THE EFFECTS OF HIGH EXPLOSIVES ON THE EAR.*

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ON HEARING.

THIS title is to be preferred to "deafness due to shell shock" for several reasons. The word "shock" has a varied significance. To the neurologist it conveys many ideas; thus von Monakow distinguishes several varieties of shock—surgical shock, psychical shock, apoplectic shock, commotio cerebri, diaschisis. Even in its best recognized form, surgical shock, the physiological mechanism which has been interfered with to produce the symptoms is unknown. The title chosen will, I hope, do away with much ambiguity.

In considering deafness due to high explosive, one should, as far as possible, dismiss from the mind preconceived ideas. Not only should one avoid the term "shock," but also one should avoid as far as possible the terms "hysteria" and "neurasthenia." To get some idea of what has happened the subject ought to be considered with an open and a critical mind.

THE RELATION OF THE EAR TO PRESSURE.

The organ we call the ear, like all peripheral organs, is a mechanism adapted to transform one form of external energy into nerve impulses. There are two separate and distinct mechanisms in the internal ear, one concerned with hearing, one concerned with equilibrium. Each of these mechanisms is adjusted and made sensitive to register minute pressures and transform them into nerve impulses. The nerve impulses are carried to the central nervous system to be there interpreted and utilized. In hearing, air vibrations (varying, say, from thirty-two double vibrations per second up to several thousand double vibrations per second) in various combinations are transmitted normally through the external auditory meatus to the drum membrane, which is finely swung to catch them. The vibrations are then transmitted through the ossicles and the middle ear to the cochlea, where they are transformed into nerve impulses. Nerve impulses may also be set up in the cochlea by vibrations through bone. The nerve impulses are conveyed along the cochlear nerve and acoustic path to the temporal lobe, where they enter in one definite bundle to be again redistributed to various parts of the cerebrum. The route from the periphery to the cortex is not one long undivided path. To put it roughly, there is not one telephone wire from the ear to the receiving and interpreting station in the cortex. The path is broken at various synapses or junctions or telephone exchanges. At these synapses connexion is made with other nerve paths, and communication can be and is established with other physiological systems. The significance of this I shall refer to later.

The other pressure mechanism in the internal ear is a system of canals containing fluid, a manometer, so designed as to register and signal to the central nervous system movements of the head. This mechanism is adjusted to indicate very small varying pressures. The information so obtained, together with information received from other sources (the eyes, joints, etc.), enables the muscular mechanism to adapt itself rapidly to varying alterations of our centre of gravity—for instance, to maintain the erect posture during movements of the head.

There are certain limits of pressure normal to these two mechanisms. Pressure beyond the normal produces disturbances which are pathological. Disturbances of hearing may cause hyperacusis, hypoacusis, or total deafness. Disturbance of the canalicular system produce disturbances of equilibrium—for instance, vertigo and nystagmus.

The effect of the high explosive is a great compression, amounting in some cases to ten tons to the square yard (10,000 kilos per square metre), followed by an equally great decompression.¹ The rôle played by each of these I do not intend to discuss.

The effect of the explosion can be best expressed by referring to a few typical cases. Cases of gradual diminu-

tion of perception of sound from long exposure to gunfire do not come within this paper. We are here concerned chiefly with the results of one severe concussion, followed by total or diminished loss of hearing. In some of the cases a previous concussion may have damaged the hearing, and the second made it worse, or resulted in total loss.

Classification of Cases.

The cases that are admitted to the hospital suffering from "nerve deafness due to shell concussion" can be divided into three groups:

1. Those with nerve deafness.
2. Those who have had nerve deafness of a varying degree, and who have still the fixed idea that they cannot hear.
3. Malingerers. Some undoubtedly have had shell concussion which has affected the hearing, but have now recovered; others are malingerers pure and simple.

It is a difficult matter to separate the second from the third. Experience, the general condition of the patient, his answers to the test questions, largely aid one. The following may be given as an example of Group 2.

Pte. De S. Severely wounded in head by bullet on May 28th, 1916; unconscious for several hours; when recovered consciousness was deaf in right ear. Admitted October 1st because of deafness. Has a depression (5.2 cm. by 1.5 cm.) in the right parietal bone near interparietal suture through which pulsation of brain can be seen. Right membrana tympani slightly retracted, otherwise normal; no hearing for voice or forks. Left membrana tympani normal; hearing for voice and forks normal. No paralyses, no anaesthesia, no astereognosis, no tremor, no Romberg.

