an adult is attacked by the severe form of this disease characterized by haemorrhages, intense jaundice, albuminuria, and conjunctival congestion the clinical differentiation of the two diseases is by no means easy. It is in these cases that the laboratory findings are of the utmost value.

In the first place, in the absence of any conditions such as a septic focus which would affect the blood picture, the differential blood count is of great importance. In catarrhal jaundice Findlay, Dunlop, and Brown (1931) found a very distinct relative mononuclear increase. A typical count would be: polymorphonuclears 51 per cent., small lymphocytes 41 per cent., large mononuclears 3 per cent., eosinophils 4 per cent., mast cells 1 per cent. In Weil's disease, however, the most striking feature of the differential count is the polymorphonuclear increase, up to 85 or 90 per cent. being by no means uncommon.

When circumstances do not permit of the actual finding and isolation of the leptospira in Weil's disease the serological reactions are very important. Antibodies as a rule appear about the sixth day of the disease, and reach their maximum three or four weeks later. One point which must always be taken into consideration is the fact that agglutinins persist in the patient's serum for many years, and an agglutination titre, even up to 1 in 300 several years after the onset of the disease, is not uncommon (Fairley, 1934 ; Brown, 1935). This fact, together with the rise in agglutinin titre which occurs during the first fortnight of the disease, must always be considered in making a diagnosis of Weil's disease.

## Case Record

The following account of a case of jaundice in a miner whose serum agglutinated *Leptospira icterohaemorrhagiae* to a dilution of 1 in 100 bears out these points.

The patient, aged 52, who had worked in a seam of a colliery in which cases of Weil's disease had occurred, was admitted to hospital on March 20th, 1936, complaining of jaundice, epistaxis, pruritus, dark-coloured urine, and putty-like stools.

The serum gave the following agglutination reactions on three occasions against a London human strain of *Leptospira icterohaemorrhagiae*:

| 1936 | 1/10 | 1/30 | 1/100 | 1/300 | 1/1,000 |
|---|---|---|---|---|---|
| March 31st ... ... | 0 | + | + | 0 | 0 |
| April 4th ... ... | + | + | + | Trace | 0 |
| April 14th ... ... | + | + | + | 0 | 0 |

It will thus be seen that there was no appreciable rise in the titre of this serum on successive examinations.

The second point to be noticed is that the titre of the serum is much lower than would be expected to be the case considering that jaundice was well marked eleven days before the first agglutination test.

The following shows a rise in titre in a typical case of Weil's disease:

| 1935 | 1/10 | 1/30 | 1/100 | 1/300 | 1/1,000 | 1/3,000 | 1/10,000 | 1/30,000 |
|---|---|---|---|---|---|---|---|---|
| Sept. 3rd | + | + | + | 0 | 0 | 0 | 0 | 0 |
| Sept. 4th | + | + | + | Trace | 0 | 0 | 0 | 0 |
| Sept. 7th | + | + | + | + | + | 0 | 0 | 0 |
| Sept. 8th | + | + | + | + | + | 0 | 0 | 0 |
| Sept. 9th | + | + | + | + | + | + | Trace | 0 |
| Sept. 11th | + | + | + | + | + | + | + | 0 |

The second point which we consider important in the diagnosis of the case under consideration is the differential blood count, which was as follows:

| | April 1st, 1936 | April 14th, 1936 |
|---|---|---|
| Polymorphonuclears | ... 48.5 per cent. | ..... 48 per cent. |
| Lymphocytes ... | ... 39.0 ,, | ..... 38 ,, |
| Large mononuclears | ... 9.0 ,, | ..... 11 ,, |
| Eosinophils ... ... | ... 3.5 ,, | ..... 3 ,, |

This is typical of the count met with in cases of catarrhal jaundice, and in that it is present in a patient in whom there is no rise in the agglutinin titre against *Leptospira icterohaemorrhagiae* as the disease progresses it leaves very little doubt that the disease in question is catarrhal jaundice, and that the low titre of agglutination is due to a previous infection with the leptospira.

We wish to thank Dr. J. C. Spence for kindly allowing us to publish this case.

BIBLIOGRAPHY

Fairley, N. H.: *British Medical Journal*, 1934, ii, 10.
Alston, J. M., *et al.*: *Lancet*, 1935, i, 806.
Alston, J. M., and Brown, H. C.: *British Medical Journal*, 1935, ii, 339.
Davidson, L. S. P., Campbell, R. M., Rae, H. J., and Smith, J.: Ibid., 1934, ii, 1137.
Buchanan, G.: Medical Research Council, Special Report Series, No. 113, London, 1927.
Swan, W. G. A., and McKeon, J. A.: *Lancet*, 1935, ii, 570.
Findlay, G. M., Dunlop, J. L., and Brown, H. C.: *Trans. Roy. Soc. Trop. Med. and Hyg.*, 1931, xxv, 7.
Brown, H. C.: *British Medical Journal*, 1935, i, 411.

