TWO CASES OF CONVULSIONS

BY

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SOME time ago I was consulted about the following case after death had taken place:

Operation for Webbed Fingers on a Child Aged Two Years.

When the mask was removed at the conclusion of the operation respiration had ceased, the pulse was not palpable, the colour of the child was fairly good. This was about 6 p.m. Resuscitation was attempted as follows:

Coramine 1 c.c. was given.
Artificial respiration with oxygen applied.
Endotracheal catheter passed—oxygen given through it, and later carbon dioxide and oxygen. Adrenalin 1 c.c. into the heart. No pulse present still. Abdomen slit—cardiac massage through the diaphragm. Heart muscle flabby. Heart started. Artificial respiration continued for a few minutes before the child took a breath properly—the colour and the pulse improved. Pupils were still dilated. The abdomen was closed. Endotracheal catheter was left in situ, and the child was returned to the ward after about 30 minutes' time, when the condition improved.

During the night the child's condition deteriorated—then came twitchings of the face and arms and the child was very restless. Nepenthe M ii. given, oxygen was administered, and coramine ½ c.c. four hourly. There was some improvement for a time, then the condition again deteriorated. About 9 a.m. there was cyanosis and twitching of the face and arms. The child was put into an oxygen tent. The child died at 10 a.m.

I immediately communicated with the pathologist who would conduct the post-mortem, and asked him to keep the
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brain. The post-mortem examination was carried out that afternoon.

There was no gross physical lesion elsewhere. The thymus was slightly overweight. There was a yellowish discoloration of part of the cerebral cortex and the caudate nucleus. Such a discoloration is noted in a few of the cases where brains have been examined after death from anoxia. Abbott and Courville note it in a case of bilateral necrosis of the globus pallidus. There was a large number of sections made, but in my ignorance of histological procedure I had failed to guard against cortical autolysis so that in all my sections simple evidence of necrosis cannot be regarded as sure evidence of ante-mortem damage.

May I say here that if an anaesthetist should get the opportunity of seizing a case like this he should remember to put the body in a refrigerator immediately after death, or pack ice all round the skull?

Fortunately, however, there is evidence of ante-mortem damage. Nearly every photograph shows an enormous engorgement of all the blood vessels.

Photograph No. 1. Nissl. 2/3 objective.

Shows two engorged vessels in the cerebral cortex with a large number of white cells between them.

Photograph No. 2. Nissl. 1/6 objective.

This is the previous slide under a high power objective showing that the cells lying between the two blood vessels are white cells. The whole slide is not in focus as it is slightly uneven.

Photograph No. 3. H. and V. G. 2/3 objective. Basal Ganglia.

Shows an enormously engorged vessel with perivascular infiltration.

Photograph No. 4. Nissl. 1/6 objective. Mid Brain.

Shows a motor nerve cell with disappearance of dendrites, "powdering" of Nissl granules and displacement of the nucleus to the side of the cell. There is considerable swelling of the axon and early satellitosis. This is not an irrecoverable condition, but undoubtedly represents at least a temporary loss of function.

Photograph No. 5. Nissl. 2/3 objective. Medulla.

Shows intensive clumping of satellite cells around the remains of (two?) nerve cells.

1/6 objective. Ditto.
DISCUSSION OF HISTOLOGICAL EVIDENCE

The engorgement of the vessels, I think, can be taken as an indication of a pathological process in the brain, and I do not think it should be confused with an engorgement at the time of asphyxia or of a reactionary engorgement immediately following asphyxia, as this is the state of the brain 16 hours afterwards.

There is evidence in several slides of slight perivascular infiltration. The two slides shown, comprising Photographs 1 to 3 are outstanding examples. It is interesting to notice that this is seen in cases of carbon monoxide poisoning, which is merely a specialized case of anoxia.

In arguing that local stagnation is the important factor which designates the local area of necrosis Courville\textsuperscript{4} points out that necrosed areas are usually around the vessels. The collection of white cells around these vessels may be significant.

Secondly, in the mid brain there is evidence of what might be termed "a very sick motor cell" which does not point to more than a loss of function but taken in conjunction with the other evidence it seems likely that this cell is dying. There are several cells with swollen axons—this is but one example.

In the third slide there is an intensive clumping of satellite cells, and there is here a remaining part of one, if not two motor cells. There is evidence of satellitosis in several other sections but this one shown is the only one to which an impartial mind can attach certain pathological importance.