Eyes.—Movements normal; fields normal; fundi normal; vision in both eyes.

October 6th.—Caloric test (cold) to right ear; immediately after heard voices in right at room length (7 yards); whisper 20 inches.

It is with the first group that this paper is concerned. To the second group belong those cases that yield to suggestion or recover hearing rapidly, often completely, after excitement or intense stimulation. To discuss the third group and how the malingerer can be detected is outside the limits of this paper.

NERVE DEAFNESS.

The cases of nerve deafness (Group 1) which have come under observation, may roughly be divided as follows:

I. Cases of nerve deafness associated with damage to the conducting mechanism.

II. Cases of nerve deafness without any visible or demonstrable lesion in the conducting mechanism when they came under observation. This does not preclude the occurrence of a demonstrable lesion at the time of the concussion; in fact, the history of many of these cases shows that this did occur.

III. Cases of nerve deafness in which there has been destruction of the cochlea and of the semicircular canals or their nerves.

In each of the first two groups the loss may be partial or total; the third always causes total loss.

It may be said in passing that the otologist has well recognized tests at his disposal which enable him to differentiate injury to the conducting mechanism from injury to the nerve mechanism. There is, however, great difficulty in saying how much of the loss is due to one, how much is due to the other.

The cases of disturbance of hearing following exposure to high explosives seen by me in West Cliff during the last two months date their onset back anywhere from eleven months to two months. We are only now receiving cases of more recent date. The average time from the date of the injury of those now under review is about three months. All have been under treatment since the injury, many in the care of competent otologists.

The symptoms associated with loss of hearing from high explosives fall within that group of nerve diseases called traumatic neuroses. In many of our cases there is a recognizable physical trauma in the head or elsewhere received during a period of mental excitement. It is well recognized that "traumatic neuroses" are specially apt to occur under such conditions. The symptoms are varied and complex. There are present, for instance, exaggeration of tendon reflexes, tremors, vasomotor disturbance, sweatings, lethargy, sleeplessness, and headaches. There

* Read before the Medical Society of the C.A.M.C. at Shorncliffe, October, 1916.

is unsteady equilibrium with vertigo. There is a concentric narrowing of the field of vision. In many of our cases fields of anaesthesia were present. In two of our cases of total deafness there was complete anaesthesia and loss of thermal sense. In one with total loss in one ear and great diminution in the other there was anaesthesia on one half of the body and hyperaesthesia on the other. Speaking generally, one may say that there are here included some symptoms which are frequently grouped under the term "hysteria," others under the term "neurasthenia." According to Dejerine, in traumatic hystero-neurasthenia the patients pretty often have hyperacusis with subjective sounds as in neurasthenia; in other cases it is auditory acuity that is diminished, sometimes on both sides, sometimes on one only, as in hysteria.²

Method of Examination.

The following case sheet is filled in on the first examination:

WEST CLIFF CANADIAN EYE AND EAR HOSPITAL,			
Folkestone,, 1917.			
Name	Reg. No.	Unit	
Age	Previous occupation		
Complaint	Previous condition		
History of present illness			
Alcohol	Tobacco		
Nose	Nasopharynx		
Right Ear.		Left Ear.	
M.T.			
Tube			
Forks:			
32 dv.			
64 dv.			
128 dv.			
C 256			
C 2			
C 3			
C 4			
Whistle			
Monochord			
Rinne	Weber	Rinne	
Schwabach	(C or C ¹)	Schwabach	
Conversation Voice			
Whisper			
Romberg			
Equilibrium in Right Foot	Equilibrium in Left Foot		
Bending Forward	Bending Backward		
Finger to Finger	Finger to Nose		
Pointing Left Hand	up	down	
Pointing Right Hand	up	down	
Ataxia	Tremor		
Reflexes	Nystagmus		
Caloric Reaction	Rotation Reaction		
Vertigo			
Diagnosis			

We have examined and treated over 80 cases of nerve deafness due to high explosives; examined as many more supposed cases. We have had three cases of total (bilateral) deafness to voices, forks, and loud noises. Of these three, after treatment, one, Pte. K., could hear in the right ear conversational voice at 18 in. before being sent to Moore Barracks because of mental hyperexcitability. The other two are now under treatment. One, Pte. H., who had complete anaesthesia and no thermal sense, can hear conversation voice through a speaking tube. The other, Pte. P., who had a gunshot wound in the region of the left temporal lobe, the R.M.T. completely destroyed, with otorrhoea, and the L.M.T. atrophic and adhesions in middle ear, can distinguish notes from a resonator along a tube to the ear, and "Tipperary" and the "Maple Leaf" when he stands close to the piano. Of the others, usually with marked deficiency on one side and total deafness on the other, with caloric reaction still present on the totally deaf side, all have improved, remarkably so in the ear with the deficient hearing.

