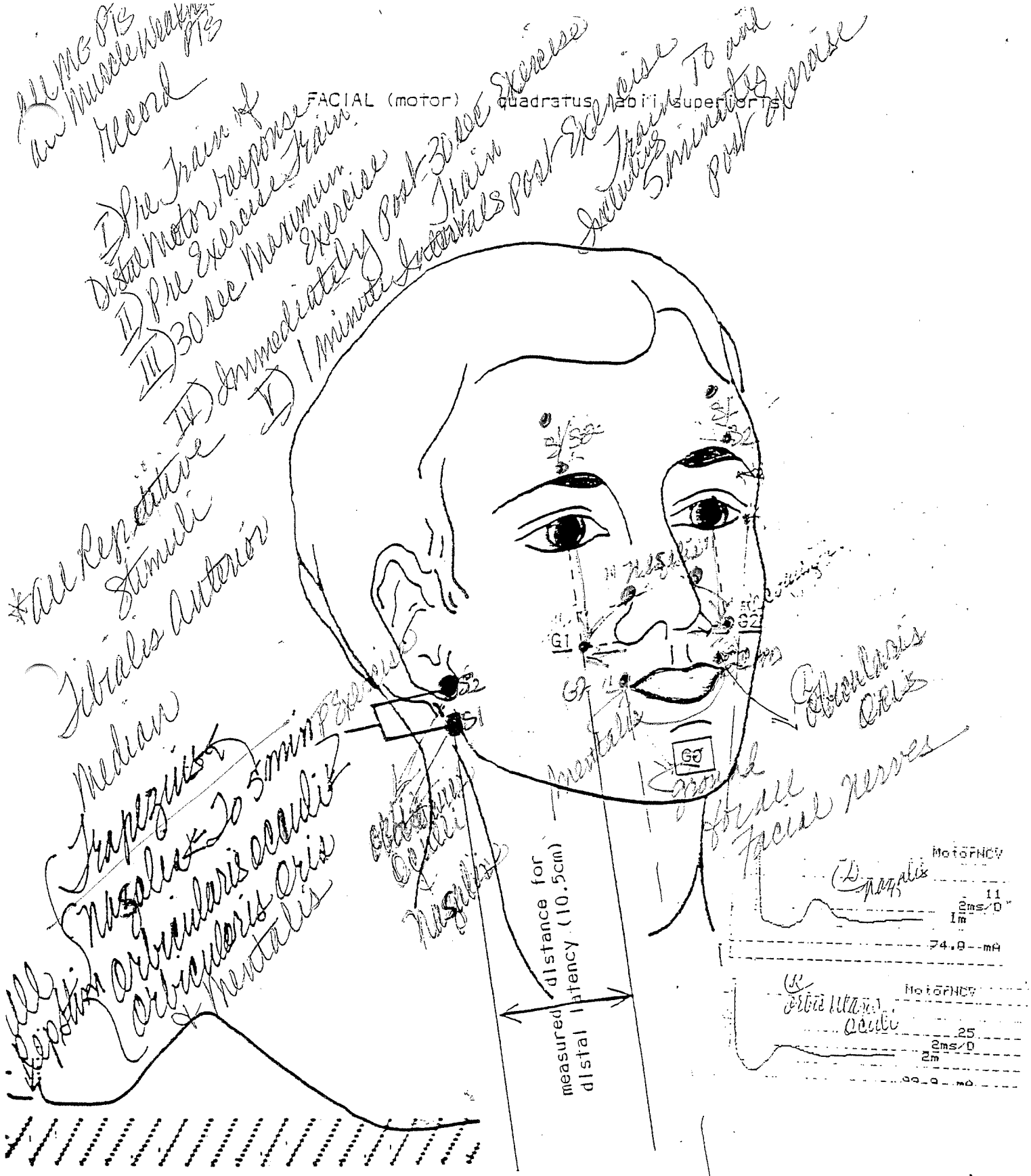
NERVE CONDUCTION STUDY SETUPS

FACIAL STUDY

Facial (motor)/quadratus labii superioris (10, 48, 50)

