anteroposterior, true lateral and right and left obliques. Each of these may be taken on 8½-in. by 6½-in. films. A Potter-Bucky or Lysholm fixed grid should be used for all projections, otherwise the pictures will not be of good quality. It is desirable to take all but the frontal picture with the patient standing or sitting, so that the shoulders may drop to the maximum.

The frontal picture should be taken last when one has examined the lateral view. One can then judge how much the central ray must be tilted in order that it passes along the plane of the intervertebral discs of the last three cervical vertebræ. These discs slope forwards and downwards quite considerably.

If the straight X-rays are normal, then a cervical disc is not present and there is no arthritis of the small joints.

If there is a diminution in the intervertebral disc spaces—and this is common from middle age onwards in the lowest three cervical vertebræ—the radiological conclusions are more difficult. The presence of arthritis of the small joints, even with considerable osteophyte proliferation invading the exit foramen, does not necessarily mean pressure on the nerve. Conversely, the absence of osteophytes does not rule out pressure on the nerve, for the periarticular tissues may be swollen and be pressing on the nerve but they may not cast a shadow on the X-ray film.

(2) *Myelography*. Owing to shortage of space this section of Dr. Bull's paper is omitted.

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[*May* 6, 1948]

Observations Upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve

By M. R. DIX, C. S. HALLPIKE and J. D. HOOD

(From the Otological Research Unit, Medical Research Council, The National Hospital, Queen Square, London)

Introduction.—The Loudness Recruitment (L.R.) phenomenon was first described in 1936 by E. P. Fowler of New York (1936), and its occurrence in certain varieties of nerve deafness has since been abundantly confirmed (Steinberg and Gardner, 1937; Huizing, 1942; de Bruine-Altes, 1946).

The phenomenon can be demonstrated most readily when the deafness is limited to one ear, and the nature of the phenomenon itself can perhaps be best appreciated from the following brief description of the simple test procedure needed for its investigation in cases of this kind.

Fig. 1 shows the test procedure being applied:

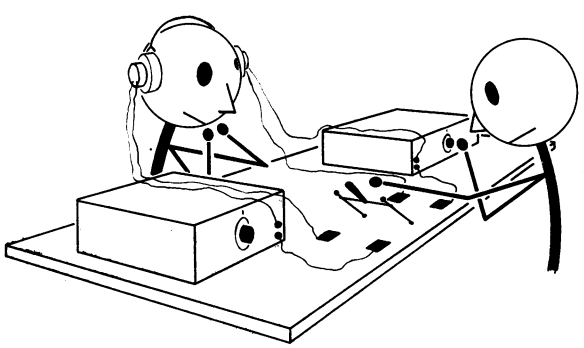

FIG. 1.

The subject wears a pair of telephone receivers, each supplied by a separate pure-tone audiometer, or preferably by a single audiometer with arrangements for independent adjustment of the intensity in the two receivers. The frequency of the sound stimulus is the same in each receiver, and the tester switches it alternately from right to left.

The audiograms of two typical cases of unilateral deafness are shown in fig. 2.

In Case 1 the deafness is due to a lesion of the conducting mechanism of the left middle ear, and in Case 2 to Menière's disease affecting the left labyrinth.

The purpose of the test is to ascertain and mark upon the two ladder diagrams two series of intensity levels, one for the right ear and one for the left, each intensity level for the right ear being connected across the diagram with a level for the left ear, found by experiment to give a sensation of equal loudness. In each case the test frequency selected is 1,000 cycles, at which point the audiogram shows a threshold shift for the affected ear of 30 db. The test starts with a stimulus of threshold intensity at the right ear ; the balancing

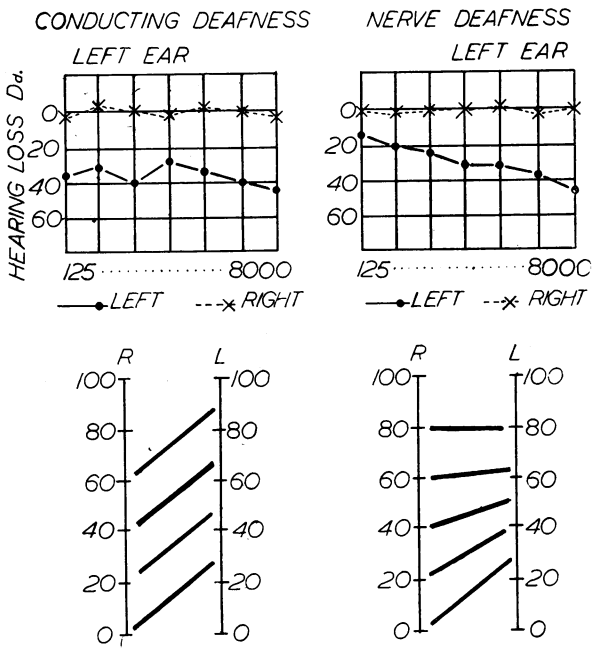

FIG. 2.—Loudness balance diagrams. Frequency 1,000 C.P.S.

intensity for the left ear will, of course, be 30 db. higher. Successive stimuli, rising in intensity in steps of 20 db., are then applied to the right ear and for each level the balancing intensity for the left ear is established by trial, the comparison being made by switching the stimulus backwards and forwards between the two ears. The result obtained in Case 1 indicates that the loss of sensitivity or deafness of the affected ear, 30 db. at threshold, remains constant at 30 db. throughout the entire intensity range.

This finding is an unvarying one in conducting deafness and is explicable on the straightforward assumption that the obstruction caused by the middle-ear disease to the sound waves on their way to the inner ear introduces an attenuation factor, in this case 30 db., which is constant at all intensities.

