From Movement to Pain: A Tribute to Professor James P. Lund

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This tribute article to Professor James P. Lund stems from 6 of the presentations delivered at the July 1, 2008, symposium that honored 3 "giants" in orofacial neuroscience: B.J. Sessle, A.G. Hannam, and J.P. Lund. It was noted that soon after his training as a dentist in Australia, Jim Lund became interested in research. At the time he decided to do a PhD, there was a lot of discussion about how rhythmic movements were programmed. The early belief, based on Sherrington's studies of motor systems, was that these movements were simply an alternating series of reflexes. In the late 1960s and early 1970s, some still shared this belief, whereas others favored Graham Brown's hypothesis that repetitive movements were centrally programmed and did not depend on reflexes triggered by sensory inputs. There was no strong evidence then for either scenario except for the rhythmic movements of respiration. Lund's pioneering work during his PhD proved the existence of a central pattern generator (CPG) for mastication in the brainstem. Since then he has been interested in understanding how CPGs function and how sensory feedback works to adjust the motor patterns that they produce. Sections in this tribute article to Lund are written by some of his close collaborators and reflect the evolution of his work throughout the years. The first 4 presentations in this article (by K.-G. Westberg, D. McFarland, A. Kolta, and C. Stohler) highlight various aspects of these interests, and the final 2 presentations (by J. Feine and A. Woda) focus especially on clinical aspects of Lund's interests. The last section of this article is a final commentary from Professor Lund. J OROFAC PAIN 2008;22:297–306

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After his dental training at the University of Adelaide in Australia, Jim Lund undertook his PhD with another Australian, Professor Peter Dellow, who had previously gone to Canada to take up an academic position at the University of Western Ontario. There his PhD research provided clear evidence for a central pattern generator (CPG) for mastication.[1] He then moved to the Université de Montréal, where for many years he has focused his research on the circuitry underlying mastication, including how the CPG modulates reflexes during movement. He also developed a keen interest in pain and its relationship to movement and motor control. Professor Lund has also served as dean of dentistry at McGill University in Montreal.

