

References

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Late blink reflex changes in lesions of thalamus and internal capsule

Article abstract—A 60-year-old man suddenly suffered from left hemiplegia with sensory loss on the left side of his face, trunk, and extremities. Brain CT disclosed hemorrhage in the right thalamus and internal capsule. Blink reflex with electrical stimulation on the left supraorbital nerve showed normal early response (R1) on the left side but absent late response (R2) on both sides, while stimulation of the right supraorbital nerve showed normal R1 on the right side and normal R2 on both sides. This observation suggests that the impulses of R2 from stimulation of the supraorbital nerve on the affected side may be blocked by contralateral lesions of the thalamus and internal capsule.

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Blink reflex evoked in the orbicularis oculi muscles by electrical stimulation of the supraorbital nerve consists of two components, an early oligosynaptic ipsilateral component, R1, and a late polysynaptic bilateral component, R2.^{1,2} The afferent limb of this reflex comes from sensory root of the trigeminal nerve and the efferent limb from the facial nerve. It is generally agreed that the central pathway of the R1 may conduct through the pons.^{1,3} The exact central pathway of the R2 is not entirely understood. It has been presumed that the longer latencies of R2 may travel along the longer pathways up to the opposite thalamus or cortex where they project down to bilateral facial nuclei.⁴ The longer latencies of R2 are suggested to include R1 latency and to-and-fro duration of somatosensory evoked potential between the upper cervical cord (N14) and the thalamus or cortex (N20) as well as the central delay of many synapses.⁴ Although R2 pathways via the lower brainstem to both facial nuclei cannot be excluded,⁵ this is not supported by the longer latencies of R2.⁴ In order to verify the pathway of late blink reflex, we studied the blink reflex of a patient with lesions in the thalamus and internal capsule.

Case report and electrophysiologic study. A 60-year-old man suddenly suffered from left hemiplegia. He was immediately sent to a local clinic, where brain CT disclosed hemorrhage in right thalamus and internal capsule, and lateral ventricles (figure 1). He was then transferred to our hospital for further evaluation.

On admission, he had normal heartbeat and respiration with an elevated blood pressure of 180/120 mm Hg. He was alert and oriented. Neurologic examination showed sensory loss of pain, touch, vibration, and joint position on left side of the face, trunk, and extremities. There was motor

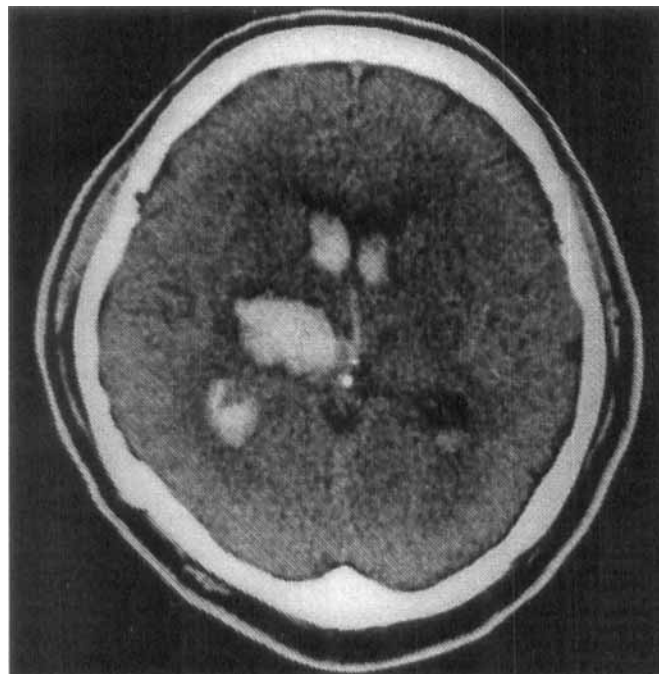


Figure 1. CT showing hemorrhage in right thalamus and internal capsule, and lateral ventricles.