A very different result is obtained in Case 2. The audiogram is substantially the same as in Case 1, with a threshold shift at 1,000 cycles of 30 db. The balancing points at threshold are identical with those of Case 1, with a 30 db. displacement upwards for the left ear. On ascending the intensity scale, however, it is found that the sensitivity loss or deafness of the left ear, 30 db. at threshold, becomes progressively less, until at 80 db. equal intensities at the two ears evoke equal loudness responses. *In other words, the deafness of the affected ear present at threshold disappears at higher intensities, and this in its simplest terms constitutes the phenomenon of Loudness Recruitment.*

A more conventional form for the graphic representation of these results is that given in fig. 3.

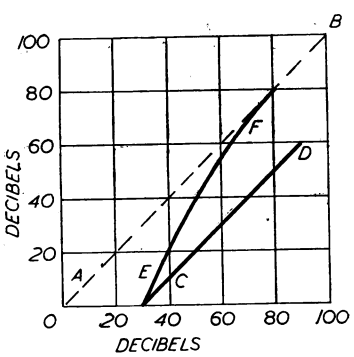

FIG. 3.

Sound intensities in decibels above the normal threshold are plotted on the vertical axis for the unaffected ear, and on the horizontal axis for the deaf ear. Equal loudness levels for the two ears are plotted on these charts as a series of points and the line AB passing through the origin connects the points which would be thus obtained in a normal individual.

CD is the corresponding line obtained in Case 1. Here the sensitivity loss at threshold of the deaf ear is represented by the displacement of the point C along the base line to the right of the origin. This sensitivity loss remains constant at higher intensities, and the line CD thus lies parallel to AB.

EF is the corresponding line obtained in Case 2. As in Case 1 the same sensitivity loss at threshold is represented by the same displacement of the point E to the right of the origin. At high intensities, however, this sensitivity loss is progressively eliminated, and the line EF approaches and finally coincides with the line AB.

There is general agreement by all who have since investigated the L.R. phenomenon that it is absent in deafness due to uncomplicated middle-ear disease, so-called conductive deafness.

On the other hand, it has frequently been demonstrated in a wide variety of disorders of the internal ear and cochlear nerve, including Menière's disease, which are collectively described as "nerve deafness", and it has come, therefore, to be regarded in a somewhat uncertain manner as a valuable indication of "nerve deafness" using the term in its widest sense.

The theoretical basis of the L.R. phenomenon has been discussed by Lorente de Nó and by Fowler (1939). Both adopt an explanation based upon certain general principles of neurophysiology and upon certain details of the finer structure of the cochlear neurones described earlier by the former. According to this explanation—a somewhat complex one—the occurrence of the phenomenon might be expected as a natural consequence of any pathological process involving a reduction in numbers of the neural elements, either of Corti's organ or of the cochlear nerve, and the matter is put by Lorente de Nó (1937) in the following terms:

> "If a number of hair cells in the ear or a number of fibres in the cochlear nerve is missing, the tones will appear to be weaker in intensity when near threshold stimuli are used; but if the intensity of the tone is increased, the more strongly activated hair cells or cochlear fibres will be sufficient to saturate, i.e. to excite with the limiting intensity the cochlear fibre or the cells of the cochlear nuclei, so that the cerebral cortex will receive the same number of impulses per second from both ears and will perceive the tone delivered to the diseased ear as strongly as the tone delivered to the normal or less affected ear.
>
> "Thus, it may be said that Fowler's phenomenon is an immediate consequence of the anatomy and physiology of the nervous system, and that, in fact, it must be pathognomonic of neural deafness".

The phenomenon of recruitment, or the variable type of deafness as they call it, is also discussed by Stevens and Davis (1938). These authors, too, accept the view that the phenomenon is attributable to "a deficiency in the total number of neural elements which normally contribute to give a tone loudness".

It must be said of the explanation that its implications are not devoid of obscurity. Moreover, in some of the cases in which the phenomenon has been found to occur, we seriously lack secure information on the nature of the pathological process. It has, therefore, been clear for some time that the confirmation of de Nó's explanation, and indeed any further developments of theory, must await a great deal of additional factual information. de Bruine-Altes (1946), in her recent monograph, has stressed very pertinently the particular need for further studies in which the results of L.R. phenomenon tests are more closely correlated with more exact information upon the underlying anatomical changes in the cochlea and cochlear neural pathways.

In the present study, therefore, we have endeavoured to throw more light upon the mechanism of the L.R. phenomenon by means of a detailed clinical study, including full investigation of the phenomenon in a number of patients referred to us by our colleagues at Queen Square and elsewhere suffering from two different and clearly defined varieties of hearing disorder, of which our knowledge both of the clinical features and morbid anatomy can now be regarded as considerable. The two disorders are Menière's disease and degeneration of the VIII nerve due to neurofibroma of the nerve and to other space-occupying lesions of the cerebello-pontine angle.

It was considered that the choice of these two disorders was likely to be particularly illuminating since in the one, Menière's disease, the primary lesion we now know to affect the endolymph system of the cochlea with its contained cochlear end-organs, while in the other, the primary lesion is of the cochlear nerve fibres within the internal auditory meatus.

Some further details may be given at this point of these anatomical changes and their correlated symptomatology.

Menière's disease.—Fig. 4 shows the histological condition of the cochleæ in a case of Menière's disease.

The subject, a man of 46, had suffered for four years from paroxysmal attacks of vertigo with deafness and tinnitus of the right ear. Otological examination revealed a severe perceptive deafness on the right side, and a defect in the right caloric responses. The tympanic membranes were normal and there were no other abnormalities in the central nervous system. The patient died of acute lymphatic leukæmia.

