

## Noise in Hospital

Recently there has been an altogether laudable and increasing concern about pollution of the countryside by motorways, motor-cars, mineral exploration, and the like; about pollution of water, rivers, and seas by dangerous effluents or dumping of chemicals; and about pollution of the air by factories, radioactive matter, and smokers. People unfortunate enough to live near an airport have also had cause for complaint about pollution by noise. But not enough has been said about pollution by noise in hospital.

Our patients could say much about the banging of doors, the rumble of lifts, the rattle of ill-maintained trolleys, the clatter from the kitchen and the sluice or service rooms, the chatter of visitors, the tapping of nurses' and doctors' heels, and the snores, groans, or flatus of fellow patients. The effect is worse if the sounds arouse fear, as groans and cries may. How much consideration is given to ambient noise in relation to the siting of new hospitals? The roar of heavy traffic outside a hospital ward may be disturbing to patients, and the reduction of noise by double-glazing of windows may be forbidden on the ground of cost. But not all hospitals have given thought to the use of sound-proof tiles or sound-deadening walls and floors.

Some parts of a hospital are much noisier than others. R. Gädeke and colleagues<sup>1</sup> recorded the noise level in children's intensive care units and studied its effect on 126 children aged 3 to 63 weeks. A level of 75 decibels led to obvious sleep disturbances, awakening two out of three children within three minutes and all within 12 minutes. R. League and colleagues,<sup>2</sup> writing from Florida, studied the noise in oxygen tents. Near the infant's ear the level was at 80 decibels or more. Noting that sounds of this magnitude are uncomfortable for most adults and that prolonged exposure to this level may damage the ear, they called for an investigation of the matter. Prematurely born babies are somewhat more likely to have defective hearing than full-term babies, though the reason is not altogether clear. Hyperbilirubinaemia, now preventable, is a possible cause but probably not the only one. Prolonged exposure to the noise of the fan and electric motor within the incubator may be a factor, but there is no evidence about it one way or the other. R. A. Shapiro and T. Berland<sup>3</sup> studied the noise levels in the operating theatre at the Michael Reese Hospital, Chicago, built in the early 1960s. They mentioned the noise from the loud-speaker calling devices, clattering of instruments, high-pitched sounds of compressed air, motorized instruments used in orthopaedic operations, the conversation

of doctors and nurses about the patient, and banter and babble about interpersonal relations, tennis, and the latest ball-game scores. They wrote: "The noise in the operating room frequently exceeds that of a freeway, and more frequently approximates that of a kitchen with a food-blender in operation, a train, or a truck. Indeed, the noises of the operating room approximate the 90 decibels (A scale) maximum permissible noise exposure (for eight hours) of the United States Federal Occupational Safety and Health Act." And again: "Operating-room noise is intense enough to provoke peripheral vasoconstriction, dilatation of the pupils, and other subtle physiologic effects, as well as interfere with speech communication and thereby provoke irritation."

S. A. Falk and N. F. Woods<sup>4</sup> measured the noise in infant incubators, a recovery room, and intensive care units, choosing these sites because they were thought to be the noisiest. They found that the noise level in all three places was sufficient to cause distinct physiological effects in the hypophyseal-adrenocortical axis, and might damage the hearing of patients receiving any of the aminoglycoside antibiotics such as gentamicin, kanamycin, or streptomycin. And it is worth noting too that noise may double or treble corticosteroid secretion rates in rats. That hospital laboratories may be uncomfortably noisy<sup>5</sup> has been the subject of comment in these columns previously.<sup>6</sup> Yet another source of noise is described by Dr. Julian M. Leigh in the *B.M.J.* this week (page 652). He finds that some but not all types of oxygen mask can be so noisy as to be a possible cause of disturbance to the user.

Not all sound is heard or noticed, and yet it may have a considerable effect on an individual. Interest is increasing in the physiological and psychological effects of infrasound—that is, frequencies below 20 Hertz. It causes discomfort and even vomiting in susceptible people,<sup>7</sup> and there is much of this type of sound in buildings. R. Brown<sup>7</sup> suggests that the discomfort, bad temper, and other responses experienced by some people in stormy or thundery weather may be explained by the effect of infrasound, which increases in these climatic conditions. We know nothing about the number of people who are sensitive to this level of sound. Both infrasound and ordinary sound may affect the behaviour of patients in hospital, and there is a real need for some down-to-earth practical investigation of the problem there.

<sup>1</sup> Gädeke, R., Döring, B., Keller, F., and Vogel, A., *Acta Paediatrica Scandinavica*, 1969, 58, 164.

