Fatigue in Human Jaw Muscles: A Review

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Muscle fatigue has been thought to be one of the causes of pain associated with temporomandibular disorders. A multitude of variables could contribute to neuromuscular fatigue when a subject attempts to sustain a given force. In studies of jaw muscles the endurance limit has been related to a failure in electrical conductivity (transmission fatigue), an increasing imbalance in the intracellular contents of muscle fibers (contraction fatigue), and the onset of pain. This review describes the principles that underlie fatigue and the results of studies of jaw muscle fatigue. Attempts are made to explain why various studies may have produced different results.
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Studies of jaw muscle fatigue may have more clinical importance than their contribution to unraveling the nature of neuromuscular fatigue. Temporomandibular disorders (TMD) have often been related to hyperactivity of the jaw muscles and to parafunctions such as bruxism. It is probable that both could cause fatigue in jaw muscles, which, in turn, could trigger some of the observed signs and symptoms associated with TMD.[1–6]

The classic definition of neuromuscular fatigue has been a failure of the tissues to maintain an expected force.[7] This is perhaps too broad a definition because it would include, for example, failure due to a sudden traumatic injury to a nerve or muscle. Other features of fatigue include that the failure develops after the given activity has been sustained for some time and that the initial level of activity returns after rest.

A chain of rapidly occurring events connects the conscious decision to contract a muscle and the actual movement during which thin filaments slide across thick filaments within a muscle fiber (cell). The chain has three major subdivisions. These are the decision itself, the electrical transmission of a signal, and the chemical reactions inside the muscle fiber that eventually lead to the mechanical movement. Any of these could be the source of fatigue (Fig 1).[8]

Central fatigue involves a failure of the command associated with the motor cortex and the upper motoneuron.[9] The central nervous system reduces its motor drive (eg, the subject is "tired of continuing"), but the peripheral elements of the nerve and the muscle fibers are unimpaired.[10] More force could be generated by voluntary effort or by directly stimulating the muscle.[11–14] Central fatigue is beyond the scope of this review.

Peripheral fatigue affects components of the command chain distal to and including the motor end plate (Fig 1). It may be caused by a disruption in either transmission or contraction. *Transmission fatigue* is caused by a failure either at the motor end plate or in the postsynaptic propagation of the electrical impulse along the cell membrane, including its extensions into the T-tubules, of a muscle

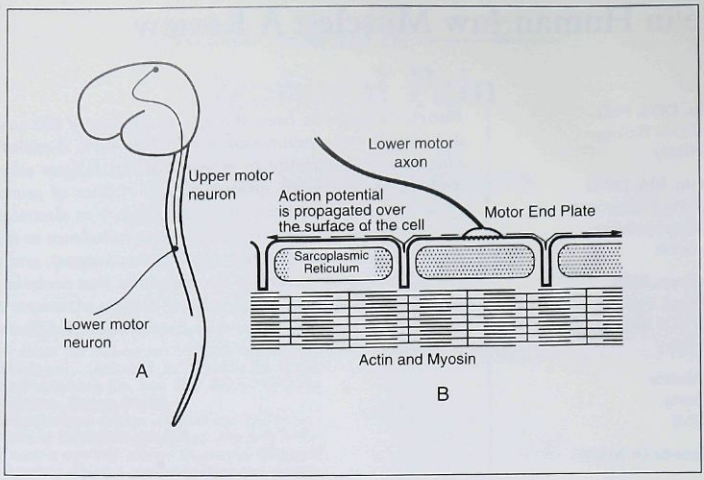

Fig 1 (A) Sites susceptible to central fatigue; (B) sites susceptible to peripheral fatigue. The arrowed lines from the motor end plate passing over the surface of the cell and down the T-tubules (between the elements of sarcoplasmic reticulum) represent the passage of an action potential: these sites are susceptible to transmission fatigue. Intracellular events are susceptible to contraction fatigue. The extracellular and intracellular events are linked by excitation-contraction coupling.

fiber. *Contraction fatigue* is due to either a failure to convert the extracellular electrical transmission into an intracellular chemical reaction or to a failed link in the chain of these intracellular reactions, the end result of which is to cause the actin and myosin filaments to slide across each other.

