Direct œsophagoscopy. At 19 cm. from teeth a white stinking mass 4 cm. long was seen and removed. After removal, pus welled up. At 24 cm. a sharp spicule of bone about 2·5 cm. long was seen impacted transversely in the œsophagus. The right end had penetrated the gullet, and the œsophagoscope was used to push the wall of the gullet gently away from this end; the fragment of bone was then removed easily. A perforation in the wall was seen, with pus welling out: it was enlarged with forceps, and the cavity douched with hydrogen peroxide solution. Two days later the patient was swallowing fluids easily without pain. Fourth day. From 3 p.m. onwards he complained of severe pain along left side of spine: temperature normal. At 4 p.m. he vomited 6 oz. blood; half an hour later the breathing became embarrassed; died suddenly. Autopsy: On right side of œsophagus, a healing abscess just above level of aortic arch; on left side, perforation of œsophagus into arch of aorta, about 8 mm. in diameter.

II.—L.G., female aged 59, was admitted under my care in *March*, 1935, with the story that eight days previously she had been eating rabbit and a bone had stuck in the throat. Since then she could take no solids at all; two days before admission she had coughed up a stinking mass from the throat, with some blood, and slight bleeding continued. She was kept on a fluid diet. Three days later (eleventh day) she vomited a mass of bright blood and fell back dead. At autopsy a perforation nearly 1 cm. area was found leading from the aorta into the œsophagus. There was no evidence of mediastinitis.

Suggested pathology in both cases: prick perforation of œsophagus, with infection and subsequent necrosis of aortic wall; later rupture of wall of aorta from internal pressure.

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

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President—DOUGLAS GUTHRIE, M.D.

Modern Views on Concussion of the Labyrinth

By DR. N. RH. BLEGVAD (Copenhagen)

IN order to illustrate the types of so-called "concussion of the labyrinth" I shall mention to-day, I will begin by quoting a couple of case histories.

I.—The patient is a man, aged 69. The hearing in his left ear has been very poor for the past thirty-three years—in his own opinion,

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because one day he ducked his head in the bath tub. The deafness did not become worse through all these years, and did not inconvenience him very much so long as the hearing of his right ear was good. Then a new telephone was installed in his house, and the first time he tried it, it gave a loud and sharp crack in his right ear—on account of a short circuit in the telephone wiring, he thinks. There was at once a marked reduction in the hearing of this ear and violent tinnitus. There has been no pain or discharge from the ear, but a spell of dizziness now and then. He was treated by a local otologist with catheterization, and this seemed rather to aggravate his deafness, and also the subjective phenomena. As the deafness did not diminish, he put in a compensation claim, and hence he was sent to a Copenhagen otologist for examination. This otologist found that the hearing of the left ear was practically absent and that the patient was able with the right ear—that is, the ear that had been damaged by the telephone—to hear the speaking voice only at a distance of four inches; whispering voice could not be heard. The bone conduction was reduced, and the upper tone limit considerably lowered. The otologist concluded that the lesion consisted in a " concussion of the labyrinth ", and that there was not much chance that the hearing would ever get better. The patient was discharged from his service with a pension, and he demanded very considerable compensation. The insurance company decided, however, that he must be examined by another otologist also, and he was referred to me.

Now it was found that even the circumstance that the man had obtained a very good pension had already brought about a considerable improvement of his hearing. It is true that he tried at first to exaggerate his deafness considerably, but his hearing grew better and better gradually, as the examination proceeded, and was repeated by my assistant, and then repeated by myself once more. The ear that had been deaf for many years was not able to hear anything at all. But with the other ear—the damaged ear—he was now able to hear whispering voice at a distance of six inches, and speaking voice at a distance of more than six yards. The bone conduction was almost normal, and the upper tone limit was not lowered more than was to be expected at his age. After the question of compensation had been settled, the hearing probably became just as good as it was before the injury.

II.—A married woman, aged 29, was struck on the right ear by her husband. She felt a little crack in the ear, and noticed at once that she had become completely deaf in that ear. She consulted an otologist, who found a traumatic rupture of the tympanic membrane. The perforation healed within two months, but the hearing was still poor. The husband was summoned before the police court, and the

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consulting otologist certified that the hearing of the right ear had practically disappeared, and would probably never be better.

Result: The man was sentenced to gaol for twenty days, and to pay his wife 1,000 kroner damages, with costs. The case was taken before a higher court, which referred the woman to me.

