

## A CASE OF SUDDEN DEAFNESS OCCURRING DURING ECLAMPSIA.

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THE following case is, I think, well worth placing on record. I have so far been unable to find one similar in the published literature, either otological or obstetric, whilst inquiries amongst obstetricians of my acquaintance have been equally unsuccessful, save that Dr. Eardley L. Holland informs me that he has met with one instance in the German literature of eclampsia. The history of the case is, briefly, as follows.

The patient, aged 40 years, was seen by me on Oct. 26th, 1909, at the request of Dr. J. B. Wallace of Clapham. She had been deaf in the right ear since October, 1907, and she stated that the deafness had come on suddenly during eclampsia. She did not think that there had been any change in the hearing since its onset. Tinnitus had been present from the beginning, at first of a roaring character, "like thunder," now more of a "continuous whizzing." There had been occasional general dizziness, which was now much less, but no specific vertigo could be elicited. There was no paracusis Willisii, hearing being better in a quiet room. On examination both tympanic membranes were of similar aspect, their texture and lustre being normal, and the light reflexes somewhat small. Both mallei moved normally to the Siegle speculum. The nasal septum was slightly deflected to the right, but caused no obstruction; the post-nasal space was small, and there was slight chronic pharyngitis. Functional tests gave the following results: Weber's test lateralised to the left. Acoumeter, spoken and whispered voice heard on the left the length of the room (over 15 feet) and the whole series of tuning forks, from 3C 16 to C<sup>4</sup> were heard well by air conduction. On the right side spoken voice was perceived at 31 inches, acoumeter and whisper *nil*. By air 3C 16 and C<sup>4</sup> were lost, the perception for the forks between these limits being greatly diminished, but the absolute length of time in seconds for which they were heard could not be accurately gauged. Rinne's reaction to C (128) was positive on both sides. Bone conduction to the C fork was, after repeated testing, apparently — 17 seconds on the right and — 8 seconds on the left. The Edelmänn Galton-pfeife was heard up to 40,000 double vibrations on the left, up to 15,000 (?) on the right. The tests for each ear were done with the opposite ear tightly plugged with cotton-wool, but it is possible that the voice and whistle results and those for some of the higher forks were due to hearing through the skull by the opposite ear, as I have had no opportunity of checking their accuracy by testing with Bárány's noise-producing apparatus. I have already pointed out this possible source of error in a published note.<sup>1</sup> There was no spontaneous nystagmus and the calorific test showed no impairment of the vestibular apparatus.

Since examining this case I have to thank Dr. Wallace and Dr. A. E. Giles for the following notes as to the original attack of eclampsia.

Dr. Wallace was called to the patient on Oct. 19th, 1907, at 4 A.M., and found her insensible. "She was removed to a nursing home and was seen by Dr. Pelham Webb. Tried chloroform, &c. Dr. Giles saw her about 9 P.M., and transfused 8 pints into her median basilic vein. All the 20th she remained insensible, being unconscious for over 48 hours and having frequent convulsions. Between 3-4 A.M. on the 21st I delivered her of a seven months' stillborn male child; on my return after breakfast she had regained consciousness. She had no more fits after this, but hung between life and death for days. The urine at first was almost solid and gradually improved. Her heart was dilated, she was badly cyanosed, and oxygen and strychnine were freely used. She gradually improved and left the home on Nov. 15th; albumin remained in the urine for some months after. It was her third pregnancy; I confined her twice before (girls both times), but nothing noteworthy occurred. She could hear before the illness."

Dr. Giles gave me the following note: "There is not much

to say clinically, but briefly the matter was as follows. I saw her at 9 P.M. on Oct. 19th, 1907, when she had had a good many fits stretching over some hours and was comatose; I at once transfused her to the extent of 8 pints of saline; labour had not advanced far, so I decided not to interfere; she had no more fits and the following morning, Sunday, she was conscious but dazed; confinement took place naturally on the Monday morning. The urine was absolutely loaded with albumin, so that we came to the conclusion that she probably had a definite nephritis; her mind remained rather dull for several weeks after the confinement."

Without entering into the etiology and pathology of eclampsia (an admirable review of which by Dr. Eardley Holland has appeared recently<sup>2</sup>) two features may be pointed out: (1) that the symptoms are those common to many toxæmias, and that eclampsia is, broadly speaking, an auto-intoxication by the toxic products of protein disintegration; (2) that eclamptic blood is characterised, among other things, by an increased coagulability, and that Dienst considers that the increase of fibrinogen in the blood of eclamptic patients is most important, and accountable for nearly all the thrombotic and other changes which occur in the body. Deafness occurring during eclampsia may, therefore, be due to toxæmia, to effusion or hæmorrhage into the cochlea, or to thrombosis or embolism. In the first case—deafness due to toxæmia—it is unlikely that the condition would be confined to one ear, since the deafness met with in other toxæmias is invariably bilateral. I am inclined to regard the present case as one of blocking of the cochlear branch of the auditory artery, since hæmorrhage or effusion into the cochlea would have been accompanied by vestibular disturbances, which appear to have been absent and which, if overlooked in the gravity of the eclamptic symptoms, would have left permanent traces recognisable on testing the vestibular apparatus.

