below upwards: that is, from the part first affected to that last). There was left in the cornea an universal thick projecting opacity. The conjunctivæ were still injected and insensible. The masticatory muscles seemed to be regaining their power. The inferior maxilla could now be moved to a slight extent towards the right side, showing some return of power in the pterygoids. The abrasions on the buccal mucous membrane were healing, as also the sponginess of the gums. *Hearing was improved.*

The patient took her discharge on May 9th, contrary to the advice of the medical officer, and much to my annoyance. I think it by no means improbable, had she remained in hospital, that, under the continued use of iodide of potassium, proper hygienic means, and good unstimulating diet, she would have improved still more, if not have been cured altogether; but I fear that, returning to her old habits—and those, I am inclined to think, neither very temperate nor moral—her symptoms will retrograde, and possibly at some future date she may be admitted to some hospital in a worse condition than at first.

REMARKS.—The case I have just related is one of very great interest, and by no means of common occurrence. We could not have a more complete and accurate exposition of the functions and distribution of the trigeminal nerve, as proved by the results of experimental physiology and clinical observation. It shows, in a remarkably clear manner, the influence this nerve exerts, directly or indirectly, over several of the special senses, viz., of vision, hearing, taste, and smell, all these being more or less implicated.

As far as my researches have extended into the literature of the subject, I have been able to find but very few undoubted cases of lesion of both portions of this nerve, uncomplicated by symptoms of encephalic disease, or the implication of other nerves.

When we consider the symptoms of the present case, and bring to bear on them the anatomical distribution of the trigeminal, the evidence derivable from experimental physiology, from pathological research, and from careful clinical observation of the various authorities I have quoted above, there remains no manner of doubt concerning the nerve affected, and the absolute localisation of the lesion to it alone.

On the first glance I had of the patient, from the mask-like appearance of the left side of the face, I was inclined to regard the case as one of "Bell's paralysis", but when I observed the condition of the cornea and examined the state of sensibility of the face, etc., the delusion was dispelled, and there was no difficulty in assigning the lesion to its proper category. On further investigation, it became quite clear that not only the sensory branches were paralysed, but also the motor portion shared a like fate. The lesion (whatever may be its nature) which produced the symptoms, I imagine to be situated somewhere along the course of the nerve between the pons and the ganglion of Gasser, or possibly involving this structure. It implicates the motor and the sensory roots; and on this account I am inclined to place it somewhere along the course of the nerve between the pons and the ganglion. The fact of the symptoms being confined to this nerve goes far towards excluding the supposition of a central pathological change causing them. A lesion situated in the medulla oblongata, the floor of the fourth ventricle, the crus cerebelli, or in the pons Varolii, would produce symptoms, graver, more diffused, and involving the functions of other nerves, except, indeed, it were strictly limited to the nuclei of this particular nerve, or confined accurately to some part of its course, before its exit from the encephalon. It is difficult to imagine a central lesion of such very limited extent as not to produce other symptoms as evidence of its existence. Caries, or some other diseased condition of that portion of the base of the skull in which are situated the foramina through which the several branches pass, may exist, so as to constrict or otherwise disorganise the nervous trunks, and thus produce the paralysis. I am inclined to think that the cause is a syphiloma or other small tumour, probably situated at that part of the dura mater pierced by the trunks before they reach the summit of the petrous portion of the temporal bone, or else some inflammatory process or other degeneration of the neurilemma behind the ganglion, or surrounding this body. Taking into account the general improvement of the patient's condition under the use of iodide of potassium, and the absence of symptoms of paralytic affections of other nerves, I am certainly in favour of the syphilitic theory.