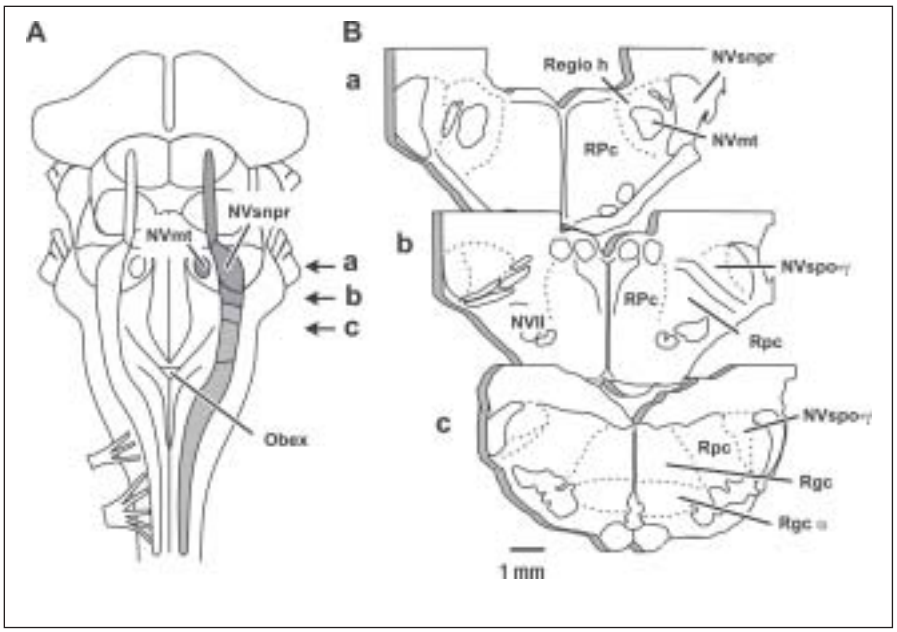

Fig 1 *(a)* Diagrams showing outlines of the trigeminal nuclei in the brainstem. Arrows mark levels of the sections shown in b. *(b)* Locations of brainstem nuclei of importance for control of masticatory movements. Abbreviations: NVsnpr = main sensory trigeminal nucleus; NVmt = trigeminal motor nucleus; NVspo-γ = subnucleus γ of the oral nucleus of the spinal trigeminal tract; Rgc-α = nucleus paragigantocellularis; Rgc = nucleus gigantocellularis; RPc = nucleus reticularis pontis caudalis; Rpc = nucleus reticularis parvocellularis; Regio h = reticular borderzone of the NVmt; NVII = facial motor nucleus.

Role of Brainstem Interneurons in Patterning Mastication

Some of Lund's early work was conducted with Kurt Olsson from Umeå, Sweden and continued later with K.-G. Westberg, Olsson's former PhD student. Westberg was the first presenter at the Lund tribute. He started by pointing out that the orofacial region, which has a special biological and emotional meaning to humans, has offered the neurobiological sciences a number of interesting model systems for experimental analysis. Mastication is one example that, like locomotion and respiration, represents a rhythmic motor behavior. The masticatory motor activity is controlled by a network of neurons, a CPG, which extends from the level of the trigeminal motor nucleus to the obex. This distributed network is assumed to contain "modules" that provide the rhythm generation ("oscillation") and burst characteristics ("spatiotemporal patterning") of the motoneuron discharge to jaw muscles.[2]

Initially, it was thought that the rhythm was generated by network interactions within a hierarchically connected ensemble of neurons caudal to the facial motor nucleus (Fig 1; nucleus gigantocellularis Rgc; nucleus paragigantocellularis, Rgc-α; see also ref 3). However, recent data indicate that the dorso-medial part of the main sensory trigeminal nucleus (NVsnpr), which contains neurons with pacemaker-like properties, may provide an alternative mechanism for the oscillatory inputs.[4] In addition to rhythm generation, the jaw motor control system also has to continuously select muscle synergies that are in harmony with the prevailing biomechanical state of the food bolus, and it has been suggested that rhythm generation and motoneuron recruitment (spatiotemporal patterning) are carried out, at least partly, by separate neuronal populations.[2] Within this model, masticatory motor outputs may be refined by integrating feedback signals from orofacial sense organs and descending commands from centers in the brain.[5] Groups of interneurons, which could fulfill this purpose, are located in the lateral brainstem between the trigeminal and facial motor nuclei[6] (Fig 1). These groups include neurons in the nucleus reticularis parvocellularis (Rpc), the reticular border zone surrounding the trigeminal motor nucleus (Regio h) and the rostral part of subnucleus-γ of the oral nucleus of the spinal trigeminal tract (NVspo-γ). Many neurons in these areas have direct axonal connections to the trigeminal motor nucleus and are recruited to rhythmic firing during "fictive" mastication evoked in anesthetized and paralyzed animals. Furthermore, when fictive motor patterns are changed, their firing also changes.[7] Some become inactive while others change in firing rate and/or duration of their bursts. This indicates that subsets of interneurons in the lateral brainstem form dynamic circuits that can provide a variety of (context dependent) drives to the motoneurons. These "burst-generating" premotoneurons are, in turn, controlled from neurons in nucleus reticularis pontis caudalis (Fig 1; RPc), which occupies medial areas of the caudal pons.[6] However, at present their roles in the shaping of jaw motor patterns are less well known.

Taken together, our knowledge on neuronal mechanisms underlying mastication has increased substantially since Sherrington[8] first suggested that masticatory movements were based on a chain of alternating jaw-opening and jaw-closing reflexes. The work of Lund and his collaborators has been especially pivotal in revealing that these movements are regulated from a flexible CPG circuitry. Understanding the cellular and network mechanisms underpinning the functioning of this motor control circuit will continue to be a demanding challenge for future research.

Coordination of Movements of Brainstem Origin

The next presenter was David McFarland, who undertook postdoctoral research with Professor Lund. This research focused on understanding how CPGs interact when coordinating mastication, deglutition, and respiration in experimental animals and humans.