# THE OTOLITHIC CATASTROPHE

## A NEW SYNDROME

BY

A. TUMARKIN, M.B., F.R.C.S.ED., D.L.O.

HONORARY AURIST, BOOTLE GENERAL HOSPITAL

Ever since Ménière gave his famous account of vertigo and allied phenomena associated with disease of the ear the title of Ménière's syndrome has been used vaguely to include a very heterogeneous collection of conditions. With better understanding of labyrinthine function, however, some order is emerging from this chaos. For instance, a comparatively clear-cut entity is now well recognized in aural vertigo, a condition characterized by:

1. Progressive deafness, usually bilateral but almost invariably much more pronounced in one ear.
2. Attacks of vertigo, with allied symptoms of pallor, nausea, sweating, etc.

It is clear that the syndrome is referable to some degeneration, whether toxic, inflammatory, or abiotrophic, of the inner ear. Whether the lesion is primarily in the neuro-epithelium of the inner ear or, as Dandy has not very convincingly maintained, in the eighth nerve itself is not yet proven. That question, while important from the point of view of surgical intervention, will not influence the present argument.

## Anatomy of the Inner Ear

The inner ear can be divided both anatomically and physiologically into:

1. The cochlea.
2. The semicircular canals.
3. The vestibule, further subdivided into: (*a*) utricle, (*b*) saccule.

It will be noted immediately that although the organ involved in aural vertigo has a triple, if not quadruple, nature yet the symptoms evoked from it are classified under only two headings. The progressive deafness is referred to the cochlea, and the vertigo, etc., to the canals. This discrepancy is due to the fact that until comparatively recently very little was known of the

functions of the third portion, the vestibule. Nevertheless, although still somewhat enshrouded in mystery, the vestibular otoliths have been ascribed the following functions, with a high degree of probability.

### THE UTRICLE

This is a tiny sac (Figs. 1A and 1B) lined by squamous epithelium and lying in a hollow in the bony vestibule. At one spot in its floor is the lapillus, a little plateau of neuro-epithelium, *a*, from which runs the utricular nerve to join the main eighth nerve. Resting on it with the intervention of a gelatinous stroma is a mass of tiny crystals of calcium carbonate, the otoconia, *b*.

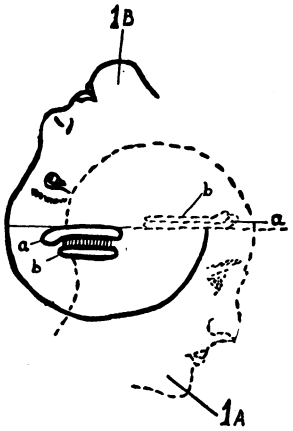


FIG. 1A AND 1B.

FIG. 2.

FIG. 3.

In the upright position (Fig. 1A) *b* is pressing on *a*. In the upside-down position (Fig. 1B) it will drag, and intermediate positions will produce intermediate effects. The right and left lapilli are synergic.

The function of this organ has been the subject of an enormous amount of research by Magnus and de Kleyn, Rademaker, Quix, Maxwell, McNally and Tait, etc. There can be no doubt that one of its functions, if not its main one, is that of influencing muscle tone. Thus experiments on decerebrate animals demonstrate quite emphatically that in its "maximum" position the lapillus augments decerebrate rigidity, and in the opposite or "minimum" position it produces flexion and flaccidity. The exact angle of each position is still in doubt; certainly it varies from one animal to another, but of the existence and importance of this phenomenon there can be no question. The effect on the human body is well shown by Figs. 2 and 3 (after Rigaud).

### THE SACCULE

The saccule (Fig. 4) is anatomically very similar to the utricle. The chief difference lies in its position in space.

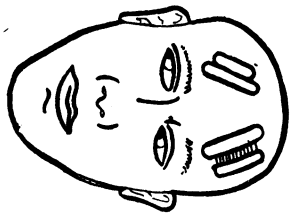

FIG. 4.

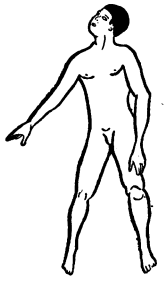

FIG. 5.

The sensitive area (sagitta) lies on the side instead of the floor of its retaining sac; furthermore the right and left sagittae are roughly at right angles to each other, so that unlike the lapilli they are opponents and not synergists.

This is not the place to discuss the work which has been done on the function of the saccule. Selective ablation experiments have seemed to indicate that it has nothing like the importance of the utricle in the maintenance of muscle tone and body posture. On the other hand, there is some reason to believe that it records sounds of low frequency by bone conduction (Ashcroft and Hallpike). From the point of view of physics and of comparative anatomy it is very likely that it subserves both functions. For the purpose of this article I shall confine myself to its influence on body posture as described by Quix, Rigaud, and others. Broadly speaking, then, the saccules act unilaterally in opposition to each other. (See Figs. 4 and 5.)