Fatigue can be induced either by sustained voluntary contractions or by electrically stimulating a motor nerve or the muscle itself. Electrical stimulation can produce two types of fatigue, depending on the frequency at which the nerve or muscle is stimulated. *Low-frequency fatigue* corresponds to contraction fatigue and is the result of stimulation below approximately 20 Hz (20 stimuli per second). It takes a long time to develop and recovery is slow. *High-frequency fatigue* corresponds to transmission fatigue and is the result of stimulation above approximately 80 Hz.[7,14] It is quickly induced and recovery is rapid.

The intensity and duration of a task are the main factors that determine the site(s) of fatigue.[7,15] Sustained maximum voluntary contraction (MVC) and high-frequency stimulation both interfere with the propagation of the action potential from the neuromuscular junction (motor end plate) and/or along the surface of the muscle fiber. On the other hand, prolonged submaximal voluntary contractions (subMVC) and low-frequency stimulation both interfere with excitation-contraction coupling (transforming the extracellular action potential into the chain of intracellular chemical reactions) and/or the energy supply and metabolic processes related to these reactions.[14,16] The relationship between different tasks and the fatigue they produce is shown in Table 1.

The type of fatigue may be identified by comparing the electromyographic (EMG) activity of a muscle with its mechanical output, ie, the force it generates. Transmission fatigue causes a parallel decline in both the force and EMG amplitude. In contraction fatigue the amplitude of the EMG activity is unchanged while the force drops, or the EMG activity is increased while the force remains unchanged.[11,12,14,17]

Electromyographic records superimpose a spectrum of frequencies because the different types of fiber in a muscle have different activation rates. Axons supplying slow muscle fibers have a slower

Table 1 Peripheral Fatigue

	Contraction (low-frequency) fatigue	Transmission (high-frequency) fatigue
Inducing task	Low intensity, subMVC	High intensity, MVC
Site of occurrence	1. Excitation-contraction coupling 2. Contractile elements	1. Neuromuscular junction 2. Muscle fiber membrane
Indicator	Force decreases EMG amplitude increases or no change EMG frequency decreases	Force decreases EMG amplitude decreases EMG frequency decreases

MVC = maximum voluntary contraction; subMVC = submaximum voluntary contraction.

conduction velocity and discharge at a lower frequency than those supplying fast fibers. By a mathematic manipulation a spectrum of the different frequencies (power spectrum) of a raw EMG record can be derived and the mean or median power frequency calculated.[18] In both contraction and transmission fatigue the mean or median power frequency (MPF) may drop because the low-frequency components increase and the high-frequency components decrease simultaneously.[18–21]

The characteristics of the above three variables (force, EMG amplitude, and MPF) in contraction and transmission fatigue are summarized in Table 1.

Cellular and Molecular Mechanisms

The factors involved in muscle contraction and fatigue are so numerous and complex that, as yet, only parts of the full picture are clear. It is unlikely that fatigue will be completely understood until it is known how electrical transmission outside the cell initiates chemical reactions inside the cell. There are many substances that might be involved in causing contraction fatigue and their roles have not been fully evaluated. For example, a fatigued muscle has low levels of Ca^{2+}, glycogen, and adenosine triphosphate (ATP), and high levels of lactate. Any of these, alone or combined, or an excess or depletion of some other substance might be the primary cause of contraction fatigue.

Although it is not the intention of this analysis to examine in detail the cellular and molecular mechanisms related to fatigue, it is necessary to describe some previous observations and conclusions in order to discuss issues relevant to jaw muscles.

Fatigue from low-intensity tasks is produced by prolonged submaximal voluntary contractions or by stimulating muscles or lower motoneurons at submaximal frequencies. During muscle contraction, calcium ions are released from the sarcoplasmic reticulum and bind to the troponin in the thin, actin-containing muscle filaments (Fig 1). When activated by Ca^{2+}, troponin indirectly initiates the formation of the cross-bridges between actin and myosin, which are required for muscle contraction. Prolonged contraction reduces the concentration of releasable Ca^{2+} in the sarcoplasmic reticulum.[14,15,22,23] The resulting (contraction) fatigue may be due to a reduction in Ca^{2+} concentration and, consequently, the number of cross-bridges between myosin and actin.[24]

