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THE RESPONSE IN THE HUMAN MASTICATORY SYSTEM TO THE SUDDEN UNLOADING OF THE JAW CLOSING MUSCLES

by

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CONTENTS

SUMMARY

When the resistance to forceful jaw closing is suddenly and unexpectedly withdrawn, the closing movement of the mandible is usually arrested before the teeth come together. An "unloading reflex" has been previously described in jaw and limb muscles where agonist inhibition and antagonist activation have been presented as a feature of this restriction of movement.

In this present study, the unloading reflex in the jaw closing muscles in man was investigated with a view to correlating the jaw closing movement with the timing of the electrical activity in the agonist and antagonist muscles. The behaviour of these muscles was studied as the force and the joint angle at the time of unloading were varied.

The results generally agree with earlier studies in that there was a similar pattern of reflex inhibition of elevator muscles and activation of the digastric muscle after unloading. However, this pattern does not adequately account for the rapid arrest of the jaw closing movement as deceleration of the mandible always started within 5 msec of unloading while the elevator muscles were still active, and well before the depressor muscles were activated. This suggested that a mechanism was acting to limit the upward movement of the mandible at a shorter-latency than could be explained by a reflexly-induced load compensation mechanism.

It was concluded that the mechanism principally responsible for the limitation of the jaw closing mechanism was a pre-programmed increase in the stiffness of the digastric muscle which opposed the movement.

This mechanism consisted of co-contraction of both the elevator and depressor muscles as the subjects attempted to overcome the resistance between their teeth.

It is proposed that at least two mechanisms exist to limit the movement around a joint after the resistance to an isometric agonist contraction is overcome. The slower reflexly-induced mechanism described in previous studies may be augmented by this rapidly acting feed forward mechanism when it is important to restrict the amount of limb movement.

Evidence of the co-contraction of digastric and elevator muscles and increased digastric activity when unloading occurs at low openings and high forces is presented in support of this pre-programmed control of jaw movement by antagonist muscle stiffness.

SIGNED STATEMENT

This thesis contains no material which has been accepted for the award of any other degree or diploma in any University. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

THOMAS M. WILKINSON

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INTRODUCTION

It is a common experience that when a brittle object is cracked between the teeth, the jaws rarely come together with sufficient force to cause damage to hard or soft tissues. This is remarkable when one considers the large forces being generated between the teeth and the small jaw separation.

It would seem to be of value for a masticatory apparatus to have the ability to crack and break up foodstuffs, which may include hard and soft plant and animal material, without causing damage to similar tissues and structures of the mouth itself. Though this may not be of great importance to present day western man, observation of a dog breaking a bone between its teeth demonstrates the effectiveness of the mechanism that has been developed to decelerate the mandible as soon as such food shatters.

The speed with which this occurs suggests that some rapidly acting mechanism must inhibit the jaw closing muscles and activate the jaw opening muscles. Such an "unloading reflex" has been demonstrated in the muscles which produce movements around other joints (Hansen and Hoffman, 1922; Angel *et al.*, 1965; Alston *et al.*, 1967). Several workers have described a similar reflex on unloading the masticatory muscles (Hannam *et al.*, 1968; Beaudreau *et al.*, 1969; Gill, 1970; Luschei and Goodwin, 1974; Lamarre and Lund, 1975) where there is rapid inhibition of jaw elevators followed closely by activation of the anterior belly of the digastric muscle.

The jaw elevators are powerful muscles, able to exert maximum closing forces of up to 600 N (Pruim *et al.*, 1978) and the tension exerted by a

contracting muscle decays only slowly when the muscle is inhibited (Grillner, 1972). This suggests that the jaw elevators would continue to provide resistance to deceleration of the mandible in the closing direction for some time after they are inhibited following unloading.

The jaw opening muscles by comparison are much weaker, as is seen when one tries to separate the teeth when they are stuck together with toffee. This suggests that it would be difficult for a burst of activity in these muscles to be able to rapidly decelerate the mandible while there was residual tension in the elevator muscles.

