controlled trials (RCTs). However, no RCTs were available (as Eden, and the guidelines to which Pyke refers, note), and we had to content ourselves with observational studies. We should remember that no evidence for safety is not the same as evidence for danger. What is important is that we did not miss any trials.

Legal issues worry many doctors, even when decisions are supported by best research evidence,[3] but, rather than pose a medicolegal threat, we believe that this evidence-based approach is more likely to *protect* doctors. Why? Failures in communication are the most common preventable cause for doctors being sued by patients.[4] Yet, taking the trouble to find empirical information such as this and then discussing it with the patient is surely the most effective way of communicating the pros and cons of different treatment strategies (including, we agree, alternatives such as those mentioned by Hitchins). In the end the patient has to decide on the basis of the risks and benefits, and the choice can often be extremely difficult. It is likely to be more dangerous to assume the patient has abdicated this responsibility to the doctor without checking first. Can doctors be sued for a "safe" decision that leaves a patient exposed to unnecessary symptoms? It may be dangerous to assume that doctors can play "safe" in any one direction.

Why do experts take exception when non-experts delve in their areas for the best evidence to manage patients? After all, a cat may look at a king.[5] Experts seem to welcome the attention, but seem to think they should be dispensing the information.

snapshot

Collapse

AN 88-YEAR-OLD MAN was admitted to hospital with "collapse", manifested as hypotension and fever. It was noted that he had gaze palsies.

He had been admitted for a three-day period, 12 days earlier, with a urinary tract infection, which was treated with intravenous, then oral, antibiotics. On this occasion he was again started on intravenous antibiotic therapy. He was seen by an ophthalmologist, and a cranial computed tomography (CT) scan was arranged.

Later that evening, the ward nurses sought further medical review, as the patient remained hypotensive, with a systolic blood pressure of 80 mmHg. On examination he was observed to have a partial left third cranial nerve palsy and complete right third cranial nerve palsy (see Box, A).

A presumptive diagnosis of pituitary apoplexy was made, and the patient was commenced immediately on intravenous fluids and hydrocortisone 100 mg, 12-hourly. The CT scan (Box, B) confirmed the diagnosis, showing a pituitary tumour measuring 12 mm in diameter. The salient feature is the variegated appearance, suggesting haemorrhage within the tumour.

In patients presenting with the constellation of collapse and gaze palsies, a diagnosis of pituitary apoplexy should be considered. The mechanism of third-nerve palsy is illustrated in the Box (C). The condition is life-threatening, but responds well to appropriate treatment. After the patient had undergone hypophysectomy, the gaze palsies took about three months to resolve completely. The patient remains active on pituitary replacement therapy only.

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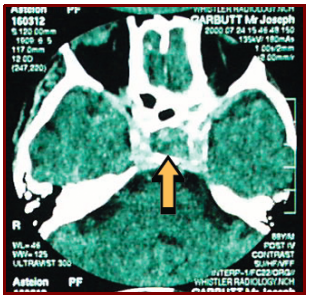
Acknowledgement: I would like to thank Dr W Ryan and the patient for allowing me to present this case. ❑

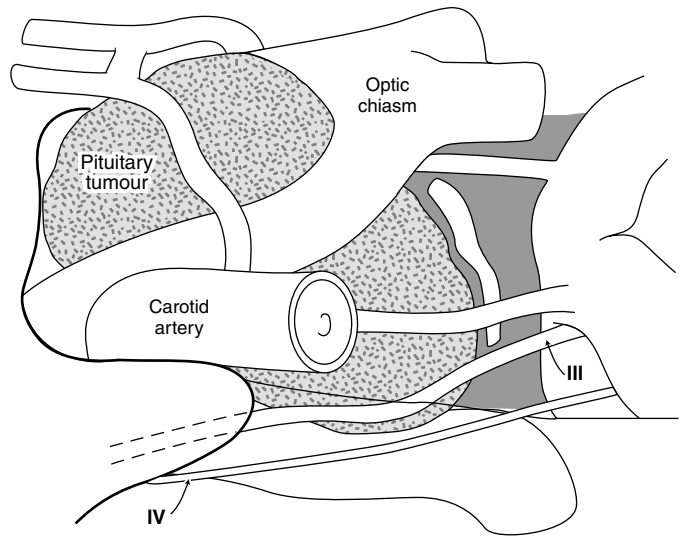
Cranial nerve palsy caused by pituitary tumour

A: Patient's eyes



B: Computed tomography scan

The picture of the patient's eyes 12 hours after commencing hydrocortisone (A) shows partial left third cranial nerve palsy and complete right third cranial nerve palsy. A computed tomography image (B) shows a 12 mm diameter pituitary tumour with haemorrhage (arrow). The mechanism of third cranial nerve palsy is illustrated in C: as the tumour expands, it involves the optic chiasm anteriorly and displaces the carotid siphon laterally. The third cranial nerve is vulnerable to lateral displacement. (Adapted from Patten J. Neurological differential diagnosis. *New York: Springer Verlag, 1982.)*



C: Mechanism of third cranial nerve palsy