The condition was unlike any I have ever seen due to nephritis, and it is more likely that the deafness was dependent for its cause on the general conditions found in eclampsia than on kidney lesion. Renal changes occur in 99 per cent. of cases of eclampsia (Schmorl in 73 necropsies only met with one instance in which the kidneys were healthy), and, in the review already alluded to, Dr. Eardley Holland says that the severe clinical symptoms of eclampsia cannot be dependent upon the renal changes.

It is somewhat surprising that no other cases of deafness arising during eclampsia have been recorded. Eclampsia is, however, so fatal a condition that it is possible that instances have passed undetected both clinically and on the post-mortem table.

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## A NOTE ON COCAINE POISONING.

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THE easily accessible accounts of the acute toxic effects of cocaine mention among the prominent symptoms those of brain excitability, mental instability, tonic and clonic convulsions. Convulsions seem not to be one of the usual effects of cocaine poisoning in man, and the description of tonic and clonic applied to them hardly agrees with my own observations in a recent case.

At 2 P.M. I was suddenly called to see a married lady, aged 40 years, suffering with the most violent spasms. The history was as follows. She had been in her usual health, but in consequence of some throat irritation she had been occasionally painting her fauces and tonsils with a 2 per cent. solution of cocaine hydrochloride. Before luncheon she had painted her throat because of a "choky feeling." Immediately after luncheon, though not feeling ill, she again had the same sensation and retired to paint her throat a second time. No sooner had she done this than the sensation became worse, and she felt as if she could not breathe. She rang for her maid who found her in violent spasms. On my arrival, perhaps five minutes from the onset of the attack, I found her half sitting, half lying in a chair, clutching at her sides with both hands, making grimaces such as often accompany intense muscular effort, quite oblivious to her surroundings, and occasionally uttering piercing cries. Many

<sup>1</sup> Journal of Laryngology, vol. xxiv., p. 251.

<sup>2</sup> Journal of Obstetrics and Gynaecology of the British Empire, vol. xvi.

of the movements were of the voluntary type, for on seizing her wrist in order to restrain her she made partial efforts to get it free.

A more careful examination was made after she had been placed on the floor; her face was intensely red, but was not moist, her eyes were more prominent than normal, her pupils were medium sized and equal, and her pulse was strong, full, regular, and slower than one would have expected. Her breathing was not obstructed except by the spasms. For a few moments she would remain somewhat quiet, then she would clench her teeth, strike out with her arms, and her whole body would become stiff with tonic muscular rigidity. During the spasms she was totally unconscious, but in the more quiet intervals she appeared to know what was going on and once inquired why she was lying on the floor. The movements were so violent and it was so difficult to prevent her from injury that chloroform was deemed advisable. A dose administered on a handkerchief quite insufficient to induce anæsthesia succeeded in allaying the convulsion, and it was then possible to place her on her bed, after which no further spasms occurred.

The whole attack did not last more than 15 or perhaps 20 minutes. The return to her normal state was extremely rapid and in some 20 minutes after being put to bed she was able to get up and relate to me her symptoms. She remembered nothing distinctly after she rang the bell for her maid until she found herself in bed. She had no recollection of my entry into the room, and the only feature that seems to have impressed itself on her memory was that she felt "full of fight." She was able to attend to her social duties in the course of the afternoon and felt no ill-effects until the next day when she lacked some of her usual energy.

The convulsions most nearly resembled those seen in hysteria, but they were more violent; they lacked entirely that avoidance of self-injury which is so conspicuous in many forms of hysteria; during their course the patient was completely unconscious; and, lastly, the patient is not a hysterical subject and has had no similar seizures before. The occurrence of the convulsions in relation to the use of cocaine as a throat application makes it most likely that they were due to that drug, especially when it is remembered that they occurred in a non-hysterical subject and, the drug having been withheld, have not been followed by any further tendency to spasm. An examination of the bottle showed that a deposit had settled to the bottom mixed with which was a fungoid growth, and it is natural to suppose that some of the solid drug or some decomposition product of it had been applied on one or both occasions of painting the throat preceding the attack.

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## THE INFLUENCE OF NARCOTICS ON PHAGOCYTOSIS.

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IN the practice of both medicine and surgery narcotics play an important part. One of the first principles taught to the medical student is the necessity of putting an injured part to rest. Simple appliances and remedies will often produce the desired result, but there are many cases in which rest, in the true sense of the term, can only be obtained by means of narcotics. A patient is suffering, we will say, from great pain, restlessness, and inability to sleep after a severe operation. A dose of morphia is given, all restlessness disappears, and the patient sleeps. Relief from pain and distress in such cases is generally considered sufficient to justify the means and no thought is taken of any ulterior effect of the drug.

