Chapter 6.1

The jaw reflexes

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Physiological background

In the reflex control of jaw movement, the jaw openers (digastric and suprahoid muscles) play a marginal role in humans, whereas in lower mammals the jaw-opening and jaw-closing muscles act in equilibrium. The jaw-closers (masseter, temporalis, and pterygoid muscles) serve both functions: for reflex jaw-closing under normal circumstances (excitation) and for reflex jaw-opening when they undergo inhibition. The jaw-closers are excited by way of the A\(_\alpha\) muscle-spindle input and strongly inhibited by way of A\(_\beta\) capsulated mechanoreceptors and possibly A\(_\delta\) free nerve endings. The powerful inhibition exerted by cutaneous and intraoral mechanoreceptors probably compensates for the unusual organisation of the jaw-closing motoneurons, which undergo inhibitory control neither by reciprocal nor by recurrent inhibition. The only jaw reflexes (or trigemino-trigeminal reflexes) amenable to clinical investigation are therefore the mandibular tendon jerk and the ‘cutaneous’ inhibitory periods.

The mandibular tendon reflex, also called jaw jerk or masseter reflex, is the only myotatic reflex in the cranial-nerve system (Fig. 1). Its reflex afferents are I\(_a\) fibres from the muscle spindles of the jaw-closing muscles. In animals, these afferents travel in the motor root. Whether they do so also in humans is unclear (Ongerboer de Visser and Cruccu 1993). Unique among the primary sensory neurons, these afferents have their cell body in the CNS (the trigeminal mesencephalic nucleus) rather than in the ganglion. Short collaterals connect monosynaptically with synergistic jaw-closing motoneurons in the pontine trigeminal motor nucleus; but no collaterals cross the midline. Owing to this anatomical arrangement, reflex testing will disclose side-to-side asymmetries even if mechanical stimuli excite receptors on both sides.

Electrical or mechanical stimuli delivered to the oral region evoke a reflex inhibition of the jaw-closing muscles, the masseter (or temporalis) inhibitory reflex (Fig. 2). On EMG recordings from contracted jaw-closers, this reflex inhibition appears as an early and a late phase of suppression, also called ES\(_1\) and ES\(_2\) exteroceptive suppressions (Godaux and Desmedt 1975), or SP\(_1\) and SP\(_2\) silent periods (Ongerboer de Visser and Cruccu 1993). Probably because electrical stimuli yield a mixed – nociceptive and non-nociceptive – input, whether the first or the second, or both components are nociceptive reflexes remains controversial (Miles and

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evidence supports the view that the afferents belong to the intermediately myelinated Aβ group (Ongerboer de Visser and Cruccu 1993). Afferent impulses reach the pons via the sensory mandibular or maxillary root of the trigeminal nerve. The first inhibitory period (10–13 ms latency) is probably mediated by one inhibitory interneuron, located close to the ipsilateral trigeminal motor nucleus. This interneuron projects onto jaw-closing motoneurons bilaterally. The whole circuit lies in the mid-pons. The afferents for the second inhibitory period (40–50 ms latency) descend in the spinal trigeminal tract and connect to a polysynaptic chain of excitatory interneurons, probably located in the medullary lateral reticular formation. The last interneuron of the chain is inhibitory and gives rise to ipsilateral and contralateral collaterals that ascend medially to the right and left spinal trigeminal complexes, to reach the trigeminal motoneurons (Ongerboer de Visser et al. 1990; Cruccu et al. 1991).

**Technical requirements**

The patient is seated upright in a chair with a headrest. The tendon reflex and the inhibitory periods are easily recorded through surface electrodes from the masseter or the anterior temporalis muscles (both recording sites yield similar

![Jaw jerk and brain-stem circuits](image1)

**Fig. 1.** Jaw jerk and brain-stem circuits. Jaw jerk responses from the masseter muscles. Four trials are superimposed. Arrows indicate onset latency. Calibration 5 ms/0.5 mV. Schematic drawing showing the reflex arc. Note that primary afferents have their cell body in the mesencephalic nucleus. In animals these afferents travel in the motor root. In humans they might travel in the mandibular sensory root. Nucl, nucleus; Mes, mesencephalic; Princ, principalis; Spin, spinalis; N V, trigeminal nerve; N III, oculomotor nerve; N VI, abducens nerve.

![Masseter inhibitory reflex and brain-stem circuits](image2)

**Fig. 2.** Masseter inhibitory reflex and brain-stem circuits. (Left) First (1) and second (2) inhibitory periods. Recording from the right masseter muscle. Stimulation of the right mental nerve. Upper traces: 8 trials are superimposed. Lower trace: rectified and averaged signal. The horizontal line indicates 80% of the background EMG level. Calibration 20 ms/200 μV. (Right) Schematic drawing of the reflex circuits. Afferents for the first inhibitory period connect with an inhibitory interneuron (1) located close to the ipsilateral trigeminal motor nucleus. The afferents for the second inhibitory period descend along the spinal trigeminal tract and connect with a multisynaptic chain of excitatory interneurons. The last interneuron is inhibitory (2) and projects bilaterally onto the jaw-closing motoneurons. Vm, trigeminal motor nucleus; Vp, trigeminal principal sensory nucleus; Sp V tract, spinal trigeminal tract; VI, abducens nucleus; VII, facial nucleus; XII, hypoglossal nucleus; Lat Ret, lateral reticular formation.
responses). Optimal recordings are obtained with the active electrode over the lower third of the belly of the masseter (motor point) and the reference electrode 2 cm below the angle of the mandible. Responses must always be recorded bilaterally.

