to bed and within an hour she became apparently delirious, and the friends called me in. On making an examination I found that the temperature was 102, the pulse was of good tension and the artery seemed well filled and normal. The respirations were 22 per minute. She had no cough nor any rales about the chest in fact I could detect no symptoms that could be referred to any organ but the brain, her pupils were slightly dilated, and the conjunctivae were injected, the tongue dirty, but no pain on percussing the head nor any retraction of the occiput. The character of her apparent delirium was that she answered questions fairly well although the answers were all of a ludicrous nature, an hour or two later she appeared to know nobody but

her eyes were wide open and she looked all right. I knew the patient very well and when I spoke to her she did not know me and when spoken to by her husband or by her children she had not the least idea of who they were. On seeing them or on hearing them speak she appeared equally oblivious of who they were. She was uttering incoherent ideas and on hearing well known incidents had forgotten all about them. She took the very light diet that was given her but did not appear to know what she was getting. The whole partook of a waking dream, but any present impression called up no idea though when speaking herself she could name people and recall some past incident, it seemed quite like that of mind blindness. She had at no time any pain: :ful discomfort. At the end of forty eight hours she fell asleep soundly and awoke with normal cerebral functions but feeling very weak and recovered slowly. At no time was her temperature above 103 and all through there was complete absence of irritability.

C A S E IV.

H.F.

A man aged 37 was admitted into the South: :land Hospital N.Z. in a semi-unconscious state, no definite history could be ascertained as he was a stranger, he spoke on admission and seemed to brighten up a little, his eyes were injected, temperature 104, pulse 65, respiration 30, pupils contracted. No pain on percussing head nor at any time irritability, he did not grind his teeth nor any of the usual signs of meningitis, he became comatose and died.

Post mortem showed nothing abnormal in the dura mater. The pia mater and arachnoid were injected as was also the gray matter. On examination the brain skowed a large nubmer of red and white cells scattered through the surface.

ACUTE ENCEPHALITIS.

I have called the four cases that I here present acute encephalitis because I think that they may be so diagnosed, the absence of so many of the usual signs that accompany meningitis, and the presence of those which appear to me to be referable to an inflammation of the encephalon purely and simply make me for the following reasons decide on that title. Scattered through what medical literature that I had access to there are references to inflammation of the brain in a casual way, but most of the text books seem to devote very little space to the subject of acute encephalitis itself. Bristowe, 8th Edition, states " That it is a mere waste of time and ingenuity to " attempt to make any differentiation on account of " there being so constantly combined that the dis: " :tinction is almost impossible." But elsewhere he says it is unquestionable that many of the symptoms of cerebral mengitis are really due to implication of the subject gray matter. Dr Hilton Fagge, 2nd Edition, devotes a page to it. The American Edition twentieth century practice in medicine recognises it

but has only two pages on the subject. Osslers last edition on medicine 1901, the article on acute encph: :alitis is less than one page. In Coats' Pathology last edition there is less than one page. I am aware that since influenza has been written above it has been mentioned, but I have nowhere seen any article written upon it solely.

There are first of all points about the physiology of the brain that make it difficult to dis: :cuss as the lymphatic system of the cerebrum has not been fully worked out. Bevan Lewis has done a lot of work on the subject and in his last book in the Pathological section has shown the significance of deiters cells or ~~spiral~~ spider cells, and all through his book on cerebral pathology leaves the impression that he has been dealing largely with a series of chronic inflammations, but many of these chronic inflammatory conditions must have been in the early stages acute, and it is the absence of any pathological observations on the acute conditions on account of the few post mortems that make it difficult to discuss. It is noted that the sub-dural space has little to do with the matter there being no connection between that and

the sub-arachnoid space, but, the pia mater and arachnoid are stated to have as a rule by some observers only arteries and veins, (Coats Pathology page 129,) as if they only served to carry the blood to the gray matter, the sub-arachnoid space has direct communication with the perivascular and periganglionic spaces, these spaces apparently receive the fluid from the blood after it has done its work in nourishing the ganglion cells and neuroglia &c with other cells which act as lymphatics, so that it seems to me that the whole spaces can have as one function purely that of an lymphatic nature like that existing elsewhere in the body. Further Fosters Physiology teaches, paragraph 699, 5th Edition, in reference to the rabbits supply to the brain the "blood supply of even the human brain must still be "small, and making every allowance for rapidity of "current the interchange between the blood and the "nervous elements must also be small, that is, the "metabolism of the brain substance is of importance "not so much on account of its quantity as of its "special quality." So that the blood supply being small, the lymphatic vessels, that is, the perivascular, periganglionic, and subarachniod spaces are not necess:

:arily such active tissues as are required, for example in the kidney, where so much more metabolism takes place. It seems to me that too much importance is placed on the leptomeninges and not enough on the underlying cerebral tissue, the fact of there being little albumen in the cerebrospinal fluid is a difficulty, but most if not all the observations have been on hydrocephalic and other chronic diseases of the brain, so that it is hard to state definitely that it is lymphatic fluid. It is also difficult to think that where red and white corpuscles are the albumen should be wanting, the lymphatics here are at least different to the other tissues.