I. Position of Patient
   A. Patient is supine on a bed.
   B. Head and neck is supported in a comfortable position at all times.
II. Equipment
   A. 2 disc electrodes (G1 active, G2 reference)
   B. 1 ground (G0)
   C. 1 bipolar stimulator (S1 cathode, S2 anode)
III. Machine Settings
   A. Sweep speed (ms/div)—2 to 5
   B. Gain—500 (μv) to 2 mv
   C. Filters—1.6(Hz), 8(KHz)
IV. Electrode Placement
   A. G0 (ground)—on the chin
   B. G1 (recording)—over the quadratus labii superioris muscle at the point where a right angle is formed by the base of the nose (x axis) and the pupil (y axis).
   C. G2 (reference)—same as G1 on the contralateral side.
V. Stimulation Sites and Measurements
   A. Ear (only stimulation site)
      1. Stimulate—S1, slightly anterior and distal to the ear lobe over the mandible bone with S2 behind the ear off the mandibular bone.
      2. Measure—distance from S1 to G1 following the contour of the face.
VI. Calculations
   A. Distal Latency—Calculate from shock artifact to the take off of the negative deflection of the distal response.
   B. Amplitude—Calculate from baseline to the peak of the negative deflection.



age = 31

Stimulation Site	Amp. mv	Latency msec	Dist. cm
ear	3.6	3.1	10.5

FACIAL NERVE STIMULATION

Clinical applications

A. Prognosis in Bell's palsy and other disorders of the facial nerve in its peripheral course.

The main role of EMG and nerve conduction in Bell's palsy is to aid in prognosis. The parameters that are most commonly measured are **latency** of the muscle response following stimulation at the stylomastoid foramen, **minimal excitability** of the facial nerve, and **amplitude** of the response.

Latency

If nerve conduction studies are performed 5 to 7 days after paralysis, three types of responses may be seen:

1. Normal response. This group of patients can be assured of a complete recovery with very little associated movements.

2. A response occurs but it may be of longer latency than the normal side, and the amplitude may be reduced. If the latency on the involved side is more than 0.6 ms longer than the uninvolved side, improvement will likely be satisfactory but the patient may develop associated movements (facial synkinesis) or crocodile-tear phenomenon due to aberrant regeneration of the facial nerve.

3. No response occurs to nerve stimulation. Although recovery is still satisfactory in most patients in this group, the incidence of facial synkinesis is high and some patients may have no recovery of function whatsoever.

Taking the entire group of patients with Bell's palsy, 70% will have a full recovery; 20% will have a partial recovery with weakness, contracture, or synkinesis complicating the recovery, and 10% will have little or no recovery (30). Nerve conduction studies done five to seven days after the onset of the paralysis is of assistance to the clinician in determining prognosis.

Threshold excitability

Some investigators have attempted to correlate prognosis of recovery with the minimal excitability of the facial nerve (31). Using a constant current duration of 0.1 ms, there is usually no greater than 2 mA difference in the normal individual on the two sides. Patients with Bell's palsy who have a normal or slightly increased excitability (3-5 mA) all have an excellent prognosis. Patients with a difference of 10 mA or more do poorly, whereas those with a 5-10 mA difference have an intermediate prognosis. This

method allows for earlier assessment of prognosis than does determination of latency since abnormalities can be detected as early as 72 hours.

Amplitude

Other investigators have attempted to correlate prognosis of recovery with the amplitude of the response obtained on facial nerve stimulation within the first three weeks after the onset of paralysis. By comparing the amplitude of the response on the paralyzed side with the uninvolved side, a fairly reliable indication as to prognosis of recovery can be obtained (32). When the compound muscle action potential amplitude on the affected side is reduced to 10% or less of the normal side, recovery may take from six months to a year, and the sequelae may be either moderate or severe. If the amplitude is reduced to between 10% and 30% of the normal side, recovery may take between two and eight months and sequelae are mild to moderate. If the amplitude is above 30% of the normal side, full recovery can be expected within two months after the onset of the palsy. This technique appears to be a simple and fairly reliable index of prognosis.

With present electrodiagnostic techniques, the clinician cannot determine with certainty which patients would benefit from facial nerve decompression. This is because there is presently no method which allows for early detection of the patients who will subsequently develop a complete lack of response to nerve stimulation. The earliest time that allows for demonstration of complete lack of response is at five days. Decompression at this stage would be too late to affect the prognosis. If a technique were available to predict at the first or second day following paralysis which patients would subsequently develop total denervation, then perhaps the status of facial nerve decompression might be reviewed. However, at the present time, there is no satisfactory electrodiagnostic method of predicting within the first day or two following paralysis which patients will subsequently develop complete loss of function of the facial nerve (33).

B. **Localization of Facial Nerve Lesions.**

By combining the results of facial nerve stimulation and blink reflex studies, a more accurate localization of a lesion affecting the facial nerve may be obtained. For example, if direct facial nerve conduction is normal when stimulating at the stylomastoid foramen but there is a delay in the blink reflex latency, this would indicate that the lesion is proximal to the stylomastoid foramen, somewhere between the facial nerve nucleus and the exit foramen.

