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Recovery Following Acute Pontine Hemorrhage

Janice M.T. Redmond, MB, MRCPI,* and B.K. Ahmad MD*

Massive pontine hemorrhage does not always carry a fatal prognosis. A case report is presented in which many of the features usually associated with a fatal outcome were seen: severe disturbance of consciousness with early onset of coma, decerebrate posturing, systolic BP ranging from 200 mm Hg to 250 mm Hg, and hypopnea. Intensive supportive therapy was instituted during the critical phase, and the patient recovered. Electrophysiological studies helped to define the neurological deficit. (Henry Ford Hosp Med J 1986;34:6-10)

Primary pontine hemorrhage accounts for 5% to 9% of all intracerebral hemorrhages. This condition is associated with a grave prognosis, but there have been occasional case reports with a favorable outcome following the advent of computed tomography (CT) brain scanning to aid accurate diagnosis. Herein, a case is reported in which many of the features usually associated with a fatal outcome were seen, including severe disturbance of consciousness with early onset of coma, decerebrate posturing, systolic BP ranging from 200 mm Hg to 250 mm Hg, and respiratory failure. The patient recovered.

Case Report

A 52-year-old man was found on the ground in a parking lot and was brought to the hospital's emergency room. On admission he was noted to be drowsy and dysarthric. His BP was 220/110. The pupils were

miotic. Ocular bobbing (intermittent downward jerks of the eyes followed by a slow return to the primary position) and skew deviation were noted. There was a left peripheral facial nerve paresis and a flaccid hemiparesis associated with a sensory loss to pin prick. The bladder was distended.

An emergency CT brain scan showed an acute hemorrhage in the pons compressing the fourth ventricle anteriorly (Fig 1). Initial management included a nitroprusside infusion for control of hypertension and the administration of penicillin to treat an aspiration pneumonitis. The patient's condition deteriorated over the next two hours. He became quadriplegic and had decerebrate posturing to painful stimuli. The pupils were fixed, 1 mm in diameter. Caloric responses could not be elicited. Artificial ventilation was required due to hypopnea. Acute hydrocephalus was considered but excluded by another CT brain scan.

The patient was transferred to the intensive care unit. Within eight hours the patient moved his left side on command and responded to his name by opening his eyes. Painful stimuli produced withdrawal of the right lower extremity. The pupils were 2 mm in diameter and reactive. Six hours later the patient could move all four extremities. Sweating occurred only on the right side. An EEG performed at that time revealed a mild diffuse slowing of background rhythms (Fig 2). Within two days of admission the patient was successfully weaned from the ventilator, and normal strength had returned to all four extremities. There was right-sided hyperreflexia and a right Babinski sign. Oral nutrition was successfully commenced with a low salt diet.

Attempts to remove an indwelling urinary catheter failed. A cystometrogram showed a hyporeflexic bladder with a residual capacity of 800 mL to 900 mL. Intermittent self-catheterization at regular intervals was recommended.

Furosemide (80 mg/d), atenolol (300 mg/d), and prazosin (10 mg/d) were required for hypertension control. Serum creatinine was 3.0 mg/dL, and urinary sediment examination was compatible with hypertensive nephrosclerosis.

Nine days after admission, brain stem auditory evoked potentials showed bilateral dysfunction at the level of the rostral pons, which was more pronounced on the left side (Fig 3). Somatosensory evoked potentials in the upper extremities were normal (Fig 4). Blink reflexes con-



Fig 1—CT brain scan on admission showing massive pontine hemorrhage (arrow).