It will have been noticed that the eye was the first to indicate the occurrence of disease within the cranium. This is probably owing to the more acute sensibility and less power of resistance to external irritants of this organ than the other parts to which the trifacial is distributed, thus acting, as it were, as a very delicate test for prognosticating the future of a given case, but one by no means applicable to every case, and difficult of a direct interpretation. However, if in any case of sloughing cornea, the common symptoms of this affection, viz., pain, lachrymation, intense sensibility of the conjunctiva (both ocular and

palpebral), photophobia, etc., were replaced by absence of pain, dryness of the eyeball, anæsthesia, and tolerance of light, I would say, in nautical phraseology, "Keep your weather eye open", and "look out for squalls" in the direction of the area of distribution of the trigeminal. Bearing on this part of our subject, Marshall Hall observes (*Lectures on the Nervous System and its Diseases*, 1836, p. 31): "If the sensation of the face be lost by paralysis, arising from disease of the brain, the eye is safe; but if the same event occur from compression or destruction of the fifth (nerve) within the cranium, by disease, or in an experiment, the eye ceases to be nourished, and becomes destroyed! In the former case, the nerve of sensation only has suffered; in the latter, the nerve of nutrition, as well as sensation, has been involved in the disease or injury."

An interesting symptom is the deafness on the affected side, and one which may possibly be explained when we have regard to the part the trifacial nerve plays in the special sense of hearing. The tensor tympani muscle derives its nervous supply through the lesser petrosal nerve—a twig from the otic ganglion. Now, the action of this muscle, as its name implies, is to render tense the tympanic membrane, "thus enabling it to vibrate in reciprocation to sounds having a great variety of fundamental notes. It appears to be antagonised by the stapedius, the contraction of which seems to diminish the tension of the membrana tympani, and to take off pressure from the fluid of the labyrinth.

In the present instance, the tensor tympani must be paralysed, and, in consequence, the sound-waves fall upon a lax insufficiently vibrating membrane. This laxity is doubtless further increased by the action of the stapedius muscle, which is antagonistic; and being in full and unopposed working order, thus contributes in no small degree to the loss of equilibration. The accumulation of mucus in the tympanic cavity may also assist, to no inconsiderable extent, to blunt the sense of hearing; such accumulation being favoured by the paralysis of the circumflexus or tensor palati muscle, whose nervous supply is derived from the otic ganglion.

The loss of power in the muscles of mastication was a condition which could not be mistaken; this perhaps was more evident in the case of the pterygoids than of the masseter and temporal, because its effects were more manifest. In testing the action of the muscles, I had the kind assistance of Dr. Pollard, who manifested much interest in the case, and corroborated my observations on the other paralytic phenomena.

The fixed and mask-like appearance of the affected side at first was calculated to make one believe that there was paresis of the facial nerve; but, on further investigation, the action of the facial muscles was found to be intact. This, as well as the depression of the angle of the mouth, I think, can be satisfactorily explained by the partial paralysis of the buccinator (the muscle being supplied by the fifth and facial nerves), and the falling of the jaw from want of support of the masticatory muscles. This condition is graphically described by Dr. Althaus, in his case of bilateral paralysis of this nerve quoted above. In a former part of this paper, I have drawn attention to the experiment of Bell on an ass, in which, after section of this nerve, the jaw "fell relaxed and powerless". Thus I hope I have proved that a "fixed and mask-like appearance" of the face may exist, in some cases at least, without any lesion whatever of the portio dura.

P U E R P E R A L S C A R L A T I N A.

By V. STONE, F.R.C.S. Ed., Laurence Kirk.

ON May 9th last, Mrs. A. B., primipara, a member of my own household, was delivered of a male child at 9 A.M. The forceps was used, as the head was inclined to stick at the bony outlet; otherwise the labour was natural. The patient went on well till about 5 P.M. on the following day, when she became sick, and vomited, and felt very ill. When I came in about 8 P.M. (having left her perfectly well in the afternoon), I found her pulse 120, and skin burning hot; she complained of severe headache; no other symptoms. I washed out the vagina and uterus freely with diluted Condyl's fluid (having carefully examined for any bit of membrane, or anything likely to give rise to irritation, and failed to find anything), and repeated the washing every four hours. The patient passed a restless night, and next morning complained of sore throat. In the full daylight, I discovered an eruption on the neck and chest, the face being very red. I at once suspected scarlatina, as it was epidemic at the time, and I had been attending several cases. I requested my friend Dr. Johnston of Fordoun to see the case, and he, without being told of my suspicion or of the existence of an epidemic, expressed his opinion that it would turn out to be scarlatina. The patient was given

regularly every four hours a tablespoonful of liquor acetatis ammoniae with five grains of carbonate of ammonia added, and had champagne, with or without soda-water, at the rate of a bottle every twenty-four hours. I may mention that this treatment and the washing out the vagina and uterus were continued throughout.