If the acceleration given to the mandible in the closing direction on unloading were high, this reflex mechanism involving the elevator and depressor muscles would have to act extremely rapidly to be an effective protective mechanism. This raises the question as to whether, after allowing for the delay in the afferent fibres signalling the unloading and in the efferent fibres in changing the muscle activity, a reflexly initiated response is the prime mechanism which arrests the upward movement of the mandible.

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1. LITERATURE REVIEW

Merton (1953) proposed that the fusimotor fibres may be a normal pathway for initiating muscular contractions. Through its effect on the muscles spindle, fusimotor discharge can lead to an increase in the firing rate of the spindle primary endings causing activation of the alpha motor neurones via the 1a monosynaptic reflex. He proposed that this mechanism may provide a servo-control by which muscles can be set reflexly to any desired length. The evidence on which he based this proposal was derived from investigations of the "silent period" which occurs in the electromyogram of a muscle contracting voluntarily when its nerve is stimulated electrically, producing a twitch. However, as Matthews (1964) has pointed out, the silent period under the conditions of Merton's experiment may result from several factors, of which the spindle pause is only one. Electrical stimulation of a mixed nerve may for example cause alpha inhibition by activation of Renshaw cells or via 1b fibres supplying Golgi tendon organs. To establish the role of spindle silence in causing this silent period it was necessary to produce the silent period by some method that did not produce a synchronous discharge of motor neurones, Renshaw cells or Golgi tendon afferents. The tendon jerk, in stretching the muscle does not exclude other possible sources of inhibition.

To avoid these objections, Angel *et al.* (1965) produced a silent period in an isometrically contracting muscle by suddenly withdrawing the resistance opposing the voluntary muscular contraction, or "unloading" the contracting muscle. In their experiments, human subjects were seated with their elbow supported on a platform and their forearm extended horizontally forward. A metal rod, supported by gimbals, hung vertically in front of the subject and was taped to the wrist. A lateral force was applied to the bar by means of a cord, pulley and a measured weight. The

subject held his hand stationary against the external force and the experimenter unexpectedly cut the wire supporting the weight. The velocity of the forearm and the electrical activity of the agonist (biceps) and the antagonist (triceps) were recorded. They found that there was a silent period in the agonist at a latency of about 40 msec after unloading and that this was followed by a burst of antagonist activity. The velocity trace showed that deceleration of the forearm started at about 50 msec after the start of this antagonist burst. Action potentials were again seen in the agonist 70 to 150 msec after the release of the weight but they had no apparent effect on the velocity curve. When flexion of the elbow was prevented by a restraining wire, the silent period did not occur and there was no discharge in the triceps. When the distal end of the radius was allowed to move 13 mm or more, the agonist silent period and the antagonist burst progressively returned as the amount of flexion allowed was increased up to 40 mm, beyond which there was no further increase in the silent period. They concluded that as inhibition from Golgi tendon organs and Renshaw cells was presumably inoperative in their experimental paradigm, the role of spindle silence in causing the silent period must assume greater prominence. They felt that reciprocal inhibition from stretching the antagonist, which required more than one synapse, was unlikely to cause the silent period since the agonist fell silent before the burst of activity in the antagonist which they considered to be a monosynaptic reflex. They stated that the return of activity in the agonist appeared to be an isotonic contraction which exerted no force on the limb but set the muscle at a new length.

Alston *et al.* (1967), using a similar experimental apparatus, studied the motor activity following the silent period after unloading by varying the initial force being opposed before unloading, the inertia of the limb

(by loading the hand with additional mass), and the amount of movement allowed to the limb (by use of a restraining wire). The EMG activity of the agonist (biceps) and the displacement and velocity of the hand were recorded. They found that action potentials in the agonist were significantly larger during the terminal volley than during the period before unloading. The size of the terminal motor volley was increased when the force being resisted before unloading was greater, whereas it was reduced as inertial loading increased. The latency of the volley was not altered by either of these variables. Their results agreed with those of Angel *et al.* (1965) in that if the movement of the limb was interrupted within 15 msec of unloading, no silent period occurred. If the interruption occurred between 30 and 100 msec after unloading, the latency of the motor volley was evidently proportional to the duration of movement and this volley occurred within 20 msec of the interruption. If the interruption occurred at a latency greater than 100 msec, there was no further increase in the latency of the volley.

