Episodic Blindness

"I do not know what importance is to be attached to temporary losses of sight when the appearances in the fundus are normal, and when there are no nervous symptoms."[1] It can scarcely be gainsaid that the physician faced with this phenomenon today is no less puzzled than was Hughlings Jackson 100 years ago. "I used to call cases of temporary failure of sight, epilepsy of the retinae; but since I cannot know that the retina is really the part at fault, I now use the term epileptiform amaurosis." Earlier the syndrome was termed "periodic amaurosis" and more recently acquired the now familiar name of amaurosis fugax.[2]

As a causative factor of episodic blindness Bright's disease was among the first to be recognized. Gowers's ophthalmoscopic observation[3] of reduction in the size of retinal arteries in some cases of Bright's disease resulted in the erroneous concept of spasm of retinal arteries—"angiospasm" is the term in vogue in ophthalmic literature. Because of their transparency the retinal vessels in the normal eye are virtually invisible. They show up by virtue of the blood they contain. The column of blood within the retinal vessels is in part determined by the ratio between intravascular and intraocular pressures and in part by the dimensions of the vascular lumen. I. C. Michaelson found that the narrowing of retinal blood vessels in hypertension usually persists after therapeutic lowering of blood pressure and inferred from this that the reduction in "size of the vessels was due to organic changes in the vessel wall."[4] "Angiospasm" also presupposes vasomotor adrenergic innervation, yet histological studies have shown that in man only few adrenergic fibres accompany the retinal vessels.[5 6] The assumption that transient cessation of retinal circulation as observed in amaurosis fugax is due to retinal "angiospasm" has therefore been criticised.[7] Episodic blindness in Bright's disease is due to the fact that the visual cortex appears to be particularly sensitive to ischaemia engendered by vasconstriction, which is a feature of all forms of arterial hypertension caused by increased peripheral resistance. Loss of vision in Bright's disease may recur for varying periods or may persist until treatment brings about an appreciable fall in blood pressure.[8] The presence of slow delta activity in the occipital leads of the encephalogram may assist the diagnosis if this is in doubt when ophthalmoscopic examination shows no abnormality.

Recently repeated transient cortical visual loss has been described in association with paroxysmal hypertension caused by what might be called autonomic hyperreflexia. When a transverse lesion of the spinal cord is above the level of the thoracolumbar sympathetic outflow—that is, above the fifth thoracic segment—stimuli such as bladder distension may evoke uninhibited sympathetic reaction resulting in intense generalized vasoconstriction and cortical blindness.[9] Episodic cortical blindness which occurs in porphyria may also have a similar basis of paroxysmal vasoconstriction.[10 11] In migraine the cortical disturbances of vision may also be due to vasoconstriction, though localized.[7] Recurrent transient loss of vision of cerebral origin is not uncommon among patients whose vertebro-basilar circulation is compromised by occlusive disease.[12 13] This episodic blindness is frequently associated with vertigo, and must be differentiated from vestibulogenic vertigo conjoined with syncopal reaction.[14]

Bilateral dimness or blindness is in some patients provoked by rising from the stooping or sitting positions. In a patient of Jonathan Hutchinson's "rising suddenly would place her for a short time in total darkness".[15] Usually this phenomenon occurs in healthy individuals but is also encountered in association with papilloedema or with obstruction of the great vessels at or near their origin from the aortic arch.[16] These patients with the so-called "aortic arch syndrome" show in addition to absence of pulses in the arteries evidence of long-standing retinal hypoxia, appearing as "venous stasis."[17 18] Measurements of intra-arterial blood pressure in persons subjected to tipping from horizontal to head-up position have shown transient and opposing pressure changes in the brachial and carotid arteries. While the pressure in the brachial artery at heart level rises by as much as 20 mm Hg, the fall in the carotid is on average 40 mm Hg.[19] Clearly this physiological fall in the intracarotid pressure when occurring under certain pathological conditions may result in transient regional ischaemia, with consequent loss of vision.[20]