The other point that seems of great import: :ance is what evidence is there that inflammation of the brain substance proper is present. In making a post mortem of the brain especially is it difficult to be sure that hyperaemia always exists, as owing even to the lapse of a few hours gravitation and other elements come into play, and on opening the dura mater the lateral sinuses and other venous cavities are filled with blood which has drained there. The

naked eye appearance of the pia-arachnoid and gray tissue is also altered especially in one particular, one of the evidences of inflammation in any tissue is the exudation the fluid especially, this, owing to the nature of the lymphatic peculiarities of the brain drains away easily from the periganglionic and peri: :vascular spaces into the subarachniod space, and although it may exist there, it does not always remain in the cerebral tissue, as an evidence of a past inflammatory condition when examined after death, and the exudation that does exist is often put down to the inflammation or otherwise of the lepto-mengies themselves, the disappearance of the exudation exists in other tissues in the same way, in acute inflammatory diseases of the skin,such as those of an erythematous type,also in cases of erysipelas,specific fevers &c when a post mortem is made the inflammatory condition of the skin has disappeared and no trace of the redness or exudation exists, unless of course haemoglobin has been effused and haematoidum or some other derivative stains the tissues, so that in acute cases owing to the lympathic structure and the easy return of blood

through the blood vessels to the sinuses and inter: :nal jugular and easy flow into the subarachnoid cisterns, the exudation, is either difficult to detect or has been removed by gravity. Even in the tissues themselves it is not always easy to detect the fluid. It is different with the solid exudation, the leucocytes and red corpuscles can be easily detected and as a sign of inflammation this appears to me to be the most definite, wherever the endothelial lining of a capillary allows an excess of red and white corpuscles to pass through it means that the cell, which is a living structure, is more or less damaged, the passage through the wall according to physiologists is not a filtration but a living process of a moleculo- chemical nature, so that the presence of these cells in a place where they are not usually found means inflammation. Coats states, page 159, "In the examination of " tissues after death the presence of leucocytes " or round cells is often the most definite evidence " of the existence of a inflammation." But it is not necessary to go even so far as all pathologists from Virchow down have classified albuminious

infiltration or cloudy swelling as a parenchymatous inflammation, and, in the kidney the tubular epithelium is stated to have suffered from inflammation if it has undergone fatty degeneration and cloudy swelling, although there may be little other evidence, so in the brain analogous states exist, the neurone may be altered, the protoplasmic processes or the axone split up, or the cell body of the neurone altered, either the cell body is contracted or its molecular disposition different to normal, or else the whole cell enlarged with altered fatty material analogous at least to a tubular kidney cell, all this seems to be evidence of some amount of irritation quite as well deserving the name of inflammation in the brain as in the kidney. In the kidney the inflammation is well known to paralyse the functions of that organ, and as this occurs in so many febrile diseases, so, the alteration in the brain cells also paralyses its function, and it may quite as well be said to be suffering from an inflammatory condition as the kidney. So that even short of the presence of red and white cells the alteration of the cell itself of the neurone is some evidence of irritation.

Leonard Hill's experiments and others where an animal was bled to death and the cortical cells examined showing, according to their statements, that the cells always went through such a definite molecular change that they could state definitely that bleeding had been the cause of death. In the Deutsche medicinische wocherschrift for 1900 experiments are described where rabbits and other animals were poisoned by arsenic, and other poisons, and after preparing the specimens taken from the cortex of the brain, a definite change was stated to have taken place so that the various poisons could be recognised, this irritation could fairly be put down as an inflammation. So that the presence of the red and white cells and the alter: :ation of the axone if found after death may stand as evidence of inflammation. If found in the liver, lungs or kidney it is always stated inflammation was present, but it is so constantly found in the brain and very little notice taken of it while the mengies get the credit of the ~~information~~ inflammation. My whole argument is that it is principally an encephalitis or inflammation of the brain substance

that is present and such I take it to be in the cases I have cited. I think [illegible], while it may be a matter of inference, that the overfilling of the arteries, &c. and slight exudation ought to be recognised as inflammation. In other cases Coats, (page 662) devotes only half a page to diffused encephalitis where it is stated that an irritant circulating in the blood causes general irritation of the brain &c, that in acute febrile diseases popoff found abundant leucocytes, and that Middleton in the brain of a delirium tremens case found the whole encephalon overrun with leucocytes and that micrococci have been found form: :ing centres of irritation with minute softenings &c. In influenza and delirium tremens this appears to be the leading factor and the fault cannot be placed on the meninges but to the brain substance itself, and is just as much a parenchynatous inflammation of the brain sub: :stance, as glomerulo-nephritis, and acute yellow atrophy of the liver.