The right tympanic membrane showed a small coagulum of blood as the only remnant of the injury. The membrane had healed completely without any visible scar. On testing with the speaking voice the patient stated at first that she could not hear anything at all with her right ear. She was asked to repeat different numerals spoken in ordinary conversational voice, but she did not even try to repeat the words; she merely declared she could not hear anything at all. But when next she was asked, in a very soft voice, to turn about, she did it promptly. Thus she was caught in the simplest trap, and then her hearing improved at once; at the end of the examination there was hearing for whisper at a distance of one yard. Hearing for the higher tones was perfectly normal in both ears. Hearing for air conduction and bone conduction was a little diminished on both sides. I requested the patient to return two days later. When she came she declared at once that her hearing now was a great deal better, and that on the whole she was feeling better. It was found that now she was able to hear a whisper at a distance of two yards without any difficulty, and a conversational voice at a distance of six yards, the greatest distance obtainable in my clinic. To counterbalance the restoration of her hearing she now complained of pain in the ear and in the surrounding region, but the examination failed to reveal anything that might explain this pain.

As a result of this change in her condition, the case turned out differently in the court of appeal. Instead of going to gaol for twenty days and paying 1,000 kroner damages, the husband was let off with a fine of 200 kroner, and 500 kroner damages.

It was these cases and two more, which I shall mention later, that interested me in concussion of the labyrinth.

When I speak of concussion of the labyrinth, I have in mind only *those lesions that are produced by a strong and sudden sound*, sometimes associated with a marked and sudden rise of the air pressure. I omit lesions of the ear associated with fracture of the temporal bone, labyrinthine hæmorrhage, etc., lesions produced by shot wounds, falling on the head or feet, with fracture of the skull, and injuries that follow a blow on the head with a hammer or club. These lesions have been studied very thoroughly, clinically and experimentally (C. Stenger, Zange, Wittmaack, Hayato Ueda). We know that such conditions produce severe injuries to the labyrinth—fractures and hæmorrhage, followed by secondary degeneration of the nerve-end apparatus in the labyrinth.

Nor do I speak of those cases of deafness that arise through a

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fall on the head and other severe head injuries, without fracture of the skull and without any demonstrable direct injury to the ear, especially without fracture of the labyrinthine capsule. Voss has reported such a case: A man tumbles down from a coal-waggon and hits the back of his head against the pavement. This results in bilateral complete deafness and abolition of all static labyrinthine reactions. No fracture of the skull or labyrinthine capsule can be demonstrated, not even through very careful X-ray examination.

Such affections are known also from shot lesions. In many cases where the bullet has grazed the mastoid process or the frontal, parietal or occipital bones without causing any injury to the bone involved, the accident has given marked deafness, sometimes on the same side, sometimes on the opposite side. Also lesions of the facial bones may give rise to affections of the ear. Opinions differ about all these lesions. This divergence of views naturally comes from the fact that pathologic-anatomical examination of these lesions in man has been carried out only very seldom, for, as a rule, nobody dies of these lesions, and also from the fact, that experimental studies have given widely different results. Some investigators (Ulrich, Nager, Körner) look upon these affections as expressions of traumatic neurosis, and do not think there are any underlying pathologic-anatomical changes in the labyrinth or in the brain. Other authors (Uffenorde, Wittmaack, Kock, Voss) hold that these affections are due to different causes:

(1) Circulatory disturbances in the labyrinth and brain, or (2) hæmorrhages in the perilymphatic spaces (especially the scala tympani and scala vestibuli), or (3) destruction of the neuro-epithelium in the cochlea and sacculus, resulting from hæmorrhage or pressure of fluid, or (4) microscopic fissures in the labyrinthine capsule.

The pathogenesis of these lesions is as yet unsettled. The various investigators now agree on the whole about the third category of traumatic labyrinthine injury (concussion), namely, *ear-lesions due to strong and sudden sound-effects*, and it is largely experiences from the World War that have brought about this consensus of opinion.

It has long been known that strong sounds may give rise to deafness, as in boilermakers, riveters, weavers, locomotive engineers, etc. The old-fashioned way of boilermaking is now replaced by the pneumatic hammer, and this is ten times worse (600 strokes per minute). Indeed the noise in such a shop is actually felt as an annoying physical discomfort, and deafness comes after employment for a relatively short time in a modern boilermaking shop. Nothing definite is known about the frequency of occupational deafness. Alt states that 10 per cent. of all affections of the internal ear are occupational. Pogány found 26 per cent.

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A single, very loud, sound (a shot close to the ear, explosion, etc.) *may also produce deafness* involving, as a rule, only the ear that is nearest to the source of the sound, but in some cases involving both ears.