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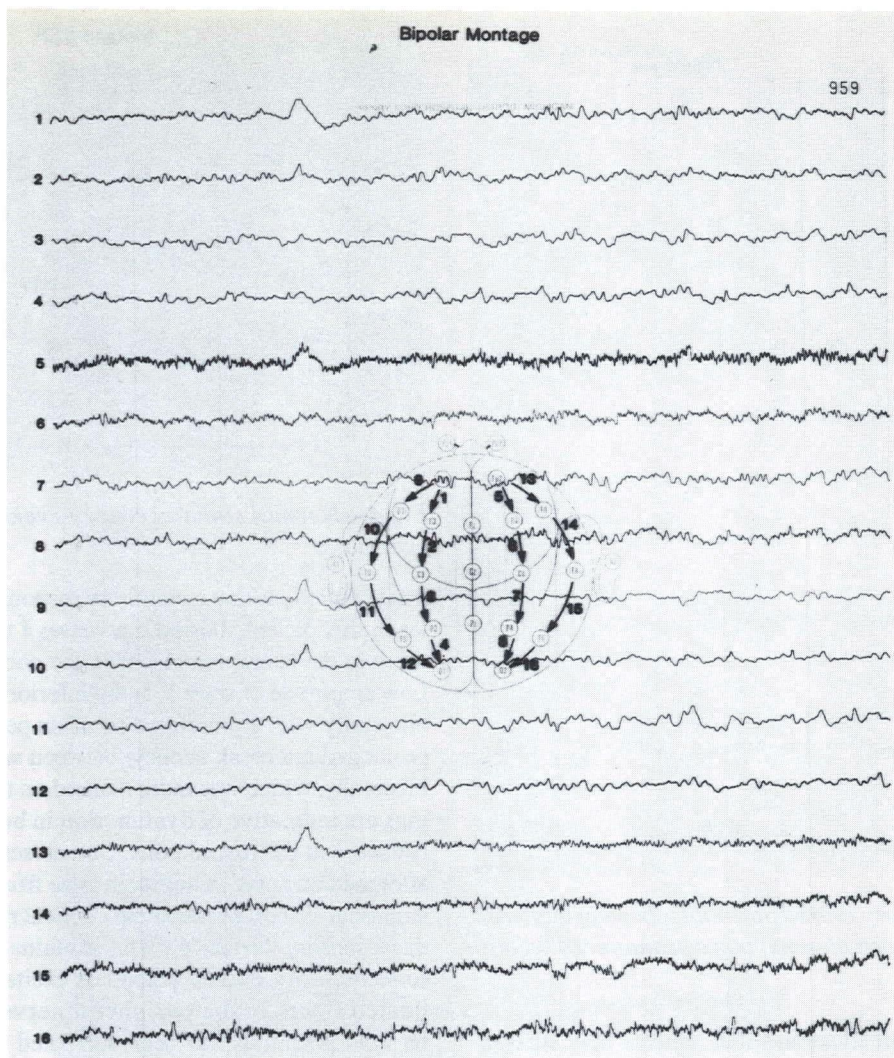


Fig 2—EEG showing mild diffuse slowing of background rhythms.

firmed an asymmetric lesion of the brain stem at the midpontine level and a peripheral lesion of the left facial nerve (Fig 5). Facial nerve studies showed a greatly reduced compound muscle action potential on the left side (Fig 6).

Gradual mobilization was commenced eight days after admission. Two weeks later, the patient could sit independently and stand with close guarding of two people. He was independent in the activities of daily living on discharge to a rehabilitation center. At that time clinical exam revealed ocular bobbing, skew deviation, left facial paralysis, and right-sided hyperreflexia. A follow-up CT brain scan before discharge showed considerable resolution of the hematoma (Fig 7).

Discussion

The earliest description of primary pontine hemorrhage was in 1812 by Cheyne, but Oppenheim first described the associated clinical features in 1900 (1). In the pre-CT brain scan era, insight into this condition was limited because primary pontine hemorrhage had to be differentiated from many conditions associated with stupor and coma, including acute subdural and epidural hemorrhage; hypertensive, uremic, and hepatic encephalopathy; diabetic coma; hypoglycemia; brain abscess and tumor;

pontine infarct; meningitis; and drug overdosage. CT brain scan can detect even a small hematoma in the brain stem and show the variations in size, shape, and location of such lesions accounting for the variety of symptoms and signs noted clinically.

An early study by Epstein in 1951 described the classical, clinical, and pathological findings of the condition in seven cases collected over a 33-year period (2). Coma, quadriplegia, and ocular pareses were followed by a fatal outcome. In 1951, Steegman also reported a similar outcome in 17 cases with a special emphasis on respiratory difficulties (3). More recently, in 1983, Nakajima published a retrospective study of 60 patients: 24 subjects were diagnosed at autopsy before the availability of CT scan, and the remaining 36 subjects were identified by CT scan (4). Of the latter group, 17 survived. The fatal cases were marked by early and severe disturbance of consciousness, quadriplegia, and decerebrate posturing. Systolic BP ranged from 200 mm Hg to 250 mm Hg in most fatal cases. Levels greater than 250 mm Hg were noted in three cases, and systolic BP was normal in five cases. Regarding the respiratory difficulties of the 43 subjects who died, 11 had apnea, 26 had irregular respirations, and only six had no disturbance of respiratory function.

