

Unilateral Fixed Dilation of the Pupil as a False-localizing Sign with Intracranial Hemorrhage: Case Report and Literature Review

Laurence A.G. Marshman, F.R.C.S.,
Charles E. Polkey, M.D., Christopher C. Penney, F.R.C.R.
Department of Neurosurgery, King's College Hospital, Denmark Hill, London, England

OBJECTIVE AND IMPORTANCE: Although other focal signs may prove "false localizing," it is a neurosurgical axiom that unilateral fixed dilation of the pupil occurs ipsilateral to a supratentorial mass.

CLINICAL PRESENTATION: A 25-year-old man collapsed with a dense right hemiplegia and a Glasgow Coma Scale score of 6 (eye opening, 1; motor, 4; verbal, 1) after rupture of a left middle cerebral artery aneurysm associated with an intrasylvian hematoma. Initially, both pupils had remained equal-sized and reactive; however, within hours, the right (contralateral) pupil became fixed and dilated (i.e., false localizing). For some time, the left (ipsilateral) pupil remained small and reactive; at emergency craniotomy, this also became fixed and equally dilated.

INTERVENTION: After evacuation of the clot and wrapping of the aneurysm, both pupils rapidly became equal-sized and reactive. Twenty-four hours later, concurrent with massive left hemispheric swelling and a midline shift, the left (ipsilateral) pupil became unilaterally fixed and dilated (i.e., false localizing). Eventually, the right (contralateral) pupil also became fixed and dilated, concurrent with cardiovascular collapse. Death occurred within 10 hours.

CONCLUSION: Unilateral fixed dilation of the pupil in patients with hemispheric mass lesions may be false localizing. Furthermore, disparate "herniating mechanisms" can arise despite mass effect emanating from the same side. Because such mechanisms cannot be witnessed, their nature remains speculative. An extensive review is contained in this article. (*Neurosurgery* 49:1251–1256, 2001)

Key words: False localizing, Iridoplegia

CASE REPORT

A 25-year-old Ukrainian male immigrant was found unconscious, slumped on a toilet. His breathing was irregular and the gag reflex was depressed. He was intubated and ventilated (without paralysis) by paramedics, and intravenous diazepam was admin-

istered for a presumed seizure. Pulse rate and blood pressure were 73 beats and 116/64 mm Hg, respectively; both pupils appeared equal in size (4 mm) and reactive to light. Routine investigation at the local Emergency Department revealed the only abnormality to be a serum potassium of 2.9 mmol/L. A loading dose of phenytoin was admin-

istered in view of the presumed seizure. Computed tomographic scanning (*Fig. 1*) demonstrated a large 3.5 × 5.5-cm hemorrhage in the left sylvian fissure, with extensive subarachnoid but modest intraventricular extension. There was significant mass effect, but only minor midline shift. No significant change occurred in cardiovascular or pupillary parameters during transfer to our center, and his core body temperature was 36.8°C. The Glasgow Coma Scale score on arrival was 6 (eye opening, 1; motor, 4; verbal, 1), with flexion to painful stimulation on the left side, and paralysis on the right. He was sedated with propofol and observed. Arterial blood gases were maintained within normal ranges, with oxygen saturations of 98 to 100% and pCO₂ values of 30 to 35 mm Hg. During this time, both pupils remained equal in size and reactive to light (directly and consensually). Each pupil, however, decreased to 2 mm over the next 2 hours, concomitant with sedation (40). At this stage, the right (contralateral) pupil became unreactive to light (directly or consensually) and progressively dilated to 6 mm over a period of 30 minutes, with a slightly oval outline. The left pupil remained unchanged (2 mm) and reactive. Valproate was administered in addition to intravenous mannitol. Repeat CT scanning (*Fig. 2*) showed a slight increase in mass effect and in midline shift as a result of swelling. Immediately preoperatively, the left pupil became fixed and dilated as well, also with a slightly oval outline. Neither pupil appeared to respond to mannitol. No significant change occurred in motor or hemodynamic parameters throughout this period and the patient remained apyrexial.