Many investigators have tried, through experimental studies, to determine the cause of this deafness and what changes take place in the ear. Previously it was thought that the effect of a single violent noise was to produce a concussion of the labyrinth, which was taken to be an organic lesion of the internal ear. What this lesion might consist in, was an open question, as no pathologic-anatomical examination of such a case had been reported.

Wittmaack's experiments on animals demonstrated that a single, brief—but strong—sound (whistle or crack) had the effect that all the cells in the organ of Corti were whirled around. With a weaker, brief sound-effect he found the injury limited to the sensory epithelium while the supporting cells were intact. The effect of a protracted strong sound was first a degeneration of the sensory cells, then degeneration of the peripheral nerve-fibres and ganglion cells. Finally the supporting cells in the organ of Corti degenerated too. The lesion was confined exclusively to the lower turns of the cochlea. Weak sounds gave no changes at all, not even when kept up for a long time. When the animal was placed in a cage with a metal floor, and the sound was produced continuously by letting a felt-muffled hammer beat on the metal floor, so that it was transmitted to the ear chiefly through bone conduction, the result was degeneration of the nervous apparatus, especially in the upper turns of the cochlea.

Wittmaack's experiments met with severe criticism and other investigators (*e.g.* Siebenmann and his pupils) arrived at other results. Recent Japanese investigations (by Takezawa), appear to show that the vibrations from the floor play the greatest rôle and, especially, that a brief, strong sound acts on the ear only through these vibrations and not through the air.

Long before the Great War it was realized that deafness resulting from concussion of the labyrinth in man may, not infrequently, diminish spontaneously, or recover completely, in some cases even quite suddenly, and then the recovery often takes place under the influence of some mental factor—fright, sudden joy, and other similar conditions. In his work on *Lesions of the Organ of Hearing*, 1905, Passow characterizes many of these cases as " hysterical ". Yet, almost all the older otological authors think that a strong, sudden, and brief sound-effect is able to produce a concussion of the labyrinth and thus give rise to an organic lesion of the organ of hearing.

The World War has completely altered our conception of concussion of the labyrinth. During the War a great number of persons

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had their organs of hearing exposed to the most violent sound-effects, especially gunners who sometimes stayed for many hours in succession close to the heavy guns under "Trommelfeuer" ("drum-fire"). Sometimes these men were penned-up together with the guns in vaults, so that they were exposed not only to the sound-effect, but also to great changes in atmospheric pressure. Impairment of the hearing could be demonstrated in all gunners who had been operating the guns for some length of time, but it subsided, as a rule, before long. Permanent impairment of the hearing was encountered only in men who had been exposed long and steadily to a strong detonation in firing the guns.

The soldiers were further exposed to a violent sound-effect, combined with high atmospheric pressure, by the explosion of shells or mines; this was particularly severe when the shells hit the places of refuge, the trenches, or marching columns. In such cases the damage was not limited to the effect of the explosion upon the ear; there were often additional, more or less extensive, injuries to the body, and in many instances the soldiers were almost buried. A study of the patients who have been exposed to such shell explosions is particularly interesting, and it is especially these studies that have impelled us to revise our conception of concussion of the labyrinth, for when these patients were extricated after an explosion they were frequently found to be completely deaf (sometimes mute, too), or, at any rate, more or less hard of hearing. Indeed, such cases were even so frequent, that Pflug states that deafness appeared in 99 per cent. of the soldiers who were buried by explosion. As is evident from the papers written on this subject during the War, it was then the prevailing view that, resulting from the violent sound-effect, these patients had a concussion of the labyrinth with marked organic changes in the internal ear, and that the prospects of recovery were minimal.

During the war, however, there were some otologists who doubted that this was really a matter of severe organic lesions. In the *Lancet*, 1917, Hurst and Peters presented the case-records of two soldiers who had been perfectly deaf after shell-shock. Many different therapeutic measures were tried on them in vain—but they were cured by a fictitious operation. Under light anæsthesia, the authors made a small incision behind the ear, hitting at the same time a plate with a hammer to simulate chiselling. The two patients showed normal vestibular reaction. From this Hurst and Peters conclude that deafness with normal reactions means hysterical deafness. The Editor of the *Lancet* remarks, quite correctly, that this point is by no means settled, as there are instances on record of "mild labyrinthitis with total deafness but in which the canal reflex could still be excited". In Logan Turner's *Textbook*, 1936, Fraser

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and Stewart subscribe to this view, but they think that deafness due to explosion may in many instances become permanent.

