

Fig. 3.67. Facial nerve conduction study in a patient with left sided Bell's palsy showing prolonged latency (3.5 ms) on left side (L) compared to right (R, 2.2 ms) on 7th day of illness. The patient had complete recovery.

pathy although one should wait for 5-7 days before interpreting the significance of amplitude change (Fig 3.66 and Fig 3.67). The guidelines for interpreting the amplitude changes of facial nerve conduction results are as follows :

1. CMAP amplitude if less than 10% compared to the healthy side, the recovery may take 6-12 months and there may be moderate to severe deficit

2. The CMAP amplitude if is 10-30% compared to the healthy side, recovery may take 2-8 months with mild to moderate deficit

3. The CMAP amplitude if more than 30% compared to healthy side, complete recovery is expected by 2 months (Olsen 1975; Dumitru et al 1988)

Latency measurements of facial CMAP are not as useful as the amplitude for prognosticating facial nerve lesions. A normal latency after 5-7 days of Bell's palsy is associated with complete recovery without aberrant connection. Prolonged latency is associated with good recovery but with some chance of synkinesis. Unrecordable facial CMAP suggests a high chance of synkinesis with no or poor recovery (Langworth and Taverner 1963).

Blink Reflex

Blink reflex is the electrical analogue of corneal reflex.



Fig. 3.68. Electrode placement for blink reflex. Ri = recording ipsilateral to stimulation, <math>Rc = recording contralateral to stimulation, S = stimulating and G = ground electrode.



Fig. 3.69. Mechanism of blink reflex. The impulse travels through the Vth nerve and generates direct R_1 response through VIIth nerve nucleus and VIIth nerve. For R_{2i} and R_{2c} responses the pontomedullary interneurons are excited which inturn stimulate bilateral VIIth nerve for ipsilateral (i) and contralateral (c) R_2 responses.

The afferent limb of blink reflex is ophthalmic division of trigeminal and efferent, the facial nerve. The recording surface electrodes are placed bilaterally inferior to the lower lid halfway between the inner and the outer edge of the orbit. The reference electrode is placed on



Fig. 3.70. Facial nerve conduction study and blink reflex in a patient with right sided Bell's palsy. A = facial nerve conduction showing pronounced attenuation of CMAP on right side (0.3 mV) compared to left (2.4 mV). B and C = prolonged latency of R₂ responses recording from right side on both left and right supra orbital nerve stimulation.

the side of nasal bone. Ground electrode is placed on the chin. Stimulation is carried out keeping the cathode on the supraorbital notch over the supraorbital nerve and the anode directed somewhat laterally (Fig 3.68). A gain of 200-500 μ V/division with a time base of 10 ms/division is used. Stimulation should be given at low rate (1 in 3 sec) and the subject is asked to keep the eyes open to avoid muscle artifact. On the side of stimulation, 2 responses R₁ and R₂ and on the contralateral side only R₂ are recorded. The R₂ response may vary and may have an unclear onset; therefore several responses (5-8) are superimposed and minimal latency is measured. Prolonged studies should be avoided lest the R₂ response gets habituated. The test requires stimulation on either side. Blink responses are present in upper but not in lower facial muscles. If aberrant innervation is suspected these may also be recorded from lower facial muscles.

On supraorbital stimulation, the impulse propagates to the trigeminal nucleus, excites the facial nucleus via oligosynaptic reflex (R_1 response) and traverses a polysynaptic pathway leading to bilateral facial nucleus excitation (R_2 response)(Fig 3.69). Blink reflex differs from direct facial nerve study by evaluating the trigeminal nerve and pons in addition to the facial nerve. Normally the R_1 response latency is less than 13 ms, ipsilateral R_2 less than 40 ms and contralateral R_2 less then 41 ms (Gilchrist 1993). Age has an important effect on blink reflex parameters, especially in premature neonates. Under 35 weeks of conceptual age, the recovery curves of blink reflex are considerably different compared to full term infants. Premature infants have little or no inhibition which suggests absence of inhibitory interneurons in premature infants (Hatanaka et al 1990).

A delayed or absent R_1 could either be due to trigeminal or facial neuropathy. Abnormal R₁ if associated with abnormal R₂ on the paretic side regardless of the side of stimulation indicates slowing in the ipsilateral facial nerve (Fig 3.70). An abnormal blink reflex showing this pattern is consistent with facial neuropathy. In a study on 144 patients with Bell's palsy, R₁ response was absent or delayed on the paretic side during the first week (Kimura et al 1976). Blink reflex does not offer further information about the prognosis of facial palsy compared to direct facial nerve conduction study. The differentiation between upper motor and lower motor neuron lesion is often based on the involvement of frontalis muscle. In rare instances, where this differentiation is not clear, blink reflex may be useful. Increased R₁ latency will suggest facial nerve lesion whereas it will be normal in supranuclear lesion (Kimura 1989).