The jaw jerk is usually evoked by tapping the patient’s chin with a tendon hammer; the closure of a microswitch triggers the oscilloscope sweep. Wide-open filters (5 Hz–2 kHz) yield an EMG response appearing as an initially negative, biphasic potential. The sweep is 20–50 ms, and the sensitivity 0.5–1 mV per division. The jaw is allowed to remain in its natural postural position; 4–8 trials are sufficient.

The masseter inhibitory reflex is usually evoked by transcutaneous electrical stimulation of the mentalis territory (to test the mandibular division) or of the infraorbital territory (to test the maxillary division), by placing the cathode on the skin overlying the mental, or the infraorbital foramen, and the anode 2 cm laterally. A stimulus lasting 0.1 ms delivered at an intensity of about 3 times the sensory threshold allows best visualisation of the first and second inhibitory periods without causing excessive discomfort. The optimal bandpass is 50 Hz–2 kHz, the sweep 100–200 ms, and the sensitivity 200 µV. The patient clenches the teeth at maximum strength, receives the stimulus and is then allowed a few seconds’ rest. At least 8 trials, but preferably 16, are repeated and superimposed. Quantitative studies of the excitability of the brainstem inhibitory interneurons require measurement of the size of responses (e.g. area of suppression): the level of background EMG activity must be kept constant, and the signal is full-wave rectified and averaged (Fig. 2). For measuring the recovery cycle with the double-shock technique (see Chapter 6.2), the optimal interval between the conditioning and test stimuli is 250 ms (Cruccu et al. 1991).

### Measurements and normal values

The peak-to-peak amplitude of the jaw jerk ranges from 0.1 mV to 4 mV but varies substantially, depending on the force and direction of the tap, position of the mandible, and occasional changes in motoneuronal excitability. It varies greatly between and within subjects. The amplitude is usually ignored. The latency is measured to the onset of the negative phase (or to the onset of the first reproducible deflection). Table 1 shows normal values. Comparisons between subjects are of little value for diagnosis. But the intraindividual latency difference between the two sides is extremely small (range 0–1 ms, mean 0.13 ± 0.17 (SD) ms in 131 normal subjects). The unilateral absence of the reflex or a side difference in latency larger than 1 ms generally implies a neurological disease. A bilaterally absent jaw jerk is a common finding in elderly patients.

The first and second components of the masseter inhibitory reflex are harder to measure, because these responses appear as phases of suppression in the ongoing EMG activity. The signals must either be full-wave rectified and averaged or examined by superimposing several trials. The onset latency should correspond to the beginning of the EMG suppression. This is usually taken at the intersection

### Table 1

<table>
<thead>
<tr>
<th>Latency (ms)</th>
<th>Jaw jerk</th>
<th>First inhibitory period (SP1 or ES1)</th>
<th>Second inhibitory period (SP2 or ES2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>6.8</td>
<td>12</td>
<td>45</td>
</tr>
<tr>
<td>Mean</td>
<td>6.8</td>
<td>11.8</td>
<td>45.1</td>
</tr>
<tr>
<td>SD</td>
<td>0.8</td>
<td>0.8</td>
<td>5.2</td>
</tr>
<tr>
<td>Range</td>
<td>5–10</td>
<td>10–13.6</td>
<td>38–60</td>
</tr>
<tr>
<td>20-year-old subjects&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.5</td>
<td>11.1</td>
<td>42</td>
</tr>
<tr>
<td>70-year-old subjects&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.0</td>
<td>12.3</td>
<td>48</td>
</tr>
</tbody>
</table>

<sup>a</sup> Standard curve calculations for age latency function.
between the inhibitory shift and a line corresponding to 80% of the mean background EMG activity (Fig. 2). The size of the response can be evaluated by measuring the area of suppression or the duration, taking the end-latency at the point when EMG returns to 80%. The onset latency is the most reliable measure for clinical applications (Table 1 shows normal values). The latency of the first inhibitory period (though not the second) has a relatively narrow range of variability, thus allowing comparisons between subjects. The intraindividual latency difference between sides is small (range 0–1.2 ms, mean 0.3 ± 0.37 ms, in 100 normal subjects). A latency difference between sides larger than 1.2 ms is abnormal. The second inhibitory period is considered abnormal if it is absent unilaterally or the latency difference between sides exceeds 8 ms. Like the jaw jerk, the second inhibitory period may be absent bilaterally in elderly patients or in patients with malocclusion.