In 1915 Tibor Szász said, in dealing with functional ear lesions in soldiers: "To begin with, these forms of deafness were taken to be simulation, but then it was noticed that they were accompanied by extensive disturbances of the sensibility." So, the author designated these cases as hysterical, or traumatic neurosis. He made a distinction between them and shell-deafness and concussion of the labyrinth, and stated that the prognosis could be made only with reservation.

After the War, however, we saw something peculiar happen: Only very few veterans were given disablement benefit on account of this form of deafness, for most of them had recovered their hearing, completely or partly. Recovery proceeded most rapidly in soldiers who were kept at the front after the explosion, because they were not wounded. In these cases the deafness subsided mostly within a few hours or days, passing sometimes through a stage of hardness of hearing until the normal hearing was gradually restored. But the patients who were transferred to hospitals at home did not fare so well. Their condition was diagnosed as "concussion of the labyrinth", "acoustic deafness", "labyrinthine hæmorrhage", "destruction of the organ of Corti", etc. (Muck), and their deafness lasted longer. On subsequent examination several of these patients were regarded as hysteria, but this was not correct, Muck says, because many of them had been robust and perfectly "free from nerves" before the explosion. A fright-neurosis had brought about a hardness of hearing which resembled organic deafness, but was not due to any organic lesion, and hence was designated "psychogenic deafness".

What has been said about complete deafness applies also to hardness of hearing. Psychogenic traumatic hardness of hearing resembles perfectly the organic labyrinthine hardness of hearing, yet Passow and Muck think there are certain characteristic features that differentiate psychogenic hardness of hearing from true labyrinthine hardness of hearing. The most characteristic feature of psychogenic hardness of hearing is that the result of the functional tests varies, so to speak, from day to day.

There is an additional symptom to which I wish to call attention —a symptom I have encountered often, not in cases of explosion deafness, it is true, but in persons with functional hardness of hearing from other causes, namely normal hearing for all high tones, by bone conduction as well as by air conduction, while the other functional tests are suggestive of a severe affection of the sound-perceiving apparatus (air and bone conduction markedly shortened, Rinne positive, hearing for whisper relatively far more diminished than hearing for conversational voice).

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As another characteristic sign of psychogenic hardness of hearing, Muck states that in the test of the hearing for whisper and speaking voice the patient may well be able to repeat the spoken word correctly, but only after some hesitation, or not until the word is repeated once or twice. Muck thinks this phenomenon is to be explained in this way: that such patients require a much longer time than normal persons to understand, i.e. to conceive the meaning of the word in question. In this condition, the affected part is the faculty of understanding, that is to say, the capacity for immediate and correct conception of the spoken word. It is obvious that such a defect will appear as hardness of hearing when it comes to connected sentences. These patients often declare: " I hear very well that you say something, but I cannot hear *what* you say."

Sometimes it is possible in these cases to demonstrate a " perception limit ", that is, a definite distance—generally not more than from 6 to 8 inches—within which all the words are perceived correctly and instantaneously. As soon as the examiner goes beyond this limit, the slow conception makes its appearance.

Examination of the static labyrinth furnishes no definite evidence from which to judge. Even in cases of complete deafness—for example, after suppurative labyrinthitis—the static labyrinth, as is well known, may preserve its full functional capacity, and hence it is not justifiable to conclude that the deafness is functional in those cases in which the static labyrinth shows normal reactions. But on the other hand if the static labyrinth gives no reaction (for instance, in the caloric test) we can be quite sure that the deafness is not functional.

So far, we have considered exclusively the diagnosis of psychogenic hardness of hearing. But if the effect of a strong and sudden sound is followed by complete bilateral deafness, its psychogenic nature can be recognized by the sudden onset. Passow says: " Sudden onset of absolute deafness without basal fracture is always due to hysteria." Also this form of deafness presents various characteristic features. For one thing, it is typical that these patients very rapidly learn lip-reading. This was discovered during the War when—what undoubtedly meant a great disadvantage to the patients—they were instructed in lip-reading, because they were thought to be permanently deaf. But even if such patients are not instructed in lip-reading, they often learn it by themselves, so that they convey no impression whatever of being deaf. In many cases, however, it is possible to ascertain that these patients really hear unconsciously, as their facility for lip-reading disappears as soon as the examiner plugs their auditory meatuses and speaks to them with whispering voice and with distinct movements of the lips.

