In Snakes, there is a small fenestra rotunda left open in the capsule. In front of this, the fenestra ovalis appears; and the rudimentary hyoid arch grows towards it, and, dilating above, gives off an epihyal; it then becomes arrested, and ossifies to form the columella. There is no tympanic cavity or annulus; and the rest of the hyoid arch is for the most part suppressed.

In such aberrant Lizards, as *Hatteria* and the Chameleons, there is no tympanic cavity nor annulus; and in the latter there is no fenestra rotunda. Moreover, in these types, the columella remains inside the quadrate, like the hyomandibular of a fish. The rest of the hyoid is developed in its lower part in the Chameleon; but in *Hatteria* there is a continuous hyo-stapedial chain from the fenestra ovalis to the floor of the face. In Chameleons, the basi-hyal is immense.

In the typical *Lacerta* and many other high types, the columella is long and continuous, having a proximal plate, a rod, a double ascending, and a tongue-shaped extrastapedial process. Ossification stops at the end of the rounded rod; and here an infrastapedial is given off, which corresponds to the interhyal of Ganoids and Teleostei. The stapedius muscle is now distinct from the digastric, and is inserted into the outer fork of the suprastapedial. The cerato-hyal and basi-hyal are continuous, the latter being a long style. There is a rudiment of a second branchial arch, and there are also two long hypobranchials supporting the larynx. In most types, at any rate, there is no annulus tympanicus. The tympanic cavity is well developed, but the bones surrounding it are not pneumatic. The epi-cerato-hyal does not unite with the infrastapedial, as in *Hatteria*.

In the *Chelonia*, the pharyngo-hyal exists as a cartilaginous rod dilating proximally to fill the fenestra ovalis, and a second joint dilated distally to form the extrastapedial, which becomes circular. There is a cartilaginous annulus tympanicus. The cerato-hyal is aborted; but there is a small hypophyal rudiment attached to the buccal plate. The first cerato-branchial is large; the second is not developed; but there are two hypobranchials forming the thro-hyals. There is a large tympanic cavity; and this is made so much the larger by the quadrate, squamosal, and even the opisthotic, becoming pneumatic. In the smaller kinds of *Emys*, the tympanic cavity is almost entirely in these bones. There is no siphonium carrying air to the mandible; but Meckel's cartilages unite in front, and nearly differentiate a broad basi-mandibular. The basi-hyobranchial is a continuous plate, apiculate in front.

In the *Crocodyles*, the columella is formed in the same manner as in the *Turtle*; but the distal segment, instead of being circular, is formed of two fan-shaped flaps—a supra- and an extra-stapedial. In a very early stage, the cartilage of the mandible and cerato-hyal is continuous. Above the cerato-hyal, there is both an inter- and an epi-hyal segment; the epi-hyal unites for a time with the suprastapedial; and the inter-hyal with the inferior angle of the extrastapedial above, and with the top of the cerato-hyal below. This hyo-stapedial chain, much like that of *Hatteria*, continues intact until after the middle of incubation; then, during the last month, the segments all separate again, and the cerato-hyal detaches itself from the articular region of the mandible. At the time of hatching, these parts, which have separated again from the columella, exist as small nuclei. There are a cartilaginous annulus tympanicus, a huge tympanic cavity formed mainly inside the large quadrate, and a median Eustachian tube between the two lateral ones, which last are forked above. These tympano-Eustachian canals open into the basis cranii, which is pneumatic both in the sphenoidal and in the occipital regions; the alisphenoid part of the bony auditory capsule, and the whole occipital arch, are burrowed by air-cavities. From the tympanic cavity, a membranous tube passes to the articular region of the jaw behind the hinge; this siphonium carries air to the pneumatic cavity in the proximal part of the lower jaw.