Calcium ions not only bind to troponin to initiate muscle contraction but also participate in the conversion of ATP to adenosine diphosphate (ADP), a process that releases the energy used to drive the contraction. The reduced Ca^{2+} concentration may be due to an excess being bonded to troponin[25] with the result that too few are available to release sufficient energy from the ATP to continue contracting the muscle.[26,27]

The ATP is also required to transport Ca^{2+} from the cytoplasm of the muscle fiber back into its sarcoplasmic reticulum. The depletion of calcium ions in the sarcoplasmic reticulum, which is associated with fatigue, may therefore be due to a reduction in the amount of ATP necessary to drive Ca^{2+} back.

Both slow and fast muscle fibers are normally involved in sustained muscle contractions. Slow fibers use oxidative phosphorylation to recycle ADP back into ATP (catalyzed by Ca^{2+}) in order to provide the energy for sustaining a contraction. If oxygen is lacking, then ATP is also depleted and the muscle fatigues because it lacks the energy to drive the contraction. Previously uncontracted fast

fibers might now be recruited to maintain the force output of the muscle.

Fast muscle fibers derive their energy supply from anaerobic metabolism by converting glucose to lactic acid, called a fatigue substance by Asmussen.[9] The presence of lactic acid causes an increase in intracellular H^+, which may compete with Ca^{2+} for the binding sites on troponin.[15,24] Fatigue could be due to a lack of sufficient Ca^{2+} to bind with troponin and sustain the contraction. Additionally, some key glycolytic enzymes are sensitive to changes in pH. The rise in lactate leads to a fall in pH, which slows down the glycolytic reaction required to convert ADP back into ATP.

Fatigue from high-intensity tasks is induced by sustained maximum voluntary contractions or by high-frequency stimulation. Unlike fatigue induced by low-intensity tasks, it is related to a failure in the transmission of the electrical impulse either across the motor end plate[25] (Fig 1) or along the muscle cell membrane and down the T-tubules.[13,16,26]

There is about 40 times greater K^+ concentration inside a resting cell than outside. There is only about 5 times the concentration of the smaller Na^+ outside the cell compared with its inside. The net imbalance is largely responsible for a resting muscle fiber being polarized (having an internal negative charge). When an action potential reaches any given point on the cell membrane, local sodium channels in the cell membrane are opened and Na^+ rushes into the cell, down their concentration gradient. At the same time potassium channels are opened and the K^+ moves more slowly out of the cell.[8,28] The net effect is to cause the inside of the cell to become positively charged (the membrane is said to be depolarized). Immediately after this, sodium is pumped out locally to restore the (polarized) resting negative charge. The wave of depolarization (the action potential) has now traveled past the previously given point. A local potassium pump moves K^+ more slowly back into the cell.

During maximum effort, the potassium channels are constantly open. Potassium ions are constantly diffusing out of the muscle cell and not being pumped back.[23,24] For example, the intracellular concentration of K^+ in a rabbit masseter fatigued by high-frequency stimulation was reduced by 33%.[29] The high positive charge that builds up outside the cell is referred to as hyperpolarization. A hyperpolarized cell membrane cannot be sufficiently depolarized to propagate an action potential. This may explain the rapid onset of (transmission) fatigue at MVC and at high-frequency stimulation.

With the onset of transmission fatigue, the action potential ceases being propagated and K^+ can now be pumped back into the cell to restore the resting polarization. This may account for the rapid recovery from fatigue induced at MVC or at high-frequency stimulation.

Fatigability of Human Jaw Muscles

The conditions used to induce fatigue in an experiment are not necessarily the same as those that induce fatigue during daily life. Christensen[30] and Lyon and Baxendale[31] argued that the usual intercuspal distance is increased with an instrument for measuring the occlusal force (an occlusal force transducer) between the occluding teeth. This stretches the length of the fibers in the jaw closing muscle and might modify the sensory input from muscle and joint receptors. Jow and Clark[32] argued that the jaw separation would simulate more naturally the conditions during mastication, whereas a clench in centric occlusion is not used to break up food. Since force is an essential variable in studying fatigue, the submaximum isometric occlusal force can only be measured with a transducer (placed between the teeth) and, as a result, the teeth must be separated.[33]