At emergency craniotomy, without angiography, copious clot was evacuated from the left sylvian fissure surrounding a large, ruptured, left middle cerebral aneurysm. The aneurysm could not be safely dissected and was wrapped with cotton wool. Within 2 hours of surgery, both pupils became equal in size (2 mm) and reactive to light, directly and consensually. The left

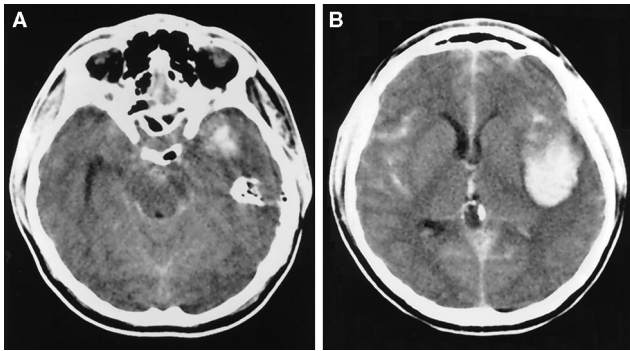


FIGURE 1. A and B, serial CT scans at presentation where both pupils were equal and reactive to light. There is extensive hemorrhage spreading around the left sylvian fissure and into the adjacent left temporal lobe. There is also considerable, and wide-

spread, subarachnoid hemorrhage, with a small amount of intraventricular hemorrhage. Moderate mass effect is apparent with minor midline shift to the right.

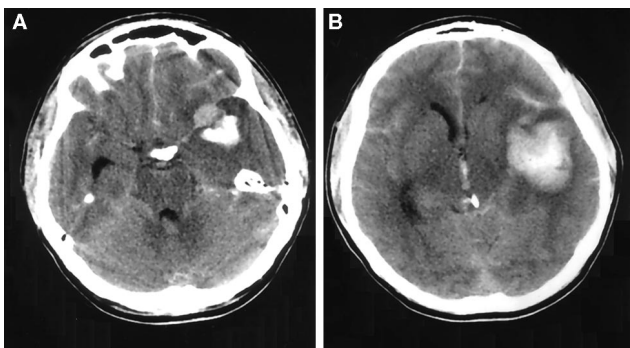


FIGURE 2. A and B, serial CT scans at the first deterioration where fixed dilation of the contralateral pupil was false localizing. Extensive hemorrhage remains in relation to the left sylvian fissure and the left temporal lobe. An underlying middle cerebral

artery aneurysm is probably visible. Local edema is apparent with mass effect distorting the left lateral ventricle; however, only minor midline displacement (particularly subfalcine) is apparent to the right. Despite this, right ventricular distention suggests that some degree of obstruction has occurred at the contralateral foramen of Monro. The left ambient cistern is clearly patent, which implies that gross ipsilateral uncal herniation has not occurred yet.

arm and leg continued to flex to painful stimuli; the right side remained hemiplegic. Clinical parameters remained stable for approximately 24 hours, after which the left (ipsilateral) pupil gradually became fixed (directly and consensually) and dilated (6 mm). No concomitant change occurred in motor or hemodynamic parameters, and the right pupil remained small (2 mm) and reactive. CT scanning demonstrated low attenuation in left middle and partial posterior cerebral arterial territories, consistent with infarction (Fig. 3). There was considerably increased mass effect with distortion of the upper brainstem. However, there was no evidence of significant further hemorrhage. Despite the administration of intravenous mannitol,

the left pupil remained fixed and dilated; no further clinical deterioration was apparent for more than 20 hours. After this time, however, the right pupil also became fixed and dilated (5 mm). The mean arterial pressure fell to 65 mm Hg, with a central venous pressure of 11 cm H₂O, despite fluid challenge and inotropic support. Cardiovascular collapse became inexorable, and the patient died 10 hours later. The patient's family refused post-mortem examination.