Fig. 4A shows the cochlea of the unaffected ear (left) and fig. 4B the cochlea of the affected ear (right).

In fig. 4A, the normal position of Reissner's membrane will be noted (arrows). Corti's organ appears normal.

The apparent disappearance of Reissner's membrane in fig. 4B has been brought about by the maximal distension of the scala media, which has thrust the membrane back upon the bony wall of the scala vestibuli and caused its herniation through the helicotrema. Arrows indicate the new position of the membrane. It should be noted, however, that the cells of the spiral ganglion appear quite normal in number and structure. Further, there is no apparent reduction in number of the cochlear nerve fibres in the modiolus, or in the osseous spiral lamina.

Figs. 5A and B show the organ of Corti of the two ears. That of the left ear, fig. 5A, is

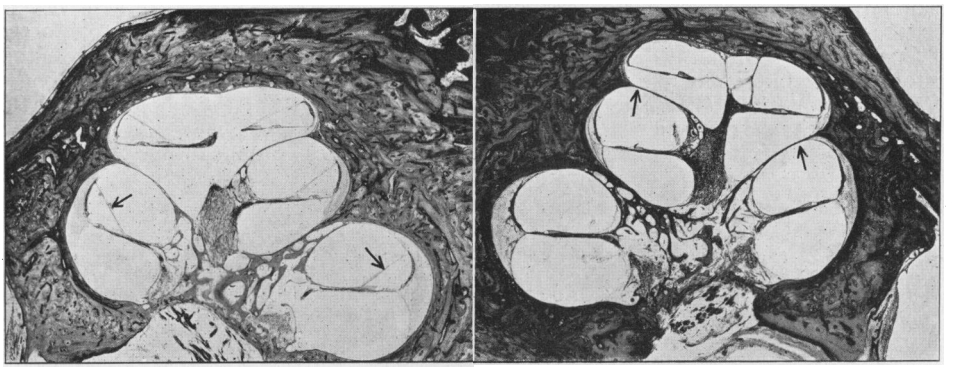

FIG. 4A.—× 7·5. FIG. 4B.—× 7·5.

fairly well preserved, and presents a normal appearance. The cell outlines are well demarcated, and the cell nuclei are clearly differentiated.

It should be noted, however, that as usual in preparations of the human cochlea the technical difficulties of fixation make it impossible to discern any details of the hair cells.

In the affected ear, fig. 5B, marked changes are to be seen in Corti's organ. The cell mass is compressed, the cell outlines obscured and the staining differentiation between nuclei and cytoplasm virtually extinguished.

This type of pathological change in the cochlea was first described by Hallpike and

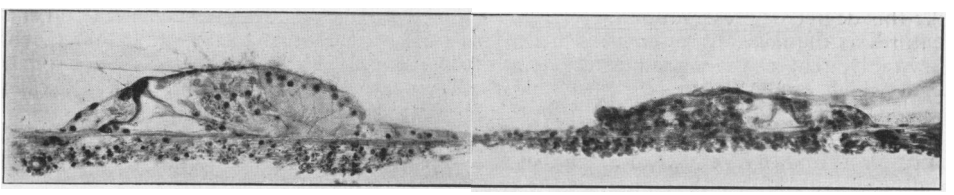

FIG. 5A.—× 200. FIG. 5B.—× 200.

(Figs. 4 and 5 are reproduced by permission of the Editor, *The Annals of Otology, Rhinology and Laryngology*.)

Cairns (1938) in two clinically characteristic cases of Menière's disease. Since then, histological studies have been possible in three further cases, in one instance in collaboration with A. J. Wright (1940), and in a second with T. E. Cawthorne (1947).

The findings in a third case have not yet been published.

In addition to these, a number of other histological examinations have been carried out elsewhere (Rollin, 1940; Lindsay, 1942; Altmann and Fowler, 1943) in a number of subjects presenting the characteristic clinical features of Menière's disease.

Whilst in all of these the presence of an endolymphatic dilatation has been established, reports have varied on the condition of Corti's organ. In two of our own series it has appeared normal, and this has also been the finding in the majority of cases reported upon elsewhere. In no less than 3 of our 5 cases, however, Corti's organ has presented abnormalities of the type described. These abnormalities are so striking in character that it would appear inevitable that they should be regarded as being distinctive of Menière's disease in certain of its phases. That they are found sometimes and not always only means, as we see it, that they represent a transient and reversible reaction on the part of the hair cells to the chemico-physical disturbance which goes with the gross distension of the endolymph system. It seems quite reasonable that this disturbance should be phasic in character, so matching the clinical course of the disease, and it is natural, too, that the morphological changes in the hair cells should vary at the same time. It follows, that whether these changes are revealed in any given case or not must be largely a matter of chance, depending upon the phase of the disease at the time of death.

Although these morphological changes may be largely reversible, and may in fact appear to be absent at death, it does not follow that a corresponding reversal need be expected of the functional loss, and, indeed, the fact that the deafness in Menière's disease persists between its active phases can only be taken to mean that while the disorder of structure *is* apparently reversible, the disorder of function *is not*. The statement that the structural

disorder in Menière's disease is *apparently* reversible requires some amplification in view of the well-known technical difficulties which beset the histological study of the human organ of Corti. Its post-mortem disintegration is rapid, and fixation difficult, and the resulting histological picture is, therefore, always an imperfect representation of the original.

This point will be made clearer from an examination of fig. 6 and its comparison with fig. 5A. Fig. 6 shows the organ of Corti in a cat, well fixed by an *intra-vitam* injection technique. Every detail of the hair cells and their associated membranes is well shown.

