during the convulsive seizures which form a prominent symptom in the disease.

Normal cerebro-spinal fluid does not contain nucleo-proteid or choline, or if these substances are present, their amount is so small that they cannot be identified. Normal cerebro-spinal fluid produces no effect on arterial pressure; neither does the alcoholic extract of normal blood or of ordinary dropical effusions.

The presence of choline in the pathological fluids will not explain the symptoms of general paralysis; for instance, it will not account for the fits just referred to. Its presence, however, is an indication that an acute disintegration of the cerebral tissues has occurred. If other poisonous substances are also present, they have still to be discovered.

Our proof that the toxic material we have specially worked with is choline rests not only on chemical tests, but also on the evidence afforded by physiological experiments; the action of the cerebro-spinal substance exactly resembles that of choline. Neurine, an alkaloid closely related to choline, is not present in the fluid; its toxic action is much more powerful, and its effects differ considerably from those of choline.

PHYSIOLOGICAL ACTION OF CHOLINE.

The doses employed were from 1 to 10 c.cm. of a 0.2 per cent. solution, either of choline or of its hydrochloride. These were injected intravenously. The fall of blood pressure is in some measure due to its action on the heart, but is mainly produced by dilatation of the peripheral vessels, especially in the intestinal area. This was demonstrated by the use of an intestinal oncometer. The limbs and kidneys are somewhat lessened in volume; this appears to be a passive effect, secondary to the fall in general blood pressure. The drug causes a marked contraction of the spleen, followed by an exaggeration of the normal curves, due to the alternate systole and diastole of that organ.

The action on the splanchnic vessels is due to the direct action of the base on the neuro-muscular mechanism of the blood vessels themselves; for after the influence of the central nervous system has been removed by section of the spinal cord, or of the splanchnic nerves, choline still causes the typical fall of blood pressure. The action of peripheral ganglia was in other experiments excluded by previous intravenous injection of a solution of nicotine.

Section of the vagi produces no effect on the results of injecting choline. We have obtained no evidence of any direct action of the base on the cerebral vessels. Choline has little or no action on nerve trunks, as tested by their electrical response to stimulation. This aspect of the subject has been taken up by Dr. Waller and Miss Sowton, who will publish their results fully in a separate paper. Choline has no effect on respiration.

The effect of choline soon passes off, and the blood pressure returns to its previous level. This is due partly to the great dilution of the substance injected by the whole volume of the blood, and may be partly due to the excretion of the alkaloid, or to its being broken up into simpler substances by metabolic processes. We could not find it in the urine.

If the animal has been given a hypodermic injection of morphine and atropine prior to the administration of the ether or A.C.E. mixture, the effect produced by choline is a rise of arterial pressure, accompanied by a rise of the lever of the intestinal oncometer. We consider this observation of some importance, for it shows how the action of one poison may be modified by the presence of another. This has some bearing on general paralysis, for the arterial tension in that disease is usually high, not low, as it would be if choline were the only toxic agent at work.

PHYSIOLOGICAL ACTION OF NEURINE.

The doses employed varied from 1 to 5 c.cm. of a 0.1 per cent. solution. These were injected intravenously.

Neurine produces a fall of arterial pressure, followed by a marked rise, and a subsequent fall to the normal level. Sometimes, especially with small doses, the preliminary fall may be absent. Sometimes, especially with large doses, by which presumably the heart is more profoundly affected, the rise is absent. The effect of neurine on the heart of both frog and mammal is much more marked than is the case with

choline; in the case of both choline and neurine, the action on the frog's heart is antagonised by atropine.

The slowing and weakening of the heart appear to account for the preliminary fall of blood pressure; in some cases this is apparently combined with a direct dilating influence on the peripheral vessels. The rise of blood pressure which occurs after the fall is due to the constriction of the peripheral vessels, evidence of which we have obtained by the use of oncometers for intestine, spleen, and kidney. After the influence of the central nervous system has been removed by section of the spinal cord, or of the splanchnic nerves, neurine still produces its typical effects. After, however, the action of peripheral ganglia has been cut off by the use of nicotine, neurine produces only a fall of blood pressure. It therefore appears that the constriction of the vessels is due to the action of the drug on the ganglia; in this, it would agree with nicotine, coniine, and piperidine. Section of the vagi produces no influence on the results of injecting neurine. In animals anaesthetised with morphine and atropine, injection of neurine causes only a rise of blood pressure, which is accompanied with constriction of peripheral vessels.