DISCUSSION

Localizing signs with supratentorial masses, in particular contralateral hemiparesis, were known to the ancient Egyptians (1). The localizing value of

the pupil, however, was not specified until the 19th century, despite pictorial evidence from 16th century woodcuts (7, 15, 30). In 1830, Bright (2) was the first to describe a case of unilateral iridoplegia, along with bradycardia and depressed consciousness, associated with an ipsilateral extradural hematoma. Macewen (25) later described similar cases in a patient series with skull fractures. Cushing (8) noted, in 1908, that miosis briefly preceded progressive mydriasis with hemispheric compression. Hutchinson (19) in 1867, specifically correlated ipsilateral iridoplegia and intracranial hemorrhage with ipsilateral oculomotor compression. Although Meyer (27) later identified uncal herniation with hemispheric compression, Reid and Cone (32) specifically correlated this with oculomotor distortion and iridoplegia. Despite concern by Jefferson (20) that iridoplegia related to brainstem distortion rather than the oculomotor nerve, he is credited with the modern concept of the "tentorial pressure cone."

Between 1953 and 1958, Sunderland (38, 39) extensively studied normal anatomic relationships at the tentorial hiatus. He found considerable variation in the size of the brainstem-tentorial aperture available for uncal herniation. The widest apertures occurred in older subjects, where atrophy prevailed. Because uncal-oculomotor contact was frequently noted in Sunderland's specimens, he proposed that ipsilateral uncal compression of the superolateral oculomotor nerve, where parasympathetic pupilloconstrictor fibers coursed, was the first event in tentorial coning. Initially this "irritated" the fibers, causing transient ipsilateral miosis (as Cushing had observed); paralysis ensued, with serial mydriasis. Further compression ultimately affected more resilient somatic fibers, leading to complete ipsilateral oculomotor paralysis (38, 39). This sequence has since become established neurosurgical dogma and has been confirmed clinically in cases of intracerebral hemorrhage (34, 35). In these studies, loss of light reflex was the first ocular sign; complete oculomotor paralysis was delayed, in exact agreement with Sunderland (38, 39).

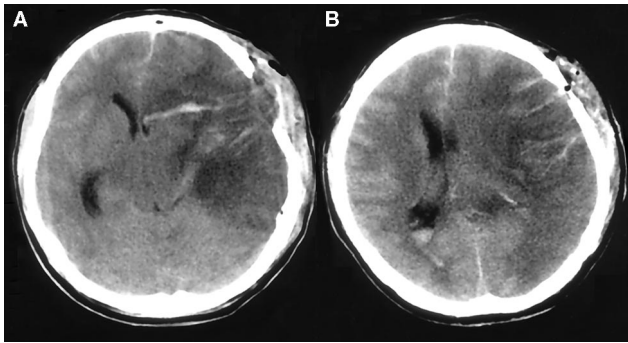


FIGURE 3. A and B, serial CT scans at the second deterioration where fixed dilation of the ipsilateral pupil was true localizing. Extensive low attenuation in the left middle (and part of the left posterior) cerebral artery territories suggests infarction in these regions.

This is associated with significantly increased mass effect and midline displacement to the right, with distortion of the upper brainstem and compromise of the contralateral quadrigeminal cistern. Both the recent craniotomy and the evacuation of the left-sided hematoma are clearly evident.

The concept of false-localizing signs with intracranial mass lesions, first categorized by Collier (6) in 1904, was later epitomized by Kernohan and Woltman (22) in 1929. Kernohan and Woltman showed that hemiparesis could be ipsilateral when, as a result of lateral brainstem shift, contralateral peduncular notching occurred at the tentorial edge. In similar fashion, other signs, such as hemianopia, could also prove false localizing; depending on brain shifts (14, 20, 31). How true localizing, then, is Hutchinson's pupil? Pevehouse et al. (31) found a larger contralateral pupil in 9 (9%) of 101 patients with acute or chronic subdural hematomas with anisocoria. More significantly, Brower (4) found a larger contralateral pupil in 29% of 132 patients with acute or chronic subdural hematomas and anisocoria. Gassel (14), in 250 patients with intracranial meningiomas, found the larger pupil to be false localizing in as many as 12 (80%) of 15 patients with anisocoria. Unfortunately, all of these studies were essentially retrospective, with little or no mention of temporal pupil reactivity, and with heterogeneous case-mixes, including both acute and chronic herniations. Furthermore, anisocoria is present in up to 20% of a normal population (40). However, Schwartz and Rosner (37) found a larger contralateral pupil with invariable loss of light reflex in 9 (50%) of 18 patients with space-occupying lesions and anisocoria. More recently, Chen et al. (5) de-

scribed a case in whom unilateral iridoplegia occurred contralateral to an acute frontal intracerebral hemorrhage.