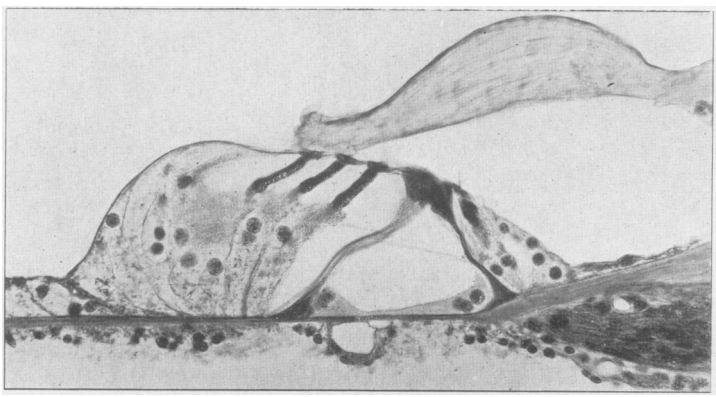

FIG. 6.—× 310.

(Reproduced from Schafer's "Essentials of Histology" by permission of Messrs. Longmans, Green & Co.)

In human material, however, of which fig. 5A is a good representative sample, nothing like this degree of preservation is possible. The hair cells are seldom well defined, and such features as the nerve fibres crossing the tunnel of Corti are never to be seen. This means, inevitably, that changes in the human hair cells, morphologically slight but functionally vital, which are likely to be responsible for the deafness during the negative phases of Menière's disease, will never be demonstrable by histological means.

For this reason, although no definite abnormalities can be recognized in Corti's organ in some cases of Menière's disease, we feel it to be very likely that such changes *are* present and obscured only by the limitations of histological technique.

To sum up the structural changes found in Menière's disease, we can say that these are limited to Corti's organ, but the nerve fibres and cells of the spiral ganglion are normal.

Degeneration of the VIII nerve due to neurofibroma, &c.—Interruption of the cochlear nerve fibres central to the spiral ganglion, whether caused by pressure from a tumour or by surgical section, leads, in contravention of the Wallerian law, to degeneration of the nerve fibres and ganglion cells peripheral to the point of interruption (Witmaack, 1911; Kaida, 1931; Hallpike and Rawdon-Smith, 1934).

Fig. 7 shows a horizontal section through the cochlea of a patient with a high degree of

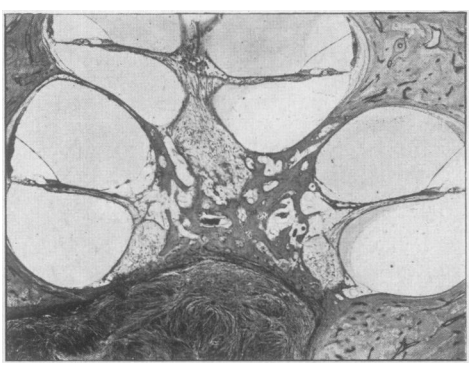

FIG. 7. —× 11

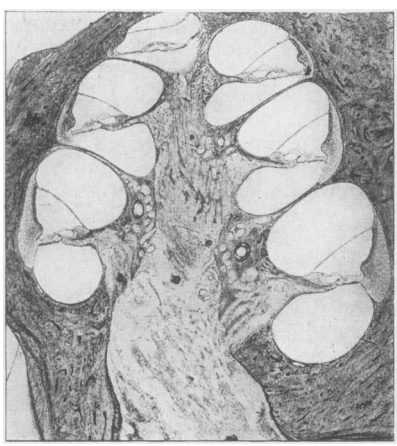

FIG. 8.—× 9.

deafness and a complete loss of the caloric responses resulting from a neurofibroma of the VIII nerve. The tumour is seen filling the meatus, and the fibres of the VIII nerve are lost to view. The main peripheral effect of this tumour is a gross reduction in the number of nerve fibres themselves and of the cells of the spiral ganglion.

Indeed, very few cells of the ganglion remain while the nerve canal of the osseous spiral lamina is virtually empty.

Corti's organ, however, is substantially normal and this is characteristically the case unless the tumour has involved the cochlear blood supply. These cochlear changes in VIII nerve tumours closely resemble in their essentials those which follow an operative section of any mammalian VIII nerve. Fig. 8 shows the cochlea of a cat twelve weeks after an intracranial section of the VIII nerve. It shows again the characteristic disturbance of the whole peripheral cochlear neurone with perfect preservation of the hair cells of Corti's organ. Figs. 9A and B show Corti's organ in these two conditions, namely: Fig. 9A, Neurofibroma of the human VIII nerve ; fig. 9B, Degeneration following intracranial section of the VIII nerve of the cat.

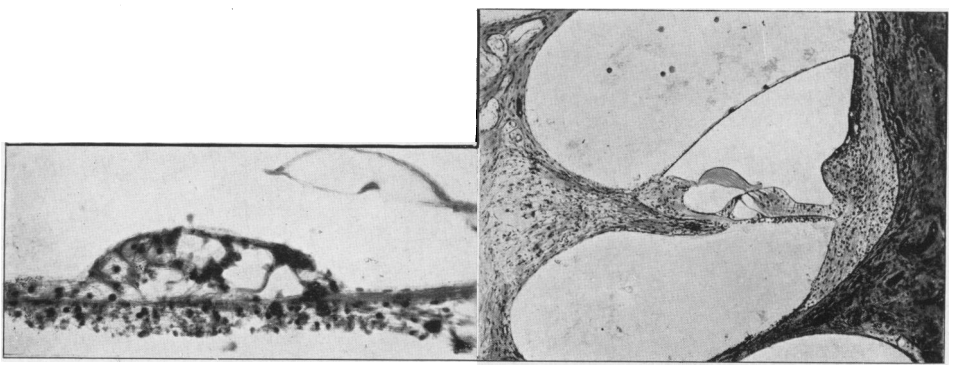

FIG. 9A. × 200.—Neurofibroma of the human VIII nerve.