Endurance Time

Dahlstrom et al[34] showed that if the maximum voluntary occlusal force (MVOF) was measured, the endurance time was about 29 seconds, but during this period the occlusal force decreased by 10% if the subject was unaware of the actual force being used (without biofeedback). At 50% MVOF the endurance time was 107 seconds. These figures for the endurance time generally agree with those of Palla and Ash[21] (about 25 to 50 seconds at maximum intercuspal clenching), Christensen[30] and Christensen and Mohamed[35] (about 30 to 40 seconds at maximum intercuspal clenching), Naeije[36] (53 to 206 seconds at 50% MVOF), and Lyons and Baxendale[31] (less than 120 seconds at 50% MVOF). Hellsing and Lindstrom[33] recorded a long endurance time (10 to 15 minutes) when 40% MVOF was sustained on a transducer placed between the incisors. They reported that some subjects felt pain after about 1 minute.

EMG Amplitude

Masseter EMG amplitude increased by about 15% during the last of four successive endurance tests.[30,35] When unilateral occlusal force was moni-

tored with a transducer, the force gradually declined and EMG activity increased in the ipsilateral masseter muscle and decreased in the contralateral.[37]

When 40% incisal MVOF was sustained, the EMG activity decreased in the masseter muscle and increased in the temporalis muscle.[33] With 50% MVOF, Lyons and Baxendale[31] found their integrated EMG results to be inconclusive.

EMG Power Spectrum

Palla and Ash[21] showed that during maximum voluntary contraction the MPF of anterior temporal muscles dropped after about 25% of the endurance time. The masseter MPF dropped when sustaining 40% MVOF[38] and when sustaining 20%, 30%, 40%, 50%, 60%, 75%, or 90% of maximum EMG activity.[36,39] In addition, MPF shifted to lower frequencies more rapidly at stronger contraction levels.[39]

Van Boxtel et al[40] compared the endurance time at 50% of maximum EMG activity of five of the muscles of facial expression with that of the masseter and temporalis muscles. The drop in MPF was most rapid in the masseter, suggesting that this muscle is the more susceptible to fatigue.

The changes in MPF, EMG amplitude, and occlusal force in many of the above studies suggest that human jaw closing muscles are susceptible to transmission and/or contraction fatigue. However, Clark and coworkers[1,41,42] suggested that jaw muscles may not be susceptible to contraction failure. In different experiments, subjects maintained various levels of occlusal force, from 25% MVOF to MVOF.[1,41,42] Each submaximal task was repeatedly alternated with brief MVOF tasks. The endurance limit was caused by pain. Even after they had reached their endurance limit, subjects were able to repeat MVOF following a brief pause. Rapid recovery is not consistent with contraction failure. The authors, therefore, concluded that contraction fatigue had not limited the endurance time.

Clark et al[43] showed that the sum of the EMG amplitude of masseter and anterior temporal muscles divided by the occlusal force magnitude (EMG/force ratio) did not change during sustained contractions at various levels of occlusal force (the ratio input/output remained constant). The MPF, however, dropped. After maintaining a MVOF until the endurance limit, most subjects could nearly repeat the original MVOF after a 30-second rest.[33] Their most important conclusion was that endurance time is limited by pain.

The masseter muscle's possible high resistance to fatigue has been demonstrated by van Steenberghe et al.[44] They compared the ability to maintain the maximum force exerted by jaw, handgrip, and arm-flexor muscles. For each exercise, subjects exerted maximum force for 5 seconds, followed by a 15-second rest. Each task was repeated six times. The masseter muscle resisted fatigue better than the two limb muscles.

Discussion

During an experiment a subject may or may not be aware of the level of fatigue. Conceivably, the level might be measured as (1) a drop in the force output with unchanged EMG activity, (2) a rise in EMG activity with maintained force, (3) a change in the pattern of muscle activity (one muscle increases its activity to compensate for the declining activity of another in the same synergistic group), (4) a change in MPF, (5) a change in features associated with the conduction of the electrical impulse, (6) a change in the balance of intracellular substances, (7) the amount of discomfort or pain, or (8) a change in the flow of blood to the muscle.