An important advance in determining the mechanism of mass-induced iridoplegia came from the experiments of Jenet and Stern in 1959 (21). In contrast to Reid and Cone (32), Jenet and Stern showed that unilateral hemispheric balloon compression could cause rapidly reversible iridoplegia without significant uncal herniation and oculomotor compression (21). Moreover, the contralateral pupil was larger in 12% (without obvious explanation at postmortem). An initial, transient miosis was not commented upon in any animal studied; while electrical conduction studies implicated the oculomotor nerve as the site of dysfunction. Questioning Reid and Cone's mechanism, Jenet and Stern envisaged oculomotor nerve "impingement" against "unyielding structures" as etiological. However, they also cited Thompson and Malina's (41) concept of "dynamic axial brainstem distortion," where supratentorial pressure causes the brainstem to buckle during its descent as a result of its caudal tethering by way of the first dentate ligament. Oculomotor dysfunction could therefore also relate to brainstem dysfunction, as Jefferson (20) had stated. Weintraub (44) and, later, Hassler (17) elaborated this concept by postulating descent-induced traction of vulnerable paramedian brainstem arteries; this occurred as a result of their rostral tether-

ing by the circle of Willis. Such traction resulted in brainstem ischemia and, with rupture in extreme cases, midbrain hemorrhages, a key pathological feature of terminal coning (28, 42). Although some have since dismissed brainstem descent as merely a late epiphenomenon (12, 13), others have clearly shown that it is both acute and proportional to the size of the supratentorial mass (43). Furthermore, abnormal pupillary responses have been correlated with critically reduced brainstem blood flow clinically (33).

The precise pathophysiology of iridoplegia with supratentorial mass lesions, necessarily speculative in any one case (5, 12, 34, 35), may clearly be either "peripheral" (oculomotor nerve [19, 21, 23, 32, 38, 39, 44]) or "central" (brainstem [12, 13, 17, 18, 30, 33]). Postmortem evidence, lacking in our case and in the recent case of Chen et al. (5), casts light mainly on terminal events (13). The presence of oval pupils does not localize the compressive focus to any particular region (11, 26). The uniqueness of our case relates to the fact that lateralization of initial iridoplegia differed completely between two clinical scenarios, despite mass effect emanating from the same side. In the first deterioration, the contralateral pupil became fixed and dilated long before any abnormality occurred in the ipsilateral pupil. In this instance, mass effect related to left sylvian fissure hemorrhage (Figs. 1 and 2). After decompressive surgery, both pupils regained normal size and reactivity, a situation remained for 24 hours. In the second clinical deterioration, the ipsilateral pupil dilated first. Mass effect here related primarily to hemispheric swelling as a result of left middle and partial posterior cerebral arterial infarction (Fig. 3). Clearly, two distinct herniating mechanisms had occurred with two, temporally distinct, but unilateral pathologies.

Lateral brainstem movement in the transverse plane slackens the nerve in the direction of translation (i.e., the contralateral oculomotor nerve) (35, 38, 39, 44). Simultaneously, it pulls taut the ipsilateral nerve, bowstringing it around the posterior clinoid process (35, 38, 39, 44). A low brainstem oculomotor origin (39, 44), with each nerve coursing up-

ward through the interpeduncular cistern, also predisposes to nerve bowstringing during brainstem descent. Moreover, since the basilar artery is tethered to the brainstem, each nerve is increasingly slung around its corresponding posterior cerebral artery as coning proceeds (44). Sunderland (38, 39) found that the upper surface of the normal oculomotor nerve was frequently grooved by either the posterior cerebral artery or the posterior communicating artery. Conversely, the lower surface was often grooved by an elevating superior cerebellar artery of high basilar origin (38, 39). More recently, Lang (24) showed that the oculomotor nerve may even be pierced at its origin by a posterior cerebral arterial branch in up to 14% of autopsies. Such normal variants clearly suggest a predisposition to oculomotor nerve tethering that could be incurred during even the mildest of brain shifts, a concept clearly compatible with Jennet and Stern's experimental findings (21). Kerr and Hollowell (23) demonstrated that the oculomotor nerve is the most pressure-sensitive intracranial structure to correlate with mydriasis; the fact that complete oculomotor paralysis is infrequently seen with mass lesions (13) may instead implicate traction as the mydriasis-evoking stimulus (21).