In Birds, the columella, at an early stage, is very similar to that of *Hatteria*; but only the epi-hyal is developed in connection with it. This connection remains; but the whole cerato-hyal region is membranous. The hypohyals meet each other at a sharp angle in front of the first basi-branchial; the second basi-branchial supports the larynx. There is one branchial arch, the upper cornua of which embrace the skull, and sometimes, as in woodpeckers and humming-birds, run forwards to the nasal region. The quadrate is pneumatic, and communicates by a foramen with the tympanic cavity, which lies principally in the hollow wing of the exoccipital. There are two Eustachian tubes, which meet in the middle line under the sella Turcica. The whole skull is pneumatic, and the tympanic cavity communicates with all the surrounding bones. In the *Passerine* birds especially, the siphonium is ringed with small bones, there being seven of these small tympanics on each side in *Corvus corone*. There is no cartilaginous annulus, and the meatus externus is becoming deeper. The concha, though imperfect in most birds, is large, but membranous in owls.

NITRO-GLYCERINE IN PUERPERAL CONVULSIONS.*

By W. E. GREEN, M.R.C.S. & L.S.A., Sandown.

Surgeon to the Isle of Wight and Newport Junction Railways.

ON August 21st, 1881, I was summoned to attend Mrs. M.; but, as I was absent, my partner, Dr. Barker, saw the patient for me. He found that she was primipara, and that a midwife had been in attendance; and, in consequence of the labour being unduly prolonged, it was causing considerable anxiety to the friends. As the head was presenting, and all seemed to be going on well, Dr. Barker left, leaving instructions to be sent for again in case of necessity. On returning from my morning round, a messenger met me with an urgent request to go immediately, as the patient was in convulsions. On my arrival, I found her lying on the floor in convulsions, which, however, were less violent than they had been. On their first appearance, the midwife had, with considerable presence of mind, applied snipisms to the calves of the legs and to the nape of the neck; the patient had been convulsed an hour. I had her placed in bed, and, finding the head of the child well down upon the perineum, I requested Dr. Barker (then present) to apply the forceps and deliver at once. The removal of the placenta was followed by a short rush of blood, which soon stopped. The convulsions had ceased before the application of the forceps, but the unconsciousness remained. The pulse was quick and of high tension; the face and eyelids puffy; the legs oedematous, and pitting deeply upon pressure, whilst the feet were cold. Leaving the case in charge of my partner, I ordered a mixture containing eight minims of a solution of nitro-glycerine (1 per cent.) in an ounce of water, with instructions to give a teaspoonful every hour until the evening. The first dose was given to the patient about two and a half hours after the commencement of the attack, the coma being then as profound as at any time; within ten minutes of taking it, the patient regained perfect consciousness, and asked questions about her confinement and child, of which, till then, she had been oblivious. Four or five doses of the mixture were given, and, when seen again during the evening, she had a soft quiet pulse, and was quite comfortable in other respects. The first urine we were able to obtain showed, upon examination, at least two-thirds of albumen; and, upon inquiry, we found that this secretion had been scanty for some months, and that there had been also considerable anasarca of the lower extremities. The patient made a tedious recovery, and it was fully six weeks before all trace of albuminuria disappeared. The pulse-rate and temperature remained abnormally high for some time, and at length it was necessary to wean the baby; after which the patient's progress was more rapid, and at the present time she is in better health than she has been for years.

Having given the history of this case, it will be well to relate by what train of reasoning I was led to select nitro-glycerine as an appropriate remedy. It was my first experience, during a period of more than twenty years, of a case of puerperal eclampsia; and, whilst watching it, I noticed how all the symptoms pointed to it as belonging to that class of convulsions which are identical with uræmic convulsions. There were the pulse of high tension, the oedematous and cold extremities, and the puffy condition of face and eyelids, which combined to show that an albuminous condition of the urine was present. In uræmic convulsions, I had seen benefit accrue from two remedies: first, from the inhalation of nitrite of amyl (such benefit being transitory, and lasting but a short time after withdrawing the remedy); and secondly from bleeding, the relief from which was permanent until a future attack.

Since the introduction of nitro-glycerine into practice as a remedy for angina pectoris, chiefly through the investigations of Drs. Brunton and Murrell, I have had considerable experience of its value as a means of reducing arterial tension, and of its power of keeping up this effect for a variable time. I therefore determined to use it when I should next meet with a case of uræmic eclampsia, and, as it were, bleed the patient into his own blood-vessels by dilating them. I believed my theory to be perfectly rational, and that it was so is, I think, fully exemplified by the success of the remedy in this particular instance; for the patient not only quickly regained perfect consciousness, but this was maintained without the least approach to a relapse.