Kimura, et al., have studied the blink reflex in 81 patients with Bell's palsy (34). It does not appear to be helpful in the early detection of patients who will subsequently develop total denervation since it can be present in the early days following an acute Bell's palsy, even though it may not be elicitable in the same group of patients after a few days have elapsed. It can be of interest in prognosis

since return of the previously absent reflex before loss of distal excitability is nearly always associated with clinical recovery.

Other diagnostic methods to assess facial nerve function such as the stapedial reflex and electrogustometry are useful in determining the location of the lesion but not in assessing the degree and extent of facial nerve involvement (35). They are beyond the scope of this discussion.

C. Repetitive stimulation in suspected disorders of the neuromuscular junction

In patients who have suspected myasthenia gravis with bulbar involvement, facial nerve stimulation may demonstrate a defect of neuromuscular transmission when repetitive stimulation of nerves in the limbs is normal. However, there may be a lot of movement artifact when stimulating the face which may make interpretation of the results difficult.

NEEDLE ELECTRODE EXAMINATION

The muscles innervated by the cranial nerves can be examined by standard concentric needle electrodes in the usual fashion, although it must be pointed out that there are certain individual features that must be kept in mind when examining these muscles. The motor unit potentials are usually more numerous, smaller in amplitude, and of shorter duration than they are in limb muscles, and, as a result, they are more difficult to analyze on an oscillograph. Some electromyographers have found that abnormal spontaneous activity seems to be of lower voltage also, and they will often look for this type of activity at an increased gain (e.g. 20 microvolts per division rather than 50 microvolts per division).

The extraocular muscles can be examined by special techniques. A fine-gauge needle must be used, and it should be inserted by an ophthalmologist. The extraocular muscles are in a state of continuous contraction and for this reason it may be very difficult to identify fibrillation potentials. In addition, the motor units themselves are much shorter than in limb muscles and their normal firing rates are much faster. Some authorities have found electromyography of extraocular muscle to be clinically useful in differentiating the cause of extraocular muscle paresis, but its use is not widespread. In most patients, one cannot distinguish the electrical abnormalities associated with myopathies from those associated with neuropathies in these muscles because the motor unit potentials are so numerous and their firing rate is so high. As a result, it is very difficult to analyze the number, size, and configuration of these potentials.

The muscles innervated by the motor root of the trigeminal nerve are easily accessible to needle examination. These include the masseter and the temporal muscle, although the latter is rarely examined because the examination is quite uncomfortable. The medial and lateral pterygoid muscles can also be examined, but examination of these muscles is uncomfortable for many patients.

The muscles innervated by the facial nerve are also relatively accessible to needle examination. These include the orbicular muscles of the mouth and eye, the frontalis, and the mentalis, as well as others that are more difficult to examine. In addition to the usual abnormalities seen in neuropathic and myopathic conditions, these muscles demonstrate unique patterns in two clearly defined clinical and electromyographic entities: facial myokymia and hemifacial spasm.

Facial myokymia is characterized clinically by the presence of continuous vermicular movements of the muscles supplied by one of the facial nerves. Its electromyographic counterpart is the presence of regularly recurring bursts of motor units, each burst firing independently of the others (Figure 17). The rate of firing within a burst is 25-60 Hz. There are three to ten units within the burst, and the burst itself recurs every 0.5-3 seconds. This pattern is usually seen in association with intrinsic brainstem lesions such as multiple sclerosis or brainstem tumors, and it has also been seen rarely with cerebellopontine angle lesions. Facial myokymia may also occur in polyradiculoneuropathies, but this association is very rare (36).

Hemifacial spasm is a distinct clinical entity with a specific electromyographic pattern (17). Brief clonic jerks of variable duration, lasting 10-200 ms, merge into a tonic spasm lasting several seconds (Figure 18). The interval between the clonic jerks varies considerably, from 20 to 225 ms. The firing rate of individual motor unit potentials within a burst is quite rapid and irregular (75-250 Hz).

In most instances, there is no serious underlying disease, and the cause is thought to be a blood vessel compressing the nerve at its exit from the brainstem. In selected cases, resolution has occurred after microvascular decompression of the facial nerve through an intracranial approach (37). As mentioned in the section on the blink reflex, a characteristic synkinetic response is present in other muscles innervated by the facial nerve after supraorbital nerve stimulation. This synkinetic response disappears after successful surgical treatment, and its disappearance parallels the clinical response (37,38).

Nielsen also presented impressive evidence that ectopic and ephaptic excitations are important pathophysiologic mechanisms in the genesis of hemifacial spasm. He demonstrated, by recording from the orbicularis oculi and mental muscles, that antidromic impulses are transmitted bidirectionally between the zygomatic and mandibular branches of the facial nerve. This phenomenon also disappears in most patients after successful vascular decompression (39,40).