Blackout, signifying temporary blindness, was a term introduced in aviation medicine in the 1914-18 war. It refers to the condition wherein as a result of positive gravitational force a person cannot see although his mind is functioning. When under these conditions the effective systolic arterial pressure falls to about 20 mm Hg, vision is lost and the retinal arteries are seen to be empty of blood.[21] It can be presumed that similar retinal circulatory disturbances occur in other conditions associated with fall in blood pressure, such as syncope, postural hypotension, and anaemia. Episodic disturbances of this nature may also occur without a fall in blood pressure in apparently healthy persons after exercise, after a hot bath, and after a heavy meal. This unusual, though physiological, response may however first appear when the optic nerves are affected by disease, especially as a sequela of retrobulbar neuritis.[22 23] Here must also be mentioned midline aneurysms and tumours—especially meningiomas and pituitary tumours. By pressing on and causing ischaemia of the chiasm or optic nerves, they may engender blindness followed by spontaneous remissions.[24 25] This is especially a feature of "apoplexy" of pituitary tumours, a condition which, however, is associated with severe headaches.[26] Such spontaneous remission of blindness may lead to erroneous diagnostic conclusions. Finally, it is worth bearing in mind that all the lesions which have been listed as causing amaurosis fugax may on occasions result in permanent blindness.

[1] Jackson, J. H., *Medical Times and Gazette*, 1871, **2**, 341.
[2] Middlemore, R., *Treatise on the Diseases of the Eye and its Appendages*. London, Longmans, 1835.
[3] Gowers, W., *British Medical Journal*, 1876, **2**, 743.
[4] Michaelson, I. C., in *Ocular Circulation in Health and Disease*, ed. J. S. Cant, p. 109. London, Kimpton, 1969.
[5] Laties, A. M., *Archives of Ophthalmology*, 1967, **77**, 405.
[6] Ehinger, B., *Investigative Ophthalmology*, 1966, **5**, 42.
[7] Behrman, S., *Archives of Ophthalmology*, 1951, **45**, 458.
[8] Lennon, P. A., *et al.*, *Australian and New Zealand Journal of Medicine*, 1971, **1**, 346.
[9] Rosenberg, R. S., Mitchell, A. M., and Lester, H. A., *Archives of Ophthalmology*, 1969, **81**, 325.
[10] Martin, W. J., and Heck, F. J., *American Journal of Medicine*, 1956, **20**, 239.
[11] Goldstein, N. P., Wartin, W. J., Brunsting, L. A., and Kirby, T. J., *Mayo Clinic Proceedings*, 1957, **32**, 82.
[12] Minor, R. H., Kearns, T. P., Millikan, C. H., Siekert, R. G., and Sayre, *Archives of Ophthalmology*, 1959, **62**, 84.
[13] Bradshaw, P., and McQuaid, P., *Quarterly Journal of Medicine*, 1963, **32**, 279.
[14] Behrman, S., *Brain*, 1955, **78**, 471.
[15] Hutchinson, J., *Archives of Surgery (London)*, 1892, **4**, 184.
[16] Ross, R. S., and McKusick, V. A., *Archives of Internal Medicine*, 1953, **92**, 701.
[17] Kearns, T. P., and Hollenhorst, R. W., *Mayo Clinic Proceedings*, 1963, **38**, 304.

[18] Font, R. L., and Naumann, G., *Archives of Ophthalmology*, 1969, **82**, 784.
[19] Loman, J., Dameshek, W., Myerson, A., and Goldman, D., *Archives of Neurology and Psychiatry*, 1936, **50**, 510.
[20] Behrman, S., *British Journal of Ophthalmology*, 1967, **51**, 269.
[21] Duane, T. D., *Archives of Ophthalmology*, 1954, **51**, 343.
[22] Earl, C. J., *Transactions of the Ophthalmological Societies*, 1964, **84**, 215.
[23] Nelson, D. A., Jeffreys, W. H., and McDowell, F., *Archives of Neurology*, 1958, **79**, 31.
[24] Rucker, C. W., and Kearns, T. P., *American Journal of Ophthalmology*, 1961, **51**, 15.
[25] Cogan, D. G., *Archives of Ophthalmology*, 1961, **66**, 180.
[26] Robinson, J. L., *Journal of Neurosurgery*, 1972, **36**, 83.

Pollution in the Operating Theatre

Though the occupational hazard to industrial workers from the inhalation of the noxious vapours of solvents such as trichloroethylene, benzene, or carbon tetrachloride is well known, the possibility of a similar risk for those working in the atmosphere of an operating theatre is less so. The suggestion that even this atmosphere might affect people's health has recently received some attention in Scandinavia and America.