There are several causes of puerperal convulsions, but the largest number of cases are the result of a high tension of the arteries, caused by their containing blood which has been imperfectly depurated, probably owing to pressure upon the renal arteries by the gravid uterus.

* Read at a meeting of the Isle of Wight District of the Southern Branch.

The next most frequent cause is sudden or exhaustive hæmorrhage. In both these cases, the convulsion is probably the result of an anæmic condition of the brain.

In looking through the literature of uræmic eclampsia, we find most observers agreeing that an anæmic condition of the brain is the cause of the symptom, and that with this we have œdema of the brain-tissues; but they differ as to the manner in which this state of things is produced. Dr. Hughlings Jackson suggests "spasm of the cerebral arteries" as a cause of this anæmia; whilst Dr. George Johnson, supported by the ophthalmoscopic observations of Dr. Gowers, advances the theory of a "stopcock action of the arterioles."

Traube, some years since, said that the nervous symptoms of Bright's disease (convulsions amongst the rest) were caused indirectly by cerebral anæmia. His idea was that the impoverished state of the blood, and the coincident cardiac hypertrophy, led to the exudation of serum through the coats of the minute vessels; that the capillaries and veins were pressed upon by this effusion; and that this pressure diminished the circulation of blood through the vessels. In some interesting papers on this subject, written by Dr. Mahomed, he adopted an entirely new theory to account for the production of the convulsions, viz., "the rupture of minute blood-vessels within the substance of the brain." My own impression is that both anæmia and œdema exist, nor is it difficult to see how this combination may be produced. The lessened calibre of the arterioles, owing to their conveying improperly depurated blood, will account for the starved condition of the brain; while serous exudation into, and between the membranes of the brain, is simply an extension of that same condition of things which we find in other parts of the body, as exemplified by the œdematous condition of the extremities and of the face and eyelids. If an anæmic state of the brain is sufficient to account for the convulsion, may not this serous effusion be the cause of the profound coma which usually accompanies this condition? The rapid and favourable action of nitro-glycerine in this case appears to me to favour the above theory, and rather to militate against the hypothesis propounded by Dr. Mahomed; for, if the cause of the convulsion were rupture of minute blood-vessels into the substance of the brain, the removal of the tension of the blood-vessels would not be likely to cause an immediate return to the normal condition.

In reviewing the results of treatment both of puerperal and of uræmic eclampsia during the past few years, the fact immediately strikes one that the successful cases have been treated with such remedies as have the property of dilating the blood-vessels, and thus freeing the circulation through the brain. The principal of these have been chloral in large doses, chloroform, opium, occasionally but not often successful; vapour-baths or wet packs; Simpson's baths; sinapisms and warmth to the lower extremities, combined with cold applications to the head; free purgation, and also bleeding. Of these remedies, the first and last have, in all probability, been more successful than all the others together.

Ergotine, also, was mentioned by more than one speaker in a discussion upon puerperal convulsions; but it is difficult to see how this could have been of benefit, and when its use was called in question by other members, all that they claimed for it was that it was serviceable in rapidly expelling the contents of the uterus.

In the treatment of one class of this affection, viz., the convulsions produced by excessive hæmorrhage, I might be induced to use this remedy; first, as a means of checking such hæmorrhage; and, secondly, in the hope that, by contracting the arterioles, a larger supply of blood might, perhaps, reach the brain, though I do not feel sure that so happy a result would be effected. I believe more would be done for such a case by warm affusions to the head, and by placing the upper portion of the body in the dependent position, which has often proved serviceable in the anæmia of brain produced by an over-dose of chloroform.

It may be long before I have a second opportunity of testing the value of this remedy in another similar case; therefore I have thought it advisable to place this one on record, not because I think a single case which has proved successful sufficient to establish the value of a remedy, but in order that others may, if they think proper, adopt what appears a rational, reliable, and safe treatment which is not open to the objections and dangers of chloral, chloroform, etc.

Since writing the foregoing paper, the following case has occurred in my practice, and as it has a distinct bearing on my other case, I think it advisable briefly to record it.