Other mechanisms are also possible. For example, the contralateral oculomotor nerve may become wedged against its adjacent tentorial edge by the posterior cerebral-superior cerebellar arterial angle during "midline shift" (23, 38, 39). This mechanism, possibly in evidence in Figure 2A, could adequately account for the contralateral iridoplegia detailed in one of the patients described by Schwartz and Rosner (37). However, brainstem rotation may also occur with hemispheric mass lesions in the sagittal, coronal, or axial plane. For example, it is possible that axial rotation may occur such that the contralateral brainstem moves posteriorly (38, 39). If so, then this would pull taut the contralateral nerve while relaxing the ipsilateral nerve. Such axial rotation, combined with a low oculomotor origin, could explain the occurrence of contralateral iridoplegia before the effects of ipsilateral uncal herniation in the first of our patient's deteriorations.

Weintraub (44) noted cases of proximal oculomotor bruising, the presumed result of posterior cerebral artery descent and compression, that were also inexplicably more marked on the contralateral side. It should be noted that stimulation of the ipsilateral hippocampus (such as may occur with herniation) also elicits contralateral mydriasis (16, 23).

It is difficult to explain why the ipsilateral pupil dilated first in the patient's second deterioration. One possibility is that, should ipsilateral uncal herniation have eventually ensued in the first deterioration, a partial herniation is likely to have persisted despite oculomotor recovery (21). This, and the fact that a nerve previously distorted possesses a lower threshold for a second insult (38, 39), would encourage earlier ipsilateral oculomotor dysfunction with reherniation. Most likely, a more important factor was that the second deterioration related to hemispheric edema rather than sylvian fissure hemorrhage. Jefferson (20) thought that, in view of the inferolateral tentorial slope, downward compression would initially encourage medial temporal lobe movement away from the tentorial edge ("... its tentlike form ... protects the midbrain"). This could explain delayed ipsilateral uncal herniation in the first deterioration. In contrast, ipsilateral uncal herniation was considered far more likely with hemispheric edema because expansion would be radial (20, 36). Thus, a residual medially displaced left uncus, along with a process more likely to encourage its herniation anyway, could conceivably account for earlier ipsilateral iridoplegia in the second deterioration. Partial left posterior cerebral artery infarction (Fig. 3) suggests that left uncal herniation had ultimately occurred here. Alternating asymmetries in brainstem blood flow (33), however, are not likely to explain *both* deteriorations (29). Furthermore, gross hemodynamic fluctuations, a tenet of "dynamic axial brainstem distortion" (34, 39), were not observed in either acute deterioration (but only, instead, terminally with bilaterally fixed and dilated pupils). Consistent contralateral hemiplegia throughout both deteriorations is probably explained by vertical straightening of

the ipsilateral peduncle as a result of localized mass effect (38, 39).

Other alternative causes of anisocoria should be acknowledged in this discussion. The possibility of corneal contamination with iris-active drugs is currently an ever-present risk in intensive care units (40). Spontaneous variations in pupil size and reactivity, reflecting balance variations in autonomic tone, may occur physiologically (3, 40) or as a result of migraines (40). Furthermore, autonomic innervation may itself be fundamentally asymmetric in head and neck structures (9). In all of these examples, the simultaneous loss of light reflex would be unusual. Nonconvulsive status epilepticus could also explain the clinical findings; however, a full loading dose of phenytoin and valproate had been administered, in addition to intravenous diazepam. One final differential diagnosis could be that of a "Behr pupil." This phenomenon relates to prolonged (often persistent) contralateral mydriasis after an ipsilateral cerebrovascular accident (18). The pathology is presumed to be cortical, or subcortical, in the contralateral hemisphere and not within the optic nerve or tract, as Behr had originally postulated (18). Importantly, rapid recovery of normal pupillary size and function after surgical decompression, by definition, excludes this possibility, as indeed, it effectively excludes all others (18). It could be, however, that some of the excess false-localizing pupils witnessed in an earlier series related to this (or other) variants.