The cases here given are selected to show the variety of cases, and not to emphasize the results. The complete results will be published later.

Sergt. L. Previous to enlistment ears normal. In France from January, 1915. On June 2nd, 1916, was hit with shrapnel, which pierced his helmet over the frontal bone, and buried; scalp bruised, bone not fractured. Not unconscious; dazed, not vertigo; no nausea; totally deaf in the left, some dullness in the right; refused to be invalided till laid off with "influenza" ten days later. Seen September 6th. R.M.T. normal; L.M.T. perforation and otorrhoea. Hearing for conversational voice, R.E., 3 yards; L.E., 0. All forks heard by right ear, none by left, though faint over mastoid. Few symptoms of traumatic neurosis. Left caloric easily obtained. Sent to the C.C.A.C. on October 10th with this report: R.M.T. normal, L.M.T. small perforation, no discharge. Hearing for conversational voice, right ear 7 yards; left ear 1 yard (with noise apparatus in right), whisper close to ear.

Pte. H. Previous to enlistment ears normal. In France nine months. On June 28th, 1916, received shrapnel wound on left side of head and blown up. Unconscious for several hours; left ear bleeding, followed by suppuration for a few days. Admitted September 15th, 1916. R.M.T. thickened and retracted; L.M.T. congested, hammer not seen; conversational voice, right ear, two yards; voice and forks in left, nil; violent headaches, great tremor, slight vertigo. October 17th: Sent to C.C.A.C., with this report: R.M.T. retracted; L.M.T. slight middle catarrh; conversational voice, right ear normal, whisper 24 in.; left ear 7 yards, whisper 15 in.

Pte. C. H. Previous to enlistment ears normal. In France four and a half months. On June 13th, 1916, shell-burst about 5 ft. away on right. Slight shrapnel wounds over left eye; not knocked down, not unconscious; heard and felt loud crack in right ear; rotation vertigo; no vomiting. Helped back to doctor's dug-out and sent to Boulogne. Totally deaf in right ear, some deficiency in left. Admitted October 1st, 1916. R.M.T. retracted and perforated; L.M.T. retracted. Hearing for voice and forks, right ear, nil; left ear, voice 7 yards, all forks. Right caloric easily obtained. October 15th: Hears voice in right ear with noise apparatus in left; easily fatigued; sweats freely during treatment. October 20th: Sent to the C.C.A.C. with this report: Right ear, drum membrane retracted, small recently healed scar, also small perforation; no discharge; hearing for conversational voice 8 in. with noise apparatus in left. Left ear, drum membrane retracted; hearing normal for voice and whisper; tremor less marked.

Lieut. S. Previous condition of ears normal. In France three weeks. On June 3rd, 1916, blown up by bursting of shell; unconscious for twenty-four hours. No ear haemorrhage; no discharge; no vomiting; vertigo not marked. Efficiency of hearing in right ear, totally deaf in left. Seen September 15th, 1916. Hears all forks and voice at 3 yards in the right; in left total deafness to forks and voice. R.M.T. displaced by adhesions; L.M.T., adhesions and marked atrophy of posterior half. Hyperaesthesia on right half of body, anaesthesia on left; concentric limitation of the fields of vision. On October 24th, 1916, sent to the C.C.A.C. with the following report: Conversational voice, in the right ear over 7 yards; in the left ear (with noise apparatus in the right) 10 in.

Gunner R. Previous to enlisting, defective hearing in left ear sufficient to exclude him from Australian Government telephone service. On July 30th struck by shell on left arm and back; blown up. Does not remember anything for three days (unconscious?), then rotation vertigo; great nausea; deaf in both ears. Hearing came back in left ear in twelve days without treatment. Admitted September 14th, 1916. Right membrane normal; no hearing for voice or forks. Left membrane normal, hearing for voice 2 yards. Severe vertigo at times. October 12th, 1916: Sent to the C.C.A.C., with this report: Conversation voice, in right ear 20 in.; in left ear 5 yards. Recommend that he be sent to convalescent home.