Next day, the skin was completely covered with the eruption, the tongue taking on the typical strawberry appearance, and the fauces being very much inflamed and painful. The pulse remained about 120, and the temperature ranged from 101 deg. to 104 deg.

Altogether, the case was one of pure unmixt scarlatina. I say pure, for from first to last there was not a single local puerperal symptom. On the third and fourth nights of the fever, there were some delirium and restlessness, but not more than one sees in even moderately mild cases of scarlatina. The case went on well; desquamation began on the eighth day, and was completed before the end of a month. The patient sat up in a chair in a fortnight from her delivery, and was out-of-doors in a month.

Now here is a case in which a sharp attack of undoubted scarlatina, setting in thirty-two hours after delivery, does not modify in any way the puerperal condition. There never was the slightest abdominal tenderness, or tympanites, or suppression of the lochial discharge; the single exception being that no milk was secreted. I may mention that there was a slight laceration of the perinaeum, which would of course have been favourable to the development of septicæmia. This case disposes satisfactorily of the proposition that scarlatina is necessarily transformed into malignant puerperal fever when it attacks a recently delivered woman.

On the other hand, several years ago, I had a case of which I kept notes, and of which I shall give a summary. Mrs. C. D., the wife of a labourer, was easily and safely delivered of her sixth child while three of her children were suffering from scarlet fever, which was epidemic at the time, almost every house in the neighbourhood being infected. The cases of the children were mild, and the mother had no anxiety about them, or alarm about herself, though she had never had fever. She went on well for two days; but on the third, complained of abdominal tenderness and tympanites; her pulse rose to 140; the lochial discharge was suppressed; the strength rapidly sank; and, on the sixth day from her delivery, the patient died of what might well be called puerperal fever. When the case first went wrong, I looked most carefully for some specific symptom of scarlatina, but could find none; and from day to day, and several times a day, I examined the skin, throat, and tongue, but not a single feature of scarlatina could I detect. I had examined and washed out the uterus and vagina at first, and afterwards, but found nothing to account for the local mischief.

Of course, one cannot say a great deal from a case like this; but it is my decided opinion that this, too, was a case of scarlatina, though there were no visible symptoms of it. I think the explanation is not very difficult. There can be no doubt that scarlatina does in most cases give rise to a set of symptoms and produce effects which are identical with those seen in puerperal fever; and the very intensity of this condition is sufficient to swamp the special features of the scarlatina itself, which would otherwise be visible. This is more especially the case when the patient dies early. This production of local symptoms is not to be wondered at when the extreme susceptibility of the recently emptied uterus to any poison, whether communicated by the blood or otherwise, is considered. A case of scarlatina may be so slight of itself as to elude detection. We have all seen cases where a member of a family, some of which have suffered, or are suffering, from fever, is not very well, but we cannot pronounce his to be a case of scarlatina till we find that he afterwards desquamates. Cases like these, occurring in the puerperal condition, might easily produce fatal results and their real nature escape detection.

In connection with this, I may mention one which has a certain bearing on the subject. A little girl, of a particularly healthy constitution, slightly nipped the fleshy part of her arm in shutting a door, and beyond a little pain at the moment suffered no injury. Two days afterwards, she was taken ill of a very mild attack of scarlatina. On the second day of the fever, the arm, which was considered quite well, became very much inflamed, and a large abscess rapidly formed. The little patient suffered severely from this, both locally and constitutionally, but I may say not at all from the fever. If I had not been in attendance on another member of the family suffering from fever at the time, I should probably not have been asked to see the patient till the arm was inflamed; and it is probable I might have overlooked the scarlatina, and put the whole down to the injury of the arm.

I think there can be no doubt that in some cases of puerperal scarlatina, the specific symptoms of the disease may be so suppressed as to escape notice, even on careful examination; but these on that ac-

count are not to be classed as malignant puerperal fever. It would be interesting and go a great way to clear up this matter, if some one who has large opportunities would bring forward some cases—if such there be—where women who have, within, say, a few years, had undoubted scarlatina have had malignant puerperal fever conveyed to them by scarlatinal infection. For my part, I should have no fear of so conveying it, any more than I should of conveying scarlatina to a non-puerperal patient from one suffering from malignant puerperal fever.