In other diseases the evidence produced at the post mortem table is not more than that in the two cases I have reported. Some years ago I made a post mortem examination on a man who died from pyaemia and evidenced amongst other symptoms complete paraplegia a few days before

death. On opening the spinal canal and taking out the spinal cord at about the dorsal region the spine for about two inches was distinctly altered being red and soft and easily torn with the fingers, that an inflammation existed there was no doubt, but not a sign of suppuration had taken place. The softness and redness which were practically the fluid exudate of red and white cells were the only evidence I had of inflammation. In transverse myelitis, and Anterior Poliomyelitis acute as far as reports go nothing else is present, in these cases the meninges have an exudate of fibrin on them, they distinctly show evidence of inflammation just as the cerebral meninges do, but the diseases are not called meningitis but myelitis and I think that where the brain is inflamed it ought to be encephalitis. I think the fact of the brain receiving comparatively so little blood, is one reason why so little is found as compared with the kidney which gets such a large supply, there the presence of diapedesis is very evident, also in the lung, and even the spinal cord receives a good supply. There is no fibrin present in the brain substance, but it deposits easily on the piaarachnoid, there is none in the cord until

it reaches the surface, so in the lung it is only when it reaches the air vesicle that fibrin comes down, in the kidney it is only when it reaches the tubule, that is, the ordinary connective tissue cell spaces seem to prevent its formation, and it seems that it is only in a traumatic case, or where blood has been effused in quantity that it forms into a haematoma &c. In some cases little importance is placed on these signs of inflammation, especially I noticed that Ossler, page 1075, in describing Acute Delirium (Bells Mania) states "There is great venous engorgement of the meninges and "gray cortex, in two cases there were peri-saccular "exudation and leucocytes in the lymph sheath and peri- "ganglionic spaces." This I should think is ample evidence of a fatal encephalitis, in cases of other diseases the evidence is no more than this and yet they are accepted as the fatal cause, Coats page 948, "In "some cases of Scarlatinal Dropsy the kidneys are to "the naked eye scarcely at all altered although the "patient may have died with symptoms of Uraemia." It is for these reasons that I think in the cases I have brought forward that the chief factor was inflam: :mation of the brain substance itself. I do not think

that imflammation of the meninges itself would produce the same order of symptoms, the meningeal tissues appear to cause great pain and generally pressure or the results of pressure symptoms, first on the brain substance itself producing coma, optic neuritis, pressure on the ocular nerves causing strdbismus, dilatation of the ventricles, slow pulse, as stated in the usual class of symptoms given in text book, while in the four cases I have given these were not present and irritability only in one for a short time. In Case II the suddenness of the attack prevented any detailed examination but a distinct aphasic condition was present, the child appeared to know perfectly what was said to her and of her own wants, if she could have spoken her ideas might have lacked intelligence but she seemed to me to understand perfectly, the absence of pain and all symptoms usually put down to meningitis made me think that it was purely and simply an encephalitis. The post mortem appearances certainly were all in its favour as round cells were found in the cortex. In examination of the brain and eye it is difficult for a general practitioner to be positive as the number of cases one gets are too scattered,

and where these happened I had no specialist to fall back upon, the intelligence appeared to me good as the tongue was put out at once without any hesitation what:ever, the tendency to coma seemed to be due to pressure. In Case IV I can add little excepting the same absence of pain with cerebral symptoms and injected conjunctivae, it seemed to resemble Case II. In the other two cases I could see no evidence of imflammation of the membranes, in Case No I the intelligence was good and the habits I ascertained afterwards were decidedly good as he gave no trouble to his employer, the suddenness of the attack in this as in all the other cases may have been due to the influenza bacillus, that, I could not be sure about as there seemed such an absence of it having attacked any other part of the body, such as cough &c or any chest symptoms. It might be that the particular toxin of the disease was the irritant in all cases, but whatever the etiology of the cases the result and only name I could think of to fit in with the appearances was an inflammation of the brain substance proper. In Case No I the leading features might be what one would expect from a case of acute insanity, but the short duration of the case, the absence of violence,