From a histological point of view this may not be weighty evidence, but I submit that when one views all these things as a whole, and remembers that the child only survived 16 hours after the fatal incident that it is remarkable that so much evidence of tissue damage in the central nervous system can be found, especially when it is remembered what a power of recovery the tissues in the young have. Furthermore, although some 30 sections were stained the chances of missing the level of a group of satellite cells like that shown in Photograph 3 must be extremely high.

If, therefore, one grants that definite damage to the nerve cells has occurred it remains to find out from what cause.
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There are two obvious causes:
1. The ether damaged these and other cells, and so caused the original cessation of respiration and the later sequelae.
2. The ether caused the cessation of respiration, etc., and the consequent anoxia did the permanent damage.

Had this been the first reported case of its kind a considerable time might be spent in arguing it, but in view of Courville's work and his comparison with deaths due to asphyxia in his series, and those due to anaemia in Gildea and Cobb's series, I think it can be accepted that the second explanation is the correct one.

There are the following points of special interest:
1. This is the first case so far as I know of nerve cell damage to be reported as low in the central nervous system as the basal ganglia and medulla from anoxia. All cases up to date have dealt with the cortex and caudate and lenticular nuclei.
2. As far as I am aware there is no such report in existence of a death which has followed ether anaesthesia. Courville recently wrote a paper on "Ether Anaesthesia and Cerebral Anoxia," in which he quotes only two cases: one alive with a decerebrate rigidity and blindness, and one in which an autopsy was performed without examination of the brain. He says "as yet the detailed cerebral changes after anoxæmia under ether anaesthesia (actually due to cardiac or respiratory failure) are unknown." This is one such case.
3. From the point of view of discussion on convulsions under anaesthesia it will be noted that there were no convulsions during anaesthesia, but twitchings of the face and arms started about 1 A.M.—seven hours later. At this time nearly all the ether administered must have been eliminated but the effects of the anoxia remained.

Conclusions

It seems to me fair to say that while the histological evidence, if taken piece-meal, must be considered inconclusive, the whole picture certainly points to nerve cell damage from anoxia and that the history of the case is almost conclusive. It seems probable that had the patient lived a little longer
and had steps been taken to fix the brain immediately, that there would have been abundant certain evidence of this.

My thanks are due to Dr. Hilda M. Dean for the notes on this case, and for the post-operative treatment of which she was responsible.

I have to thank Dr. E. S. Duthie for securing the specimen for me, Dr. Ruby Stern for her willing help with, and instruction on, the histological appearances, and Mr. Ashford who made the photo-micrographs.

The second case that I wish to record is one in which convulsions followed a loss of blood.

I was asked to see a patient, aged 75, with a view to giving him an anaesthetic for a per-urethral resection of prostate.

His general condition was poor. Myocardium poor. Lungs clear. B.P. 148/92. He was a well-educated man, who stated that he had an idiosyncrasy to morphine. In view of this no preliminary sedative was given, but ephedrine gr. 1 was given ten minutes before operation. At 3.37 p.m. he was given 10 c.c. 1/1500 percaine intrathecally. It is my practice not to use a perineal anaesthesia but to get analgesia as high as the umbilicus in case a suprapubic cystotomy should be deemed advisable. This obviates the necessity of having, for any reason, to supplement the original anaesthetic. At 3.50 the operation was commenced, ½ c.c. veritol having been given intramuscularly at 3.45. At 4.30 p.m. the patient was looking a little pale and a further ½ c.c. of veritol was given intramuscularly. At this time the surgeon told me that he had "just about finished." A few minutes later the surgeon warned me in a whisper that the last cut which he had intended to make with the diathermy had apparently cut across a large vessel. At this stage the patient said he was very comfortable but "a little tired." In spite of copious washing with hot lotion through the endoscope it was impossible to allay the bleeding sufficiently to see a bleeding point which could be treated with a diathermy. The bleeding was profuse and it was decided to do a suprapubic cystotomy immediately. The patient was put in position, and the towels had been put on when, just before the surgeon made the incision, the patient had what obviously
would have been a generalized convulsion if his legs had not been paralysed. His eyes twitched, his jaw made chewing movements consisting chiefly of clonic masseter contractions which were so violent that the nurse whom I had instructed to hold the jaw was unable to keep her fingers on it. The hands and arms moved violently in clonic contractions. Before this occurred I had put some coramine in a syringe, and now asked the surgeon to proceed immediately to secure the bleeding point. As I pulled out the arm, the patient gave a last shudder and became flaccid. The eyeballs rotated upwards and the pupils became fully dilated, not reacting to light. There was no corneal reflex. The pulse could not be felt, respiration ceased. I injected $\frac{1}{4}$ c.c. of coramine into a large vein in the arm, told the nurse to hold the arm up in the hope that the coramine would be returned to the heart, and gave the patient's chest a few rhythmic squeezes. In the meantime the surgeon had opened the bladder which contained well over a pint of blood and had packed gauze firmly in the prostatic cavity. The artificial respiration restored normal respiration and the pulse and eye reflexes returned. The bleeding point was secured and the operation completed without further incident, except that as the patient partially recovered consciousness he vomited.

