ing labyrinth. There have been no further episodes of meningitis, and the boy was discharged well one year after surgery.

Comment

Leaks of cerebrospinal fluid from the ear usually follow trauma and have been classified as open or closed, depending on whether or not the tympanic membrane is intact.³ Rarely, a spontaneous leak of cerebrospinal fluid from the ear may occur, and recurrent attacks of meningitis have complicated nearly all the reported cases. When the tympanic membrane is intact and the leak of cerebrospinal fluid is only small or intermittent tracer studies may not show the leak, and failure to locate accurately the anatomical defect may result in fruitless neurosurgical exploration. Most congenital cerebrospinal fluid fistulas in the region of the petrous temporal bone, and especially those that occur spontaneously, are associated with abnormalities of the bony labyrinth and internal auditory meatus.⁴ Complex motion tomography or high resolution computed tomography will show

these abnormalities of the inner ear and should be considered for any deaf child with a suspected leak of cerebrospinal fluid or unexplained meningitis. The high pressure of cerebrospinal fluid against the oval window eventually causes a rupture, allowing the egress of fluid and the ingress of pathological organisms. Once the defect has been located surgery to plug the leak will prevent further attacks of meningitis.

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Clinical Topics

Anticonvulsant drugs and advice about driving after head injury and intracranial surgery

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In 1976 driving licences in the United Kingdom became valid until the driver is aged 70. Since then drivers have had a statutory duty to report both relevant and prospective disabilities—conditions that might affect their fitness to drive.¹ ² Epilepsy is a relevant disability which is, however, compatible with holding an ordinary driving licence under certain conditions that mainly relate to a stipulated period of freedom from attacks. The risk that epilepsy may begin after a head injury or craniotomy may constitute a prospective disability. Patients whose licence is revoked even temporarily in such circumstances, however, may be very resentful; their surgeons often share this resentment³ ⁴ and will sometimes support an appeal against the Driving Vehicle Licensing Centre. The surgeon then usually states that the risk of epilepsy in the patient is minimal, yet it often transpires that he has prescribed prophylactic anticonvulsant drugs.

This paper reports the results of a survey of current practice among a sample of British neurosurgeons in respect of prescribing anticonvulsant drugs and advice about driving.

Methods

A questionnaire was sent to all full and associate members of the Society of British Neurological Surgeons. Replies were received from about half, but these 55 surgeons represented almost all the regional units in the United Kingdom. For several diagnostic categories surgeons were asked whether after performing a craniotomy they prescribed anticonvulsants always, sometimes, or never. When their reply was "sometimes" they were asked what preoperative, operative, and postoperative features influenced the decision to give anticonvulsants. They were then invited to indicate separately what levels of a risk of epilepsy in the first year they considered to justify either prescribing anticonvulsants or advising against driving; bands of risk from which they chose were 5-10%, 10-20%, 20-30%, 30-40%, and more than 40%. Further questions related to details of prescribing practice. The study was designed primarily to discover how neurosurgeons ranked the relative risk of epilepsy in different circumstances, not to canvass opinion about whether or not anticonvulsants should be given or whether they are believed to be effective when there is a risk of epilepsy.

Results

All but one of the respondents always prescribed anticonvulsants for some of their patients. The proportion of surgeons who always gave postoperative drugs in various conditions showed a reasonable correspondence with the relative risks of epilepsy reported for these conditions (table I). Asked what factors other than the diagnosis led them to recommend anticonvulsants, almost all surgeons cited the occurrence of preoperative or early (first week) postoperative epilepsy. Other preoperative features that influenced about half the surgeons included

focal neurological signs, involvement of the cerebral cortex, and intracerebral haematoma. When the two last factors became obvious at operation (whether the result of the primary lesion or of the surgical procedure) about half the surgeons then recommended anticonvulsants. Postoperative features other than early epilepsy that led to prophylactic drugs were complications such as intracranial haematoma or infection, cerebral infarction, prolonged coma, or focal neurological signs.

A third of surgeons gave antiepileptic drugs before or during operation, and almost half began treatment immediately after surgery. A third of surgeons recommended drugs for only six months, but 40% did so for more than a year; only a third routinely checked the blood concentrations of drugs.