Any of the above might be used to measure fatigue. One could, for example, theoretically describe the amount of transmission fatigue as a percentage increase in extracellular K^+, or the amount of contraction fatigue as the intracellular concentration of Ca^{2+} or lactate. Fatigue is a progressive process.[8,45] When the amount of fatigue reaches a critical level, it forces a subject to end a task. This is the failure point. The time between the beginning of a task and the failure point is the endurance time. Endurance time may be limited by transmission failure (the critical level of transmission fatigue), contraction failure, pain (Clark and coworkers[1,41,42]), or some other unknown cause.

Changes in EMG amplitude and MPF develop before the failure point is reached. The MPF drops early during a sustained task, while the EMG amplitude changes near the end of the task in certain limb muscles.[18,19] Differences in defining the failure point may account for some differences between results. In some studies it was reported that the subject ended the test because there was no power in the jaw muscle, in some the test was ended with the onset of discomfort, and in others it was ended by pain. Kroon et al[39] indicated that contraction was terminated because of a lack of power in jaw muscles at high isometric contraction levels and because of pain at low levels. We can (personally) confirm that an otherwise normal, but dedicated, subject who continues sustaining an occlusal force

for as long as possible does indeed experience considerable muscular pain. The pain, which develops in response to sustained effort, is transient rather than prolonged.[46,47]

Studies of jaw muscle fatigue have, as yet, been limited. All members of a synergistic group of muscles should be simultaneously monitored because a reduced activity in one may be compensated by an increased activity in another.[33] If only the occlusal force is being measured (and sustained) then, prior to the failure point, other signs are hidden and the only indication of progressive fatigue is discomfort or pain. Medial pterygoid activity has rarely been studied because it is only accessible to needle electrodes. Furthermore, all the jaw closing muscles contain differently oriented elements that can operate independently.[48,49] The deep elements are also inaccessible to surface EMG measurements.

Occlusal forces have only been monitored with unidirectional force transducers, which measure the component of the force parallel to a measuring axis. Using a three-dimensional occlusal force transducer Osborn and Mao[50] have shown that the early anterior direction of an incisal occlusal force changes to nearly vertical at the end of the maximum voluntary contraction. An apparent change in the magnitude of the occlusal force measured by a unidirectional transducer may in reality be partially due to a change in its direction. This may also account for some discrepancies between the results of previous studies.

Human jaw muscles contain different proportions of fast and slow fibers.[51,52] Henneman's size principle states that when a muscle contracts with increasing force, slow motor units are recruited first, followed by fast motor units.[53,54] This orderly recruitment pattern has been shown to exist in jaw muscles.[55–58] The fast fibers in jaw muscles are usually recruited for stronger forces.

Histochemical studies have consistently suggested that human jaw closing muscles contain rather more fatigue-resistant slow fibers (type I) than fatigue-susceptible fast fibers (type IIB).[47,51] The electrical responses to fatigue of these two types of fiber in jaw muscles, however, have only been tested once.[59] In limb muscles type I and type II fibers have been shown to have different glycogen depletion patterns and this suggests that the rate at which they lose force (ie, their fatigability) is also different.[60]

Histochemical studies have also indicated that there are very few fast-contracting, fatigue-resistant (type IIA) fibers in human jaw closing muscles.[51] These are abundant in ruminants such as cattle and sheep, animals that spend much of their waking life chewing. The near absence of type IIA fibers from human jaw muscles seems to suggest that they would be readily susceptible to fatigue during sustained effort at stronger force levels because fast, fatigue-susceptible (type IIB) fibers must be recruited at these levels.

The considerable differences in the cell populations and structure between human jaw and limb muscles[61] may account for some of the differences observed between them in studies of fatigue. Mao et al[47] suggested that the existing proportions of fiber types in human jaw muscles may be recent and associated with the change to an increasingly softer and less challenging diet. It could have been important for earlier human populations to be able to maintain prolonged jaw activity while breaking up tough low-energy food or while chewing on leather to soften it. Part of the change would have been a decrease in the number of fast, fatigue-resistant, type IIA fibers. The pain described by Clark and coworkers could be a protective mechanism, related to this decrease, which prematurely affects some temporomandibular disorder subjects.