Pte. H. No previous ear trouble. On June 18th, 1916, after two months in France, "blown over parapet"; unconscious for some hours; totally deaf in both ears. Admitted September 4th, 1916. Right membrane congested; left congested and retracted. Totally deaf in both ears to voice, forks, and all sounds. Can distinguish and differentiate C¹ and C² on right mastoid by summation of stimuli. Mentally clear and intelligent. Bilateral anaesthesia; no thermal sense for heat and cold. Deficient senses of position for elbow, wrists, and fingers. Easily fatigued; irritable; if eyes are closed drops off to sleep. Taste accurate but weak; concentric limitation of vision. Vertigo; staggers if eyes are shut. Frequently complains of tingling and itching in the ears. (N.B.—This is a frequent complaint in these cases.) September 4th, 1916: Gets notes on the piano by right ear, and all forks over right mastoid. Galton whistle at right ear feels like the prick of a needle. (N.B.—Because of the tendency to rapid fatigue, treatment at first was confined to right ear.) October 4th, 1916: Hears all forks over mastoid in both ears; and in right from resonator 16 in. away connected to his ear by a tube. Sings tunelessly and rhythmically. October 20th, 1916: Hears in right ear short sentences by the aid of a speaking tube, and through resonator in left. Caloric in left gives reaction with 4 oz. of water at 66° F. Still under treatment.

When the caloric and rotation tests give no reaction in a totally deaf ear no treatment is persevered in. We may give one example:

Pte. S. No previous ear trouble. On June 2nd, 1916, was buried by shell fire; dug out unconscious and remained so for four hours; then deaf in left ear, with severe vertigo and vomiting. If he attempted to sit up had rotation vertigo and fell over. Severe headaches. Admitted August 20th, 1916. Totally deaf in left ear to forks and voice; vertigo on stooping or turning, with nausea. Not able, because of his condition, to test caloric. Right ear, hearing normal. Headaches less. September 10th, 1916: No hearing in right ear. Vertigo less. Caloric in left gives no reaction. Rotation gives a duration from right stimulation about half of that of left stimulation. Drum membrane normal, tube open. Diagnosis: Destruction of left internal ear.

Pathology.

The pathology of nerve deafness from high explosives is unknown. Mr. Sydney Scott of London has reported a case in which, after a bullet wound fracturing the vertex of the skull, with no paralysis but great bilateral though

not total deafness, the *post-mortem* examination showed intact tympanic membrane, no injury to stapes, or annular ligament, or membrana secundaria; no haemorrhage into the labyrinth; blood clot in both middle ears; no fracture of the base.⁸

Various explanations of the causation of traumatic neurosis, based on pathological findings, have been offered. For instance, changes in the small cranial blood vessels, changes in the myeline sheaths, small haemorrhages, air emboli following decompression as a result of high explosives, etc.; finally, as morbid anatomy throws so little light on the subject, it is suggested that we have here molecular changes in the nerve and nerve cell.⁴ It will be a great injustice to the thousands who suffer from this malady, as well as a great loss to science, if something be not attempted at this time to enlighten us on the pathological changes present. Rational treatment demands information; and rational treatment, not haphazard empiricism, ought to be given to those suffering from these distressing symptoms.

Basis of Treatment.

The treatment is founded on the following anatomical and physiological basis:

1. At the synapses there is structural discontinuity of the nerves.

2. The results produced by impulses travelling in a nerve depend on the way the fibre ends and not on any difference in the impulses themselves. "Impulses produced in the optic nerve by light are identical with those produced in the auditory nerve by sound." "The difference recognized by the organism must be due to analysers in the central nervous system."⁵

3. At the synapses different physiological systems come into touch with each other, and so co-ordinate action in diverse systems is possible.

4. At the synapses there is always a spread of the nerve impulse, and the greater the impulse the greater the spread.

With this basis we are working in the following hypothesis:

As a result of the high explosive with enormous and sudden increase of pressure in the ear, there occurs a dissolution of the permanent auditory pathway, and a spread of the nerve impulse into other adjacent paths. The auditory stimulus no longer reaches its goal and deafness results. Such a dissolution may occur at one or at all the synapses. It may not be complete, and a maximal stimulus may be still able to get through.

To re-establish connexion two things have to be done: (1) To connect up the permanent part, and (2) to stop dissipation; but dissipation is like a bad habit, the longer it has been going on the worse it is to break.

To help, we have the fact that habit and association of activity facilitate transmission of impulses. Nerve impulses tend to pass over an accustomed and long-established path, so the most favourable cases are those in which complete dissolution has not occurred. The most unfavourable are those where there has been complete dissolution and total deafness to tuning-fork, voice, and noise, no matter the intensity or pitch. It may be that this dissipation of the nerve impulses will help to account for the associated nervous phenomena.

