

Masseter Reflex

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October 1, 2025

RECOMMENDED CITATION

mohammad looti (2025). *Masseter Reflex*. PSYCHOLOGICAL SCALES. Retrieved from <https://scales.arabpsychology.com/?p=32201>

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Primary Disciplinary Field(s): Neurology, Clinical Assessment, Anatomy, Neurophysiology

1. Core Definition

The **masseter reflex**, also frequently referred to as the **jaw jerk reflex** or **mandibular tendon reflex**, represents a crucial diagnostic tool within neurological examinations. Fundamentally, it is a deep tendon reflex that elicits an involuntary contraction of the masticatory muscles, specifically the masseter and temporalis muscles, in response to a sudden stretch stimulus. This reflex arc is considered a monosynaptic stretch reflex, meaning it involves only two neurons and one synapse, making it one of the simplest and fastest reflex pathways in the human body. When the lower jaw is abruptly tapped, typically on the chin, it causes a momentary stretch of the masseter and temporalis muscles. This stretch is detected by muscle spindles within these muscles, which then transmit sensory information via the trigeminal nerve to the brainstem. The subsequent motor response involves the rapid upward movement of the jaw due to the contraction of the same stretched muscles.

The anatomical location of the masseter muscle, situated prominently at the posterior aspect of the cheeks, along with the fan-shaped temporalis muscle on either side of the skull, highlights their primary role in mastication. The reflex, therefore, directly assesses the integrity of the motor and sensory components responsible for jaw movement. Its significance in clinical practice stems from its ability to provide insights into the functional status of specific segments of the central nervous system (CNS), particularly pathways involving the trigeminal nerve and its connections within the brainstem. An evaluation of this reflex is a standard component of a comprehensive neurological assessment, offering valuable diagnostic clues.

The physiological basis of the masseter reflex, like other stretch reflexes, lies in the feedback mechanism designed to maintain muscle length and tone. When a muscle is suddenly stretched, the muscle spindles are activated, sending afferent signals to the CNS. These signals synapse directly with alpha motor neurons in the brainstem, which then send efferent signals back to the same muscle, causing it to contract. This rapid, involuntary response is protective, preventing overstretching of the muscles and contributing to postural stability of the jaw. The efficiency and magnitude of this reflex are finely tuned by descending pathways from higher brain centers, which can either facilitate or inhibit the reflex arc, thereby influencing its clinical presentation.

2. Etymology and Historical Development

The terms "masseter reflex," "jaw jerk reflex," and "mandibular tendon reflex" are descriptive of the anatomical structures and the type of stimulus involved. "Masseter" refers to the primary muscle whose contraction is observed, while "jaw jerk" vividly describes the quick, involuntary upward

movement of the mandible. "Mandibular tendon reflex" points to the location of the stimulus application, typically over the mentalis muscle or tendon near the chin, which indirectly stretches the masticatory muscles. The understanding and clinical application of deep tendon reflexes, including the masseter reflex, began to gain prominence in the late 19th and early 20th centuries as neurology emerged as a distinct medical specialty.

Early neurologists, such as Jean-Martin Charcot and Wilhelm Heinrich Erb, systematized the examination of reflexes as a cornerstone of neurological diagnosis. They recognized that deviations from normal reflex responses could localize lesions within the nervous system. The masseter reflex, specifically, was identified as an indicator of upper motor neuron integrity related to the trigeminal nerve pathway. Its relatively simple reflex arc, involving the pontine region of the brainstem, made it a valuable tool for assessing conditions affecting this critical area.

Over time, the methodology for eliciting and interpreting the masseter reflex has been refined. Standardized techniques, such as instructing the patient to slightly open their mouth and relax their jaw, and using a reflex hammer to tap the chin, ensure consistency in examination. The interpretation of findings, particularly the significance of an exaggerated or absent reflex, has become more nuanced with advancements in neurophysiology and imaging techniques, allowing for a more precise correlation between reflex findings and specific neurological pathologies.

3. Key Characteristics

The masseter reflex exhibits several key characteristics that define its physiological nature and clinical utility. Firstly, it is a **monosynaptic stretch reflex**, distinguishing it from polysynaptic reflexes that involve multiple interneurons. This direct connection between the sensory neuron (from the muscle spindle) and the motor neuron (to the masseter and temporalis muscles) results in a rapid response, making it one of the fastest reflexes in the body. The afferent and efferent limbs of this reflex arc are carried by the trigeminal nerve (cranial nerve V), specifically its mandibular division, with the integration center located in the pons of the brainstem.