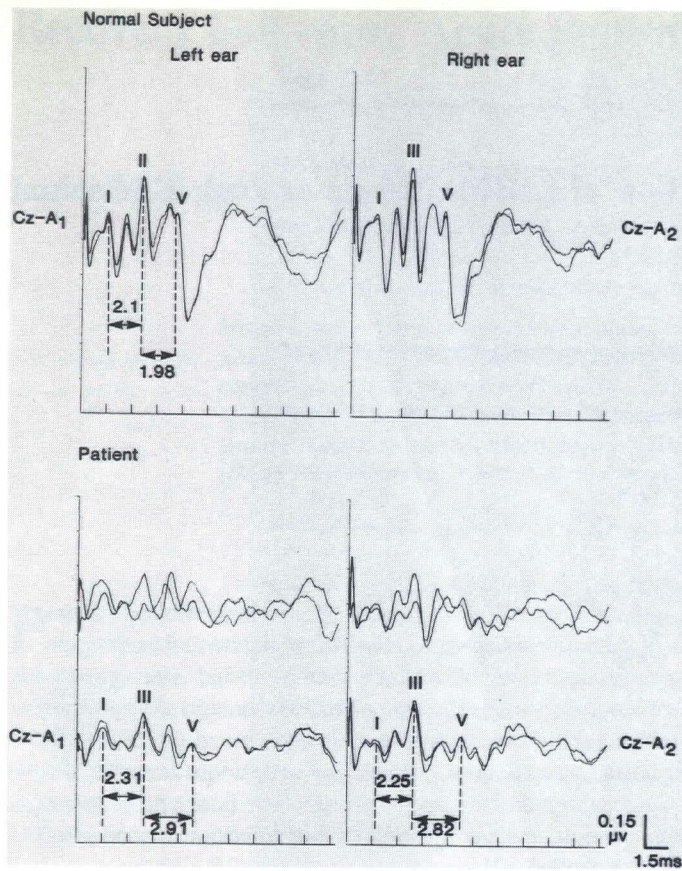


Fig 3—Brain stem auditory evoked potentials showing bilateral dysfunction at level of rostral pons, more pronounced on left side.

The current case report illustrates that patients may survive a massive primary pontine hemorrhage. Paralysis of all four limbs, an early manifestation in our patient, is the most common motor disturbance in fatal cases (4). The severity of the damage was also reflected by the occurrence of decerebrate posturing, disturbance of consciousness, and hypopnea requiring artificial ventilation. Full supportive measures during this critical period permitted resolution of the hemorrhage with considerable recovery. A transverse section of the caudal pons delineating the structures that were damaged at this level is shown in Fig 8. Electrophysiologic studies helped to localize the diverse and complex neurological deficits and thereby explain the clinical characteristics of this case.

A 16-channel EEG using both referential and bipolar montages was performed (Fig 2). The background activity consisted predominantly of 6.5 Hz to 7 Hz activity seen rather diffusely without a well-defined posterior dominant rhythm. This finding was probably related to the patient's drowsiness.

Evoked potential studies (both brain stem auditory and somatosensory) were also carried out. An evoked potential is an electrical manifestation of the brain's reception of, and response to, an external stimulus. Brain stem evoked potentials are a physiological technique that can be used to evaluate the auditory pathways between the site of reception of an auditory stimulus and the high brain stem. Brain stem evoked potentials are often abnormal with intrinsic brain stem lesions (5). An advantage of

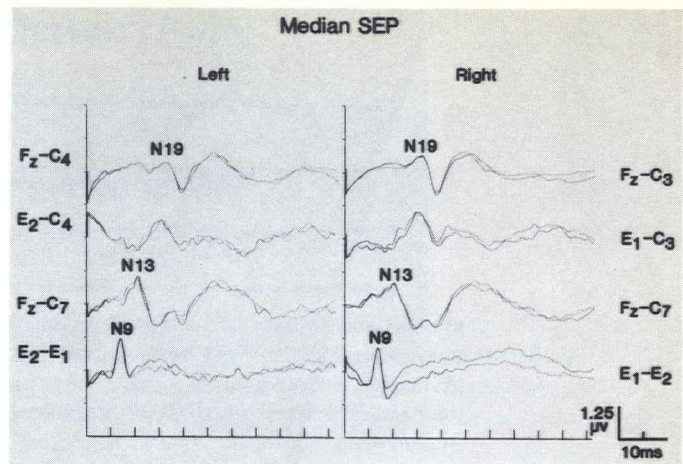


Fig 4—Normal somatosensory evoked potentials in upper extremities.