Treatment.

In the totally deaf, either in the one or both ears, the method pursued is briefly as follows:

1. Tuning-forks are applied: (a) To the bone (for example, mastoid); or (b) through resonators, attached to the ear by tube; (c) through the air. In our worst cases the time given to (a), (b), or (c) varies with the amount of deafness.

2. The voice is used: (a) Through resonators with tube in ear; (b) through speaking tubes; (c) without any aid.

3. Each period of treatment is very short, for fatigue is rapidly produced. Thus we may have headaches, vertigo, sweatings, etc., and occasionally pain is complained of, if the treatment be too long.

4. As soon as possible carefully graduated physical exercises are given. In exercise the two essentials are short duration and no bending. The treatment (except the drill) is given twice a day in the worst cases; it ought to be given twice in all. In the worst cases, when it is difficult to pass from bone to resonator, we use the piano. We find that at each successive stage we have to use summa-

tion of stimuli; thus, not only must the fork on the resonator be kept up for some time, but with the voice we have to repeat the word, but even then there is a marked delay before the response comes. In the later stages the delay is still present. There it appears to be not so much that he does not hear, but that he hesitates to attach the word to the sound, for, if asked what he thinks it is, he frequently answers correctly.

5. As early as possible we ascertain if the semicircular canals are acting, because when there is reaction to the caloric or rotation tests in totally deaf cases we feel justified in continuing treatment for some time.

SUMMARY.

1. The normal stimulus (musical notes or voice) is an adequate stimulus for the nerve and is the best stimulus. Electricity is contraindicated and likely to do harm since it so easily produces vertigo.

2. In the totally deaf, bone conduction is perceived before air conduction. It is essential to differentiate vibrations from musical notes.

3. In these cases summation of stimuli plays an important part in the perception of sound.

4. There is a marked diminution of the duration of hearing along the whole series of forks, both through bone and air. This corresponds and exists *pari passu* with concentric limitation of the fields of vision. Often both improve together. Frequently the field of vision is more retracted on the side having the greater deficiency of hearing.

5. If the conducting mechanism is damaged or destroyed it not only takes longer to get improvement but complete recovery cannot be expected.

6. Prognosis is good as a rule, especially in cases where there is no trauma demonstrable in the peripheral organ, no history of aural vertigo, and a normal caloric reaction. The most noteworthy exception met with so far is damage to the seventh nerve. In these cases hearing returns but slowly, and, so far as we have observed, not perfectly, even with a normal drum membrane, little if any signs of middle-ear inflammation, and a caloric reaction present.

7. As a result of the concussion due to high explosives there is frequently a trauma demonstrable in the ear. This may be accompanied by neurosis (traumatic neurosis), especially headaches and vertigo. The perception of sound is diminished over the whole normal range; the diminution may be so great as to totally abolish perception of sound. I have not seen a case yet associated with tone islands. What I do find is a diminution all along the scale both for bone and air conduction.

8. As the deafness diminishes there may persist for a long time an inability to grasp intelligently what is said or to retain the memory of it. Thus a word may have to be repeated two or three times before the patient gets it; or, if he be asked to repeat two or three numbers given consecutively, he will repeat the last one; he knows that there were others but did not get them.

REFERENCES.

- ¹ Mott, Lettsomian Lectures, *Trans. Roy. Soc. Med.*, 1916, vol. xxxix, p. 172. ² Dejerine, *Sémiologie des affections du système nerveux*, Paris, 1914, p. 1123. ³ *Proc. Roy. Soc. Med.*, Otolological Section, March, 1916, p. 29. ⁴ See Mott, loc. cit. ⁵ Bayliss, *Principles of General Physiology*, pp. 387, 512, 513.

RESULTS OF SCOPOLAMINE-MORPHINE TREATMENT DURING LABOUR IN 150 CONSECUTIVE CASES.

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THE majority of the cases in the present series were conducted at a private maternity nursing home; the remainder at the patients' own homes. I have no experience of this treatment in hospital wards where other patients were present. But from the observations of others it would appear that the results obtained in such wards are not the best; there would seem to be no added risks to mother or infant, but the condition aimed at, namely, complete amnesia, is not attained nearly so often. Thus, Gauss's percentage of complete amnesia is 86, a great many of the cases having been treated in hospital wards. Sir J. Halliday Croom tells me that his hospital cases are not