A LESSON OF THE WAR: SUPPURATIVE MIDDLE-EAR DISEASE.

A PLEA FOR TREATMENT IN ITS EARLIEST STAGES.

BY

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OUT of 5,000 odd cases under my care at the Throat, Nose, and Ear Department of the Military Hospital, Tidworth, during the year ending October 31st, 1917, no fewer than 890 were suffering from chronic suppurative ear disease—practically a battalion; the period of their disability ranged from a few months to many years, or in the patient's words, "as long as I can remember." A large majority of these cases were only fit for Category C, as it was at that time, whatever some people might say to the contrary. They all suffered from deafness, more or less, and were not fit for the firing line; most of them were in very indifferent health, were anaemic from prolonged suppuration, and consequently a fitting soil for every microbic disease.

A large number were most of their time in the medical officer's hands for treatment, a treatment of syringing and drops, which was only marking time, for little short of a mastoid operation would cure such cases, and space and time would not permit this.

The greatest number of those cases, if not all, would have been "A" men, as far as their ears were concerned, had systematic and energetic treatment been carried out in the earliest days of the onset of the disease. Chronic suppurative ear cases could be enormously reduced if we adopted systematic prophylactic methods and did not wait until the disease was far advanced.

Every fever hospital should have an otologist attached, or at least a medical officer in charge who has a fair knowledge of ear diseases, and who would always be on the alert for its early onset, for these institutions are a fruitful source of suppurating ears, and, what is more, these cases can act as carriers of a specific fever, thus becoming a danger to others coming in contact with them. As bearing on this the following will suffice:

A patient was admitted to a ward in a military hospital of which I had charge in 1915, suffering from slight fever and a profuse discharge from the left ear. Though at the time of admission he had no throat trouble, there was a history of such some three weeks previously. The discharge being profuse and not abated by treatment, a swab from the ear was examined and Klebs-Loeffler bacilli were found in large numbers. The case was promptly isolated. Two days later the patients in the beds on either side of this case developed diphtheria. A day later I myself was a victim to the same disease. No doubt the throat trouble which the ear case had had three weeks before admission had been diphtheria, but probably on account of slight physical local signs had been overlooked, and the disease had in the meantime spread to the ear.

It is in the earliest stages of any disease we have the best chance of obtaining permanent cure; I have invariably admitted to hospital every case of acute otitis media, and every case of suppurating ear when the disease had lasted less than two months, with the certainty of effecting a cure by careful and energetic treatment. Speaking generally, cases beyond this period cannot be so favourably regarded; also they are too numerous to be treated as in-patients, and in the majority permanent damage, more or less, has already been done to the structures in the middle ear, with consequent loss in the hearing power.

During the period mentioned there were 54 cases of acute or subacute otitis media, all of whom left hospital with the discharge ceased, the rent in the drumhead healed, and the hearing restored. Our object must be to gain at once the upper hand in the early stage, and to restore the middle ear to a healthy state before serious changes take place, such as involvement of the antrum and mastoid cells, lateral sinus, or meninges. Even should the patient escape these, there is always, if the suppuration continue for an extended time and finally dry up with the perforation in the drum healed, marked fibrous change in the lining membrane of the middle ear, which must impair the hearing, varying from absolute deafness to loss of the acuteness of hearing.

The pathology of acute middle-ear disease is similar to that of disease of other mucous membranes: dilatation of the vessels, with swelling and lessening the lumen of the

cavity. The starting point is invariably the posterior nares, and oftener than not follows the acute febrile diseases—scarlet fever, measles, mumps, influenza, etc. Among the predisposing causes the most important are adenoids, enlarged tonsils, and nasal disease. Inflammation or catarrh in the neighbourhood of the Eustachian tube easily spreads up the tube to the middle ear, and, should its lining membrane become thickened by inflammation, its walls soon come together and block the lumen. The middle ear then becomes an enclosed cavity, with no outlet for the serous fluid thrown out by the engorged vessels, and the patient then experiences fullness in the ear and deafness. The drum membrane has lost its lustre, and in all probability is indrawn, for part of the air in the middle ear has been absorbed, and the external air pressure is greater than that within. If there is much fluid in the middle ear there will be bulging in the posterior quadrant of the drum, and should the dam in the Eustachian tube be allowed to continue many things may happen: the inflammation may slowly resolve, but often it does not, and unless a knife is put into the bulging membrane and politizerization carried out the serous exudation in the middle ear becomes septic, and an abscess forms. The drum becomes intensely red and bulging, and the patient is in great pain until relieved by the abscess bursting into the external auditory canal, leaving a small jagged opening in the membrane. In some cases following the acute infectious diseases, such as scarlet fever, the inflammation spreads so rapidly that the antrum and mastoid cells are acutely attacked before the abscess can burrow through the drum, and an acute mastoid abscess develops, or the inflammation may spread to the meninges or lateral sinus, with, naturally, very grave consequences. It is therefore advisable in all these cases to incise the bulging drum early and so establish drainage as soon as it is certain that the fluid in the middle ear is not subsiding.