A third of neurosurgeons did not suggest any restrictions on driving to their patients who had recently recovered after craniotomy. A quarter told them not to drive for a few weeks, but only a fifth proposed that driving should be postponed for more than three months. Nevertheless, almost half the surgeons indicated that they considered that a risk level of less than 10% justified stopping someone driving a private vehicle, while over 80% would stop such patients driving a heavy goods or public service vehicle (table II).

TABLE I—Prescribing of anticonvulsant drugs after craniotomy for various conditions: proportion of neurosurgeons who always recommended drugs postoperatively

75%	Brain abscess
60%	Meningioma
	Arteriovenous malformation (meningioma)
55%	Subarachnoid haemorrhage
	Middle cerebral artery aneurysm
	Compound depressed skull fracture
45%	Intracerebral haematoma
	Anterior or posterior communicating artery aneurysm
	Glioma
	Repair for anterior fossa fracture
35%	Pituitary tumour
15%	Chronic subdural haematoma
15%	For all these conditions
18%	For all except subdural
2%	For none of these conditions

TABLE II—Advice related to risk of epilepsy

Risk of epilepsy (%)	% of neurosurgeons recommending				
	Anticonvulsant drugs		No private driving		No driving of heavy goods or public service vehicles
	Cumulative		Cumulative		Cumulative
<5	8		6		25
5-10	45	53	39	45	57
10-20	25	78	21	66	8
20-40	11	89	19	85	10
>40	11	100	15	100	100

Discussion

There is clearly widespread awareness by neurosurgeons that head injury and intracranial surgical procedures carry a risk of producing epilepsy. Indeed, many more British neurosurgeons are now claiming to prescribe anticonvulsant drugs after head injury than were found to be doing so in a study conducted in the 1950s and 1960s⁵; the proportion using drugs in that study was similar to the professed practice of American neurosurgeons who were surveyed in 1972.⁶ None the less, neurosurgeons may still underestimate the actual risk of epilepsy in their patients, particularly in those who have had operations for non-traumatic conditions. Not every neurosurgeon carries out systematic follow up of all his patients, many of whom in Britain are transferred only temporarily to the neurosurgical unit from other consultant services; late complications that do not require surgery may not be reported to the neurosurgeon. Lack of awareness of the degree of risk may therefore influence the advice that neurosurgeons give to patients soon after operation, and later to employers or to the Driving Vehicle Licensing Centre.

Evidence is accumulating about the risk of epilepsy after different types of head injury⁵ and after intracranial surgery for non-traumatic conditions.⁷ The risk varies widely according to the pathological condition and the surgery performed, but its level may be estimated when the patient leaves hospital. Those who have a substantial risk of epilepsy need to be told about this, and that their future as drivers is in doubt. If they are vocational drivers in charge of heavy goods or public service vehicles the sooner they consider changing their jobs the better. For private drivers a self imposed ban for a year may be sufficient, but they should be advised to inform the Driving Vehicle Licensing Centre and perhaps also their insurance company (which might refuse to meet claims if it considered that a relevant disability had been concealed). The estimated risk that an adult of working age will de novo develop epilepsy appears to be about 0.03% each year for the age group 20-59 years, judging from findings in several different populations studied at the Mayo Clinic.⁸ Compared with that, a prediction that epilepsy will develop in 15% or 30% of certain neurosurgical patients represents an increased risk of 500 and 1000 times; even at 5% the increase is 167-fold. These levels of risk are in fact quite common in postoperative and post-traumatic patients, even when allowance is made only for fits occurring in the first year.

Whether anticonvulsant drugs effectively reduce this risk has not yet been established. Several reports maintain that epilepsy occurred despite drugs or after they were discontinued. A recent trial from Australia, in which care was taken to maintain adequate treatment with phenytoin for a full year after operation, showed no appreciable benefit in the treated cases at the end of one year, and additional patients developed epilepsy during the second year once medication had been discontinued.⁹ Until there is reliable evidence for effective prophylaxis it remains important for neurosurgeons to ensure that patients and their doctors are informed of the risk of epilepsy after head injury or intracranial surgery.

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Is there any evidence to support the popular belief that food taken within an hour or so of bedtime is more likely to be laid down as permanent fat than food taken early in the day?

There is little real evidence to show that the timing of meals affects body weight but there is some evidence that the loss of heat as dietary induced thermogenesis is increased by physical activity, so that this loss would be reduced after a meal late at night.—D A T SOUTHGATE, head, Division of Nutrition and Food Quality, Norwich.