In order to examine whether the deafness is psychogenic, it is often useful to apply the method of Barth. The deaf patient is

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requested, in writing, to sing a song he knows very well. He is now requested to repeat his song, but this time the examiner gives the initial note on a piano just before the beginning of the song. If now the patient starts singing in this tone, it is practically proved that his deafness is psychogenic. To avoid a mistake, the examiner asks him to sing once more and gives again the initial tone, but now a little higher or lower than before. If the patient again starts singing in the given tone, it is proved that he has an unconscious hearing, i.e. that the deafness is psychogenic.

Colledge mentions that during the war he had excellent aid from Captain Crampton in detecting psychogenic deafness in soldiers. This officer had a series of questions written on a paper, the last question on the back of the paper. He put the paper before the soldier, spoke the questions with ordinary voice and had the soldier answer them. If the deafness was psychogenic, the patient also answered the last question, which he could not read, although the question on the back of the paper was different from the spoken.

Also the acustico-motor-reflex may be used as a diagnostic measure, as an unexpected strong acoustic irritation elicits a blinking of the eyes or a startled movement of the limbs. Finally, the psychogenic deafness is nearly always accompanied by sensory disturbances in the surroundings of the ear or over greater parts of the body surface. As a rule the sensibility to pain is impaired while the sensibility to touch is preserved.

The *treatment* of psychogenic deafness may be rather difficult. Hypnosis may be employed, and light ether anæsthesia may prove serviceable, but it is important above all to see that the economic aspects are settled, so that the patient does not profit by permanent deafness.

In conclusion I will quote two further case records :

I.—A man, aged 33, domiciled in Slesvig (that part of Jutland which was re-united with Denmark after the World War). During the War he served on the German side.

One day, while he was marching with his company, a shell burst among them, and many of the soldiers were killed and buried. Towards evening the sanitary troops came to carry the dead away. They noticed that this man, who was lying in a heap with many dead soldiers, moved his arm. He was unconscious and was brought to the field hospital. When he woke up, he was deaf and dumb. The dumbness subsided soon, but he remained deaf. He went from one hospital to another, but no treatment did him any good. He remained completely deaf in both ears, and the German army surgeons considered him incurable. He was therefore given disablement benefit.

After the War, when Slesvig returned to Denmark, the Danish Government took over the payment of his disablement benefit. He

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worked as a hairdresser, and seven years later a local otologist discovered that there was hearing in the left ear while the right was still deaf. The Invalidity Insurance Court referred him to me.

Examination of the static labyrinth showed no abnormality whatever. In Romberg's test the patient swayed back and forth, but his whole conduct during this test was very theatrical and after its conclusion he clasped his head with both hands.

The acoustic examination showed some impairment of hearing in the left ear. The patient said that he was completely deaf in the right ear, being unable to hear tuning-fork, whisper, or conversational voice. On further examination, however, he walked at once into the simplest trap. He was requested to turn his right side towards the examiner and stop the left ear firmly with a finger, and then repeat what was said. He said that he was unable to hear a word, even when spoken in a loud voice. Then the examiner spoke with ordinary conversational voice, asking him to shut his eyes, and he did so promptly, just as later he opened his eyes again when told to do so, in the same ordinary, conversational voice. It was then pointed out to him that it was necessary, for his own sake, that he must be accurate in his statements, and he was told to take a walk and return after a couple of hours.

After that the functional tests were repeated, and it was found that he was able to hear with his right ear, not only conversational voice but also whisper. He was catheterized and told to return the next day. When the examination was repeated on the following day he was able to hear tuning-fork a_1 and Galton's whistle, and after repeated tests of hearing it was found that he could hear a whisper at a distance of five yards, in the right ear as well as in the left. His answers are sluggish and slow—a feature pointed out by Muck as characteristic of psychogenic hardness of hearing.

II.—A man, aged 23, a cadet at a military academy.

About two months ago a gun-shot was fired right by his side. There was at once considerable hardness of hearing in the right ear, and this has increased since, so that he is now completely deaf in that ear. There is no dizziness. On examination the static labyrinth is found to react normally. Hearing test for conversational voice gives at first the impression that there is no hearing at all in the right ear, but when Bárány's noise-apparatus is applied to the left ear, there is hearing for loud speaking voice. The hearing of the left ear is normal. When the examiner places his hand quite lightly on the left ear, the patient states he is still unable to hear in the right ear. Thus it is established that his deafness of the right ear is psychogenic, and this finding is further corroborated by repetition of the functional tests, for then he is able to hear the tuning-fork a_1, through air conduction as well as bone conduction,

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and he can hear the highest tones in Galton's whistle. After six weeks' treatment by suggestion and with vibration massage with Breitung's apparatus, he is able with his right ear to hear a whisper at a distance of 1 yard, and conversational voice at 6 yards.