Secondly, the reflex is elicited by a specific mechanical stimulus: a sudden, brisk tap on the chin while the mouth is slightly open. This tap causes a quick stretch of the masseter and temporalis muscles. The response is an equally brisk, involuntary contraction of these same muscles, leading to an upward movement of the mandible. The magnitude of this contraction can vary among individuals, but a clearly discernible, yet not overly forceful, movement is considered normal. The absence or exaggeration of this response is of significant clinical concern, pointing towards neurological dysfunction.

Thirdly, the masseter reflex serves as an invaluable tool for assessing the integrity of the upper motor neuron (UMN) pathways that descend from the cerebral cortex to the trigeminal motor nucleus in the brainstem. These descending pathways typically exert an inhibitory influence on

stretch reflexes. Therefore, damage to these UMN pathways, such as in cases of stroke, multiple sclerosis, or other demyelinating diseases, can release this inhibition, leading to an exaggerated or hyperactive masseter reflex. Conversely, damage to the trigeminal nerve itself (lower motor neuron lesion), or severe lesions within the brainstem affecting the reflex arc, can result in a diminished or absent reflex. This characteristic makes it a localizing sign for neurological lesions.

4. Clinical Significance and Assessment

The masseter reflex holds significant clinical importance as a rapid and non-invasive method for evaluating the functional status of specific neurological pathways. Its primary role is to help assess the integrity of the trigeminal nerve and the upper motor neuron pathways projecting to the trigeminal motor nucleus in the pons. When a physician performs this test, they typically instruct the patient to slightly open their mouth, allowing the jaw muscles to be relaxed. The examiner then places a finger on the patient's chin and taps their own finger with a reflex hammer, or directly taps the chin with the hammer, triggering the stretch of the masseter and temporalis muscles. A normal response is a subtle, brisk upward movement of the jaw.

The interpretation of the masseter reflex response provides critical diagnostic clues. An **exaggerated masseter reflex**, characterized by a particularly brisk and forceful jaw closure, is a hallmark sign of upper motor neuron (UMN) lesions affecting the corticobulbar tracts that descend to the trigeminal motor nucleus. Such lesions can result from conditions like stroke, amyotrophic lateral sclerosis (ALS), multiple sclerosis, or brain tumors. The hyperreflexia occurs because the inhibitory influence normally exerted by the UMNs on the reflex arc is lost, leading to an unchecked motor response. This can also be observed in conditions like pseudobulbar palsy.

Conversely, a **diminished or absent masseter reflex** typically indicates a lesion involving the reflex arc itself. This could be a lower motor neuron (LMN) lesion affecting the trigeminal nerve (either its sensory or motor components), damage to the trigeminal ganglion, or a lesion within the brainstem affecting the trigeminal motor nucleus or its interconnections. Conditions such as trigeminal neuropathy, peripheral nerve damage, brainstem infarction, or severe myopathies affecting the masticatory muscles can present with a hyporeflexic or absent masseter reflex. Therefore, the masseter reflex serves as a localized indicator of neurological impairment, guiding further diagnostic investigations.

5. Associated Neurological Pathways

The masseter reflex arc is an elegant, yet fundamental, example of a simple neural circuit, primarily mediated by the trigeminal nerve (CN V). Understanding its associated pathways is crucial for interpreting clinical findings. The reflex begins with the activation of muscle spindles located within the masseter and temporalis muscles. These specialized stretch receptors detect

changes in muscle length and the rate of change in length. When the jaw is tapped, these spindles are stretched, generating an afferent (sensory) signal.

The afferent signals from the muscle spindles travel via the sensory fibers of the mandibular division of the trigeminal nerve. Unlike most sensory fibers that have their cell bodies in the trigeminal ganglion, the cell bodies for the muscle spindle afferents of the jaw muscles are uniquely located in the mesencephalic nucleus of the trigeminal nerve, situated in the midbrain and upper pons. This nucleus essentially acts as a sensory ganglion within the central nervous system. From the mesencephalic nucleus, the central processes of these primary afferent neurons project directly to the trigeminal motor nucleus, which is located in the mid-pons.

This direct connection between the sensory neuron in the mesencephalic nucleus and the motor neuron in the trigeminal motor nucleus forms the monosynaptic arc, which is the hallmark of the masseter reflex. The motor neurons within the trigeminal motor nucleus are alpha motor neurons whose axons constitute the efferent (motor) limb of the reflex. These efferent fibers exit the brainstem via the mandibular division of the trigeminal nerve and innervate the masseter and temporalis muscles, causing their contraction and the characteristic upward jaw movement. This entire circuit demonstrates how a simple mechanical stimulus is rapidly converted into a motor response, ensuring the protective function of jaw muscles and their contribution to mastication.