this technique is that a voluntary response is not required. Studies in this patient showed that waves I to IV, from the acoustic nerve to the lateral lemniscus in the pons, were normal (Fig 3). Low amplitude of wave V at the inferior colliculus was obtained bilaterally with a prolonged absolute peak latency. There was a prolonged interpeak latency, between waves I to V and III to V bilaterally, which was more marked on the left side. These findings are indicative of dysfunction in both brain stem auditory pathways at the rostral pons. Somatosensory evoked potentials after median nerve stimulation arise from the posterior column-lemniscal system between the cervical route entry zone and primary sensory cortex (6). The stimulus intensity employed for somatosensory evoked potentials excites only the largest myelinated fibers in the peripheral nerve. The scalp-recorded far field potentials has been attributed to synchronized volleys of action potentials in the peripheral nerve (N9), the cervical cord root entry zone or nearby posterior columns (N11), the dorsal column and nucleus cuneatus (N13), the thalamus or thalamocortical radiation (N18), and the primary sensory cortex (P22). Accordingly, somatosensory evoked potentials allow localization of conduction defects due to structural abnormalities in the brain stem, and they are resistant to metabolic abnormalities. The preservation of these components in our patient is presumably related to the fact that the cervical-medullary junction is less subject to increased intracranial pressure than those structures confined to the cranium. It also confirms that the stimulus has been adequate, that a volley has reached the central nervous system, and that the pathway of the medial lemniscus through the central pons is intact. The origin of reversible coma in this case was probably due to temporary compression of the ascending reticular activating system situated above the cranial nerve nuclei in the dorsal region of the pons.

Electrical stimulation of the supraorbital nerve, which is analogous to the corneal reflex, elicits two temporally separate responses from the orbicularis oculi: an early R_1 and a late R_2 component (7). The R_1 response is unilateral via an oligosynaptic reflex, while the R_2 response is bilateral and seen late due to a more complex polysynaptic pathway through the pons and lateral medulla. R_1 shows a prolonged latency in diseases associated with peripheral or central lesions of the trigeminal nerve,