Neurine produces no direct results, so far as we could ascertain, on the cerebral blood vessels. Neurine is intensely toxic to nerve trunks (Dr. Waller and Miss Sowton). It produces a marked effect on the respiration. This is first greatly increased; but with each successive dose the effect is less, and ultimately the respiration becomes weaker and ceases altogether. The animal can still be kept alive by artificial respiration.

The exacerbation of respiratory movements will not account for the rise of arterial pressure; the two events are usually not synchronous, and an intense rise of arterial pressure—due, as previously stated, to contraction of peripheral blood vessels—may occur when there is little or no increase of respiratory activity or during artificial respiration.

As confirmatory of Cervello's statement that neurine acts like curare on the nerve endings of voluntary muscle, and to which he attributes the cessation of respiration, we may mention that after an animal has been poisoned with neurine asphyxiation causes little or none of the usual convulsions.

The full paper contains references to previous work on the subject, and complete details of the methods used, and the cases investigated; it is illustrated by reproductions of numerous tracings.

[Note added April 20th, 1899.—It should be mentioned that in the case of brain atrophy referred to the cerebro-spinal fluid was removed soon after death. Since the foregoing abstract was written we have, however, by the kindness of Dr. Turner, had the opportunity of examining two specimens removed during life by lumbar puncture, and the results of our experiments with these corroborate the conclusions previously arrived at.]

TRAUMATIC EPILEPSY, STATUS EPILEPTICUS : TREPHING : RECOVERY.

By NATHAN RAW, M.D.,

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THE following case is a good example of what may be accomplished by an exploratory operation in an apparently hopeless case.

J. A., aged 25, labourer, was admitted into the Mill Road Infirmary, Liverpool, in an unconscious condition on September 2nd, 1898. Ten months previously he had fallen a distance of 18 feet upon his forehead. He was picked up unconscious and taken to a hospital, where he remained in an unconscious state for six days. He slowly recovered, and was discharged improved after six weeks. He resumed his work after a few months, and appeared in his usual health until two weeks before his admission here, when he was suddenly seized with a "fit." The fits continued at intervals for ten days. On the day before admission he had fifteen "fits" in rapid succession.

On admission the man was quite unconscious; his face was flushed, his pupils were unequal, the right dilated and fixed; there appeared to be some slight left facial paralysis, and the left arm and left leg were a little more flaccid than the right. The deep reflexes were present. There was no scar or other

indication of the position of his injury. After a few hours he was able to understand simple questions, but could not give any information regarding his accident. The convulsions always commenced with flexion of the left thumb, rapidly spreading to the arm, and then becoming general. The head and eyes were always turned to the left side. There was well-marked optic neuritis on the right side.

He gradually passed into a condition of status epilepticus with stertorous breathing, profuse sweating, and complete coma. The temperature was 102° F., and the pulse 130. He was apparently dying, and as there was no definite scar or depression to indicate the position of the injury, it was decided not to operate; but his wife begged that something should be done, and I reluctantly consented.

On September 6th, his head was shaved, and the following operation performed. As he was quite unconscious, no anæsthetic was required. A large semicircular incision was made, so as to reflect downwards the whole of the scalp of the forehead. When this was done it was seen that the right frontal bone and half of the right parietal bone were depressed to the extent of quite one-eighth of an inch. A disc of bone was removed with a 1-inch trephine. The dura bulged to the level of the outer table, so great was the intracranial pressure. Another disc of bone was removed, and then I chipped away with forceps the greater part of the right frontal and the anterior portion of the right parietal bones, leaving an opening measuring $3\frac{1}{2}$ inches by $2\frac{1}{2}$ inches. The dura mater was not opened, and the bone was not replaced, but the wound was stitched up and a drainage tube inserted.

He recovered consciousness in six hours; the temperature rapidly dropped to normal; the pulse, which was 140 when he left the theatre, had slowed down to 100 by the evening, and after a natural sleep, he was able next morning to express himself as feeling better.

He made an uninterrupted recovery; the wound healed by first intention; and in eleven days he was walking about the ward.

On March 12th, 1899, he presented himself for inspection. He had been at regular work ever since his discharge on October 12th, and was feeling perfectly well, and had had no fit or twitching since the operation. He was directed to wear an ordinary cap with a large thick pad to protect his head.