This case also illustrates the particular feature I mentioned before, as characteristic of psychogenic hardness of hearing, namely, normal upper tone limit together with a marked decrease of the hearing for tuning-fork and whisper.

I believe that now—especially on the basis of experiences from the War—we may consider it established that *the hardness of hearing after a violent and sudden sound-effect—with or without a simultaneous increase of the atmospheric pressure—is not due to organic changes in the ear, but has its seat in the cortex of the brain, i.e. it is psychogenic.*

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DISCUSSION

Mr. SYDNEY SCOTT said that he recalled a case described by Sir Charles Ballance,* that of a sleeping soldier awakened by a pistol discharged close to his ear by a companion, as a practical joke. The wretched man was very giddy and deaf, and blood escaped, but what ultimately happened Sir Charles did not tell.

He had seen a similar injury precisely a year ago. A patient had come the day after Guy Fawkes' celebrations, with an injury caused by the explosion of a powerful firework at close quarters. The right tympanic membrane was at first hidden by a blood-clot which he did not disturb. There was total deafness to the low tones of Bezold-Edelmann forks, and to all tones of the monochord by air and bone conduction. The left uninjured ear was occluded by Bárány's noise-machine when the tests were carried out. A week later when the clot had become inspissated, a large reniform perforation of the tympanic membrane—about 5 mm. in diameter—could be seen.

Three weeks later the perforation was smaller and nearly circular —about 2·5 mm. in diameter. The patient could now hear the voice close to the ear as well as Bezold forks down to 36 cycles and high tones 2,048 to 4,096 cycles.

Eight weeks from the day of injury he was astonished to find that the perforation had completely closed. The Bezold fork, 16 cycles, was now audible in both ears. A quiet ticking watch was heard at the normal average distance from the right ear, and the conversational voice was actually better in the right than in the left ear. The patient had been in the Artillery and accustomed to a sporting gun which, he knew, had affected hearing for the higher notes in the left ear for many years. He was re-tested six months later and could inflate the right tympanum normally. Regeneration of the membrane could not be attributed to treatment, for nothing

* Ballance, Sir Charles, and Green, C. D., *The Surgery of the Temporal Bone*, London, 1919.

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had been done except to avoid syringing, and to warn him against the application of drops.

Another case which Dr. Blegvad's paper recalled was that of a musician who had been knocked down unconscious. After several days, when he seemed to have recovered, he was distressed to find disharmony of certain musical notes. The speaker tested him with Bezold-Edelmann tuning-forks, and discovered that three consecutive notes in each octave sounded higher in pitch in one ear than in the other, whereas the other five notes in each octave sounded alike in both ears. Did this indicate that there had been an injury to certain fibres of the auditory nerve? It was not known whether the defect has persisted.

Mr. LIONEL COLLEDGE said that Dr. Blegvad had recalled experiences of the War. He himself had seen during the War, as aural surgeon to the First Army in France, many patients who suffered from the effects of gunfire, and in consequence had commotio labyrinthi. At that time he considered that the cases could be divided into two main groups. In the first group were those who served with the field artillery and so were exposed to continuous gunfire from field-guns which produced sounds of relatively high pitch, but not of extremely great intensity. These artillery men, especially officers, who probably served for longer periods, were suffering from what was either labyrinthine or nerve deafness, with loss of appreciation of high tones. Their deafness was of a chronic character and showed very little tendency to improve. In the years succeeding the War he had seen a number of such patients—both the actual patients seen in the War and others afflicted in the same way—and he was convinced that they showed a tendency to the onset of senile deafness at an unusually early age—i.e. from 40 to 50.

There was a second large group, in whom the injury to the ear was caused by a single very loud explosion—a shell bursting close at hand, or the effect of being blown up by a mine. Those cases could be subdivided into two classes: (*a*) Those in whom there was no perforation of the tympanic membrane, but on examination of the ear punctate hæmorrhages could be seen on the surface of the membrane. It was known, from the observations of Mott, that those who succumbed to such explosions showed similar punctate hæmorrhages in the brain which produced the so-called shell-shock. If punctate hæmorrhages ensued in the tympanic membrane and in the brain, it was not a far-fetched assumption that the same kind of punctate hæmorrhages would probably occur in the labyrinth. That kind of case usually, he believed, made a very good recovery. The second sub-division included those cases in which the tympanic membrane was blown out; it might be expected to be blown in, but the edges of the perforation were everted, and before the patients were seen, the ear, in many cases, had already been syringed,

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and so the case was turned into one of suppurative otitis media. That, however, was not always the case, but a circular was sent round to medical officers by the D.M.S. of the Army asking them to refrain from interference in that way. Where the drum membrane was perforated in that way it apparently provided some sort of protection to the labyrinth from the effect of the explosion. Those who had this perforation did not suffer so severely from internal ear deafness as did those whose tympanic membranes remained intact. They were comparable to the case which Mr. Sydney Scott had just described and for the most part they made a very good recovery.