- <sup>2</sup> League, R., Parker, J., and Robertson, M., *Lancet*, 1970, 2, 978.  
<sup>3</sup> Shapiro, R. A., and Berland, T., *New England Journal of Medicine*, 1972, 287, 1236.  
<sup>4</sup> Falk, S. A., and Woods, N. F., *New England Journal of Medicine*, 1973, 289, 774.  
<sup>5</sup> Griffiths, P. D., Kell, R. L., and Taylor, W., *Journal of Clinical Pathology*, 1970, 23, 445.  
<sup>6</sup> *British Medical Journal*, 1970, 3, 662.  
<sup>7</sup> Brown, R., *New Scientist*, 1973, 60, 414.

## Signs of Multiple Sclerosis

Walter Moxon (1836-1886), who presented the first two cases of "insular sclerosis" in Great Britain, was guided in the diagnosis by Charcot's triad of nystagmus, scanned speech, and intention tremor.<sup>1</sup> Charcot's approach implied that symptoms are related to the plaques in the nervous system. This view is still widely held, as the following admonition indicates: "We must refuse to diagnose multiple sclerosis if multiplicity be absent."<sup>2</sup> But clinical experience sometimes counters this rule, since cases are seen in which signs and symptoms are indicative of but a single focal lesion. Thus, some 50% of cases of classic retrobulbar neuritis due to the disease never developed any other neurological signs during follow-up periods exceeding ten years.<sup>3-6</sup>

The following are some other well-recognized forms of mono-symptomatic presentation of multiple sclerosis: paresis of abducens or facial nerves; facial myokymia;<sup>7</sup> trigeminal neuralgia;<sup>9,10</sup> various forms of "internuclear ophthalmoplegia"; vertigo; progressive spastic paraplegia;<sup>11</sup> sensory disturbances, often of symmetrical distribution and not showing any obvious sensory deficit; and Lhermitte's signs (the development of electric-like shocks down the body when the patient flexes the head).<sup>12</sup> Despite even advanced and typical pathological changes in the central nervous system, signs and symptoms may be entirely absent. In some 18% of cases of multiple sclerosis discovered in the course of a large series of consecutive necropsies no relevant symptoms had been noted during life.<sup>13</sup> Again, neuropathological evidence of long-standing disease may be in striking contrast to the brevity of the clinically active phase.<sup>14</sup>

Sometimes such silent cases of multiple sclerosis may betray themselves only during periods of rise of body temperature, whether caused by muscular exercise or fever. It is now generally recognized that transient temperature-related impairment of vision—unilateral or bilateral—must be regarded as a symptom of multiple sclerosis even when other clinical features of this disease are absent.<sup>15,16</sup> This effect of a rise in body temperature intensifying symptoms of multiple sclerosis or provoking new ones forms the basis of a test.<sup>17</sup> Lowering of body temperature may in contrast give temporary relief of symptoms.

Since experimental heating and cooling of nerve tissue result in functional changes opposite to those observed in multiple sclerosis, it has been suggested that the phenomena observed in this disease in response to changes of temperature are due to hormonal influences.<sup>18,19</sup> It is assumed that the hormones concerned are those released during thermal regulation, and it is postulated that they may reduce or increase conduction in regions of central demyelination.<sup>20,21</sup> After G. D. Dawson devised a technique of recording cerebral action potentials it became possible to estimate nerve conduction in man's central nervous system.<sup>22</sup> A delay of cortical responses on stimulation of the median nerve has been detected in 75% of cases of multiple sclerosis.<sup>23-25</sup> A similar

delay of cortical responses was also found in cases of multiple sclerosis when a flash of light was used as a visual stimulus.<sup>26,27</sup> A stimulus engendered by the reversal of a checker board pattern has now been used for the study of cortical potential responses in multiple sclerosis, and its value as a diagnostic test is reported on by Dr. A. M. Halliday and colleagues in our Medical Practice section (page 660) this week. A direct comparison of the results obtained with flash and pattern stimuli has previously shown many advantages gained when the pattern stimulus is used,<sup>28</sup> and 93% of patients with multiple sclerosis who gave no history of optic neuritis were found to have delayed cortical responses.

It is difficult to explain these findings, as it is some other clinical phenomena in multiple sclerosis, on the basis of Charcot's "insular" concept. The plaques may perhaps be regarded as representing the "last of a series of changes," some of which may be more widespread than light microscopy would suggest.<sup>29</sup> Indirect support for this inference may be found in investigations proceeding along several different lines.