McFarland first noted that during his PhD research in speech physiology at Purdue University in the 1980s he first became aware of the work of the 3 giants, and the papers of Lund (as well as Sessle and Hannam) were required reading. Although speech physiology was and is a well-established scientific discipline, he remarked that the work of these orofacial physiologists in nonhuman animal systems have contributed to building its scientific base and helped orient work in speech systems. Indeed, much of this work laid the foundation for some of McFarland's early publications.[9–13]

The collaborative work between McFarland and Lund focused on, and continues to center around, cross-system coordination of the central pattern-generated behaviors of breathing, chewing, and swallowing. This work emerged out of Lund's pioneering work in the control and coordination of mastication.[1] Overlaying the closely linked systems of respiration and swallowing was a natural progression. Their early work using chronically prepared rabbits chewing freely revealed several important findings.[14] First, they documented a strong temporal linkage and coordination between mastication and swallowing and respiration and swallowing, but there was little evidence of a direct coupling of respiration and mastication; rather, they hypothesized that the requisite coordination to accommodate swallowing appeared to be controlled by the swallowing pattern generator.[14] They then extended this work to adult humans swallowing upright and found that, similar to the rabbits, swallowing tended to occur in the early opening phase of the masticatory cycle and that there was only minimal evidence of direct coupling between mastication and respiration.[14,15] Nonetheless, a striking difference between the species was that swallowing tended to occur in the late expiratory phase of the breathing cycle of humans as contrasted to inspiration in rabbits, and they provided evidence that some of the differences in respiratory-swallowing coordination between humans and most other animal species may be due, at least in part, to whole body posture while chewing and swallowing. Furthermore, mechanisms coordinating respiration and swallowing are sensitive to external mechanical constraints and gravitational forces acting on the food bolus.[16]

Their collaborative studies also found that the requisite coordination of breathing and swallowing in humans is extremely stable despite major changes to upper airway structure and function,[15] as laryngectomized patients continue to swallow in the expiratory phase of the breathing cycle despite the fact that the airway does not need to be protected. They also provided evidence and arguments that this highly stable respiratory-swallowing patterning imparts important mechanical advantages to swallowing and airway protection related to the cyclic activation of the diaphragm during spontaneous breathing.

McFarland's research on respiratory-swallowing coordination continues with recent collaborative investigations focusing on sleep apnea in infants and adults.[17,18] A recent experiment with preterm infants studied at term has shown a highly consistent respiratory-swallowing coordination pattern during sleep and wakefulness very similar to that observed in the adult human. Further, swallowing appears to have a potentially important protective function during sleep and participates in resolving apnea and encouraging appropriate arousal mechanisms during sleep apnea.[18] His postdoctoral studies with Lund on the interactions between centrally patterned behaviors set up an ideal theoretical and clinical context to look at interactions between other fundamental behaviors, including interpersonal interaction and synchrony[19] and speech and swallowing interactions.[20]

McFarland concluded his presentation by describing several examples of how Lund had influenced his scientific career. These included his very broad vision of fundamental and clinically focused research and his fostering of interactions between scientific disciplines, such as dentistry and basic neuroscience, orofacial physiology, and speech research. McFarland remarked that this notion of

incorporating insight from a variety of different theoretical and clinical perspectives has had a profound influence on his own research career, and through his own teaching, it has also influenced the careers of a whole new generation of speech language pathologists.