There was also a third, and different, category—that of psychogenic or hysterical deafness—cases of which were described by Dr. Blegvad. That kind, he quite recognized, could only be treated successfully by psycho-therapeutic means.

Dr. T. RITCHIE RODGER said he was interested to hear what Dr. Blegvad had said concerning the recent work done in Japan, re-instating Wittmaack's observations of twenty-five years ago. In 1915 he (Dr. Rodger) had reviewed the literature on the subject of the experiments conducted by Wittmaack and those who followed him, and it seemed to be established then that Wittmaack was wrong in his conclusion that bone conduction was the essential force in bringing about the internal ear destruction. Von Eicken, Siebenmann and Yoshii disproved this conclusion by substituting rubber mats for the metal lining and floor of the cages of the animals experimented upon, and proved that the same destruction of the internal ear ensued as Wittmaack had described. Some of those workers were still living, and he would like to know whether they had accepted this reversal of their earlier conclusions.

Mr. GAVIN YOUNG said he was in a position to corroborate what had been said about accentuation of labyrinthine shock when it had been conveyed by bone conduction. Since 1913 he had several times been up in an aeroplane, and had been conscious of the fact that he was increasingly liable to " air-sickness ". The last time he went up was in a " Moth ", and he was connected with the pilot by ear-phones. Accordingly all hearing by air-conduction was cut out, and so he received all the vibration of the plane straight into his labyrinth, and he was dragged out of the plane very much more shocked than he had ever been before.

Mr. F. C. ORMEROD said he wished to thank Dr. Blegvad for having raised the question of internal-ear deafness due to the proximity to gunfire. He could corroborate what had been said about cases that had sustained middle-ear deafness. One saw patients recover after the War, no internal-ear deafness being left; but some, because of the suppuration which had ensued, were left with middle-ear deafness. Many of those patients were blown up in dug-outs and trenches, and little but middle-ear deafness remained

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in them. His impression of the gunners—those who were regularly serving guns—was that their nerve-deafness was temporary ; they had at the time a good deal of nerve-deafness, but it passed off in two or three days. If, however, the experience was repeated many times and continued for long periods, the deafness tended to become permanent. Among the applicants for War Pensions on account of nerve-deafness, gunners did not predominate ; there were more cases among infantrymen who were blown up in trenches and who had resulting middle-ear deafness. His (the speaker's) experience supported what Dr. Blegvad said as to the nerve-deafness being temporary, there being no organic destruction or lesion of the internal ear.

Sir JAMES DUNDAS-GRANT said that during the Great War he had seen large numbers of these cases and they conformed to what Dr. Blegvad had said. There was an Institute for the treatment of deafened men, and two years after the patients had been seen, advertisements were issued inviting them to come again for further help. To those advertisements, however, there was only one reply, and that was from a man who had no right to compensation at all, recovery appearing to be the rule.

Rupture of the membrane seemed to take the pressure off the labyrinth—there was little room to doubt that. In a lecture which he (Sir James) had given at the Royal Institution during the War he had carried out an experiment with a long drainpipe, at the end of which was a parchment tambourine on a hinge. He fired a revolver near the open end, and while the membrane was intact there was a violent flap. When a thin, moistened membrane was substituted and the revolver fired, the membrane gave way, and there was scarcely any flapping movement.

Mr. H. V. FORSTER said that during seven post-war years, as a medical officer to a territorial medium heavy-artillery brigade, he had received few complaints of gun deafness at practice-camps, but remembered very clearly two members of a 6-inch-howitzer gun team who reported within twenty-four hours of range practice. There was ecchymosis of the drumheads in each case, with deafness answering to both middle- and inner-ear routine tests.