A great deal of work has been done of recent years on the subject of immunity, and thanks to Metchnikoff, Ehrlich, Wright, and many others, we now appreciate the great importance of the natural resistance of the body to disease. Before deciding that narcotics may be used with advantage it would be well to consider what effect, if any, these drugs have on the phagocytes. In this paper the effect of morphia only will be discussed.

In 1898 Cantacuzène published<sup>1</sup> the results of some

interesting experiments on the effect of opium on phagocytosis. A guinea-pig immunised against cholera was given tincture of opium subcutaneously, followed by a sublethal dose of cholera vibrios introduced into the peritoneal cavity. He found that in spite of vascular dilatation diapedesis did not take place. The vibrios retained their form and motility and multiplied rapidly. As the effect of the opium passed off diapedesis began, and in a short time large numbers of leucocytes appeared on the scene, which rapidly enveloped the vibrios. The animal invariably died in spite of the fact that no free vibrios were present in the exudate. On examining the exudate it was found that some of the leucocytes, showing marked signs of degeneration, were literally stuffed with vibrios. Cantacuzène explained this in the following way. Some of the leucocytes, enfeebled by the opium, although capable of enveloping the vibrios, are killed by the toxins produced by the latter, and serve as a suitable medium in which the vibrios multiply. He states that this never takes place unless the animal be under the influence of opium. In another series of experiments tincture of opium was given, followed by a mixture of immune serum and cholera vibrios in such proportion that the control survived. He found that Pfeiffer's reaction took place but phagocytosis was retarded and incomplete; some of the vibrios escaped, multiplied rapidly, and caused the death of the animal.

Metchnikoff was the first to call attention to the phagocytic power of the leucocytes and fixed connective tissue cells, and to the important part this phenomenon plays in saving the tissues from invasion by pathogenic organisms. In 1894 Pfeiffer described the phenomenon named after him, and maintained that the destruction of bacteria in the living body was brought about by substances contained in the blood serum and was not due to any influence exerted by the leucocytes. In reply to Pfeiffer the above-mentioned experiments were advanced by Cantacuzène in support of Metchnikoff's theory, and apparently no attempt has been made to apply the knowledge gained to the practice of medicine.

I have undertaken the following experiments with the object of throwing further light on this important question.

*Experiment 1.*—An emulsion was made from a 24 hours growth of staphylococcus aureus on agar slope in 1 per cent. sodium citrate. A dog was given half a grain of morphia subcutaneously. One volume of dog's blood and one volume of citrate emulsion were mixed together and incubated at 37° for 15 minutes. The number of bacteria was counted in 25 polymorph leucocytes: before morphia, 123; half hour after, 64; one and a half hours after, 29. After the morphia injection the dog became slightly drowsy.

*Experiment 2.*—A guinea-pig was given half a grain of morphia subcutaneously. One hour later two cubic centimetres of broth were injected into the peritoneal cavity. Three hours after giving morphia one cubic centimetre of an emulsion of staphylococcus aureus in normal saline, killed by heat, was injected into the peritoneal cavity. Fluid was withdrawn from the peritoneal cavity half an hour after giving the emulsion. The bacteria in 25 polymorph cells were counted: morphia guinea-pig, 89; control, 168.

Experiment repeated. 60 cells were counted: morphia guinea-pig, 59; control, 162.

Fluid was much more easily withdrawn from the control than from the morphia guinea-pig, and the exudate from the former was much richer in leucocytes. After 24 hours the exudate was again examined. In the control the cocci in the leucocytes were for the most part swollen up and stained badly, while those in the white cells of the morphia guinea-pig showed little change. After giving morphia the guinea-pig showed slight signs of narcosis.

*Experiment 3.*—A guinea-pig was given one-quarter grain of morphia hypodermically. Three-quarters of an hour later two cubic centimetres of broth were injected into the peritoneal cavity. Peritoneal fluid was removed after two hours and mixed with 2 per cent. sodium citrate, and the leucocytes were washed twice in normal saline. One volume of washed cells, one volume of own serum, and one volume of emulsion of staphylococcus aureus were mixed together and incubated at 37° C. for 15 minutes. Morphia guinea-pig, 2 in 30 cells; control, 21 in 30 cells.

Experiments were also made to see if morphia inhibited the growth of staphylococcus aureus. It was found that a plentiful growth took place in 10 cubic centimetres of broth containing 1-10th of a grain of morphia.

From the above experiments it would seem that morphia exerts a marked influence on the leucocytes. Not only does it check diapedesis, but phagocytosis is diminished in a marked degree. The growth of bacteria, on the other hand, is not appreciably affected.

Let us consider for a moment what bearing this has on the practice of medicine and surgery. It is probable that in most surgical operations a certain number of pathogenic

<sup>1</sup> Annales de l'Institut Pasteur, 1898.