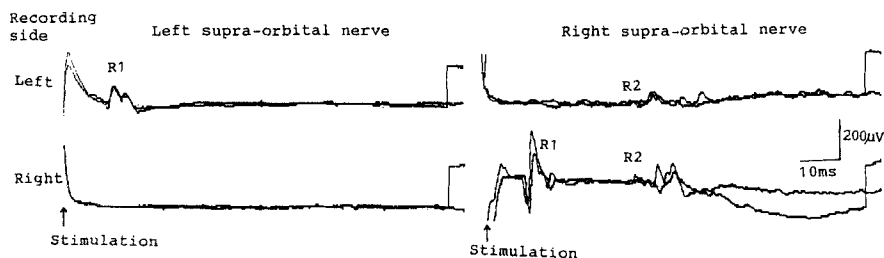


Figure 2. Blink reflex showing normal R1 (10.8 ms) on left side, but absent R2 on both sides, as stimulation on left supraorbital nerve; normal R1 (10.4 ms) on right side and normal R2 (32.1 ms, left, and 32.4 ms, right) on both sides, as stimulation on right supraorbital nerve.

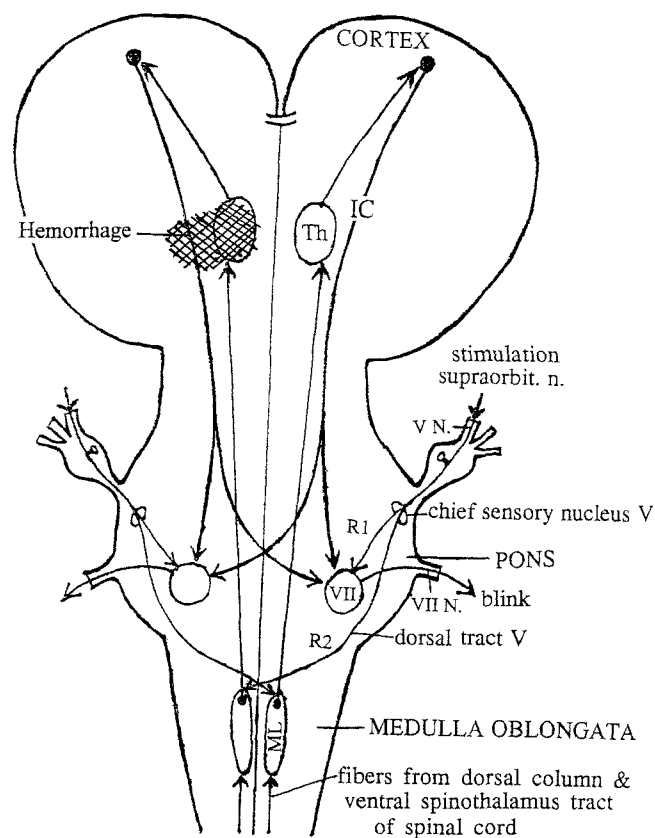


Figure 3. Diagram showing the presumed location of brainstem-thalamus (internal capsule)-cortex interneurons interpreting the two components of blink reflex. The shaded area (hemorrhage) in right thalamus and internal capsule involves R2 from stimulation of left supraorbital nerve. R1 (early reflex, ipsilateral), R2 (late reflex, bilateral), Th (thalamus), IC (internal capsule), ML (medial lemniscus).

weakness on left extremities, especially on the arm. Tendon reflexes were present with slight increase on left side. Extensor plantar responses were present on left side. His upward gaze was slightly limited. Dysarthria and left central facial palsy were noted. Corneal reflex was impaired on left side. He was known to have hypertension without regular treatment for about 1 year, but denied history of diabetes mellitus, heart, liver, or renal diseases. Results from routine laboratory tests of blood, urine, and ECG were all normal.

Electrically elicited blink reflex⁴ was studied with a DISA electromyograph on the fourth hospital day. Blink reflex with electrical stimulation on left supraorbital nerve showed normal R1 (10.8 msec) on left side, but absent R2 on both sides; stimulation on right supraorbital nerve

showed normal R1 (10.4 msec) on the right side and normal R2 (32.4 msec, right; and 32.1 msec, left) on both sides (figure 2).

Discussion. It has been suggested that R2 blink reflex is conducted via polysynaptic pathways in the lower brainstem, from the spinal trigeminal nucleus to both facial nuclei.⁵ However, it is hard to explain why, over the short distance in the lower brainstem, R2 latency (25–40 msec) is so much longer than R1 latency (8–12 msec). It seems reasonable to assume that the impulses of R2 may travel along the longer pathways up to the opposite thalamus or cortex where they project down to bilateral facial nuclei.⁴ The longer pathway up to the thalamus for R2 is also suggested by animal experiments.⁶ In the present case, the impulses of R2 from stimulation of left supraorbital nerve were blocked by hemorrhage in right thalamus and internal capsule (figure 3).