Trigeminal Circuits: From Rhythmogenesis to Chronic Muscle Pain

Arlette Kolta next provided an overview of her work with Professor Lund on how sensory feedback can modify motor patterns. She undertook her PhD studies in the late 1980s under the supervision of Lund because she was interested in the mechanisms by which the central nervous system programs rhythmical movements, and he had conducted pioneering work on mastication and shown that the CPG for this movement is located in the brainstem. The subject of her PhD thesis was to find out how the CPG interacted with sensory inputs. It was then known that the CPGs in other rhythmic movements interrupted synaptic transmission from primary afferent neurons during some phases of the movement to avoid generation of reflexes that would perturb the movement. This had been shown to occur by a presynaptic action on the terminals of primary afferents during locomotion and respiration but had not been investigated in trigeminal afferents innervating the jaw muscles during mastication. These afferents are peculiar because their cell bodies are located centrally in the trigeminal mesencephalic nucleus (MesV) and receive synaptic inputs, which is not the case of other primary afferents. The presynaptic inhibition of primary afferents was thought to be mediated by GABA, which produces a depolarization referred to as primary afferent depolarization (PAD) that is sometimes strong enough to produce action potentials that are propagated backwards (that is, antidromically) toward the cell body and the periphery and appear in dorsal root ganglia or rootlets. Even when they caused these action potentials, PADs are still translated functionally as an inhibition because synaptic transmission was reduced at the central endings of the sensory afferents.

Kolta and Lund set out to test whether this occurred in MesV but found no evidence of antidromic action potentials when recording near the cell body. Instead they found a phasic inhibition during the jaw-opening phase that occurred directly at the soma and achieved the same effect on synaptic transmission from the primary afferents as PAD did.[21] However, later work with Westberg showed that antidromic spikes are indeed generated near the central terminals and travel to anterior parts of the axon but somehow fail to reach the cell body, as if the axonal tree was divided in different compartments.[22] They later conducted work in vitro on a brainstem slice preparation and showed that GABAergic synapses control the traffic of action potentials along the axons of these afferents, thereby allowing differents parts of the neuron to display different firing patterns simultaneously.[23]

While conducting this work, they realized that these afferents have resonant properties that generate high-frequency subthreshold membrane oscillations (SMO) that often occur spontaneously, but that can also be triggered, amplified, and synchronized by synaptic inputs to the cell body.[24] Similar oscillations are seen in other large primary afferents in pathological pain conditions, and an increase in their incidence and amplitude leads to ectopic firing (that is, firing generated outside of the normal loci). Normally, pain is mediated by activation of nociceptors, small-caliber primary afferents which then activate nociceptive pathways in the spinal cord or brainstem to the thalamus and cortex. So they asked, how can ectopic firing in large-diameter primary afferents that signal muscle stretch lead to activation of nociceptive pathways? They hypothesized that if ectopic firing is generated in Mes V cells due to an increase in their SMO, then it may travel antidromically toward the muscle and cause release of glutamate, their neurotransmitter, within the receptor structure, the muscle spindle, which normally responds to stretch of the muscle and not to noxious stimuli. However, if nociceptors were found in proximity of these glutamate-releasing fibers, then they may be activated, because nociceptors carry glutamate receptors. To test this hypothesis, they used a model of chronic muscle pain, in which acid-saline (pH 4.0) injections are made twice into the muscle. When this is done in the masseter muscles, the animal develops a mild allodynia and mechanical hyperalgesia that last about a month and are accompanied by an increase in amplitude of SMO in Mes V cells and a decrease of the potential at which they appear and at which firing is triggered. Moreover, about 20% of these cells become spontaneously active (unpublished observations). These electrophysiological changes also last for about a month. Using immunohistochemistry, they obtained evidence that the peripheral branch of these cells have the machinery to release glutamate in the spindles where nociceptive fibers carrying metabotropic glutamate receptors could be seen in close proximity.

