

psychogenic facial tics, with which in the first instance the condition is most often confused. Often the movements continue during sleep, and it is characteristic that they are accentuated by tension and anxiety. In some cases, after many years, some facial weakness ensues on the affected side. Pain accompanying the spasmodic movements is rare.<sup>2</sup>

The cause of the condition is often unknown, though an identical syndrome can rarely be a symptom of neoplasm in the cerebello-pontine angle,<sup>3</sup> aneurysm of the basilar artery,<sup>4</sup> or arachnoiditis of the posterior fossa.<sup>5</sup> Sometimes the condition also occurs as a sequel to Bell's palsy, though more often in cases of the latter condition showing incomplete recovery fixed facial contracture rather than spasm occurs. Electromyographic recording from the affected facial muscles usually shows groups of two or three motor unit action potentials recurring spasmodically in time with the spasms, and this finding has been interpreted as indicating a state of chronic irritation of the facial nerve. K. J. Zülch<sup>6</sup> has reviewed the evidence for its aetiology in detail and has concluded that the condition probably results from an unusually narrow facial canal, which causes slight but lasting damage to the nerve, with formation of an artificial synapse at the point of compression and thus to permanent irritation. He admits that the lesion so postulated has not been confirmed histologically, but the occasional occurrence of the condition after incomplete recovery from a Bell's palsy does give some support to this view, as does evidence indicating that in some patients surgical decompression of the affected facial nerve in the Fallopian canal produces considerable improvement in the spasm.<sup>7 8</sup> T. Cawthorne<sup>8</sup> operated on 13 cases. The operation was followed by facial paresis lasting for a few weeks, but the spasms ceased, and as facial movement recovered they did not return in some cases for as long as 18 months. Others have found this procedure less successful. Thus J. M. Curtin<sup>1</sup> noted that the spasms ceased in less than half his patients. Miehke<sup>9</sup> suggested as an alternative method that 50% of each of the main branches of the nerve should be divided at the first point of branching after leaving the stylomastoid foramen, and H. Diamant and colleagues<sup>2</sup> operated on four patients, using this technique. These authors found that all patients developed some degree of facial palsy, and, though the spasms returned as this recovered in three cases, they were not so severe as before in two of them.

J. Potter<sup>10</sup> has now reported on two patients treated by selective division of *terminal* branches of the facial nerve. These were located by making a vertical skin incision at the anterior border of the masseter just below the malar eminence, separating the subcutaneous tissue by opening out artery forceps in a horizontal plane, and detecting the fine nerve twiglets by use of a facial nerve stimulator coupled to a diathermy needle. Small filaments, which when stimulated produced contraction of the lower part of the orbicularis oculi and elevation of the upper lip, were then divided. In his two patients there was no detectable facial palsy after the operation, and he attributed this finding to the anastomoses which occur between terminal branches of the facial nerve and to the fact that the muscles concerned are also supplied by the lower buccal and zygomatic branches of the nerve; yet the extent and severity of the spasm were substantially reduced. The duration of follow-up was relatively short at the time of writing, but it seems that the technique of surgical treatment which Potter described would be worth an extended trial in further cases. As already stated, the condition is so benign and self-limiting in many patients that simple sedative treatment with diazepam or similar remedies is often successful in damping down the movements sufficiently to render them

tolerable. But in those few patients in whom the movements become so severe as to constitute a major embarrassment surgical treatment deserves serious consideration.

- <sup>1</sup> Curtin, J. M., *Proceedings of the Irish Otolaryngological Society*, Tenth Annual Meeting, 1969, p. 23.
- <sup>2</sup> Diamant, H., Enfors, B., and Wiberg, A., *Laryngoscope*, 1967, 77, 350.
- <sup>3</sup> Cushing, H., *Journal of Nervous and Mental Disease*, 1916, 44, 312.
- <sup>4</sup> Campbell, E., and Keedy, C., *Journal of Neurosurgery*, 1947, 4, 342.
- <sup>5</sup> Laine, E., *Revue Neurologique*, 1948, 80, 38.
- <sup>6</sup> Zülch, K. J., in *Handbook of Clinical Neurology*, ed. P. J. Vinken and G. W. Bruyn, vol. 8, p. 277. Amsterdam, North-Holland Publishing Company, 1970.
- <sup>7</sup> Woltman, H. W., Williams, H. L., and Lambert, H. H., *Proceedings of the Mayo Clinic*, 1951, 26, 236.
- <sup>8</sup> Cawthorne, T., *Archives of Otolaryngology*, 1965, 81, 504.
- <sup>9</sup> Miehke, A., *Langenbecks Archiv für klinische Chirurgie*, 1961, 298, 923.
- <sup>10</sup> Potter, J., *Journal of Laryngology and Otology*, 1972, 86, 889.