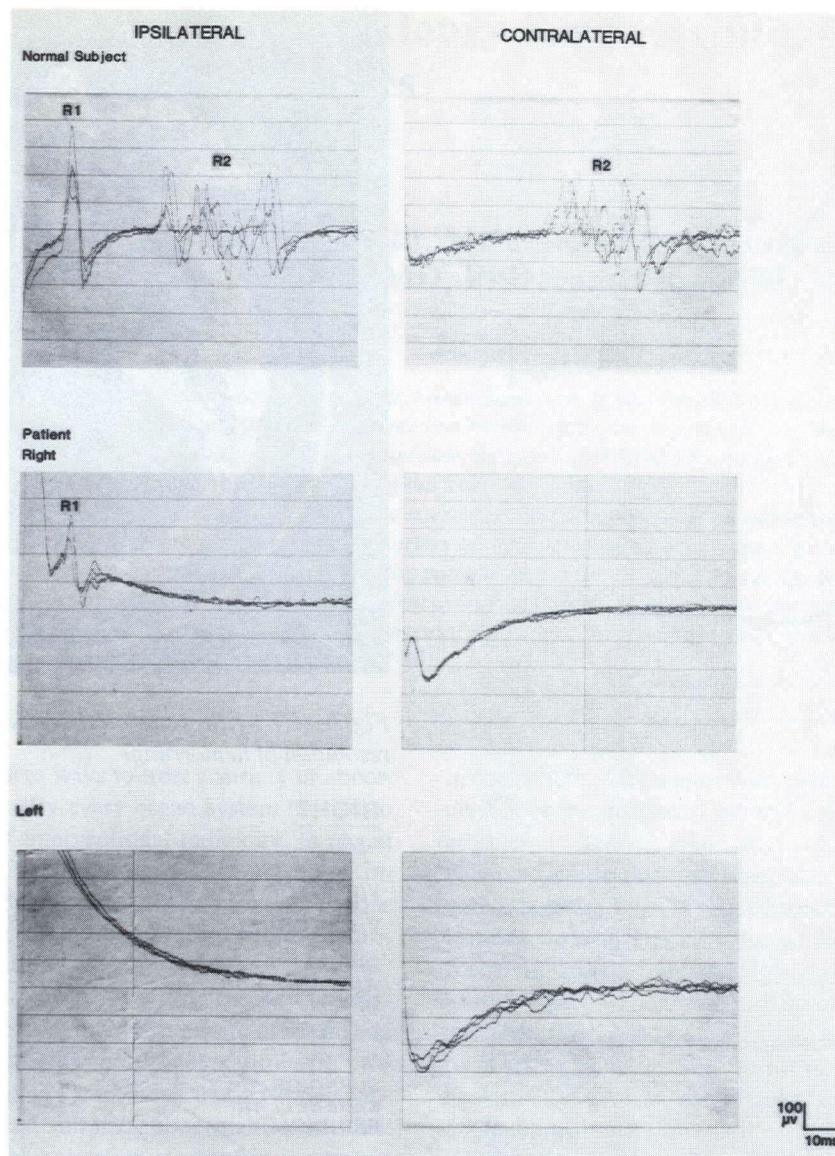


Fig 5—Blink reflexes showing asymmetric lesion of brain stem at midpontine level and peripheral lesion of left facial nerve.

facial nerve, or both. A reversible block of R_1 may occur in comatose patients when the brain stem is severely depressed with acute supratentorial lesions or massive drug intoxications. Abnormalities of R_2 can be categorized as either afferent or efferent in type according to the site of disruption of the polysynaptic reflex. R_2 is also influenced by the excitability of the polysynaptic connections and thus may be absent, diminished, or delayed in any comatose state regardless of the site of the lesion. A lesion of the facial nerve alters R_1 and R_2 only on the affected side, regardless of the side of stimulation.

A normal R_1 response latency was obtained in our patient on stimulation of the right supraorbital nerve; neither ipsilateral nor contralateral R_2 responses were elicited (Fig 5). On stimulation of the left supraorbital nerve, the R_1 and the ipsilateral and contralateral R_2 responses were absent. These findings indicate an asymmetric lesion of the brain stem in the pons and a peripheral lesion of the left facial nerve. Needle exam of the left facial mus-

cles revealed normal, spontaneous activity at rest but greatly reduced numbers of motor unit potentials firing at fast rates (Fig 6). Because the amplitude of the compound muscle action potential of the left facial nerve was only 10% when compared to the right, prognosis for recovery of function was guarded.

The ocular findings in this case were compatible with pontine damage. Miotic pupils are due to dysfunction of the sympathetic pupillary pathways in the pontine tegmentum (8). Ocular bobbing was originally observed in patients with extensive structural pontine disease, but the underlying mechanism is unclear (9). It is hypothesized that the pontine pathways for horizontal gaze are selectively damaged with preservation of the most rostral input for vertical gaze (10). Although skew deviation has been observed at a number of different sites in the brain stem, it is of no further localizing value.

Destruction of the reticular formation in the brain stem or of the autonomic pathways in the pons probably accounted for the