The absence of R2 may be explained on the basis of loss of hemispherical influences.^{7–10} However, it is difficult to interpret the present case, which shows the absence of bilateral R2, with the presence of ipsilateral R1. Because hemispherical lesions, which do not involve the brainstem reflex centers, affect the brainstem trigeminal and facial systems of both R1 and R2, the dissociation of R1 and R2 behavior cannot be explained entirely by hemispherical influences. In the present case, the absence of bilateral R2 and the presence of ipsilateral R1 suggest that contralateral lesions of the thalamus and internal capsule block the R2 pathways.

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Cat-scratch encephalopathy

Article abstract—We present a case of cat-scratch disease in a 9-year-old girl, complicated by encephalopathy and seizures. *Bartonella* (formerly *Rochalimaea*) *henselae* is the causative agent in cat-scratch disease; methods now available for detection of this pleomorphic, gram-negative bacterium, including polymerase chain reaction amplification and indirect fluorescence antibody testing, may lead to changes in standard criteria used to verify a diagnosis of cat-scratch disease.

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Cat-scratch disease (CSD) is a bacillary infection that occurs predominantly in children and adolescents. The illness is typically self-limited, characterized by regional lymphadenitis following cutaneous inoculation, and variably accompanied by low-grade fever, malaise, and headache. Neurologic complications associated with CSD are rare, with encephalopathy accounting for the majority of these cases. *Bartonella* (formerly *Rochalimaea*) *henselae*, a pleomorphic, gram-negative bacillus, is the etiologic agent of CSD.¹⁻³ Methods now available for detection of this bacterium, including polymerase chain reaction (PCR) amplification, as well as indirect fluorescence antibody testing, may lead to changes in standard criteria used to verify a diagnosis of CSD.

We report a case of a child with CSD complicated by encephalopathy and briefly review the neurologic manifestations of this disease.

Case report. A previously healthy 9-year-old girl was admitted to the hospital with a 2-week history of right-sided cervical adenitis and a 2-day history of low-grade fevers, diarrhea, and headache. Treatment with oral antibiotics over an 11-day period prior to admission (cephalexin over 3 days, followed by rifampin for 7 days, and amoxicillin-potassium clavulanate during the final 4 days) produced no discernible symptomatic improvement. Two episodes of emesis were temporally associated with ingestion of amoxicillin-potassium clavulanate. Purified protein derivative (PPD) testing was negative, and there was no history of recent travel. Although she had four kittens at home, there were no known recent scratches or bites from cats.

Following needle aspiration of the right cervical mass, the patient was hospitalized for intravenous antibiotics (cefuroxime, gentamicin) plus oral rifampin. On initial examination, vital signs were normal except for a tempera-

ture of 38.0 °C. A 5 × 3-cm, tender superior right cervical mass, and a 1-cm nontender left midcervical mass, were noted. There was no other adenopathy or organomegaly. Neurologic examination was normal.

Ten hours after admission, the patient had a generalized tonic-clonic seizure lasting 2 minutes, which aborted with administration of intravenous lorazepam. Postictal combativeness and delirium were followed by stupor and coma. Temperature was 39.2 °C. Neurologic examination demonstrated slight withdrawal of the limbs to noxious stimulation, with slow roving eye movements, extensor plantar responses, and bilateral ankle clonus.

Noninfusion CT brain imaging was negative. EEG revealed generalized theta and delta frequencies, with no evidence of epileptiform activity. Serum WBC, electrolytes, BUN, creatinine, and liver function tests were normal. Erythrocyte sedimentation rate was elevated at 73 mm/h. Studies of CSF were normal except for increased protein of 72 mg/dl. Cultures of blood, urine, and CSF were negative. Serologies for Epstein-Barr virus, tularemia, and syphilis were negative.

After 24 hours, the patient's mental status began to improve. No further seizures occurred, and no anticonvulsant medication was given. By the fifth hospital day, neurologic examination was normal. She had intermittent fever until the sixth hospital day and was discharged the following afternoon.

Two weeks later, incision and drainage of the right cervical node was performed. The necrotic tissue obtained at surgery was negative for organisms on special staining, and cultures for bacteria, acid-fast bacilli, and fungi were negative. Serum *B henselae* serology was positive at 1:8192. PCR analysis of frozen lymph node tissue, performed by the Centers for Disease Control and Prevention, Atlanta, GA, was positive for *B henselae*.

The patient has remained seizure-free, with a normal neurologic examination and normal EEG 30 months after initial presentation.

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