We are now in a position to visualize the missing third element in the syndrome as follows.

*The Utricle.*—Accesses of rigidity may be produced (Fig. 3), but, far more important, the utricle may produce sudden flexor spasm (Fig. 2). We have only to remember the sudden collapse produced by tapping a standing individual suddenly on the hamstrings to realize the effect of this flexor tone. The patient will double up and collapse backwards like an empty suit of clothes.

*The Saccule.*—Here similarly collapse is to be expected, but to one or other side rather than backward.

It only remains to describe cases which will illustrate these points.

### UTRICULAR AFFECTION

Mr. X. was sent to me with a history of giddiness and a discharging ear of several months' duration. He described typical attacks of giddiness in which "things went round," but in which he was always able to steady himself by clutching something or by getting to a chair to lie down. These I refer to the semicircular canals. In addition, however, he described an entirely different attack, of which the following is typical. One day he was standing at his desk talking to a client when suddenly he slumped to the floor. He had no vertigo, no loss of consciousness, and no malaise. The thing came like a bolt from the blue, but he was able immediately to assure onlookers that he was all right, and almost immediately got up and carried on. That is exactly what one would anticipate from Fig. 2.

### SACCULAR AFFECTION

Mr. Y. was sent to me on account of "giddiness." He was a typical case of aural vertigo, giving the usual account of giddy attacks in which, however, he was always able to steady himself and to draw in his van to the edge of the road (he was a motor driver). On one occasion he was standing in his van receiving packages when suddenly he felt "as if the wheel had collapsed under him." He crashed down on his side. Here again the effect was instantaneous, but passed off very rapidly. There was no true disorientation, no loss of consciousness, and no vomiting. This picture bears a close resemblance to Fig. 5.

### COMBINED CONDITION

Miss Z., another case of aural vertigo, also suffered with pansinusitis. She describes identical attacks of instantaneous onset and short duration. She dare not go down stairs, and can usually show a "black eye" or some similar result of her latest fall. She describes sagittal as well as coronal collapses, but also speaks of occasional spasms in which her arms jerk up, much as one would expect from Fig. 3.

## Conclusion

It would thus seem to be justifiable to distinguish a "canal" syndrome from an "otolith" syndrome, as in the conjoined table.

| | Canal | Vestibule |
|---|---|---|
| Onset ... ... ... ... | Ingravescent | Instantaneous |
| Duration ... ... ... ... | May be minutes up to days | A minute or less |
| Nausea, pallor, sweating, etc. | Yes | No |
| Giddiness... ... ... ... | Yes | No |
| Deafness ... ... ... ... | Variable in type; may be little or none | Definite reduction of bone conduction |

Apart from the academic interest of this phenomenon I should like to draw attention to the following points.

1. The rare fatal fall which occurs in Ménière's syndrome is almost certainly to be ascribed to the otolithic apoplexy rather than to the canals.

2. It is possible that the aural origin of some cases is not being recognized. In the absence of obvious deafness or giddiness one might easily be tempted to label a pure otolith collapse as hysterical or even petit mal.

So far as I am aware this differentiation of symptoms on an anatomical basis has not been proposed before, although the dramatic collapse has frequently been noted. Mygind and Dederding say: "Les attaques purement apoplectiformes sont rares. Ordinairement il y a des prodromes avertissants. . . . En général, l'attaque essentiellement apoplectiforme est momentanée. . . . Dans une telle attaque, le malade peut tomber par terre comme foudroye."

# Clinical Memoranda

## Sympathectomy for Dysmenorrhoea

Special interest attaches to the following case of resection of the presacral nerve and the inferior mesenteric ganglion for dysmenorrhoea complicated by severe constipation (achalasia of the pelvi-rectal sphincter).

A patient aged 33, sent by Dr. Kirby of Robertstown, Kildare, was admitted under my care to the Kildare Infirmary in January, 1933, on account of dysmenorrhoea and severe constipation. She could not remember having ever been free from constipation. During the last few years, however, it had been increasing to such an extent that her whole time had been devoted to the requirements of the bowel. For months before admission to the Infirmary an action of the bowel could only be obtained by daily self-administered enemata. To add to her misery a new train of symptoms had developed: pain and discomfort after meals mimicking the "immediate" pain of gastric ulcer rather than the "delayed" or hunger pain of duodenal ulcer; dysmenorrhoea, which was so severe that the patient was obliged to remain in bed until the pain had subsided; and bladder spasm of a most annoying character. Now and then attacks of abdominal colic occurred. The abdomen was at times slightly distended and tender to the touch. There was, however, no localized tenderness.