In discussing the neural mechanisms responsible for the return of agonist activity after the silent period, they considered that the silencing of alpha motor neurones after unloading could lead to a cessation of Renshaw cell discharge at a fixed latency. The removal of Renshaw cell inhibition may then make the alpha motor neurones more responsive to stimulation, resulting in a motor volley. They felt that this hypothesis was not supported by their results as the volley did not occur at a fixed latency but depended more on the motion of the limb.

If the motor discharge were due to unloading of the tendons, it should presumably be eliminated if the tendon were subject to a sudden stretching force such as occurs when the restraining wire arrests movement

of the forearm. In fact, the after-volley occurred appreciably sooner in the presence of the mechanical block. As a result of their experiments, they proposed a "fusimotor reflex" to explain this after-volley. Their suggestion was that gamma activity during the silent period, activated by stimuli from the limb, caused contraction of the intrafusal fibres. At some point in the ballistic movement, the intrafusal fibres became shorter than the extrafusal fibres and an alpha motor discharge, more synchronous than that before the silent period, caused the after-volley. This "fusimotor reflex" would explain why the muscle frequently responded within 20 msec of the blocking of limb movement, this being the latency that they reported for a stretch reflex in the biceps. This reflex would operate through the 1a fibres when the contracting intrafusal fibres became shorter than the arrested extrafusal fibres. They emphasised that this interpretation was purely speculative and that their data were equally consistent with the fusimotor reflex being excited by the joint receptors, the tendon organs or even stretch receptors in the antagonist. They considered that presence of two or more silent periods in 50% or more of the trials, especially those where the movement was restricted, suggests that the underlying mechanism has a tendency to "overshoot" and oscillate.

The question of the role of the antagonist spindles was studied further in a series of experiments by the same group by comparing the pattern of muscular responses to unloading of the forearm before and after the motor and sensory nerves to the antagonist muscle were blocked by local anaesthetic (Angel *et al.*, 1973). The first hypothesis they wished to test was that the silent period in the agonist after unloading resulted from reciprocal inhibition bought about by stretching of the antagonist Their second hypothesis was that the return of the agonist activity after

the silent period was due to a fusimotor reflex initiated by stretching of the antagonist muscle. For this experiment, the triceps was the agonist and contracted against the resistance of a coil spring. The unloading occurred when an electromagnet holding the spring was deactivated. It was presumed that the 1a afferent fibres were blocked together with the alpha and gamma motor fibres, when a subject was unable to flex his arm voluntarily.

They found that only 2 out of 7 subjects showed any significant delay in the onset of the silent period during this antagonist blockade and in no case was the silent period abolished or modified in general appearance. They concluded that reciprocal inhibition is not necessary to cause a silent period and that their results were consistent with the hypothesis that the silent period is due to a decrease of excitatory inflow from the spindles of the agonist muscle. In no instance did the nerve block produce a significant decrease in the motor activity following the silent period which is inconsistent with the hypothesis that the fusimotor reflex is initiated by antagonist stretch.

They considered other possible explanations for the excitation of the gamma motor neurones during the silent period. If alpha motor activity were inhibiting gamma motor neurones via recurrent collaterals, alpha silence on unloading would remove this inhibition. They also considered that shortening of the agonist on unloading may remove an inhibitory influence on the gamma motor neurones which then increase their rate of firing and produce the volley through the stretch reflex loop.

This latter mechanism was discussed by Angel and Lewitt (1978).