CONCLUSION

Unilateral fixed dilation of the pupil with hemispheric mass lesions can be "false localizing." More than one "herniating" mechanism can occur in the same patient despite mass effect emanating from the same side.

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Reprint requests: Laurence A.G. Marshman, F.R.C.S., Department of Neurosurgery, King's College Hospital, Denmark Hill, London SE5 9RS, England. Email: l.a.g.marshman@btinternet.com

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COMMENTS

This is a good review of the mechanisms of pupillary dilation secondary to herniation. As reported from the Traumatic Coma Data Bank (1) and from the Medical College of Virginia (2), it is not uncommon for pupillary dilation to serve as a false-localizing sign of an intracranial hematoma. Hence, its occurrence per se is not surprising. What remains enigmatic, however, is the mechanism by which this phenomenon occurs. Marshman et al. have reviewed the proposed explanations nicely. However, I must confess that I still do not clearly picture in my mind how these mechanisms actually work.

Raj K. Narayan
Philadelphia, Pennsylvania

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This article provides a nice review of pupillary inequality as a false-localizing sign. It is important to realize that mass lesions can be heralded by contralateral pupil dilation in up to 10% of cases. With the advent of near universal access to rapid computed tomographic scanning, this is not as great a problem as in prior decades; however, there are still cases where even the time taken for a fast computed tomographic scanning is inappropriate and the surgeon must rely on clinical localizing findings. This article will heighten awareness of this possibility. A sidelight is the clinical management of this patient which, I suspect, might vary in other centers.

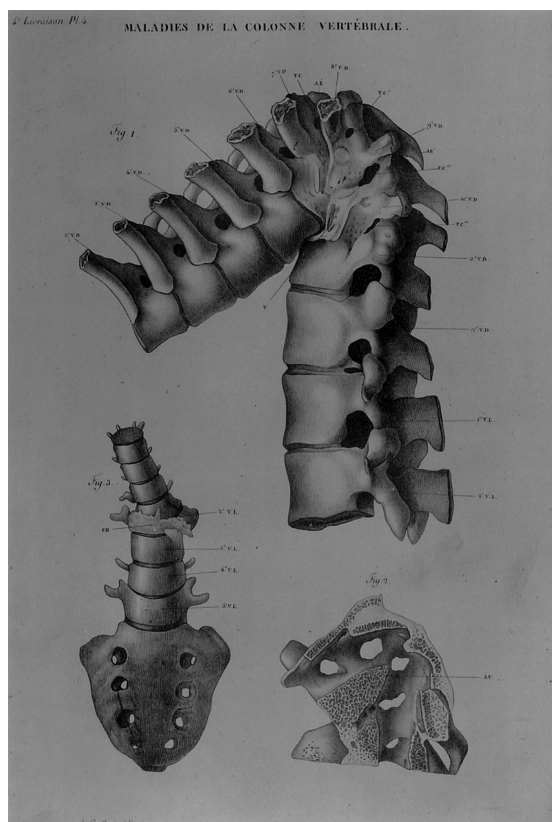
Charles J. Hodge, Jr.
Syracuse, New York

Marshman et al. have provided an interesting and well written review regarding the localizing value of the pupil in the assessment of intracranial masses. They concentrate in particular on the possible causes for ipsilateral versus

contralateral compression. They present a case report of a patient who developed both ipsilateral and contralateral papillary dilation. Unfortunately, in the absence of postmortem examination, it is impossible to determine whether the

speculation as to the causes in this patient is correct.

Steven E. Feldon
Neuro-ophthalmologist
Los Angeles, California



Examples of bony destruction of the vertebral column. From, Jean Cruveilhier, *Anatomie pathologique du corps humain, ou Descriptions, avec figures lithographiées et coloriées, des diverses altérations morbides dont le corps humain est susceptible.* Paris, Baillière, 1829–1842, vol. 1. (Courtesy, Rare Book Room, Norris Medical Library, Keck School of Medicine, University of Southern California, Los Angeles, California.)