6. Differential Diagnosis and Related Conditions

An abnormal masseter reflex can be a key indicator in the differential diagnosis of various neurological conditions. The primary distinction made based on the reflex's response is between an exaggerated (hyperreflexic) and a diminished or absent (hyporeflexic/areflexic) state, each pointing to different types of neurological lesions. An **exaggerated masseter reflex** is highly suggestive of an upper motor neuron (UMN) lesion affecting the corticobulbar tracts that descend from the cerebral cortex to the trigeminal motor nucleus. Conditions associated with hyperreflexia include cerebrovascular accidents (stroke), particularly those involving the brainstem or cerebral hemispheres, multiple sclerosis, amyotrophic lateral sclerosis (ALS), brain tumors, and various forms of spasticity. The hyperreflexia results from the loss of inhibitory control normally exerted by these descending pathways, leading to an overactive reflex response. It can also be a component of pseudobulbar palsy, a condition characterized by emotional lability, dysarthria, and dysphagia, often seen in bilateral UMN lesions of the brainstem.

Conversely, a **diminished or absent masseter reflex** points towards a lesion within the reflex arc itself, typically an lower motor neuron (LMN) lesion. This could involve damage to the trigeminal nerve (cranial nerve V) along its course, from the brainstem nucleus to the muscles of mastication. Causes include trigeminal neuropathy (e.g., due to trauma, compression, inflammation), Guillain-Barré syndrome affecting cranial nerves, brainstem lesions (e.g., infarction, tumor) directly

impacting the trigeminal motor nucleus or its sensory afferents, and certain myopathies affecting the masseter and temporalis muscles. Peripheral nerve damage due to trauma or other pathologies can also disrupt the afferent or efferent limbs of the reflex, leading to a hyporeflexic state. Thus, the masseter reflex provides valuable information for localizing neurological pathology, helping clinicians narrow down the potential causes of a patient's symptoms and guide further diagnostic imaging or laboratory tests.

7. Debates and Criticisms

While the masseter reflex is a well-established component of neurological examination, its interpretation and utility, like many clinical signs, are subject to certain nuances and considerations that can be viewed as "debates" or "criticisms" in a broader sense. One primary point of discussion revolves around the subjectivity inherent in assessing reflex responses. The degree of "briskness" or "exaggeration" can vary among examiners, potentially leading to inconsistencies in diagnosis. Standardization of technique and consistent training are critical to minimize this variability, yet subtle differences in patient positioning, muscle relaxation, and hammer tapping can influence the perceived response. This highlights the importance of integrating reflex findings with a comprehensive neurological examination and other diagnostic tests.

Another aspect concerns the sensitivity and specificity of the masseter reflex as a diagnostic tool. While an exaggerated reflex is strongly indicative of UMN pathology, it is not always present in all UMN lesions, nor is its absence definitive proof against certain LMN conditions. Furthermore, factors such as anxiety, muscle tension, or even fatigue can influence reflex responses, potentially leading to false positives or false negatives if not carefully controlled. For instance, a highly anxious patient might inadvertently tense their jaw muscles, which could subtly alter the reflex response. Therefore, clinicians must consider the patient's overall clinical picture, including other neurological signs and symptoms, when interpreting the masseter reflex.

Finally, while the masseter reflex is valuable for localizing lesions to the trigeminal pathways and descending corticobulbar tracts, it does not provide information about the specific etiology of the lesion. An exaggerated reflex indicates a UMN problem, but it cannot differentiate between a stroke, a tumor, or a demyelinating disease without further investigation. Similarly, a diminished reflex points to an LMN issue, but not the cause. Thus, the masseter reflex serves as an important screening tool that guides further, more specific diagnostic evaluations rather than providing a definitive diagnosis on its own. Its utility is maximized when combined with advanced imaging, electrophysiological studies, and laboratory tests to reach an accurate and comprehensive understanding of the patient's neurological condition.

Further Reading

[Masseter Reflex - Wikipedia](#)

[Trigeminal Nerve - Wikipedia](#)

[Deep Tendon Reflex - Wikipedia](#)

[Upper Motor Neuron Lesion - Wikipedia](#)

[Lower Motor Neuron Lesion - Wikipedia](#)

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