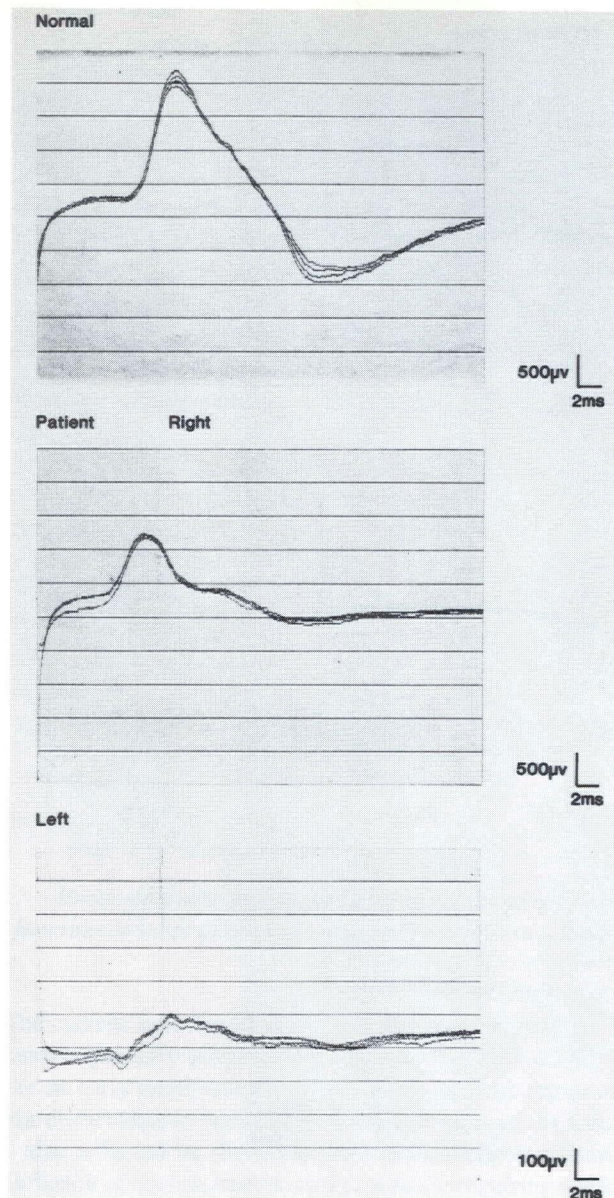


Fig 6—Facial nerve studies showing greatly reduced compound muscle action potential on left side.

patient's neurogenic bladder (4). Abnormal sweating was noted in this case, but hyperthermia did not occur. Visual hallucinations and gastrointestinal bleeding reported in other series were not seen in this case.

Since massive pontine hemorrhage is not uniformly fatal, full supportive therapy should be offered to all patients.

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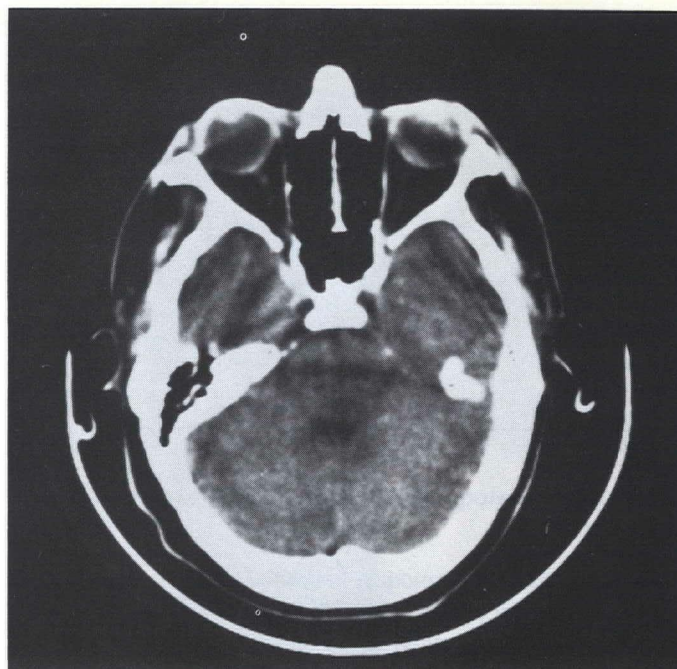


Fig 7—CT brain scan on discharge showing considerable resolution of hemorrhage.

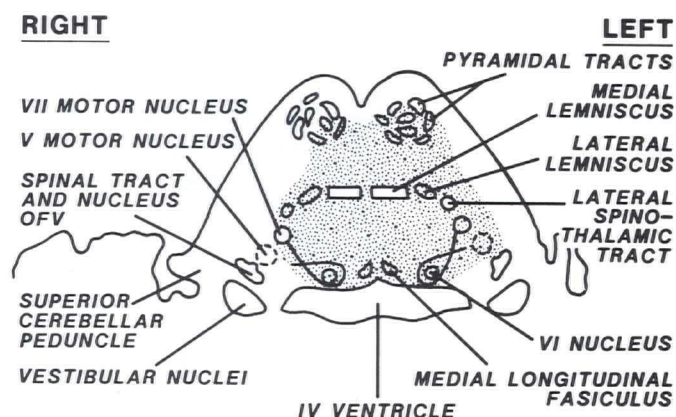


Fig 8—Diagram of pons showing transverse section at level of cranial nerve nuclei VI and VII. Motor nucleus of V is represented by a dotted line since it is situated at a higher level. Orientation of diagram corresponds with that of CT brain scan in Figs 1 and 7. Shaded area corresponds to region of hemorrhage.

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