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References

1. Clark GT, Beemsterboer PL, Jacobson R. The effect of sustained submaximal clenching on maximum bite force in myofascial pain dysfunction patients. J Oral Rehabil 1984;11:387–391.
2. Helkimo M. Epidemiological surveys of dysfunction of the masticatory system. In: Zarb GF, Carlsson GE (eds). Temporomandibular Joint, Function and Dysfunction. Copenhagen: Munksgaard, 1979.
3. Katz JO, Rugh JD, Hatch JP, Langlais RP, Terezhalmy GT, Borcherding SH. Effect of experimental stress on masseter and temporalis muscle activity in human subjects with temporomandibular disorders. Arch Oral Biol 1989; 34:393–398.
4. Laskin D. Etiology of the pain dysfunction syndrome. J Am Dent Assoc 1969;79:147–153.
5. Sheikholeslam A, Møller E, Lous I. Pain, tenderness and strength of human mandibular elevators. Scand J Dent Res 1980;88:60–66.
6. Wanman A, Agerberg G. Relationship between signs and symptoms of mandibular dysfunction in adolescents. Community Dent Oral Epidemiol 1986;14:225–230.
7. Edwards RHT. Human muscle function and fatigue. In: Porter R, Whelan J (eds). Human Muscle Fatigue: Physiological Mechanisms. London: Pitman Medical, 1981.