While all this work took them on a path quite different from what Kolta said she had in mind initially, they never lost sight of their original interest in rhythmogenetic mechanisms, which they kept investigating in parallel. These studies were in tissue slices that included the nucleus pontis caudalis in the medial bulbar reticular formation, the peritrigeminal area containing last-order interneurons surrounding the trigeminal motor nucleus and projecting to it, and laterally the trigeminal principal sensory nucleus (NVsnpr) at this level. All of these areas contained neurons that were rhythmically active during mastication, even when the motor pattern was induced in animals in which movement was prevented by paralysis, a condition referred to as fictive mastication.[25–27] However, only those of NVsnpr were found to have intrinsic ability to generate rhythmic firing. This property depends on a sodium persistent conductance (INap) that appears at the same time as the first masticatory movements during the second postnatal week.[28] They found that the amplitude and duration of the current flowing through INap is not only voltage-dependent, but is also modulated by the extracellular concentration of Ca++ ([Ca++]e); the lower the latter, the greater is INap.[28] Therefore, they asked whether [Ca++]e drops occur under physiologic conditions in the masticatory CPG. Their current working hypothesis posits that sustained activity of afferent fibers and NVsnpr neurons causes a fall in [Ca++]e, in part through the release of K+ and glutamate, both of which activate glial cells which can in turn pump the Ca++ from the extracellular space. To begin to test this hypothesis they first showed that repetitive stimulation of the trigeminal sensory tract induces rhythmic bursting in some NVsnpr neurons, an action that is blocked by NMDA receptor antagonists and mimicked by NMDA itself (unpublished observations). Both procedures, sensory tract stimulation and NMDA, also activate glial cells in NVsnpr and promote coupling between them, and may help synchronize neurons that are surrounded by the glial syncytium. Kolta concluded by stating that they are certainly not at "the end of the road" yet and that many years of fruitful collaboration lay ahead.

Pain and Movement Interactions

Much of the above work conducted on in vivo and in vitro animal models eventually led Professor Lund to formulate a conceptual model about how sensory inputs from nociceptors altered movements. He developed the "pain adaptation model"[29] and began to test it in humans in collaboration with Christian Stohler. At the start of his presentation, Stohler made the point that this model had far-reaching implications for the care of patients with jaw and masticatory muscle pain insofar that it debunked the prevailing idea of muscle hyperactivity being the source of pain. With the demise of this "Vicious Cycle Theory," the treatment focus shifted from muscle dysfunction and muscle hyperactivity to the management of pain. Adaptive strategies adopted in painful function explain the basis of the clinically observable changes in muscle function.

Since the model's original formulation in the early 1990s, most clinical and experimental data obtained in humans have been consistent with the model's prediction regarding the function of the masticatory musculature or—broadly stated—the truncal muscles in the presence of pain. Muscles functioning as agonists are significantly inhibited under heavy loads; under the conditions of very light and light loads, the inhibition may not always reach statistically significant levels. Regarding the function of antagonists in pain, their facilitation is most prominent under the conditions of moderate to major muscle extension. However, at rest, with jaw muscles exhibiting postural activity, signals from adjacent or overlying muscles, such as the muscles of facial expression ("that betray—according to Darwin—our true feelings despite our efforts to conceal them") may confound electromyographic (EMG) recordings. Expressive actions may not be limited to the muscles of facial expression but also directly include—although at very low levels of activity—the muscles of mastication, such as in pain, fear, anxiety, or grief as the position of the lower jaw appears to be part of the display of the respective affect. Regarding the model prediction, there is no difference in the effect of pain, eg, skin, muscle, joint, ligament and/or visceral tissues within which the painful stimulus originates, consistent with the "Pain Adaptation Model."

Since Lund's model has been shown to be robust in clinical and experimental contexts, Stohler asked what new knowledge has been gained since its original formulation in 1991 that would allow its alignment with new trends in science? And, how is a valid model refreshed so that it can again become a guiding impetus for the generation of exciting science? He noted that although already known to Darwin, the muscles of facial expression exhibit the pain-related affect, an important part of the clinical phenomenology. More recent data

also demonstrate that for truncal muscles other than the muscles of mastication, motoric changes often occur together with disturbances of mood, such as "akinesia" with stroke, dementia, and depression. Other examples that he brought up included manic mood states that are associated with "hyperkinesias," and an increasing body of literature associates psychoactive drugs, such as lithium, tricyclics, benzodiazepines, and others with extrapyramidal effects. In sum, an increasing body of literature emphasizes the functional interconnection of mood disorders and motor disturbances. Regarding this interconnection, the modulatory effect of pain on vocal, respiratory, and facial muscles appears to exceed the effect encountered on the muscles of mastication, or the truncal musculature in general.