ON THE ATROPHIC MARASMUS OF NURSLINGS (SO-CALLED HOSPITALISM OR ATHREPSY OF PARROT).*

By ADOLF BAGINSKY, M.D.,

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AMONG the multifarious diseases of children connected with disturbances of the digestive apparatus there is one that has for some time especially attracted the attention of physicians. It is characterised by a persistent decrease in weight, with gradual lessening of bodily powers and progressive exhaustion, with the occurrence of more or less marked complications of the respiratory organs, the skin, and the kidneys. The disease has been noted from time immemorial to occur in institutions where nurslings are taken care of. The affection has always been a source of terror to those in charge of foundling asylums, children's nurseries, and the like, on account of the extremely high mortality which has at times made it appear questionable whether it was worth while to attempt the rearing of children in such asylums at all. Charles West, in his *Lectures on the Diseases of Infancy and Childhood* (fourth edition, London, 1859) expressed the opinion that the affection was due to the exhaustion of the various organs.

Since the important advances which have been made in recent years in children's diseases, especially since the extension of our knowledge of anatomy, physiology, and epidemiology, various attempts have been made to make the clinical and pathological anatomical relations clearer. Investigators seem at the present moment to be of two opinions. One group of writers directs special attention to chronic catarrhal processes in the intestinal tract with their ultimate consequences.

They attribute these catarrhal conditions to insufficient or faulty nutrition and to faulty nursing and care. The other group of writers lay more weight on bacterial infection, and above all on the influence of toxic substances. These toxins are the products of intestinal bacteria, and may be absorbed without any real lesion of the intestinal mucous membrane existing, and it is thought that by a process of chronic intoxication they seriously disturb the vegetative functions. The clinical aspects of the whole question were very well set forth by the French writer Parrot some years ago in a series of very comprehensive articles to which very little of importance can be added. To the affection Parrot gave the new name "athrepsia."

I will spare you the exact description of the affection, which is unfortunately only too well known, and will content myself with showing you, for the purpose of recalling some of the more exaggerated features of the disease, a few photographs from my clinical cases. You will recognise the *macies maxima*—the deep sunken eyes, the wrinkled face, the piteous expression of countenance, the withered appearance of the skin as it covers in folds the atrophied muscles, while the fatty subcutaneous layer has practically entirely disappeared. There are, however, no rachitic changes in the bones, which are, on the contrary, well formed and without beading.

The first question that arises for our consideration is whether, since the disease is so notoriously common in children's asylums, we have to think of it as an infection or not. In answer to this question I may say that I have never seen this progressive atrophic condition spread from bed to bed in a hospital, but have always found that cases occur sporadically. When cases are observed side by side the relation is merely an incidental one; the children fall away gradually but very slowly, and at different rates of progress, not as though affected by the same pathological conditions, but finally reaching the same pitiable goal. Especially we find chronic dyspeptic disturbances occurring with varying degrees of severity, with occasional mild exacerbations, diarrhoea that is not severe running its course with or without vomiting, sometimes even constipation or normal-seeming motions, while furuncles may occur on the skin, and otitis media duplex may supervene.

As may well be imagined all this brings the children down in condition more and more, at times one complication, at times another seeming responsible for the deterioration in health. Remissions, however, may take place, and there may even be a gain in weight, which, however, is usually lost again in a short time.

No one can doubt that in all varieties of the affection bacterial influences have a place, and that the bacillus coli, the streptococcus, staphylococcus, pyocyanus, and pneumococcus play a certain rôle in the etiology; yet these influences are not such that we can attribute to them any specificity in the production of the anomalies of nutrition that result. The really efficient cause of the condition in children's asylums is the same as that which is at work in the homes of the poor: It is the want of proper and sufficient care. The errors are, perhaps, only slight, but they suffice, when added together, to produce the condition. It may be that the child is let lie too long in damp or dirty wrappings, that it does not receive thorough cleaning immediately after vomiting, so that minute particles of vomited material are carried by aspiration into the lungs. When the child is fed by the bottle, it may be that the milk is given, now warm, now cold, or with the child lying down, so that again opportunity for the aspiration of food particles into the lungs is afforded. Even when apparently the greatest care is taken as to cleanliness of both bottle and nipple, other causes remain at work. In short, it is to a series of defects in care that the serious disturbances that are ultimately produced are really due. If each child could receive special attention for itself these defects might be avoided. Of course, I cannot deny that in a nursing department the communication of disease by child to child may easily take place, but for this the hospital as such is not responsible, but it is the fault of the nurses, who are either too few in number, or lacking in proper training, or who do not conscientiously fulfil their duties.

What are, then, the physiological and the anatomical conditions found *post mortem*? With the help of my chemical assistant, Dr. Summerfield, I have recently made a series of

* Read in the Section of Diseases of Children at the Annual Meeting of the British Medical Association, held at Edinburgh, July, 1898.