FIG. 9B. × 50.—Degeneration following intracranial section of the VIII nerve of the cat.

This evidence of animal pathology is particularly useful since it indicates that in the human subject there is no reason for supposing that an interruption in the course of the VIII nerve by a tumour would cause any structural changes in Corti's organ, even of a kind which would be obscured by defects of histological technique. The difference in the anatomical findings in the cochlea in these two conditions, Menière's disease and VIII nerve neurofibroma, may thus be summarized as follows:

	Organ of Corti	*Nerve fibres and cells of spiral ganglion*
Menière's disease	May show gross compression of cells and loss of staining reactions. These changes are not always found. Residual changes, however, are likely to be present and account for the deafness. They are obscured by limitations of histological technique	Normal in number and structure
Degeneration of VIII nerve due to tumour pressure	Normal, unless cochlear blood supply is interfered with	Much reduced in number and may be completely eliminated

The results of the Loudness Recruitment tests obtained in these two groups of cases will now be described.

RESULTS OF LOUDNESS RECRUITMENT TESTS IN MENIÈRE'S DISEASE AND VIII NERVE DEGENERATION

(1) *Menière's disease.*—The number of cases examined was 30. All gave a characteristic history of paroxysmal vertigo, deafness and tinnitus. In the great majority the deafness was substantially limited to one ear, and in all the pattern of the caloric reactions revealed one of the various characteristic abnormalities upon the side of the deaf ear (Cawthorne, Fitzgerald and Hallpike, 1942). None gave any history of head injury, and general neurological examination revealed no abnormalities apart from the octavus system.

In all of the 30 *cases, Loudness Recruitment was marked.*

Fig. 10 shows the Loudness Recruitment patterns of 3 representative cases with their audiograms. It will be seen that in every one the sensitivity loss at threshold of the affected ear is completely eliminated at higher intensities. In the third case, well-marked over-recruitment is seen; that is to say, at high intensities the loudness function of the affected ear exceeds that of the normal ear. Tests at other frequencies than 1,000 cycles yielded comparable results.

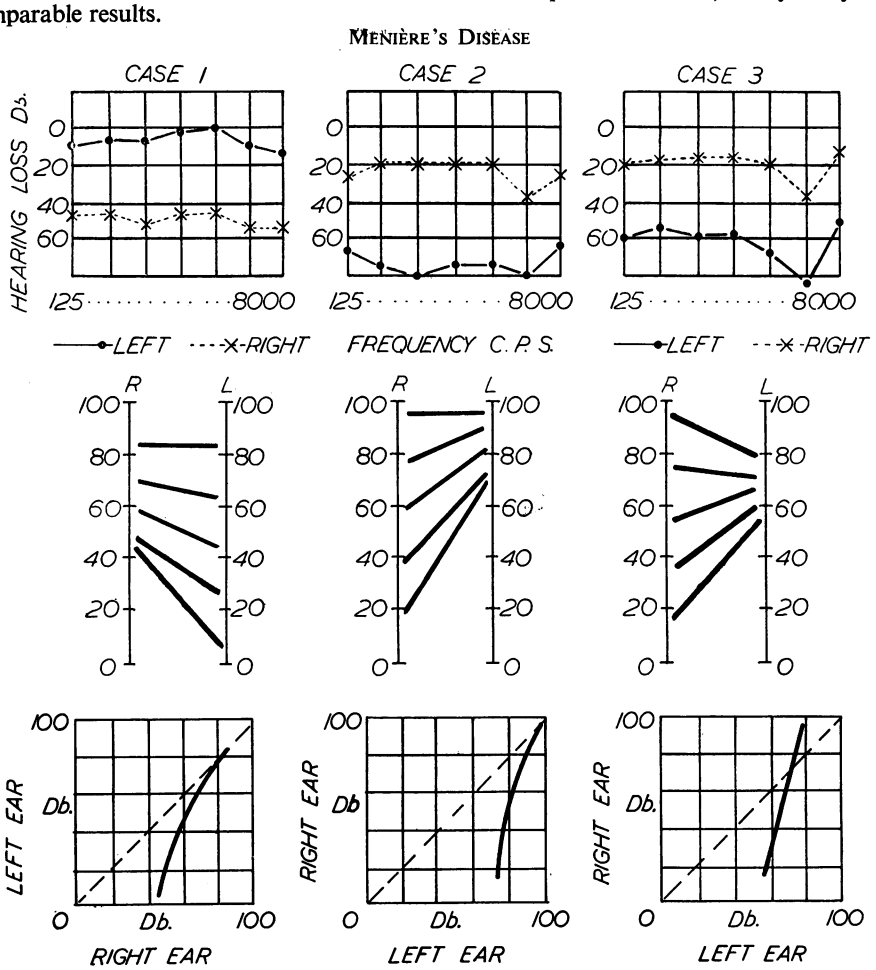

FIG. 10.—Loudness balance diagrams. Frequency 1,000 C.P.S.