In his concluding remarks, Stohler mentioned that Lund's Pain Adaptation Model continues to be appealing, not just due to its predictive power to explain clinically and experimentally important motor symptoms, but also in terms of broadening the conceptual framework, permitting the study of the involvement of higher brain centers, notably the interconnection of movement and mood.

Clinical Assessment of Therapies for Rehabilitation and Pain

Professor Lund became interested in understanding how sensory inputs other than nociceptive inputs may affect mastication. Since then, he has worked in close collaboration with Jocelyne Feine to document the effects of loss of teeth on the efficiency of mastication, and the effects of rehabilitation on mastication and nutrition.

In her presentation, Feine first pointed out that Lund had been her husband and mentor for 22 years. She first met him in 1985 when he was lecturing in Texas and was quite concerned that patients were being exposed to unsubstantiated treatments. Although he was quite occupied with his basic science studies, he was strongly motivated to step into the clinical world. She pointed out that his first battles concerned diagnostic tools that he and his colleagues showed may misdiagnose healthy people as ill[30] or may be founded upon faulty physiological concepts.[31]

From their investigations of these tools, Feine said that she learned her first big lesson from Lund, namely that one should never hesitate to question the person in the "white coat" with an authoritative manner and an important position. Everyone and everything is open to question.

Together they also studied implant rehabilitation for edentulous populations. Their interest turned from studying complex, multiple implant restorations to those that could be more accessible to the large populations of edentulous patients with limited incomes. This led to the second major lesson that she learned from Lund, being the importance of collaborating with others, and over the years, they have carried out studies with many collaborators from various domains.[32,33]

Feine next remarked on Lund's mentoring skills. He manages to maintain a kind persona, while demanding the highest quality work from his trainees and other collaborators. This has been her lesson #3 from Lund: "Nurture the next generation."

Feine then turned her attention to the concept of "knowledge transfer," which emphasizes the importance of communicating the results of scientific findings to the groups for whom the findings are most relevant. This means, of course, that researchers should not always publish in the highest cited journals, particularly if doing so will reduce the time in which the findings can be made available to the necessary groups. For many years, Lund has guided her in these principles, and they have published in a variety of journals, based on the relevance of their findings to that journal's readership.[34] This has been her lesson #4: Share your findings with those who need to know.

Her lesson #5 is: Ask important questions. Rather than concentrating on specific details, Lund has enabled her to step back from an issue and look at it from many angles. This thought process has invariably allowed her to see the problem very differently, leading to new directions and hypotheses.[35,36]

As a follow-up, her lesson #6 is to always consider issues from different perspectives. This has allowed them to introduce new perspectives into their research, from studying patients' treatment preferences[37] to assessing sex differences in ratings of postsurgical pain[38] to measuring nutritional outcomes in implant studies[39] and evaluating the impact of implant support of dentures on social and sexual activities of edentulous adults.[40]

Feine also noted that one of the delights of being a scientist is being able to share, to disagree, to raise issues, and to exchange ideas. Having a scientific mind includes being open to changing one's outlook when new and legitimate concepts are introduced. By example, Lund taught her lesson #7: Never be afraid to learn or to change. She gave the example of EMG recordings and using a 3-D real-time optical recording system measuring jaw movement to study chewing ability in human subjects who rated their chewing ability on question-

naires. Discrepancies in the outcomes between the 2 approaches taught Feine and Lund that the most valid useful measure of chewing ability in a clinical trial of oral prostheses is the individual's perception, rated on a questionnaire.[41,42]

Feine's concluding remarks focused on their mission as scientists, which is to seek information to improve human health. One frustration is that, although we may provide substantial information that should change practice, it can take many years, if ever, for the public to receive the benefit. Health technology assessment and transfer is the field that works to decrease this delay. This has been her lesson #8: "Effect change." Therefore, as part of their efforts to assist in the transfer of the scientific knowledge that they produced in the field of implant overdentures for edentulous populations, she and Lund are beginning to carry out technology assessment studies.[43,44]

Mastication in Humans

Alain Woda's long-time interactions with Professor Lund stemmed from Lund and Feine's evaluation of the effects of the type of treatment in the edentulous patients on masticatory function and the need to develop methods to measure the efficacy of mastication. Woda's presentation emphasized 4 main points related to early and more recent research on the control of mastication in humans.