## Liquor Licensing and Public Health

The Report of the Departmental Committee on Liquor Licensing<sup>1</sup> (chairman, Lord Erroll of Hale) appeared last week and has had a mixed reception. This is doubtless because most people enjoy an alcoholic drink at some time but all are aware that the abuse of alcohol is a considerable public health problem. In fact there is uneasiness about what is believed to be its increase in recent years. Certainly there is evidence in the report that the consumption of alcohol is increasing. The consequences to health should be the first consideration of Parliament when it comes to consider whether the law needs to be changed.

Among the report's main recommendations is one that pubs should, at the discretion of the licensee, have the right to stay open for any part or all of the hours between 10 a.m. and midnight; the statutory afternoon break would be abolished. The age limit for drinking alcohol at the bar would be lowered from 18 to 17. The most radical suggestion relates perhaps to the notion of the "café pub," which is envisaged as an institution where coffee and snacks could be served as well as alcohol and as being a place where mother and father could take along their children of any age.

These and other suggestions are made against a background of statistics of which the committee was aware and which indeed find a place in its report. In Britain the average consumption of both beer and spirits per person has shown an annual increase for every year since 1962, while consumption of wine has been steadily increasing since 1965. The committee accepts the probable validity of evidence<sup>2</sup> which suggests that any overall increase in national drinking is associated with an increase in the number of heavy drinkers rather than simply with the recruitment of a greater number of moderate drinkers. It notes that the rate of arrest for public drunkenness per head of population has been increasing each year since 1967, with the rate among young people aged under 18 rising by over 50% during the course of five years. The report also presents data on post-mortem levels of blood alcohol among drivers killed in traffic accidents. The percentage of such victims with blood levels over 80 mg/100 ml is now back to the 25% before the breathalyser was introduced, and the gains from the 1967 Road Traffic Act have in that respect seemingly been lost. The committee acknowledges the difficulties which beset any attempt to obtain an estimate of general rates of national alcoholism or to interpret any of the indices already in use. But it felt able to conclude that this country

has a total of between 200,000 and 400,000 alcoholics, and that the death rate from alcoholism has for the last 20 years been rising.

Given the committee's knowledge of a threatening public health situation, what did it envisage as the likely impact on health of its proposals? It says that the health consequences need watching and suggests that there should be continued research monitoring, but it goes on to conclude that the recommendations as a whole will probably not have any adverse effect on health. In an important passage (p. 46) the committee makes an assertion which shows something of its approach to these questions: it states that at one extreme total permissiveness would have a large adverse effect on health, while at the other extreme unacceptable repressive policies would have considerable impact of a contrary sort. Then it makes this key assertion, "Any solution which fell well within these limits would, we feel, be largely neutral in its impact on consumption."

The impression is given that the committee is set on policies of relaxation for reasons other than those concerning the public health. From the public health point of view the report's main proposals must be condemned as untimely. Their adoption in practice would be to risk a further increase in alcoholism, with its attendant dangers to harmony in the home and life on the roads.

<sup>1</sup> Report of the Departmental Committee on Liquor Licensing. London, H.M.S.O., 1972.

<sup>2</sup> Lederman, S., *Alcool—Alcoolisme—Alcoolisation: Données Scientifiques de caractère et physiologique, économique et sociale*. Paris, Presse Universitaires de France, 1956.

cularly now that more modern diagnostic techniques are available, including the raised level of gammaglobulin in the cerebrospinal fluid.<sup>14</sup> It might reasonably be questioned whether the sheathing could have any aetiological significance in relation to multiple sclerosis, since the nerves in the retina are not myelinated. However, the presence of an abnormality of the veins gives some support to the long-standing suggestion that plaques of multiple sclerosis have their origin in the first place round veins in the central nervous system.<sup>15 16</sup> No relationship to the pathological changes has yet been found in the veins showing sheathing in life. T. Fog<sup>16</sup> illustrated inflammatory cell infiltration of a retinal vein in an eye removed from a patient, though unfortunately there was no observation of retinal sheathing during life.

<sup>1</sup> Pattison, E. M., *Archives of Neurology*, 1965, 12, 197.

<sup>2</sup> Daroff, J., and Waldman, A. L., *Journal of Neurology, Neurosurgery and Psychiatry*, 1965, 28, 375.

<sup>3</sup> Fisher, C. M., *Journal of Neurology, Neurosurgery and Psychiatry*, 1967, 30, 383.