(2) *VIII nerve degeneration.*—11 cases of neurofibroma of the VIII nerve were studied, in all of which the clinical diagnosis was confirmed by operative or post-mortem findings. In addition, 9 cases were examined of other varieties of cerebello-pontine angle lesion exhibiting well-marked involvement of VIII nerve function. In 6 of these cases the diagnosis was confirmed by operative or post-mortem findings. It is necessary to stress here that loudness balance tests for the Loudness Recruitment phenomenon can only be carried out satisfactorily when the deafness of the affected ear is of moderate degree. Very often, however, the deafness in cases of VIII nerve neurofibroma and cerebello-pontine angle tumour is severe and not infrequently complete. It has, therefore, been a matter of considerable difficulty to find cases in which the deafness of the affected ear was not too severe for the test. That we were able to find the cases, we owe to the good offices of a number of our colleagues both at Queen Square and elsewhere who have kindly referred their cases to us, and to whom we are greatly indebted.

In the majority of these 20 cases, tuning fork tests showed the typical findings of nerve deafness, reduced bone conduction with a positive Rinne. Bone conduction was, in fact, reduced in all, but in a few the Rinne test was definitely negative in spite of careful masking of the opposite ear.

The results of the loudness balance tests in 14 cases of this group were constant in showing a complete absence of recruitment.

Fig. 11 shows the test results in 3 characteristic cases, together with their audiograms.

VIII NERVE DEGENERATION

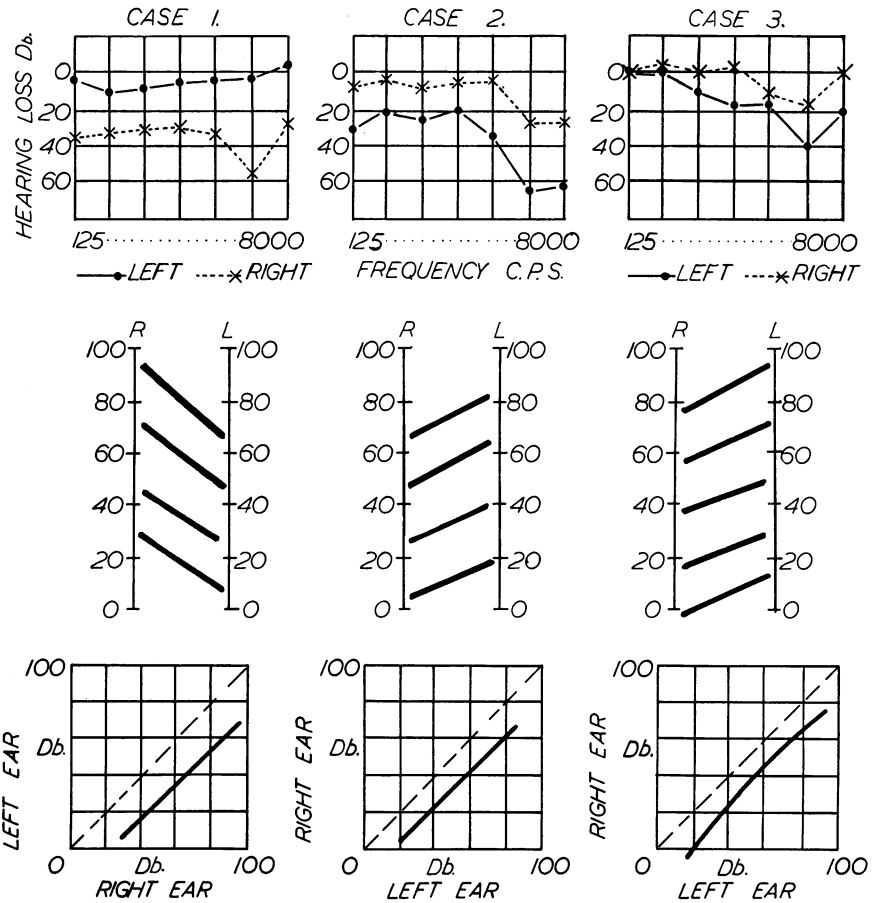

FIG. 11.—Loudness balance diagrams. Frequency 1,000 C.P.S.

It will be seen that in all 3 the sensitivity loss of the affected ear at threshold is maintained at all intensity levels. In the remaining 6 cases of this group a slight amount of recruitment was present. In some of these there was found a fixed upper loudness limit in the affected ear. That is to say, loudness appeared to increase in the affected ear up to a certain intensity level, and thereafter to increase no more. This phenomenon is shown in the second case of fig. 12. Tests at other frequencies than 1,000 cycles yielded comparable results.

SUMMARY OF EXPERIMENTAL FINDINGS

Loudness Recruitment was present and complete in all of 30 cases of Menière's disease, a primary affection of Corti's organ. In 20 cases of degeneration of the VIII nerve, due to pressure or infiltration by tumours, Loudness Recruitment was completely absent in 14. In the remaining 6 cases slight recruitment was present.

It would appear, therefore, that in this condition, an example par excellence of nerve deafness, Loudness Recruitment hitherto described as being uniquely distinctive of nerve deafness is characteristically not present at all. Instead, we have a type of response which is identical with that found in middle-ear deafness.

DISCUSSION

From the viewpoint of practical oto-neurology, the experimental results described make it clear that in the loudness balance test we have at our disposal a test procedure which should prove of great value in making clear the difficult and important distinction between end-organ deafness and nerve-fibre deafness. It seems likely, too, that the findings will prove to have a bearing upon a number of problems of auditory theory. The finding that the recruitment phenomenon is an unvarying occurrence in Menière's disease, a disorder

of the end-organ of hearing, appears to be related in an interesting way to the recent experimental work of Pumphrey and Gold (1948). According to Gold's theory, based upon this work, the microphonic potentials of the cochlea arise somewhere in Corti's organ and play a vital part in determining both its sensitivity and frequency selectivity. We should, therefore, certainly expect that a disorder of this microphonic mechanism, i.e. Corti's organ, would lead to deafness and to certain disorders of pitch sense, i.e. paracusis dysharmonica, well known to be characteristic of Menière's disease. It is pertinent to recall that the very nature of the recruitment phenomenon corresponds very well with a type of derangement well known in certain microphones. Thus a carbon microphone in good order yields an electric response which is linear over a wide range of sound pressures. In a faulty microphone of this kind, however, the response at low sound pressures may be very defective, while approximating at high pressures to something nearer its normal value. It is clear that this variety of non-linearity in the response of a faulty microphone reproduces in essentials the characteristic of the Loudness Recruitment phenomenon, and it seems possible, therefore, that Gold's theory of the cochlear mechanism may provide us with a clearer explanation than any at present available of the occurrence of the Loudness Recruitment phenomenon in Menière's disease.