8. Clamann EP. Changes that occur in motor units during activity—causes and amelioration of fatigue. In: Binder MD, Mendell LM (eds). The Segmental Motor System. New York: Oxford University Press, 1990:239–257.
9. Asmussen E. Muscle fatigue. Med Sci Sports 1979;11: 313–321.
10. Edwards RHT, Jones DA. Diseases of the skeletal muscle. In: Peachey LD (ed) Handbook of Physiology. Bethesda, MD: American Physiological Society, 1983:633–652.
11. Bigland-Ritchie B. EMG/force relations and fatigue of human voluntary contractions. Exerc Sport Sci Rev 1981; 9:757–117.
12. Bigland-Ritchie B. EMG and fatigue in human voluntary and stimulated contractions. In: Porter R, Whelan J (eds). Human Muscle Fatigue: Physiological Mechanisms. London: Pitman Medical, 1981.
13. Bigland-Ritchie B, Jones DA, Woods JJ. Excitation frequency and muscle fatigue: Electrical responses during human voluntary and stimulated contractions. Exp Neurol 1979;64:414–427.
14. Bigland-Ritchie B, Bellemare F, Woods JJ. Excitation frequencies and sites of fatigue. In: Jones NL, Mccartney N, McComas AJ (eds). Human Muscle Power. Champaign, IL: Human Kinetics Publishers, 1986:197–214.
15. Roberts D, Smith DJ. Biochemical aspects of peripheral muscle fatigue: A review. Sports Med 1989;7:125–138.
16. Stephens JA, Taylor A. Fatigue of maintained voluntary muscle contraction in man. J Physiol (Lond) 1972;220:1–18.
17. Edwards RG, Lippold OCJ. The relation between force and integrated electrical activity in fatigued muscle. J Physiol (Lond) 1956;132:677–681.
18. Basmajian JV, De Luca CJ. Muscles Alive, Their Functions Revealed by Electromyography. Baltimore: Williams & Wilkins, 1985:65–100.
19. De Luca CJ. Myoelectrical manifestations of localized muscular fatigue in humans. Crit Rev Biomed Eng 1985; 11:251–279.
20. Lindstrom L, Magnusson R, Petersen I. Muscular fatigue and action potential conduction velocity studied with frequency analysis of EMG signals. Electromyography 1970; 4:341–356.
21. Palla S, Ash MM Jr. Power spectral analysis of the surface electromyogram of human jaw muscles during fatigue. Arch Oral Biol 1981;26:547–553.
22. Belcastro AN, Maclean I, Gilchrist J. Biochemical basis of muscular fatigue associated with repetitious contractions of skeletal muscle. Int J Biochem 1985;17:447–453.
23. Vollestrad NK, Sejersted OM. Biochemical correlates of fatigue. A brief review. Eur J Appl Physiol 1988;57:336–347.
24. Hultman E, Sjoholm H: Biochemical causes of fatigue. In: Jones NL, Mccartney N, McComas AJ (eds). Human Muscle Power. Champaign, IL: Human Kinetics, 1986:215–238.
25. Jones DA. Muscle fatigue due to changes beyond the neuromuscular junction. In: Porter R, Whelan J (eds). Human Muscle Fatigue: Physiological Mechanisms. London: Pitman Medical, 1981.
26. Bergstrom J, Hermansen L, Hultman E, Saltin B. Diet, muscle glycogen and physical performance. Acta Physiol Scand 1967;71:140–150.
27. Karlsson J, Jodin B, Jacobs I, Kiser P. Relevance of muscle fibre type to fatigue of short intense, and prolonged exercise in man. In: Porter R, Whelan J (eds). Human Muscle Fatigue: Physiological Mechanisms. Turnbridge Wells, London: Pitman Medical, 1981.
28. Sjogaard G. Exercise-induced muscle fatigue: The significance of potassium. Acta Physiol Scand 1990;40(suppl 593):1–63.
29. Engel MB, Bressman JK. The effect of fatigue on essential element distribution in the rabbit masseter. Scanning Microsc 1988;2:337–344.
30. Christensen LV. Cumulative electromyography of the human masseter muscle during fatiguing isometric contractions. J Oral Rehabil 1984;11:341–349.
31. Lyon MF, Baxendale RH. A preliminary electromyographic study of bite force and jaw-closing muscle fatigue in human subjects with advanced tooth wear. J Oral Rehabil 1990;17:311–318.
32. Jow RW, Clark GT. Endurance and recovery from a sustained isometric contraction in human jaw-elevating muscles. Arch Oral Biol 1989;34:857–862.
33. Hellsing G, Lindstrom L. Rotation of synergistic activity during isometric muscle contraction in man. Acta Physiol Scand 1983;118:203–207.
34. Dahlstrom L, Tzakis M, Haraldson T. Endurance tests of the masticatory system on different bite force levels. Scand J Dent Res 1988;96:137–142.
35. Christensen LV, Mohamed SE. Contractile activity of the masseter muscle in experimental clenching and grinding of the teeth in man. J Oral Rehabil 1984;11:191–199.
36. Naeije M. Correlation between surface electromyograms and the susceptibility to fatigue of the human masseter muscle. Arch Oral Biol 1984:29:865–870.
37. Haraldson T, Carlsson GE, Dahlstrom L, Jansson T. Relationship between myoelectric activity in masticatory muscles and bite force. Scand J Dent Res 1985;93:539–545.
38. Lindstrom L, Hellsing G. Masseter muscle fatigue in man objectively quantified by analysis of myoelectric signals. Arch Oral Biol 1983;28:297–301.
39. Kroon GW, Naeije M, Hansson TL. Electromyographic power-spectrum changes during repeated fatiguing contractions of human masseter muscle. Arch Oral Biol 1986;31:603–608.
40. Van Boxtel A, Goudswaard P, van der Molen GM, van den Bosch WE. Changes in electromyogram power spectra of facial and jaw-elevator muscles during fatigue. J Appl Physiol 1983;54:51–58.
41. Clark GT, Carter MC. Electromyographic study of human jaw-closing muscle endurance, fatigue and recovery at various isometric force levels. Arch Oral Biol 1985;30:563–569.
42. Clark GT, Adler RC. Retrusive endurance, fatigue and recovery of human jaw muscles at various isometric force levels. Arch Oral Biol 1987;32:61–65.
43. Clark GT, Carter MC, Beemsterboer PL. Analysis of electromyographic signals in human jaw closing muscles at various isometric force levels. Arch Oral Biol 1988; 33:833–837.
44. Van Steenberghe D, De Vries H, Hollander AR. Resistance of jaw closing muscles to fatigue during repetitive maximal voluntary clenching efforts in man. Arch Oral Biol 1978;23:697–701.
45. Bigland-Ritchie B, Cafarelli E, Vollestad NK. Fatigue of submaximal static contractions. Acta Physiol Scand 1986;128(suppl 556):137–148.
46. Clark GT, Jow RW, Lee JJ. Jaw pain and stiffness levels after repeated maximum voluntary clenching. J Dent Res 1989;68:69–71.
47. Clark GT, Adler RC, Lee JJ. Jaw pain and tenderness levels after repeated sustained maximum voluntary protrusion. Pain 1991;45:17–22.