**Factors affecting the investigation and suggestions**

Given the variability of the jaw jerk between trials, only simultaneous bilateral recordings allow a reliable assessment of the latency difference between sides. Because this reflex is strongly influenced by dental occlusion and can be asymmetrical or even absent in patients with temporo-mandibular disorders (Cruccu et al. 1997), whenever the jaw-jerk is the only abnormal trigeminal reflex it should be tested not only in the postural position but also in intercuspal occlusion or during clenching: changing the position of the mandible or the level of pre-innervation may strongly reduce or worsen the asymmetry in patients with dental problems, though not in patients with a lesion along the reflex arc.

The masseter inhibitory reflex is best assessed after rectifying and averaging the EMG signal. If these procedures are unavailable, a high-frequency background EMG should be obtained, by asking the patient to clench the teeth as strongly as possible, and allowing longer rests between trials. This decreases the likelihood of measuring an unduly short latency should the motoneurons fire at a low frequency. Alternatively, if the patient is able to produce only a weak EMG, numerous trials should be collected and the longest – not the mean – latency measured. In these patients, the best way of seeking an abnormality along the afferent pathways is to compare responses to right-side and left-side stimulation in the same muscle (direct and crossed responses are symmetrical and have the same latency), and to take the latency at a clearly reproducible point, e.g. at the last negative peak before the pause. Finally, in some patients, no break-through EMG activity separates the two inhibitory periods (so-called merged silent periods): in these recordings the latency measured when the background EMG activity finally returns represents an end-latency, thus providing a measure of the second inhibitory period.

**Typical clinical applications**

The jaw reflexes, being trigemino-trigeminal reflexes, examine the sensory and motor trigeminal fibres. Unlike the blink reflex, they are not influenced by facial-nerve function and provide information on the maxillary and mandibular divisions.

The jaw jerk is unaffected by suprasegmental lesions. It is chiefly useful for disclosing unilateral lesions. It is most sensitive to focal extra-axial compression, for example from vascular anomalies or tumours in the posterior fossa, probably because it is supplied by a small number of afferents and pressure damages the largest fibres first (Ongerboer de Visser and Cruccu 1993). The jaw jerk also provides information on the rostral brain-stem. In our experience, it is the trigeminal reflex that is most frequently abnormal in patients with demyelinating lesions in the brain-stem. The jaw jerk is also useful for differentiating axonopathies from neuronopathies; in neuronopathy it is the only reflex spared, possibly because the cell body of the primary afferents lies within the CNS rather than within the ganglion (Valls-Solé et al. 1990). Although an asymmetrical jaw jerk is a frequent finding in patients with temporo-mandibular disorders, it is by no means diagnostic (Cruccu et al. 1997).

Brain-stem inhibitory reflexes cannot be tested by clinical procedures alone. In some patients, clin-
ical examination discloses no signs of trigeminal impairment, yet testing the masseter inhibitory reflex reveals trigeminal or brain-stem dysfunction. As in blink reflex studies (see Chapter 6.2), the pattern of abnormality (afferent, mixed or efferent) provides information on the site of the lesion (Ongerboer de Visser et al. 1990). Nevertheless, except in rare conditions such as a purely motor trigeminal neuropathy and hemimasticatory spasm, the ‘efferent’ type of abnormality (abnormal responses confined to the muscle on one side, regardless of the side of stimulation) is extremely uncommon (Crucu et al. 1991).

The two inhibitory periods of the masseter inhibitory reflex have distinct EMG features and clinical applications. The first inhibitory period appears to be insensitive to peripheral conditioning and suprasegmental modulation, its latency varies little, and it is probably mediated by a small number of afferents. For these reasons it is the best available response for assessing function of the maxillary and mandibular afferents, in focal and in generalised diseases. Recent evidence shows that the first mandibular inhibitory period is extremely delayed (even > 20 ms) in patients with demyelinating polyneuropathies and also in patients with severe diabetic polyneuropathy (Auger 1996; Crucu et al. 1998). In patients with symptomatic trigeminal neuralgia or focal lesions within the pons, it has a diagnostic sensitivity similar to that of the R1 blink reflex (Ongerboer de Visser and Crucu 1993).

The second inhibitory period is far less sensitive than the first to lesions along the reflex arc. Being mediated by a multisynaptic chain of interneurons of the lateral reticular formation, however, it is modulated by suprasegmental influences. Similarly to the R2 blink reflex, the second inhibitory period shows a strongly enhanced recovery cycle in patients with extrapyramidal disorders such as Parkinson’s disease and dystonia and conversely, an increased habituation in hemiplegia (Crucu et al. 1991). The second inhibitory period (recorded from the temporalis muscle and called ES2) is a focus of research in several centres for patients with headache. Although the findings are still controversial, some data suggest that this response might help in differentiating tension headaches from vasomotor headaches (Schoenen et al. 1987).

References