CASE OF ATROPINE-POISONING.

By A. S. GREENWAY, M.B.,
Assistant Medical Officer, Fulham Hospital.

CHARLES S., aged 45, a gardener, was brought to me about 1.25 P.M. on August 6th, 1878, said to have swallowed at least a teaspoonful of liquor atropiæ sulphatis (B. P.). He told me that he was attending an ophthalmic hospital for an affection of the eye, and received some drops and a larger bottle for internal use. He was in the habit of pouring out his daily allowance of the latter into an ounce-and-a-half phial, and carrying it in the same pocket with the atropine-bottle. About 1 P.M. (dinner-time), he left work, and, when he took his medicine, by mistake he drank from the wrong bottle, although the atropine-bottle was fluted, etc.—in fact, a regular poison-bottle. He felt nothing particular, but told the clerk who brought him home what he had done. He was then quite sensible; there were no symptoms or signs whatever except excessive nervousness. The left pupil was dilated from a previous application of the atropine. I gave him an emetic of ipecacuanha wine; and, it not acting quickly enough, I gave twenty grains of sulphate of zinc in mustard and water, and told him to walk about. At 1.39 P.M., his pulse was 140, small and regular; temperature 98 deg. He staggered as he walked, and complained of giddiness. The pupils were not affected. No sickness supervening, I used the stomach-pump and washed out the stomach. He lost power over his legs, and became quite helpless and unconscious; his pulse became excessively weak and irregular; and I gave him a drachm of tincture of digitalis in some brandy. At 2.15, he commenced to talk quickly, and looked flushed. At 2.45, the pulse was 148, full; and the skin dry. The face was very much flushed. He had very violent delirium, requiring three men to hold him. The pupils had gradually enlarged till they were nearly fully dilated. The tongue was dry and brown; he could swallow after some compulsion. Vomiting continued at intervals, and black coffee was given him now and then. His feet became cold; mustard was applied to them. At 3.15 P.M., the pulse was 156, smaller; temperature 99 deg. He was quieter, and had a tendency to sleep. The pupils were the same, and insensible to light. He did not retract his legs when they were pricked or pinched. When spoken to, he did not seem to hear, being evidently quite unconscious. At 4 P.M., the pulse was 144. He seemed generally the same, except that, when his name was shouted in his ear, he opened his eyes and turned his head in the direction of the sound. For the first time, the respiration seemed labouring and quick, accompanied by flapping of the cheeks when he was permitted to sleep. He now moved both arms and legs at times, and twitching of some of the muscles was noticed. He had a mustard poultice over his cardiac region, and small quantities of brandy were administered. An enema of castor-oil was given. At 7 P.M., the pulse was 140; respirations 40 per minute. He was more conscious. The tongue was the same, the face less flushed, and the skin still quite dry. He was decidedly more conscious, as, when told to put out his tongue, he put it out, and said "Yes" when called. He caught at imaginary objects. On account of his precarious state, I asked Dr. Milner Fothergill to see him, which he very kindly did, and advised me to continue the line of treatment, as he was recovering.

Dr. Fothergill spoke to him, and he answered him fairly intelligibly. At 9.15 P.M., the pulse was 140, small and regular; temperature 102 deg.; respiration was 50, labouring. On examining cursorily the chest, I found a few moist râles about the bases. He recognised some friends who came to see him. The skin and tongue were in the same state as said in last report. As soon as he fell asleep, he commenced to talk unintelligibly. The pupils were the same. Neither bowels nor bladder had acted. At 10.35 P.M., the pulse was 140, full and regular; temperature 102.4 deg.; respirations 60. The bowels had acted slightly. He was inclined to become violently delirious again, as Dr. Fothergill had foretold. Generally, he was in the same state as at the last report. He had been having eggs, coffee, milk, etc., with a little brandy at intervals. At 11.45, I passed the catheter; but the bladder was empty, and there was no dulness over the pubes. He had considerable tympanites, and tenderness over the abdomen.