With regard to the effect upon the ear of exposure to continuous noise : A Russian worker, Losanov,[1] had carried out an investigation into the effects of industrial noise at the Lenin nail-and-bolt factory at Saratov—a very noisy place because of the sound produced by the nail-stamping machines. He found that nose and accessory sinus disease acted as aggravating factors in the injury to the phono-receptory apparatus caused by the noise. This was confirmed in experiments on rabbits, in which artificially produced conditions of nasal obstruction were found to contribute definitely to the marked damage found in the cochlea.

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Another Russian worker, Temkine,[2] had concluded that those workers in noisy trades who already had incipient defects in the auditory organ suffered earlier than normal healthy workers.

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[1] Losanov, N. (Saratov), *Acta Oto-Laryngologica*, 1930, xiv, 393.
[2] Temkine, Jacques (Moscow), *Idem*, 1934, xix, 13.

Major W. A. D. Drummond said that he had had some experience of the deafness resulting from the newer types of machine guns. He found that in the cases investigated the tympanic membrane was indrawn and engorged, and that hearing by both air and bone conduction was reduced. Caloric tests revealed no obvious abnormality of the labyrinth. Inflation did not affect the deafness either way. The disability lasted, on the average, four days and then cleared up, but when they were at a height of 6,000 feet the men suffered more and the deafness might last up to a fortnight.

He did not think that there was a psychical element in these cases, as the men enjoyed this type of training, and earned more pay by proficiency.

It appeared that the rapid vibration of the gun, together with the noises of explosion, induced a hyperæmia of the middle ear which damped down the conductivity through this area and temporarily diminished the sensitivity of the cochlea.

Dr. Blegvad (in reply) said that the remarks of Mr. Sydney Scott proved the truth of what he had advanced in the paper, namely, that when a man was blown up in the trenches and the membrane was ruptured the patient would recover without treatment, or with only psychological treatment. He agreed with Mr. Colledge that continuous or prolonged concussion was apt to produce hardness of hearing, as it was known to do in the case of boiler-makers, and they might sustain damage to the nerve-endings in the labyrinth. As to the effect of a sudden heavy sound, the War experiences showed that the effect was always psychical, and that was supported by the telephone cases. Many years ago he had examined all the telephone operators in Copenhagen, to ascertain whether the hearing had been damaged by their work, and he found that it had not—even in cases whose hearing had been poor before they took up telephone work. In the cases in which complaint was made of hardness of hearing as a result of a trauma—e.g. a lightning-stroke in the telephone wiring or other severe acoustic shock—he had found no organic damage: the disability was a psychical one.

Both Wittmaack and von Eicken were still living, but he (Dr. Blegvad) did not know their views on the recent Japanese experiments. These latter were very carefully conducted, and the microscopical technique was very fine, but the cases were difficult to work out in a convincing way—there were so many possible

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sources of error. Based upon the theory of Wittmaack that it was essentially due to the vibrations from the floor, carried through the body to the ear, that the labyrinth was injured, many efforts had been made in Germany to isolate the different floors in the machinery shops from each other. It was—for economic reasons—very important to decide whether the lesion was due to the effect on the ear of sounds coming through the body, or to that of sounds coming from the air.

During the War it was noticed that after the men had been near discharging guns for several hours a retraction of the drum membrane occurred, but it soon disappeared.

ABSTRACTS

EAR

Osteomyelitis of the Inferior Surface of the Petrous Pyramid. WELLS P. EAGLETON. (Newark, N.J.) (*Jour. A.M.A.*, August 15th, 1936, cvii, 7.)

Because of the peculiar nature of the bone, the pathological process in the vast majority of cases has a tendency to spontaneous cure. In about 15 per cent. suppuration takes place, and well directed treatment may be successful, while in about 5 per cent. of invasive forms of thrombophlebitis the disease is uniformly fatal.

The petrous apex, from a pathological and surgical standpoint, is divisible into three sections. First, the marrow-filled osseous matrix itself with its dural coverings on the anterior and posterior surfaces; second, the domain of the carotid canal containing the carotid artery and the carotid venous plexus; third, the inferior "cribriform" perforated plate on the pharyngeal surface of the apex. Surgery of the apex resolves itself into two distinct lesions, namely, abscess-formation, which requires evacuation, and infective thrombophlebitis of small vessels, which requires expectant treatment until it in time either undergoes resolution or breaks down into pus. Enlargement of any fistula found in the region of the tympano-mastoid usually furnishes adequate drainage.

Mastoiditis in infants runs the course of a true suppurative osteomyelitis, that is, suppuration within bone marrow spaces. After the third year of life no trace of fat marrow is found within the region of the mastoid apophysis and mastoiditis after the second year is generally a local osteitis. Glandular enlargements and lateral

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