On January 2nd, I was called to see Mrs. J. W., aged 63. Her friends gave me the following account of this patient. For some months previously she had seemed in better health than usual, but had complained of shortness of breath when walking, and of occasional attacks of giddiness. The day before I was sent for, not feeling well,

she had taken an aperient dose, which had freely operated. The patient lived alone, and was an early riser. On the morning of my visit her son had gone as usual, to call her; but, after knocking at the front door, he could hear no movement within. Later on, he heard a fumbling at the bolts, and on pushing open the door, found his mother insensible on the floor. She was carried to bed, and was in the same condition when seen by me about 10 a.m. She could be roused, but not sufficiently to reply to questions. She was able to put out her tongue, and pointed to her head as being in pain. The pupils of her eyes were contracted irregularly. The extremities were warm, and there was no paralysis, though the symptoms were those of compression, but from what cause I was unable to judge. The pulse was quick, small, irregular, and hardly countable, being over 180 per minute. Digitaline was given, which reduced the pulse to 160, but did not improve its regularity.

January 3rd. The patient was worse. Pulse still 160. The temperature was normal. She could not be roused. The bowels had not acted since the 1st instant. During the day I was able to procure some urine for examination; it was scanty, and contained two-thirds of albumen. I, therefore, considered the case was one of uræmic coma, and gave a very unfavourable prognosis. One-minim doses of solution of nitro-glycerine were administered every four hours, and nothing but skimmed milk given as food. Her friends wishing to have Dr. Coghill's opinion, he saw her with me the following morning, and confirmed my diagnosis. The heart being weak and irregular (though stronger than on the previous day), he recommended ten minims of tincture of belladonna and ten grains of iodide of potassium to be given every four hours, and the nitro-glycerine with every alternate dose. The bowels had acted twice since commencing the nitro-glycerine, but a powder of calomel was to be given at night if required. At my night visit I found it necessary to stop the mixture, on account of its having produced unpleasant physiological results. The powder was not given, as several free actions of the bowels had taken place. The nitro-glycerine was ordered to be given every six hours.

On January 5th, the patient was improved; more conscious; she took milk freely. Pulse 120, stronger and more regular. The bowels were freely open. On January 6th, she was better in every respect. On January 7th, she was improved; pulse 108, stronger. She was ordered to take digitaline and arseniate of strychnine three times a day, in addition to the nitro-glycerine. The bowels acted several times a day freely, without medicine. On January 10th the patient had steadily improved, was quite conscious, and able to converse. The bowels not acting well, a dose of sulphate of soda was ordered to be taken every morning. The nitro-glycerine was given less frequently. Milk was freely taken. The secretion of urine was much greater, and the albumen was reduced to one-sixteenth. On January 13th, she had improved steadily, but was becoming tired of milk; she was allowed to have some fish. The nitro-glycerine was ordered to be taken every twelve hours only. The pulse was reduced to 84, steady and full. In the evening, whilst drinking a cup of milk, she suddenly fell backwards unconscious, and with complete paralysis of the right side. Neither medicines nor food could be taken, and the patient gradually sank, and died on the morning of the 16th, fourteen days after the first symptoms had appeared. The friends not permitting a *post mortem* examination, I was unable to find out if the second attack was but an extension of the first mischief, or the rupture of a blood-vessel. I rather incline to the latter view of the case.

One further remark I wish to make with regard to the power of nitro-glycerine in causing free evacuations of the bowels. I believe it to have been useful in the case I have mentioned, on this account, as well as in dilating the blood-vessels. That it has an aperient action in certain cases, I have verified in several instances.

CASE OF STRYCHNINE-POISONING: RECOVERY.

By R. COCKBURN, M.R.C.S.

Surgeon-General (retired) Bengal Medical Service.

THE article by Dr. Robert Barnes, in the BRITISH MEDICAL JOURNAL for April 1st, on "The Antidotes for Strychnine," reminds me of a case of accidental poisoning by strychnine, which came under my care several years ago, while I was Civil Surgeon of Benares in the Bengal Presidency; and the publication of the following notes of the case may prove interesting, affording, as they do, additional proof to that supplied by Dr. Barnes' case, of the possibility of recovering from a poisonous dose of the drug, if treated on the lines indicated by him. Knowing of no antidote that would "destroy or neutralise the poison in the stomach, or in the system," I adopted the only means, which occurred to me at the time, for counteracting the effects of the poison (these being in accordance with the first and third indications for treatment