In biopsy material obtained from the brain of patients suffering from multiple sclerosis thinly myelinated axons with widened nodal gaps have been identified by means of electron-microscopy.<sup>30</sup> Histochemistry has shown abnormal forms of myelin in multiple sclerosis.<sup>31</sup> Reactions produced on cultures of brain tissue by the addition of sera from patients with multiple sclerosis indicate that such sera contain highly specific factors which are toxic to myelin and oligodendrocytes, and also have an inhibitory effect on synapses.<sup>29,32</sup> Whether speculations on these lines will eventually come to be substantiated or refuted the observations made by Halliday and colleagues on visually evoked responses in multiple sclerosis, when scrutinized as they have suggested, should prove of great value in diagnosis—"an art which consists largely of balancing probabilities."<sup>33</sup>

- <sup>1</sup> Hospital Practice, *Lancet*, 1875, 1, 471 and 609.  
<sup>2</sup> Kennedy, F., *Research Publications Association for Research in Nervous and Mental Diseases*, 1950, 28, 524.  
<sup>3</sup> Kurland, L. T., Auth, T. L., Beebe, G. M., and Kurtzke, J. F., *Transactions of the American Neurological Association*, 1963, 88, 231.  
<sup>4</sup> Collis, W. J., *Archives of Neurology*, 1965, 13, 409.  
<sup>5</sup> Bradley, W. G., and Whitty, C. W. M., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1968, 31, 10.  
<sup>6</sup> Taub, R. G., and Rucker, C. W., *American Journal of Ophthalmology*, 1954, 37, 494.  
<sup>7</sup> Andermann, F., Cosgrove, J. B. R., Lloyd-Smith, D. L., Gloor, P., and McNaughton, F. L., *Brain*, 1961, 84, 31.  
<sup>8</sup> Matthews, W. B., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1966, 29, 35.  
<sup>9</sup> Olafson, R. A., Rushton, J. G., and Sayre, G. P., *Journal of Neurosurgery*, 1966, 24, 755.  
<sup>10</sup> Chakravorty, B. G., *Archives of Neurology*, 1966, 14, 95.  
<sup>11</sup> Bramwell, B., and Dawson, J. W., *Edinburgh Medical Journal*, 1916, 17, 411.  
<sup>12</sup> Brody, I. A., and Wilkins, R. H., *Archives of Neurology*, 1969, 21, 338.  
<sup>13</sup> Georgi, W., *Schweizerische medizinische Wochenschrift*, 1961, 91, 605.  
<sup>14</sup> Russell, D. S., *Lancet*, 1964, 1, 978.  
<sup>15</sup> Earl, C. J., *Transactions of the Ophthalmological Societies of the United Kingdom*, 1964, 84, 215.  
<sup>16</sup> *British Medical Journal*, 1972, 2, 122.  
<sup>17</sup> Davis, F. A., *Journal of the Mount Sinai Hospital*, 1966, 33, 280.  
<sup>18</sup> Paintal, A. S., *Journal of Physiology*, 1965, 180, 20.  
<sup>19</sup> Heiss, W. D., Heilig, P., Heyer, J., *Experimental Brain Research*, 1968, 4, 321.  
<sup>20</sup> Hopper, C. L., Matthews, C. G., and Clelland, C. S., *Neurology*, 1972, 22, 142.  
<sup>21</sup> McDonald, W. I., and Sears, T. A., *Brain*, 1970, 93, 583.  
<sup>22</sup> Dawson, G. D., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1947, 10, 137.  
<sup>23</sup> Namerow, N. S., *Bulletin of the Los Angeles Neurological Society*, 1968, 33, 74.  
<sup>24</sup> Baker, J. B., Larson, S. J., Sances, A., and White, P. T., *Neurology*, 1968, 18, 286.  
<sup>25</sup> Namerow, N. S., *Neurology*, 1968, 18, 1197.  
<sup>26</sup> Richey, E. T., Kooi, K. A., and Tourtelotte, W. W., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1971, 34, 275.  
<sup>27</sup> Namerow, N. S., and Enns, N., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1972, 35, 829.  
<sup>28</sup> Halliday, A. M., McDonald, W. I., and Mushin, J., *Lancet*, 1972, 1, 982.  
<sup>29</sup> Bornstein, M. B., in *Central Nervous System*, ed. O. T. Bailey and D. E. Smith, p. 70. Baltimore, Williams and Wilkins, 1968.