48. Herring SW, Grimm AF, Grimm BR. Functional heterogeneity in a multipinnate muscle. Am J Anat 1979; 154:563–576.
49. Osborn JW, Baragar FA. Predicted pattern of human muscle activity during clenching derived from a computer assisted model: Symmetric vertical bite forces. J Biomech 1985;18:599–612.
50. Osborn JW, Mao J. A thin force transducer capable of measuring bite force in three dimensions (in press).
51. Eriksson P-O, Thornell LE. Histochemical and morphological muscle-fibre characteristics of the human masseter, the medial pterygoid and the temporal muscles. Arch Oral Biol 1983;28:781–795.
52. Mao J, Osborn JW, Stein RB. The fiber type and composition of mammalian jaw muscles; a review. J Craniomandib Disord Facial Oral Pain 1992;6:192–201.
53. Henneman E. Relations between size of neurons and their susceptibility to discharge. Science 1957;126:1345–1346.
54. Henneman E, Somjen EG, Carpenter DO. Functional significance of cell size in spinal motoneurons. J Neurophysiol 1965;28:560–580.
55. Desmedt JE, Godaux E. Recruitment patterns of single motor units in the human masseter muscle during brisk jaw clenching. Arch Oral Biol 1979;24:171–178.
56. Goldberg LJ, Derfler B. Relationship among recruitment order, spike amplitude, and twitch tension of single motor units in human masseter muscle. J Neurophysiol 1977; 40:879–890.
57. Yemm R. The orderly recruitment of motor units of the masseter and temporal muscles during voluntary isometric contraction in man. J Physiol (Lond) 1977;265:163–174.
58. Kawazoe Y, Kotani H, Hamada T. Relation between integrated electromyographic activity and biting force during voluntary isometric contraction in human masticatory muscles. J Dent Res 1979;58:1440–1449.
59. Nordstrom MA, Miles TS. Fatigue of single motor units in human masseter. J Appl Physiol 1990;68:26–34.
60. Jacobs I. Lactate, muscle glycogen and exercise performance in man. Acta Physiol Scand 1981;(suppl 495):1–35.
61. English AW. Limbs vs. jaws: Can they be compared? Am Zool 1985;25:351–363.

Resumen

Revisión sobre la fatiga en los músculos mandibulares de los humanos

La fatiga muscular parece ser una de las causas de dolor asociado con los desórdenes temporomandibulares. Un sinnúmero de variables podrían contribuir a la fatiga neuromuscular cuando una persona intenta sostener una fuerza determinada. En los estudios de los músculos mandibulares el límite de resistencia ha sido relacionado a una falla en la conductividad eléctrica (fatiga en la transmisión), a una desproporción creciente en el contenido intercelular de las fibras musculares (fatiga en la contracción), y al umbral del dolor. Esta revisión describe los principios que sirven de fundamento a la fatiga y los resultados de los estudios de la fatiga de los músculos mandibulares. Se intenta explicar la razón por la cual varios estudios pueden haber reportado resultados diferentes.

Zusammenfassung

Ermüdung der menschlichen Kaumuskulatur Eine Übersicht

Es wurde postuliert, dass Schmerzen im Zusammenhang mit Myoarthropathien des Kausystems durch Muskelermüdung verursacht werden. Verschiedene Faktoren können zur neuromuskulären Ermüdung beitragen, die während der Ausübung einer langanhaltenden Muskelkontraktion entsteht. Das Auftreten einer Ermüdung der Kaumuskeln wurde mit einer Störung der elektrischen Leitfähigkeit (Transmissionsermüdung), mit biochemischen Vorgängen in den Muskelfasern (Kontraktionsermüdung) und mit dem Einsetzen von Schmerzen in Verbindung gebracht. Diese Übersicht beschreibt die Mechanismen, welche der Ermüdung zugrunde liegen und die Resultate von Arbeiten über Ermüdung in der Kaumuskulatur. Es wird auch versucht zu erklären, warum verschiedene Studien zu unterschiedlichen Resultaten gekommen sind.