Every case of acute middle-ear inflammation should be put to bed and given a brisk calomel purge. If there is any nose or throat trouble this should have appropriate attention. Should the drum membrane be bulging, the external auditory canal should be thoroughly disinfected by swabbing with 1 in 4,000 flavine or 1 in 20 carbolic, and the membrane freely incised under general anaesthesia; the incision should be made in the posterior quadrant just behind the handle of the malleus and running crescentically down to below the tip. Gentle use of the Politzer bag will open the Eustachian tube and force the fluid out of the middle ear. This fluid is then mopped out of the canal; a pledget of cotton-wool soaked in flavine is placed close up to the drum, but must cause no pressure. It should be changed every few hours. I have sometimes plugged the canal lightly with wool soaked in 50 per cent. rectified spirit, which can be increased in strength if it causes no excoriation. At each dressing politizerization should be carried out or the fluid extracted from the middle ear by gentle suction.

For the pain, which is sometimes very acute, glycerin of carbolic, 10 grains to 1 oz., is useful; a few drops warmed in a teaspoon and instilled into the ear, or a few drops of chloroform, will give relief; hot fomentation over the mastoid and auricle is always very soothing to the patient. There is always some post-nasal catarrh, and a warm inhalation of compound tincture of benzoin every two hours will relieve the congestion of the lining of the mouth of the Eustachian tube and help the necessary drainage.

Pain in the ear should never be passed over. It may only have its origin in a carious molar tooth or hard cerumen in the external auditory canal, but it is Nature's danger signal, and should ever be regarded.

The object of this paper is to draw attention to the large number of cases of chronic suppurative ear in England to-day, and to urge that everything in our power should be done to lessen it. A man with ear disease loses from 10 to 70 per cent. value in the labour world.

A large and increasing number of cases is now coming under my notice originating in rupture of the drum by high explosives and secondary infection of the middle ear, which, for lack of continuous and expert treatment, have become chronic. The drum is torn by the terrific concussions of high explosives, and though the man may escape all other injury he is so dazed that he does not notice he is deaf, and possibly has some slight bleeding from one or other ear. A day or so later there is pain in the ear and

a slight muco-purulent discharge. In other cases there was no pain in the ear and only a slight discharge, and the men, thinking it only trivial, did not complain until it became profuse and was accompanied by marked deafness.

During the last twelve months I have examined some hundreds of cases of deafness in varying degree for pension purposes, and I now, as never before, realize at what a disadvantage a man with even a partial loss of hearing is. There are many trades in which good hearing is indispensable. The miner has again and again told me he can no longer get work down the pit on account of his loss of hearing, because he may fail to hear sounds in the pit which portend disaster or death to himself or his comrades. The same applies to many other trades, and the man has to seek other work, each succeeding class of work is more precarious for him, and he is finally driven to casual labour. The poorer classes take but casual notice of the condition of the ear, and this applies especially to suppurative otitis media. Pain is not a frequent concomitant, otherwise they would oftener seek advice. I cannot do better than quote Dr. Mygind of Copenhagen in his book on deaf-mutism. He writes: "It is to be hoped that the recognition which is by degrees, though slowly, being yielded to otology by the medical profession, will make itself felt in the prevention of deaf-mutism, by opening the eyes of the practitioner to the importance of ear diseases and their treatment, and also that the general public may be led to form other opinions upon the subject than those now prevalent."

If by propaganda the poor and so-called working classes could be taught to look upon their hearing as precious, and realize that a "running ear" may lead to the loss of that power and calls at the earliest onset for advice, the efficiency of the nation in general would be increased.

STRANGULATED UMBILICAL HERNIA :

RESECTION OF GANGRENOUS ILEUM AT THE AGE OF 69.

BY

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SUCCESSFUL resection of gangrenous gut in inguinal and femoral herniae are by no means rare, but in umbilical herniae successful resection is less common. The following case is thought to be of interest because success was attained in spite of the patient's age.

M. P., a widow aged 69, was admitted to the South Devon and East Cornwall Hospital on the afternoon of January 21st, 1920, complaining of severe abdominal pain and vomiting.

Her history was difficult to elicit, and was probably inaccurate. So far as could be discovered she had had an umbilical hernia for many years. She stated that she had had pain in the hernia for more than a week, associated with vomiting. During this time the bowels had been very slightly opened after aperients, and for three or four days had not moved at all. She did not call a doctor until the day of admission, and he at once sent her to hospital.