They cited the description by many workers of a similar pattern of agonist silence with a return of motor activity when a muscle is passively shortened, the so called "shortening response" (Burke *et al.*, 1971; Andrews and Burke, 1972; Andrews *et al.*, 1973). They considered that after a contracting muscle was unloaded, it would be passively shortened during the final part of the silent period as the limb would be moving by inertia alone. They designed a study to test the hypothesis that the contraction following the silent period after unloading and the contraction after passive shortening are both produced by the same neural mechanism. As this shortening reaction is known to be exaggerated in Parkinsonism (Andrews *et al.*, 1972), it would be predicted from this hypothesis that the amount of agonist contraction after unloading would also be exaggerated. They studied the unloading response in a subject with asymmetrical Parkinsonism, predicting that the contraction after unloading would be greater on the more severely afflicted side. They found this to be so and concluded that the response to unloading and the shortening reaction share a common neural mechanism. They considered that a possible function of these reactions may be to "take up the slack" in the muscle to prepare it for efficient contraction at the new, shorter length.

A similar unloading reflex was first described in the masticatory muscles by Hannam *et al.* (1968). Their experimental apparatus consisted of a device which held two horizontal bars, the upper one being a perspex tube and the lower one a brass bar. Six human subjects were asked to fracture the tube by biting with their upper premolar teeth on the tube and their lower premolar teeth on the lower bar. The depth of a notch cut in the perspex tube determined the force necessary to cause its fracture and strain gauges cemented to the lower metal bar recorded the

force being applied between the teeth. The initial separation of the tube and bar could be varied. The electrical activity of the masseter, temporalis and anterior digastric muscles on the working side was recorded by surface electrodes. In one subject a needle electrode placed into the anterior belly of the digastric muscle confirmed that the surface electrodes were recording its activity faithfully. Vertical movement of the mandible relative to the maxilla was recorded by a transducer in which a primary coil fixed to the head induced a current in a secondary coil attached to the lower anterior teeth. The magnitude of the current depended on the separation of the coils. This transducer was non-linear and was calibrated at each experiment by placing blocks of known thickness between the premolar teeth.

They found that irrespective of the initial separation of the jaws or the force necessary to fracture the tube, the upward movement of the mandible was always arrested at least 5 mm from the closed position. The pattern of muscle responses was similar to that seen in the unloading reflex of the muscles of the forearm in that the masseter and temporalis were inhibited approximately 20 msec after the moment of fracture, the digastric being activated at about the same time. Variations in the separation of the jaws or the force applied did not essentially change these responses although, when the initial separation and the applied force were both large, the digastric muscle was active before the moment of fracture and its responses following fracture were seen as an increase in its activity. On occasion, the silent period of the elevator muscles was followed after approximately 50 msec by a sudden return of activity. They concluded that this unloading reflex played an important part in arresting jaw movement before tooth contact occurred, though the identity of sensory input which triggers the reflex was not

clear. They considered the trigger signal may have come from muscle spindles or from joint, tendon or periodontal ligament receptors.

Beaudreau *et al.* (1969) also noted the unloading reflex as part of an investigation of motor pauses in masticatory muscles. Weights ranging from 1 to 5 kg were suspended by a cord looped over the mandibular cuspid teeth. The subject was instructed to close his eyes and the weight was released by cutting the cord. Recordings were made of the electrical activity of the anterior belly of the digastric, anterior and posterior temporalis, cephalic part of the trapezius, sternomastoid and biceps brachii muscles of six adult men. The instant of unloading was determined by a piezo-electric transducer which signalled vibration of the head sensor. They found a silent period in the agonist muscles on unloading at a mean latency of about 35 msec for the masseter and temporalis and 69 msec for the trapezius muscle. The digastric acted in a variable manner, being electrically active as an "agonist" during support of the weight before unloading in 5 trials and electrically inactive in the remaining 13 trials. When the digastric acted as an agonist before unloading, it showed a silent period at a mean latency of 38 msec after release of the weight. It is stated in their text that, when the digastric was active before unloading, a burst of digastric activity followed unloading at a mean latency of 94 msec. The sternomastoid acted in a manner similar to that of the antagonists, with electrical silence before unloading and a burst of activity after unloading. There is a major discrepancy between the latencies cited in the text and those presented in their Fig. 6. This discrepancy suggests that either the latencies given in the table and the text have been inadvertently multiplied five times, or that the time calibration given in the diagram as 20 msec should be 100 msec. If the first proposal is accepted, their

results would then agree with those of the previous investigators. The output of the movement sensor in their Fig. 6 showed an oscillation almost immediately after unloading and this rapidly reduced to zero amplitude within 15 msec when measured against the given 20 msec time calibration.