1. The ready-to-swallow food boluses produced by different individuals display similar particle size distributions. Indeed, several converging studies have pointed to the small variability of the particle size distribution in the pre-swallow food bolus, despite the great variability of physiologic parameters (number of cycles, total muscle work[45–48]). This weak variability could be explained by the need to reach by any means the required pre-swallow food bolus state because of the danger of swallowing an unprepared bolus.[49] The study of food bolus may serve as a milestone for understanding mastication in humans. However, Woda considered that there are no standard methods for determining of food bolus characteristics, and there are 3 unsolved problems that future research will have to solve. First, the result of granulometric studies cannot be compared between studies, even if there is a general agreement to use the d50 value to express particle size distribution, because the result depends on many factors, such as the choice of the test food,[45] the chosen tools for granulometric analysis, and the method of preparation of the samples by the experimenters. Second, particle size is not the only important property of the pre-swallow food bolus since the rheological properties of the bolus also participate to the triggering of deglutition.[50] However, knowledge about the bolus rheology is sparse.[51] Third, the study of food recovered from the mouth bolus does not tell the whole story, since about half of the bolus mass is lost when the ready-to-swallow bolus is collected,[52] probably because of intermediary deglutitions.[53]
2. The values of the physiological parameters of mastication are simpler to standardize than food bolus particle size measurement, but these physiological parameters vary widely from one individual to another. In a single individual, their modifications reflect the adaptation of mastication to the size of the food bolus, the hardness and other rheological characteristics of the food. In a population, these parameters are also influenced by general characteristics such as age, gender, loss of teeth, and characteristics of prosthetic replacements. Figure 2 displays a summary of the adaptations that have been observed in subjects with healthy and impaired mastication.
3. Because they focus on the physiological variables, the methods proposed to evaluate masticatory function[54] may lead to a very effective mastication being classified as total failure. There is therefore a need to differentiate 2 groups of subjects: Those who succeed in preparing all foods for a safe swallow and another group of subjects who cannot do this and who either swallow poorly prepared food boluses or who develop another strategy (changing their diet, food preparation, etc). This emphasizes the need to define and differentiate 2 concepts. The first is the ability (or inability) to produce a safe ready-to-swallow bolus, whatever the masticatory strategy needed to reach the goal. The ability to produce a satisfactory bolus separates individuals who have either a healthy or unhealthy mastication. The second concept is the efficiency with which the bolus is prepared. This is usually measured by analyzing the particle size of a food bolus obtained by expectoration at a given number of cycles before the natural swallow. The significance of this property is unclear, since there is no certainty that the fast chewers are healthier than the slow chewers. Perhaps a more important indicator of masticatory health is the adaptability of the system. An

