as a consequence of axonal injury produced by tensile strain in human DAI. Membrane channels opened at the time of injury may therefore be a mechanism by which an influx of calcium occurs at the damaged node. Thus in the next few years, there should be concerted efforts to identify treatments which prevent calcium mediated secondary axonal degenerations. We suggest that study of voltage dependent calcium channels is likely to be the most rewarding since present evidence indicates that receptor mediated channels are limited to the cell soma and possibly dendrites. Other possibilities include the blockade of voltage dependent calcium channels, the inhibition of calcium activated proteases and the mitigation of the effects of the cascade of events that result from calcium mediated phospholipase activation, especially involving free radicle events.

Unless some progress can be made along these lines the outlook for patients who sustain severe diffuse axonal injury will remain bleak.

J HUME ADAMS
D I GRAHAM
Institute of Neurological Sciences,
Glasgow
Department of Neurosurgery,
University of Pennsylvania
W L MAXWELL
Department of Anatomy,
University of Glasgow

6 Nevin NC. Neuropathological changes in the white matter following head injury. J Neuropath Exp Neurol 1967;26:77-84.

Neurological stamp

Rhazes (850–923)

Named after the town where he was born (Rai, near modern Tehran), Rhazes was one of the great figures in Arabian medicine. His most noteworthy contributions were the distinction between smallpox and measles, and the use of animal gut in sutures. In a treatise on anatomy he described the recurrent laryngeal nerve.

Rhazes practiced in the town of his birth and later in Baghdad. It is said that when he was asked to choose a site for the hospital there, he hung pieces of meat at various points in the city, and selected the place at which putrefaction was longest delayed. He was honoured on a stamp issued by Syria in 1968 (Gibbons No 995, Scott No C414).