HISTORICAL NOTE

James Collier (1870–1935) and uncus herniation

This note outlines the origins of the recognition of brain herniation caused by raised pressure. Observers in the 19th century failed to recognise the importance of the signs accompanying brain herniation. James Collier described cerebellar tonsillar herniation in 1904. He observed false localising signs in consecutive cases of intracranial tumour that were examined clinically and pathologically. Later accounts of Kernohan's notch were followed by the newer concept that decreased alertness corresponded to a distortion of the brain by horizontal rather than vertical displacement.

The clinical signs and physical mechanisms underlying intracranial hypertension and brain swelling were not understood until the mid-20th century. Why patients, subsequently shown to harbour brain swellings, suddenly became comatose and then usually perished was a mystery. One of the first references to causal brain herniation was made by Collier,¹ who clearly described cerebellar tonsillar herniation in 1904. He observed accompanying false localising signs in 20 of 161 (12.4%) consecutive cases of intracranial tumour examined clinically and pathologically. He commented:

In many cases of intracranial tumour of long duration, it was found postmortem that the posterior inferior part of the cerebellum had been pushed down and backwards into the foramen magnum and the medulla itself somewhat caudally displaced, the 2 structures together forming a cone-shaped plug tightly filling up the foramen magnum.

Supratentorial lesions were the commonest cause. Collier thought that earlier diagnosis reduced the frequency of false localising signs, although an incidence of 12.4% was substantial. He deduced that tonsillar herniation was therefore a late sign. A year after Collier, Alquier² described cerebellar coning in patients with brain tumours: "heterotopie du cervelet dans le canal la rachidien [heterotopia of the cerebellum into the spinal canal]". Alquier noted that the autopsy showed tissue displacement, "forced to migrate due to the pressure ...". He also quoted the opposite and uncharacteristically erroneous opinion of Pierre Marie (1853–1940), who believed that herniation was a postmortem artefact:

I had the occasion to see a good number of such cases; in my opinion, the trauma of autopsy projects a portion of the cerebellum through the occipital foramen, it is thus a postmortem lesion.²

Eminent observers had failed to recognise the importance of the signs accompanying brain herniation. For example, in 1867, Jonathan Hutchinson (1828–1913) described unilateral pupillary dilatation (Hutchinson's pupil) but did not relate it to transtentorial descent, and William Macewen (1848–1924) considered pupillary dilatation to be a sign of nerve irritation, paralysis or vascular changes in connection with the cerebral lesions.³ In 1896, Hill documented the production of a transtentorial pressure gradient in dogs by instilling fluid into a pouch in the supratentorial space. Hill showed a pressure dissociation between the supratentorial and infratentorial compartments and concluded that two distinct tamponade points occurred at the foramen magnum and the tentorial notch.⁴ After Collier, Meyer (1866–1950), in 1920, observed a supratentorial tumour-causing tentorial herniation, which

impinged on the posterior cerebral artery and caused a hemianopia. He reported:⁵

The falx and tentorium constitute an important protection against any sudden impacts of pressure by keeping apart heavy portions of the brain, but they also provide an opportunity for trouble in case of swelling or need of displacement.

Brain herniation classically causes false localising signs⁶ when the free edge of the tentorium compresses the contralateral crus cerebri in the midbrain, causing Kernohan's notch.^{4,7} A tumour, haematoma, abscess or oedema initiates herniation of the temporal lobe through the tentorial incisura, detectable by CT or MRI. Early signs of uncus herniation in a CT scan include encroachment on the suprasellar cistern, displacement of the brain stem, enlargement of the ipsilateral crural subarachnoid space and compression of the contralateral cerebral peduncle.⁸

More recent observations on cerebellar pressure coning have raised doubts about its reputed lethal connotations.⁹ Ropper¹⁰ regards uncus herniation as a passive rather than an active process because the mesencephalon is twisted by the expanding cerebral mass. Contrary to traditional concepts, early stupor or coma correspond to distortion of the brain by lateral displacement rather than vertical transtentorial herniation with brainstem compression. Fisher⁹ states that herniation is a harmless accompaniment. Bilateral brain stem compression in acute bilateral cases must be distinguished from herniation. Upward cerebellar herniation is the only sign of an overfull posterior fossa. Subfalcine herniation is tolerated unless lateral displacement is excessive.

James Stansfield Collier (1870–1935)

James Collier, son of a doctor, studied medicine at St Mary's Hospital, London, graduating MB in 1894 and MD in 1896. After junior appointments, he became assistant physician to the National Hospital for the Paralysed and Epileptic, London, in 1902, and a full physician in 1921. In 1903 he was elected assistant physician at St George's Hospital, London, UK. Honours followed: the Royal College of Physicians, Lumleian Lecture 1928, FitzPatrick Lectures 1931–2 and Harveian oration ("Inventions and the outlook in neurology") in 1934. He was a frequent contributor to *Brain* and wrote chapters in Allbutt and Rolleston's *System of medicine*. With his friend WJ Adie (1886–1935), he was responsible for the section on neurology in *Price's textbook of medicine*. At a time when Queen Square was replete with colossi of clinical neurology, Collier attracted students from far and near. He used¹¹

... every histrionic trick of display, eloquence, gesture and emphasis ... despite a weakness for exaggeration, he possessed an uncanny clinical flair and diagnostic brilliance ... the elegant phrases, falling to a whisper as with the mannerisms of a magician, ...

His practice and teaching were inspiring, thanks to his prodigious clinical memory, penetrating insight and theatricality. In his leisure he busied himself in fly fishing, stamp collecting and archaeology. He lived and died in his house in Wimpole Street, London.

His most esteemed work is his comprehensive analysis of *Subacute combined degeneration of the spinal cord*,¹² written in

1900 in collaboration with JSR Russell and FE Batten. He wrote on cerebral diplegia, Babinski's sign, epilepsy, aphasia, agnosia, cerebral aneurysms and peripheral neuritis. Collier clarified the mechanism of clinical signs in total transverse spinal cord lesions. He pointed out the combination of migraine and tension headache (transformed migraine) in 1922.

Denny-Brown's¹¹ accolade is his lasting memorial:

Without doubt Collier did more to mold medical opinion and contributed more to the betterment of clinical neurological ability of internists in general than any other man of his time.

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