<sup>4</sup> Crews, S. J., *Hospital Medicine*, 1967, 2, 543.

<sup>5</sup> Harcourt, R. B., *British Journal of Hospital Medicine*, 1969, 2, 831.

<sup>6</sup> Holden, W. A., in *Multiple Sclerosis. Association for Research in Nervous and Mental Diseases*, 1921, 2, 102.

<sup>7</sup> McAlpine, D., Lumsden, C. E., and Acheson, E. D., *Multiple Sclerosis, A Reappraisal*. London, Livingstone, 1965.

<sup>8</sup> Leibowitz, U., and Alter, M., *Acta Neurologica Scandinavica*, 1968, 44, 70.

<sup>9</sup> Rucker, C. W., *Mayo Clinic Proceedings*, 1944, 19, 176.

<sup>10</sup> Rucker, C. W., *Association for Research in Nervous and Mental Disease*, 1950, 28, 396.

<sup>11</sup> Rucker, C. W., *Mayo Clinic Proceedings*, 1972, 47, 335.

<sup>12</sup> Field, E. J., and Foster, J. B., *Journal of Neurology, Neurosurgery and Psychiatry*, 1962, 25, 269.

<sup>13</sup> Lössner, J., Müller, J., and Bachmann, H., *Ophthalmic Literature*, 1968, 22, 734.

<sup>14</sup> Princeas, J., Teasdale, G., Latner, A. L., and Miller, H., *British Medical Journal*, 1966, 2, 922.

<sup>15</sup> Putman, T. J., and Adler, A., *Archives of Neurology and Psychiatry*, 1937, 38, 1.

<sup>16</sup> Fog, T., *Acta Neurologica Scandinavica*, 1965, Supplement No. 15, 41.

## Retinal Veins in Multiple Sclerosis

The eyes, those windows on the soul, may often give an indication of an underlying general or neurological disease.<sup>1-5</sup> Multiple sclerosis presents with optic neuritis in 10-20% of patients and with some ocular symptoms in about 30% of patients.<sup>6-8</sup>

One ocular feature of multiple sclerosis which has caused interest and discussion over the years is the sheathing of the retinal veins first described by Wilbur Rucker in 1944.<sup>9 10</sup> He has recently reviewed his own and others' experience of this condition.<sup>11</sup> Sheathing is seen as a fine, opaque border to the veins of the retina, and is thought to be of significance in relation to multiple sclerosis only if it occurs at a distance from the optic nerve head. Many conditions such as papillitis and papilloedema may cause sheathing of the veins at the nerve head. Other changes also included within the term of "sheathing" are soft, opaque clouds in the vitreous over the veins, which may be more acute phenomena. The parallel sheathing of veins may be due to slight thickening and increased visibility of the vein walls, and may remain for many years.

These changes are seen only with difficulty, and require that the pupil be fully dilated and the ophthalmoscope light not too bright. Though more than 500 cases have now been reported, other workers have failed to find evidence of retinal-vein sheathing despite diligent search.<sup>12 13</sup>

The sheathing of the retinal veins never causes symptoms, and is therefore of little importance in itself. But it may on occasions be of help in suggesting a diagnosis of multiple sclerosis, though few would rely on it, parti-

## Transmission of Cholera

The recent outbreak of cholera among passengers on an airliner travelling from Great Britain to Australia was confirmed as due to the *el tor* vibrio. The Australian authorities were quickly led to suspect the *smörgåsbord* that was taken on board for economy class passengers at Bahrain as the vehicle of infection. The water from the aircraft was shown to be perfectly satisfactory. Only economy class passengers were affected; none of the crew and first-class passengers had the *smörgåsbord*. The recognition of two other cases among passengers on a flight in the opposite direction, which had also taken on *smörgåsbord* about the same time, helped to confirm this. All the evidence was that this was a food-borne outbreak. Thus cholera, traditionally considered to be the classic example of a water-borne disease, was presenting as a food-borne infection. Many questions may be asked about the method of spread of *el tor* cholera and the significance of the cholera carrier today.

It is certainly true that countries where sewage disposal is inadequate and where a clean water supply is lacking may suffer from water-borne outbreaks. In many areas of the world water is drawn for drinking and cooking from the very rivers and ponds into which defecation takes place. But in the present pandemic of *el tor* cholera there has been much evidence of case-to-case spread. In the Philippines<sup>1</sup> the infection was found to be transmitted from person to person until a water supply was contaminated, as a result of which an explosive outbreak occurred. An explosive outbreak in Istanbul was rapidly brought under control by the