and the peculiar nature of his intelligence would scarcely fit in with acute mania, he had that cunning which is exhibited by the insane but his moral nature was so suddenly altered and the peculiar nature of it with the manner in which he used to insult the nurses always waiting until he knew I was out of the way when he became particularly filthy. He was very similar to a previous case which I had, where a horse had kicked a boy twelve years old, and a compound fracture of the skull resulted, over the frontal bone, some of the anterior part of the frontal lobe was extruding and had been torn away in the kick, other parts of the brain substance I had to remove but I returned as much as was possible, and sewed up in the usual way, he did very well with the exception of a complete change in his moral nature, he did not steal or anything of that description but morally his language and ideas were always exceedingly filthy, this must also have been an encephalitis although of traumatic origin, and the change in his intelligence seemed to me similar to Case No I which I have reported. Other changes in Case No I were those of space and time which were quite like the peculiar condition which is often shown by patients

suffering from alcoholic neuritis, he did not know where he was, nor where he had come from, nor where he had lived, nor could he remember the day of the week or how long he had been in his last situation. The pupil reactions were sluggish and the pulse slower that one would expect considering the temperature but otherwise he showed none of the usual symptoms of meningitis, it is interesting that he became insane two years later and I presume there must have been some degree of sclerosis of the connective tissue analogous to that described by Bevan Lewis in his insane cases. Case No III presented somewhat similar symptoms but the disorder to her intelligence was somewhat different. Her husband, children and intimate friends she treated as if they were absolute strangers, I may say that I have known her very well and never at any time saw or heard of the least evidence of hysteria, as far as her relations to the external world were concerned she seemed quite oblivious, the sensations produced on her retina, or her tympanum, she could not co-relate with any past experience, her own name and the name of her children meant nothing to her. Her memory of past events was not quite obliterated but she mixed up

events and persons in a ludicrous fashion, as far as I could make out as one would do in a dream, but if asked a direct question concerning those she had spoken about she could give no reasonable answer, the nature of the delirium was so far peculiar, when she recovered she recovered completely, not like a person with delirium tremens who may keep up a fixed idea for a few days, and her memory of her delirium was very slight, the nature of her food was also a blank to her, all the special senses seemed blunted to anything involving the idea of relationship. This seemed like a case of mind blindness reported by MacEwen where pressure on the cortical tissue, at the angular gyrus has caused mind blindness and recovery when the bone was truphined. I presume that the unusual presence of the exudation would cause deranged function, as the pressure inside the skull is considerable, palpating the fontanelle of a child shows how great the pressure is and what would happen if the finger were rough bone, on opening the skull at any time the meninges bulge considerably, this together with the unusual presence of the foreign bodies such as the exudate, would materially affect

the function of the cortical cell. Why the toxins or whatever caused the inflammation should select one part of the brain more than the other is a difficult question, in lead poisoning the fact that the supinator longus is not paralysed, and that in the system diseases of the spinal cord one more than the other is selected has received as yet no adequate explanation, although the embryological evidence is of some value, and in this respect the brain shows different times at which medullation takes place. I think that Flechsigs researches are of interest here, in his "Gehirn und "Seele" where he states that only one third of the cortex is devoted to sensori-motor centres, and that the association areas are different and he thinks subserve different psychical processes, the anterior to higher functions, and the posterior to those which have to do with the knowledge of the external world. In Case No I the anterior part of the brain seemed, on this hypothesis affected, and in the Case of No III the posterior part of the brain seemed inflamed. I do not think that at present much can be said about it, but I think the evidence points to involvement distinctly of the cerebral tissue itself. I have

been endeavouring to place on paper, evidence to show that far too much importance is sometimes placed on the meninges and not enough onthe tissue beneath, the small space allotted to inflammation of the brain sub: :stance itself in some of the well known taxt books has been my excuse for bringing it forward. I know that there are plenty of cases of encephalitis complicating other conditions, for instance that it follows a traumatic meningitis and that suppuration in ear diseases causes abscesses in the brain, but these are always complicated being associated with the symptoms connected with disease of some other part of the body. I fully understand that the meninges may also be affected, even in cases where the brain substance itself is affected, but what I principally contend for is that they are only indirectly affected, the essential condition being inflammation of the brain substance itself. In the twentieth century practice of medicine in the article on influenza, reference is made to cerebral symptoms coming on in that disease and proving fatal, and some cases of isolated symptoms which apparently could only be placed under the category of influenzal encephalitis, but the references are small and occupy but a small