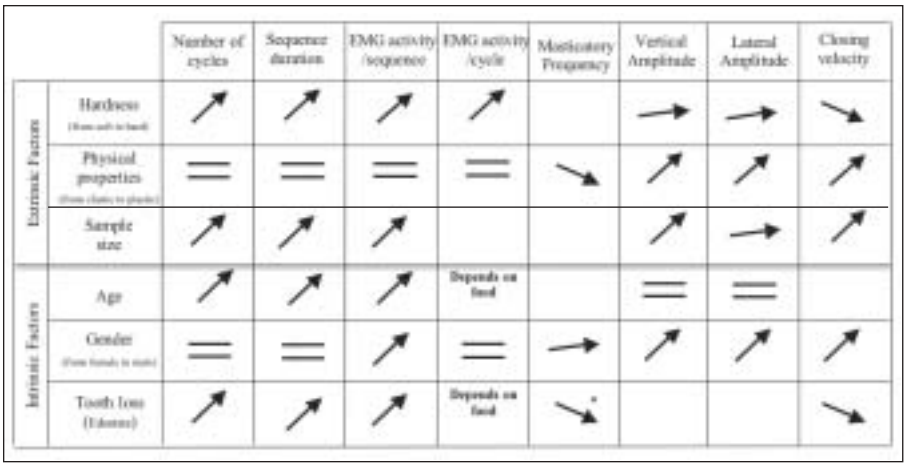

Fig 2 Schematic representation of the adaptation of mastication. Responses of the major electromyographic and kinematic parameters to the main extrinsic (3 top lines) and intrinsic (3 bottom lines) factors. Four signs are used to display the effects of these factors on masticatory parameters: downward pointing arrows indicate a decrease, slightly or strongly upward pointing arrows indicate a slight or strong increase in the values of the masticatory parameters. An equal sign indicates no change. Figure from Woda et al (2006).[60]

example of an adaptation that appears to maintain masticatory ability is given by aged dentate subjects who, compared to younger subjects, increase the number of chewing cycles. Therefore, they succeed in making a good food bolus although they appear to expend more energy because they increase total EMG activity.[55] An example of a failed adaptation leading to poor masticatory ability is given by edentate subjects. Even if compensated for by complete dentures, loss of teeth hinders the formation of a normal bolus,[56] although the number of cycles is increased compared to similarly aged dentate subjects.[52] However, it has been shown that implant-supported overdentures improve the deficient mastication of denture wearers.[54] Although some people have deficient mastication and good general health, there is much evidence that poor mastication is linked to poor nutrition and poor general health.[57,58]

4. The impaired mastication observed in denture wearers approaches the masticatory disabilities found in persons with neuromotor deficiencies. In some populations, bad oral health amplifies a pre-existing disability. This situation is observed in special care units where rehabilitation of mastication can have a much greater impact on the general health than the standard dental care given to healthy individuals.[59]

Lund Response

Professor Lund first remarked that the series of papers was written by some of his very best collaborators who are also among his best friends, and that it was very kind of them all to join with Drs Lavigne, De Laat, Peck, and Kato and others in organizing this tribute to Sessle, Hannam, and himself. He said that he could not think of a more pleasant way to spend a couple of hours than listening to friends say nice things about one's science and recalling the great times that they have spent together.

He also said that the papers written by Westberg, McFarland, and Kolta each describe a series of studies that they conceived and carried out together, mainly in the laboratories in the centre de recherche en sciences neurologiques at the Université de Montréal. Westberg and McFarland had come as postdoctoral fellows, and Lund noted how pleased he was that they have been able to continue their collaborations after they moved on to higher callings. Kolta, he said, began as a student in Montreal, left for the good life in California for a while, then came back to keep the family farm running. He remarked that without her, not much would have come out of Lund and co for the last 15 years. When all 3 were young, and he was youngish, they had the stamina to carry out long experiments.

With the passing of the years, they switched to human studies, which are necessarily less complicated and are limited in time by the ethical rules, and to in vitro work, which is easier to delegate and to end in time for dinner. Lund stated that just as form follows function, experimental design follows diminished function and earlier bed-times!

He thought that Stohler was far too generous in giving him credit for the work and the discussions that they had together, a lot of it in Stohler's laboratories at the University of Michigan. It was Lund's hope that Stohler will be able to find a little time in his packed schedule so that they can take another look at pain and movement in the coming months.

Lund also mentioned Woda's description of the work that he and his group, including Marie-Agnes Peyron, who spent time in Montreal, have been carrying out in Clermont-Ferrand, France. Lund said he had been very lucky to have been included in some of these studies and on Woda's walks into the mountains of the Auvergne; on most occasions, he had been able to bring them out again!

Lund directed his concluding remarks to his dear wife Jocelyne, who painted a very fine picture of their collaboration, and insisted that she learned a lot from him. However, he said that anyone who knows Jocelyne will agree that this is an absurd simplification. Without her, her ideas and drive, none of the clinical research that she describes would ever have been done.

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