The particular aspect of ill health studied in two recent reports concerned the incidence of spontaneous abortion among married women working in operating theatres. By means of a questionnaire V. Askrog and B. Harvald,[1] in Copenhagen, found that among nurse anaesthetists there were 10 abortions plus perinatal deaths out of 85 pregnancies (12%) before they entered this employment, and there were 44 out of 229 (19%) after they entered it. The difference is not significant at the 5% level, but when similar figures for women anaesthetists and even the wives of men anaesthetists (which showed a similar disparity) were added in, the result was a significant difference at the 0·1% level. Out of 212 pregnancies before employment in anaesthetics 21 ended in abortion or perinatal death (10%), whereas out of 392 pregnancies afterwards 80 ended in abortion of perinatal death (21%). There was also a significantly greater number of premature deliveries among the women who gave birth after taking up anaesthetic work. A study at Stanford University School of Medicine in California gave similar results to the Danish one. E. N. Cohen and colleagues[2] obtained information by personal interview from 67 operating-room nurses and 92 general duty nurses, and found that in 1966-70 30% of pregnancies in the first group ended in spontaneous miscarriage, while 9% in the control group did so. By means of questionnaires they obtained confirmatory figures in a comparison between practising anaesthetists and doctors in other specialties. In both these series the women at risk were a little more than three years older than the control group, and there may also have been undisclosed differences between them. Despite these doubts the statistical differences found between those exposed and those not exposed to the atmosphere of the theatre is notable.

Apart from these reports about specific effects many people who work in operating theatres experience the discomfort of a stuffy atmosphere. Anaesthetists in particular are well aware of the consequences of a long day spent in close proximity to their patients' expired breath. There is a widespread, if vague, feeling among them that headache and fatigue, resulting perhaps in inattention, are common in poorly ventilated theatres.[3] In addition it is known that anaesthetists absorb measurable amounts of anaesthetics. Occasionally these may be sufficient to show up an existing illness. Two anaesthetists are reported to have developed sensitivity to halothane, suffering jaundice as a result of their occupational exposure.[4] A nurse-anaesthetist developed signs of myasthenia gravis after administering methoxyflurane but was unaffected by some other drugs.[5] There is also the possibility that the incidence of malignant tumours of lymphoid tissue is higher in anaesthetists than in the general population.[6]

Anaesthetic drugs are potent poisons. In experimental animals some of them are teratogenic, but only after long exposure to concentrations within the range used clinically in man. Similarly, fetal death can be produced in experimental animals. In man it has proved impossible to determine the incidence of teratogenic effects or abortion after anaesthesia and surgery.

The reports about abortion provide some evidence of a possible occupational hazard, but more information is needed and is being sought. Whatever conclusion is reached on the validity of these data, there seems to be enough evidence already to suggest that a general improvement in the standards of ventilation of operating theatres, with removal of anaesthetic and other gases or vapours, is urgently required. Perhaps in addition anaesthetists and nurses should spend only limited periods of time in the operating theatre.

[1] Askrog, V. F., and Harvald, B., *Nordisk Medicin*, 1970, **83**, 498.
[2] Cohen, E. N., Bellville, J. W., and Brown, B. W., *Anesthesiology*, 1971, **35**, 343.
[3] Whitcher, C. E., Cohen, E. N., and Trudell, J. R., *Anesthesiology*, 1971, **35**, 348.
[4] Klatskin, G., and Kimberg, D. V., *New England Journal of Medicine*, 1969, **280**, 515.
[5] Elder, B. F., Beal, H., DeWald, W., and Cobb, S., *Anesthesia and Analgesia*, 1971, **50**, 383.
[6] Bruce, D. L., Eide, K. A., Linde, H. W., and Eckenhoff, J. E., *Anesthesiology*, 1968, **29**, 565.

Chondromalacia Patellae

The term chondromalacia patellae is used to denote a condition affecting fit persons in which pain arises from the posterior aspect of the patella. The earliest recognized account of the clinical condition[1] described changes in the cartilage of the patella which were thought to be due to trauma, and despite a later account of 640 cases[2] in which evidence for trauma was found in two-thirds the exact cause of the syndrome remains a mystery.

Disorders of the cartilage on the posterior aspect of the patella give rise to characteristic symptoms and physical signs. The main symptom is of pain in the region of the patella, sometimes associated with a sensation of "giving-way" or "locking"—though these seldom actually occur. The pain is worst when the quadriceps muscle contracts most strongly, notably when descending stairs or rising from a chair. The knee itself may look quite normal, though quadriceps wasting is frequent and joint effusions have been described. The range of movement is full. The posteromedial and posterolateral aspects of the patella can be palpated when it is displaced and are tender, and clinical tests involving compression of the patella against the femoral condyles are painful. Radiographs are usually normal,