The patient did not become fully conscious until 8.20 p.m., and had no precise memory of the operation—remembering only being given the spinal anaesthetic and lying on the table for a while. His recovery was not rapid, but was complete, although it was jeopardized by hiccup which lasted for five days.

The convulsion was a typical asphyxial convulsion, the like of which I have only seen when "pushing" nitrous oxide in an alcoholic, when I unintentionally employed the secondary saturation technique. Extensor spasms of a clonic type were followed by a temporary cardiac and respiratory failure.

Cerebral anoxia was undoubtedly produced by the sudden reduction in the quantity of circulating blood in addition to a previous comparative anoxia and was probably only combated by the combined effect of the 4.30 intramuscular dose of veritol, together with the 4.45 intravenous dose
of coramine which, presumably, together raised the blood pressure sufficiently to allow the cerebral circulation to recover.

It will be noticed that the convulsions were of "central" origin as they only reached down to the level of the spinal anaesthesia.

Barcroft, in 1920, divided anoxæmia, or more correctly, anoxia, into three types:

1. The anoxic. 2. The anaemic. 3. The stagnant.

In the first class Barcroft includes normal respiration in an atmosphere deficient in oxygen, and normal respiration where the atmosphere has not normal access to the blood, as in lung disease. I should think he would also have added reduced respiration as one sees it in spinal anaesthesia which results in insufficient oxygen being admitted to the blood, although there is here something of the third, or stagnant, type.

In his third or stagnant type he includes blood supplied to the tissues in quantities insufficient to allow of adequate oxygenation as in shock, lowered blood pressure and haemorrhage.

In my second case haemorrhage followed insufficient ventilation of the lungs and resulted in a very violent convolution which to my mind is typical of anoxia. Why the average case of haemorrhage without anaesthesia usually results in fainting or loss of consciousness without convulsions is a difficult question, but it is possibly due to the fact that the tissues normally have a reasonable reserve of oxygen.

The only case comparable to this that I can quote is one in which Batten and Courville described mental disturbance in a patient following a profuse haemorrhage while under nitrous oxide anaesthesia. Only slight cyanosis was noticed at the time, and convulsions followed 15 minutes later.

Beecher* quotes Gildea and Cobbs' observations on convulsions due to cerebral anæmia and says "experienced anaesthetists who have had the misfortune to witness 'ether convulsions' will recognize that Gildea and Cobb have given unintentionally a remarkable description of this cata-
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In his earlier works Courville was inclined to stress the fact that the damage due to anoxia was in the presence of nitrous oxide, although later he admits that the effect is purely one of anoxia.

To summarize we have:

1. Numerous reported cases of so-called "ether convulsions"—i.e. convulsions occurring during ether anaesthesia, in many of which the patient's colour is referred to as "poor."
2. Convulsions occurring due to cerebral anæmia as reported by Gildea and Cobb.
3. Convulsions occurring during nitrous oxide anaesthesia and resulting in permanent cerebral tissue damage.
4. Convulsions occurring long after ether has been withdrawn, but after damage has been inflicted on the tissues of the central nervous system, as in my first case.
5. Convulsions occurring after haemorrhage where no ether or other general anaesthetic has been used, as in my second case.

The only explanation that I can see that fits all these cases is that the convulsions are due to anoxia and that permanent damage due to anoxia has been proved in cases where the victim has survived sufficiently long for its effects to be established.

REFERENCES