Rarely, more serious pathologic lesions have been implicated in hemifacial spasm, including epidermoid tumors, arteriovenous malformations, and vertebral artery aneurysms. Fortunately these secondary causes are distinctly uncommon.

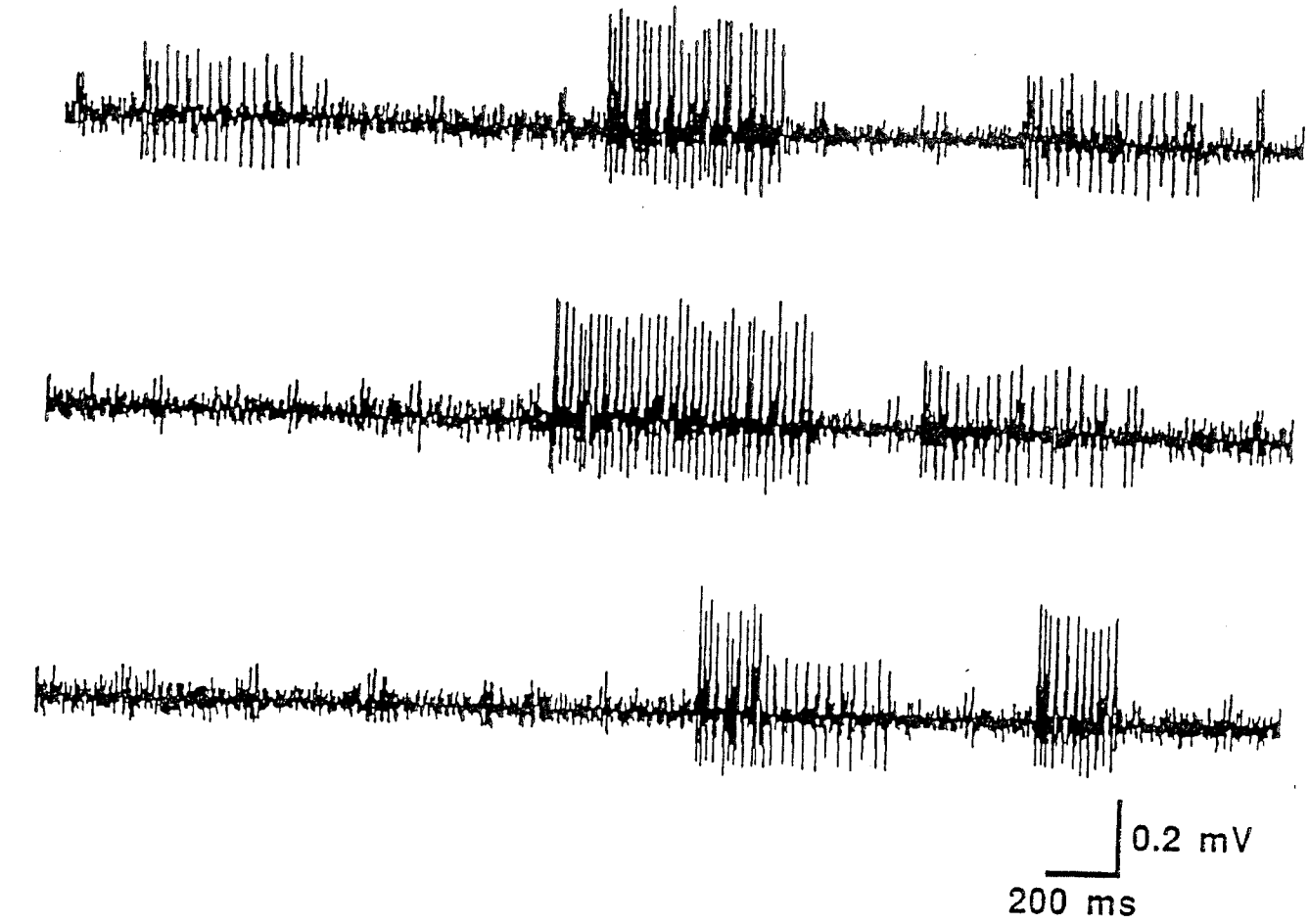


Figure 17

Hemifacial spasm

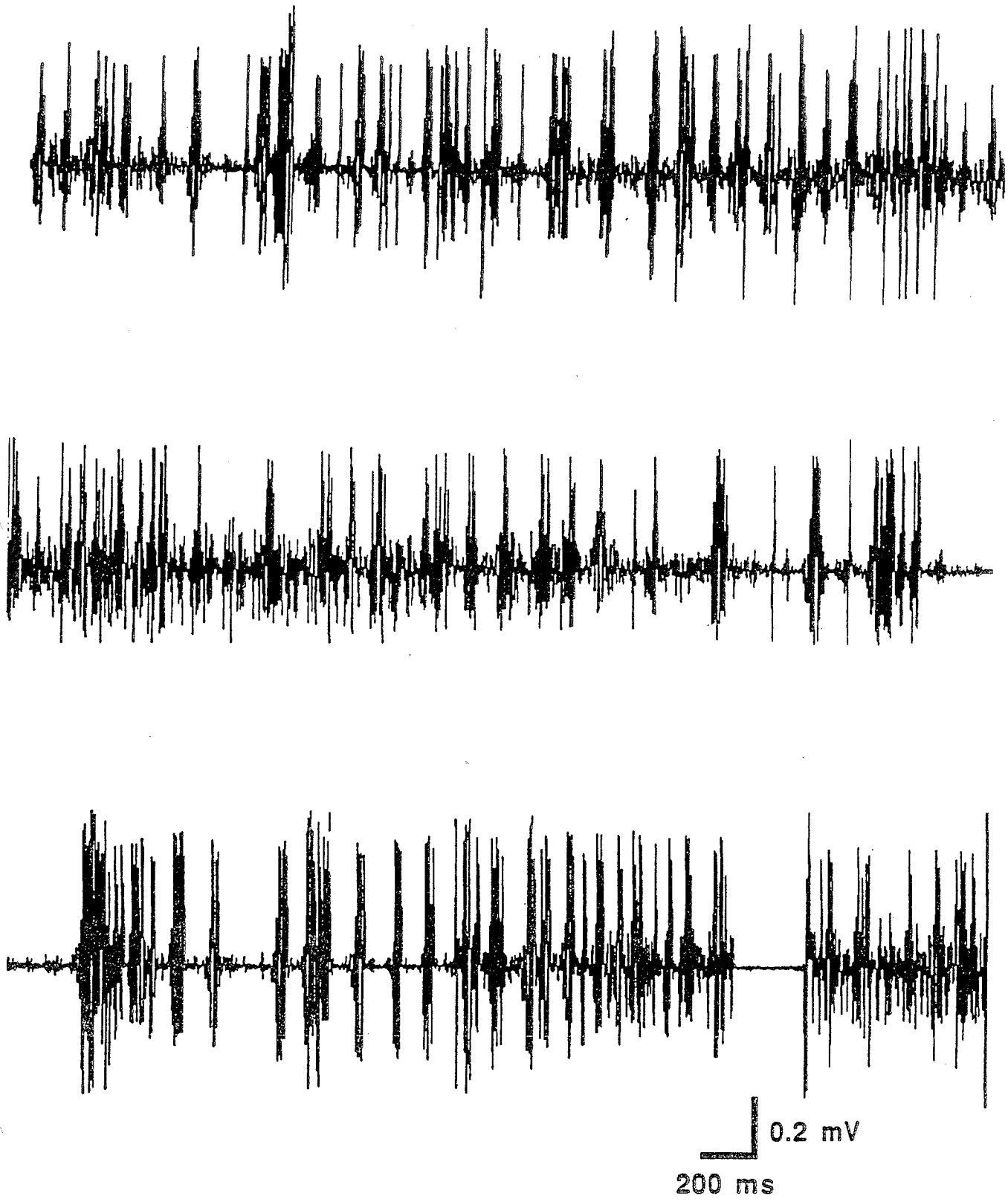


Figure 18

The laryngeal muscles (supplied by the vagus nerve) can be examined with a concentric needle electrode. This technique can be used to study unusual bulbar palsies or atypical recurrent laryngeal nerve lesions.

Muscles supplied by the spinal accessory nerve are easily accessible to examination. These include the sternocleidomastoid and the trapezius. The muscles of the tongue may be examined when a lesion is suspected to involve the hypoglossal nerve or its motor nucleus in the brainstem. The muscles of the tongue are often involved in motor neuron disease, and the finding of abnormal spontaneous activity in this muscle provides further evidence in favor of a diffuse denervating process when this is a diagnostic possibility. Needle examination of the tongue is not unduly uncomfortable to the patient; when such an examination is performed by an experienced electromyographer, abnormal spontaneous activity can be detected in the relaxed tongue. The examiner must be wary, however, because the motor unit potentials are normally numerous and of small amplitude, and they may be confused with spontaneous activity, particularly in the patient who finds it difficult to relax the tongue.