space considering its importance, there are no differentiations given as to the symptoms, or how to distinguish it from others,but it distinctly states it as a separate entity. To attempt to make a diagnosis on the supposed difference that exists between various intellectual states may seem a stretch of imagination, but in Jacksonian Epilepsy and other brain diseases it is not very difficult to effect a fair localisation in many cases, mostly in the motor area, but cases have also been placed correctly in both the anterior and posterior lobes, especially at the angular gyrus and supramarginal gyrus, so that the differentiin roughly stating where the inflammation is may not be so difficult as we know more about the brain itself. If there were a complication of the meninges themselves it might be a much more difficult matter to locate any inflammation of the brain substance itself as most likely by this time pain and pressure symptoms would become prominent. At the post mortem table one is usually struck with the slight amount of damage the coverings to the brain undergo, when distinct evidence of inflammation is present, while the pleura, peritoneum

and pericardium show evidence of much more inflammatory changes than the meninges, possibly this is owing to the visceral coverings requiring as part of their function a good deal more active absorption, whereas the pia mater and arachnoid have not this to do to such an extent. From all I have read and seen it is a rare thing to find the meninges affected without the brain substance to a very slight extent, but I think the converse that the brain substance is often affected without the meninges more common, in cases particularly of tubercular meningitis, although of course the meninges may show evidence of deposit of the tubercle bacillus it is along the blood vessels and penetrating the brain to a slight extent that they are so often found, and cases are often reported where nothing but cloudyness of the meninges are noted, but the disease is always labelled meningitis. As regards the question of etiology I think it is interesting to read the accounts given of this disease in Hirsch's History of Diseases, in his account he states that in cases of influenza there were some of sudden death, (possibly cerebral pressure symptoms) gastro-intestinal cases, paralysis (neuritis), and that in some epidemics cerebral symptoms

were prominent, if this is true then the disease has not undergone such a change of type, but must have breed fairly truely. One epidemic we had in New Zealand of influenza there were three cases sent by me to the Asylum, all the history of symptoms previous to admission that we could obtain from the friends fitted in with influenza, following this violent delirium in the three cases which had all the characters of acute mania, they all lasted over three months, in these cases the meninges may have been affected, but the nature of their delirium and their intelligence was quite different from the cases I have given, they were all of a busy maniacal type and had to be restrained. Possibly also the cerebral symptoms of pneumonia may be due to either the toxins or the micro-organism it: :self irritating the cells and causing a transient inflammation. There is a distinct difference between diseases which are purely functional and those which are organic, but in all other organs of the body the covering has a certain amount of elasticity, whereas in the brain a small functional lesion may mean the pouring out of an extra amount of fluid, and owing to the closed cavity it may exert an injurious effect out

of all proportion to its amount, so that pressure symptoms are easily produced, and these would complicate even an encephalitis pure and simple. Having giving all my reasons for thinking that it should receive a dis: :tinct place and that inflammation of the brain exists quite as much as a separate entity as that which occurred in acute anterior poliomyelitis, and that it is not infrequent, and particularly that it is not the meninges that are affected, and if so,that they are only affected secondarily. In the cases I have given influenza may have been the start although no cough was present or any of the usual signs of that disease still it seems a likely factor. If any differential diagnosis could be made I should say that the absence of pain and irritation with the presence of mental symptoms of an intellectual nature quite well recognised in other diseases as of an aphasis description, say simple aphasis, or mind blindness, or affections of the higher psychic functions would point to an involvment of the brain substance. In Osslers Book on inflammation of the brain, page 1025, he states that headache,coma, somnolence, and all those symptoms which are common to acute brain trouble existed, but in the four cases I

had most of these were absent, I think the intellectual symptoms an important part of the diagnosis, what alter: :ations may take place in the blood I am unable to say.

As regards treatment little or none is mentioned by any authors beyond the usual brain treat: :ment of rest and quietness &c. Lumbar puncture is said to be safe and might give some evidence of a micro-organism but personally I have not tried it. In those cases of high temperature the cold water bath, wet pack &c would certainly be tried, but in those cases which are apparently hopeless I think on purely physological grounds it would be a perfectly fair thing to trephine the skull and give relief to pressure symptoms, as these are apparently the immediate cause of death, the skull could be trephined and the dura mater opened or not as the case may be, with all the present antiseptic appliances this would not involve much extra risk, the bleeding at the operation could be encouraged and would act like ordinary venesection, it seems a rational treatment where the case appears otherwise hopeless. Drugs to relieve arterial tension, calomel &c would also act well.