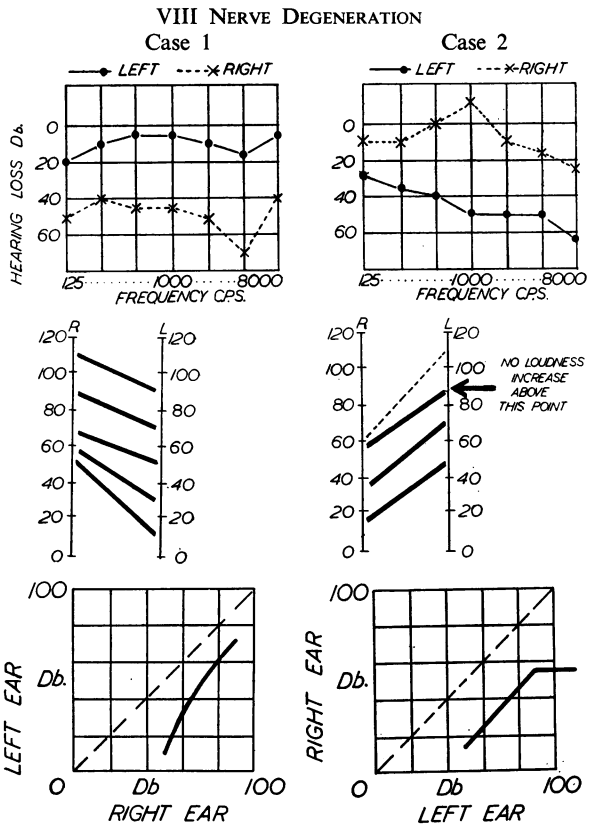

FIG. 12.—Loudness balance diagrams.

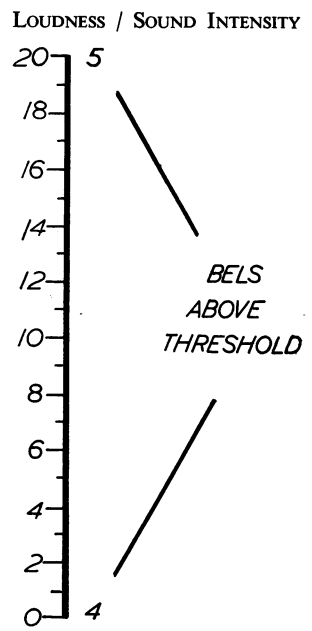

FIG. 13.—Distinguishable loudness steps (each step corresponds to an intensity increase of about 13%).

The finding that recruitment is characteristically absent in cases of VIII nerve tumour has been a very surprising one. Clearly it controverts decisively the theory of its neurological mechanism advanced by Lorente de Nó and Fowler. It would seem quite possible, however, to explain this finding in a different and more simple way if the following assumptions be made: first, that the nerve degeneration engendered by tumour pressure or infiltration is an evenly diffused one with a definite fibre survival rate; secondly, that the relationship between stimulus intensity and loudness sensation depends upon a simple type of formula governing the numerical increase of active cochlear nerve fibres which accompanies each ascending step of the loudness scale.

Such a formula may be outlined as follows:

The intensity range on the scale shown in fig. 13 lies between four and five bels above the threshold of hearing for a tone of 1,000 cycles, that is to say, it covers the middle range of audible intensity for this frequency, and the total intensity change of 1 bel between the top and bottom of the scale is a tenfold one. Between these points there are twenty just distinguishable increments of loudness, each corresponding to an intensity increment of about 13%, this being the so-called Weber fraction, and according to the data of Shower and Biddulph (1931) the value of this fraction for frequencies between 1,000 and 4,000 cycles approximates very closely to the value given, i.e. 13% for a great part of the loudness scale.

Concerning the manner in which the steps on this loudness scale are related to the number of active nerve fibres, no precise quantitative details are known. We have, however, available a variety of well-known data derived from the electro-physiological recording of cochlear action potentials and from experiments on masking and auditory fatigue, which all indicate that as stimulus intensity is increased there occurs a spatial spread within the cochlea, with progressive activation of additional nerve fibres.

It is suggested that our present experimental finding, i.e. that Loudness Recruitment is absent in diffuse degeneration of the VIII nerve, would be adequately explained upon the following simple hypothesis: That each step on the loudness scale shown in fig. 13 *corresponds to a definite fractional increase in the number of activated cochlear nerve fibres.* The manner in which this hypothesis might be applied to the working of the normal and degenerate cochlear nerves is shown schematically in fig. 14.