On admission she looked ill, and senile beyond her years. The pulse was 94 but weak, the temperature 98°. At the umbilicus was a tense tender red—almost purple—swelling about the size of a cricket ball. There was no impulse on coughing. The rest of the abdomen was somewhat distended, but not tender. The vomit was bile-stained and foul, but hardly "faecal."

Under a chloroform and ether mixture the hernia was surrounded by a transverse elliptical incision, which was deepened to the rectus sheath. The hernia was then lifted off the rectus sheath until the neck was exposed all round. The abdomen was then opened just clear of the neck of the hernia. The hernia was then isolated by extending this incision completely round, but just clear of, the neck. The abdomen was then packed off, and the sac opened by splitting up the neck from what had been its peritoneal aspect. The sac contained a quantity of very foul, almost black, purulent fluid, a mass of adherent and gangrenous omentum, which was ligatured off and removed with the sac, and about four inches of gangrenous ileum. The centre of this length of small gut was of very doubtful viability, but at either end, where it had been nipped, it was reduced to little more than a slough. In order to leave a good margin on either side of the damaged gut about eight or nine inches of gut was resected. Continuity was restored by end to end anastomosis, the collapsed distal portion of the gut being split along its anti-mesenteric border so as to give a sufficient lumen for an anastomosis to the proximal distended gut.

The abdomen was closed in layers by mattress sutures placed transversely (modified Mayo's method). A rubber drain was placed at either angle of the wound, as the subcutaneous fat was considered likely to be infected.

On the day following operation the temperature was 97.4°, the pulse 80, and respirations 22. There was considerable flatulence. The bowels were opened by a turpentine enema on the following day. Thereafter convalescence was smooth but for trifling superficial sepsis necessitating removal of a couple of skin sutures.

The patient left hospital for a convalescent home on February 24th, 1920. She was then in her normal state of health.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SHINGLES AND CHICKEN-POX.

ALMOST immediately after reading Dr. Taylor's neurological jottings in your issue of February 28th, a girl of 16 came to my surgery for some medicine for her father, who had developed shingles about a fortnight before. She asked me about a "rash" which had appeared on her body on the previous day. I found that she was in an early stage of chicken-pox, which subsequently developed, with a temperature of 102°, into a good crop of vesicles. There is no other case of chicken-pox in the neighbourhood. Her father is an instructor in gardening at a boys' home, but there have been no cases amongst the boys, probably because of the work being entirely in the open air.

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ACUTE OEDEMA OF THE LUNGS.

I HAVE been much interested in the correspondence on this subject, as, with Dr. Stewart McNaughton (February 28th, p. 293) I believe that the disease is more common than is usually thought. In the autumn of 1897 I reported a case of dramatic severity in the *Lancet*.

The victim was a man aged about 50, who was seized with respiratory distress after a bicycle ride against a cold head wind. He died within an hour of his first symptoms, and for the last few minutes of his life poured out pints of bright pink foam. Auscultation revealed the presence of fine crepitant râles, commencing at the bases and very rapidly spreading upwards. There was a history of some previous renal mischief.

Within a few months I saw a woman of much the same age who fell ill suddenly after cleaning out a flooded basement. Her condition was less acute. The expectoration was aerated, bright pink, but not so great in quantity, nor so frothy. She lived a few hours.

Again, a man of 60 was placed under my care with a story of recurring suffocative attacks at night. He had aortic regurgitation. The urine was of low gravity, and contained a trace of albumin. During a period of perhaps two months he called me some half-dozen times, always a little before midnight. He was invariably in great distress, standing with both hands grasping the bed rail. The sputum was more or less frothy and coloured red. A hypodermic injection of atropine would relieve him at once, allow him to go back to bed, and in the morning he would be quite recovered. The fine râle was very evident in this case, and the rapidity of the upward extension remarkable. His last attack was more severe and I was longer in reaching him. This time atropine failed to check the secretion, and he died expelling much frothy sputum.

Two other cases of recurrent oedema occur to me. One in a woman of over 70, suffering from mitral regurgitation; the other in a woman of 60, suffering from myocardial degeneration. In each instance the crises were spread over a period of two or three years at varying intervals of months. In each there was the great distress, the rapidly ascending râle, and the tinged aerated sputum. Atropine acted well in both and promptly relieved the condition.

The older patient eventually, after her cardiac condition had badly deteriorated, died in an attack, in spite of the usual dose. The other died of gradual heart failure and generalized oedema. The last time I saw the condition was in the following case:

A man of 46. He suffered from extreme arterial high tension, and gave a history of a former but milder bout.

I was called in the night to find him intensely dyspnoeic, unable to move or lie down. There was the usual type of sputum; but for the first time in my experience the râles were the most abundant at the upper part of the lungs. A dose of atropine relieved at once and allowed him to return to bed. In the morning he was quite well.

There are differences in all these cases, but they clearly belong to one group. It is noticeable that where a history