Before her admission to the Kildare Infirmary the patient's appendix had been removed and her cervix dilated, without any improvement in her menstrual or abdominal symptoms. On admission to the Infirmary the patient was put upon special dietary. Drugs of various sorts were administered, with little or no improvement in her condition. An *x*-ray examination of the gall-bladder and of the entire alimentary tract was then carried out Cholecystography revealed a gall-bladder which appeared to function normally. Further *x*-ray examination revealed some delay in the passage of a bismuth meal from the stomach, the contour of which appeared to be quite normal. The barium meal appeared to reach the head of the caecum in the normal time, and there was no evidence of ptosis of the ascending colon or caecum. There was considerable delay of the barium meal in the descending and pelvic colon. A fractional test meal revealed an increase in the total acidity and free HCl of the stomach. A trace of starch was found two and a half hours after the test meal had been administered. An *x*-ray examination after a barium enema revealed a slightly dilated pelvic colon and a smooth-walled descending colon showing no signs of peristalsis. An interesting point was that several pints of the enema were administered without discomfort. The case, in other words, presented the characteristic features of megacolon as it appears in the adult.

The patient was then brought to the operating theatre and spinal anaesthesia was administered. It had been my intention then to have the patient immediately transferred to the *x*-ray room, in order to take a second photograph, but the enema was evacuated before the patient left the operating table—the first spontaneous evacuation for months. The induction of splanchnic paresis by means of spinal anaesthesia was a clear indication for abdomino-pelvic sympathectomy. The intractable nature of the dysmenorrhoea was an additional reason for this procedure.

*Operation.*—The presacral nerve was divided over the promontory of the sacrum at the point where it divides into the hypogastric nerves. It was lifted off the fifth and fourth lumbar vertebrae and the bifurcation and the interior aspect of the aorta as high as the origin of the inferior mesenteric artery. The sympathetic fibres converging upon the origin of this vessel from above and from the sides were divided, and the proximal inch of the artery was completely denuded.

The immediate results of the operation were a painless menstruation, a relief of the bladder symptoms, and the cessation of attacks of abdominal colic. The gastric symptoms, however, persisted, and a daily enema was still the only effective method of securing an evacuation of the bowel. Three weeks after the operation the bowels could be moved by strong purgatives. The gastric symptoms were less frequent, and the patient was able to partake of a more generous dietary. At the end of three months she was discharged.

Shortly after her discharge she wrote to say that she occasionally required opening medicine; otherwise she had never felt so well in her life, and her weight had increased by a stone and a half. Quite recently she wrote, in reply to my queries, that she feels no gastric discomfort; that she does not require any purgatives; and that there has been no return of her menstrual or bladder symptoms.

The case described is of interest alike to the gynaecologist, the physician, and the general surgeon. Its special interest lies in the light which it appears to throw upon the relationship of the abdomino-pelvic sympathetic system to the special type of dysmenorrhoea which the case exemplifies. It is impossible within the limits of a short communication to elaborate upon this interesting relationship.

When rectal spasm with consequent constipation accompanies dysmenorrhoea it is due, as a rule, to achalasia of the anal sphincter. It may, however, as in the case described, be due to achalasia of the pelvi-rectal sphincter. These conditions can be distinguished only by *x*-ray examination of the pelvic and descending colon after the administration of a barium enema. The distinction is vital. Since most of the sympathetic fibres of the distal colon are supplied by the inferior mesenteric plexus resection of the presacral nerve alone may not completely restore bowel functions. The inferior mesenteric plexus should be resected as well.

D. J. CANNON, M.B., B.Ch., M.A.O.
Surgeon and Gynaecologist to Kildare County Infirmary, and the Drogheda Memorial Hospital, Curragh.

## A Resectoscope Result

A bricklayer, aged 46, was sent to see me at the Nottingham General Hospital with the history that four years ago a vesical calculus had been removed by suprapubic operation. The wound had healed well, but the man had never passed any urine naturally after the operation. He was entirely dependent on a No. 9 silver catheter, which he carried in his pocket wrapped up in a handkerchief along with a bottle containing liquid paraffin for lubricant.

Cystoscopy showed a trabeculated bladder with mild cystitis. The prostate could be seen as a slight elevation surrounding the internal meatus, and it appeared probable that this was the cause of the inability to micturate.

The man was admitted and endoscopic prostatic resection done, using a MacCarthy resectoscope; a catheter was tied in. On the fifth day the catheter was removed, and five hours later the man passed naturally 22 oz. of urine all at once and without effort, but with great delight.

When he was seen three weeks later as an out-patient his stream had remained undammed and his joy undimmed.

Nottingham. J. LLEWELLYN DAVIES, F.R.C.S.