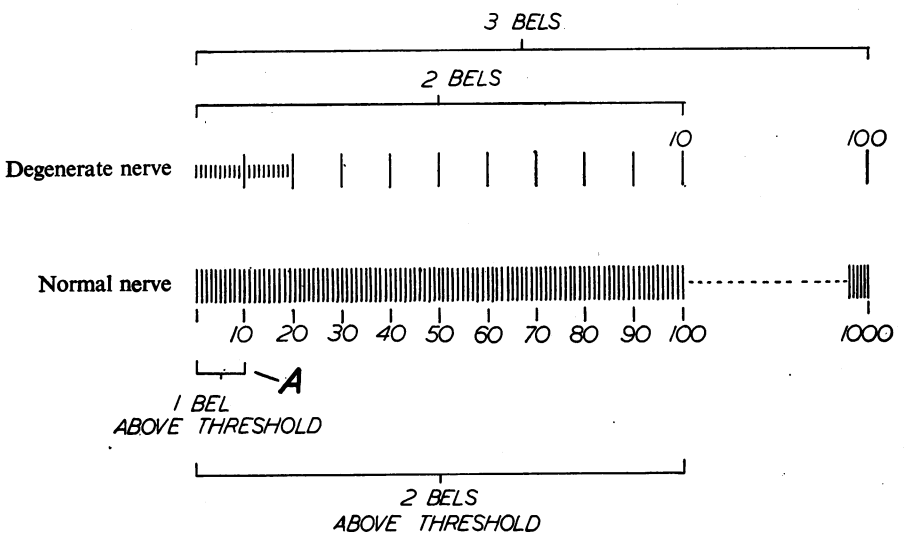

FIG. 14.

This represents the fibres of two cochlear nerves arranged in linear fashion as they lie in the osseous spiral lamina of the cochlea.

The lower set of fibres is that of a normal nerve, while the upper set is that of a degenerate nerve. The degeneration is diffuse, and the long strokes indicate the surviving fibres. The survival rate is one in ten. The small bracket "A" is taken to indicate the number of fibres stimulated by a sound stimulus of intensity 1 bel above threshold. The number of fibres is conveniently taken as ten. This stimulus exceeds by tenfold the threshold intensity. Correspondingly, stimulus intensities of two and three bels exceed it by one hundred- and one thousand-fold. Now, it is supposed that such changes of stimulus intensity involve corresponding changes in the number of active nerve fibres. This means, in the case of the normal nerve, that the 2 bel stimulus will activate 100 fibres and the 3 bel stimulus 1,000 fibres.

It will be noted that the loudness sensation elicited in the normal nerve by the 1 bel stimulus arises from 10 fibres, and to match this sensation in the degenerate nerve, 10 active fibres will also be required. It is clear that with a fibre survival rate of one in ten, a 2 bel stimulus will be required to bring these into action. In other words, a tenfold intensity increment, 1 bel, is required to compensate for this particular degree of nerve degeneration.

With a stimulus intensity of 2 bels, it will be seen that 100 fibres are activated in the normal nerve. To activate the same number in the degenerate nerve, we require again a tenfold increase of stimulus intensity, i.e. a 3 bel stimulus, and it follows that the same rule will apply as we go further up the intensity scale. In other words, given a constant fibre survival

rate in the degenerate nerve, this would be compensated by a constant fractional increment of stimulus intensity at all points of the intensity scale.

This argument, therefore, leads us to expect that the sensitivity loss, or deafness of the affected ear in a case of diffuse degeneration of the VIII nerve, would be constant throughout the intensity range. In fact, the Loudness Recruitment phenomenon would be absent in accordance with our own experimental findings.

It is necessary to add two amendments to this general statement of theory.

It is unlikely to apply at low intensity levels near threshold, where the sensitivity of the ear to small intensity differences is changing rapidly. At high levels, too, it would seem inevitable that a saturation point would be reached very soon in the case of the degenerate nerve, above which no further increase of loudness perception would be obtainable.

This latter point is in good agreement with the finding in some of our cases of a fixed upper limit of loudness in the affected ear.

The highly schematic character of our hypothesis need not be stressed. It would indeed appear evident that loudness grading depends upon a grading not only of fibre numbers but of discharge frequency in the fibres themselves, the resultant being a grading of the total number of nerve impulses reaching the cochlear centres per unit time. While this may be the case, it is doubtful whether it would seriously affect the general validity of our hypothesis.

Thus, while a certain degree of nerve degeneration may lead to a definite fractional decrease in the number of cochlear fibres activated by a sound stimulus of given intensity, it is likely, also, to lead to a definite fractional decrease in the discharge frequency of these surviving fibres, and the combination of these two effects will result in a fixed fractional decrease in the total number of action potentials reaching the cochlear centres per unit time.

This decrease will be compensated and loudness loss restored in the manner of our experimental findings by a fixed fractional increase of stimulus intensity over a wide range of the intensity scale.

SUMMARY

Loudness balance tests were carried out in

(*a*) 30 cases of unilateral deafness due to Menière's disease.

(*b*) 20 cases of degeneration of the VIII nerve, due to neurofibroma of the VIII nerve and other varieties of space-occupying lesions of the cerebello-pontine angle.

Results.—Loudness Recruitment was found to be present and complete in all 30 cases of Menière's disease.

Loudness Recruitment was found to be absent in 14 of the 20 cases of VIII nerve degeneration. In the remaining 6 cases it was present but incomplete.

Loudness Recruitment was thus shown to be characteristically present in a disorder of the end-organ of hearing and to be characteristically absent in a disorder of the cochlear nerve fibres.

This latter finding sharply controverts existing views upon the neurological mechanism of recruitment. The practical and theoretical significance of the experimental results is discussed. New hypotheses are advanced in explanation of these results and are based upon recent experimental work on the physiology of hearing and upon the known pathology of the cochlea and VIII nerve.

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The following took part in the subsequent discussion:

Mr. T. Gold; Mr. E. D. D. Davis; Mr. D. W. C. Northfield; Dr. C. Worster-Drought; Dr. C. H. Edwards; Dr. N. G. Hulbert; Dr. G. D. Dawson.