The unloading reflex in the jaw muscles in both dentate and edentulous subjects was briefly reported by Gill (1970). A strain gauge was used to record the force being applied to a brittle disc, and sudden release of the force as the disc fractured indicated the moment of unloading of the active muscles. A displacement transducer attached to the lower anterior teeth measured mandibular movement. A motor pause of up to 40 msec was reported in the elevator muscles and this was terminated by a sudden return of the muscle action potential. The initial rapid closing movement of the mandible was terminated without contact of the opposing teeth and mandibular opening had commenced within 200 msec of unloading. The responses of the jaw muscles to unloading during voluntary contraction were the same for dentate and edentulous subjects.

Luschei and Goodwin (1974) studied the pattern of mandibular movements and jaw muscle activity during mastication in monkeys. As an incidental finding they were able to show that the unloading reflex was a common occurrence during normal mastication of foodstuffs. They recorded jaw movements and associated EMG activity in the jaw closing muscles of monkeys. Each animal's head was rigidly immobilised by means of stabilising lugs attached to the skull. Jaw movements were monitored by attaching an arm, which moved a light-emitting diode in front of a light-sensitive position transducer, to a mandibular implant. This faithfully recorded jaw movements but there was said to be a response

lag of up to 5 msec during rapid upward movements of the mandible as the arm carrying the diode was not completely rigid. Sudden rapid movements of the mandible were often observed during the initial chewing cycles, presumably because of breaking of the piece of biscuit. These events were associated with a sudden, transient cessation of EMG activity in the closing muscles. After allowing for the assumed 5 msec mechanical lag in the movement transducer, this cessation of activity had a latency of 5 to 10 msec.

Goodwin and Luschei (1974) then studied these movements in monkeys in which the tract of the mesencephalic nucleus of the fifth nerve had been destroyed by a lesion in the brain stem. They found that this lesion, which destroyed the cell bodies of muscle spindle afferents, had no effect on the muscular response to sudden unloading, even though it abolished the jaw jerk reflex. They suggested that, because of its short latency, this unloading response must be a segmental reflex, but that it could not be attributed to the known monosynaptic pathway from muscle afferents or to any other reflex currently known from the jaw musculature.

The most systematic investigation of the unloading reflex was made by Lamarre and Lund (1975), as part of an investigation of load compensation in the human masseter muscle. Subjects were asked to perform tasks while they bit with their upper and lower teeth on two metal bars. The upper bar was fixed while the lower was attached to the spindle of a torque motor. A weight ("inertial load") was hung from the lower bar to load the jaw closing muscles. A potentiometer attached to the shaft of the motor monitored movement in the vertical plane and its output was displayed as a spot moving in the vertical axis on an oscilloscope

screen in front of the subject. In some experiments, the subject was asked to maintain or close his jaws at the same speed as a moving spot on an oscilloscope screen which was controlled by the experimenter. Bipolar surface EMG recordings were made from masseter and digastric muscles. In the first unloading experiment the subjects were asked to maintain their jaw position as their jaw elevator muscles supported the weight hanging from the lower bar. During randomly selected trials, a torque pulse was applied in the closing direction. The EMG recordings from the masseter were full-wave rectified and the EMG and angular displacement records for at least eight control and eight unloading trials were averaged. It was found that if the torque pulse were greater than the inertial load and complete unloading occurred, the masseter activity was depressed after a latency between 6.5 to 11 msec (mean 8.5 msec) and stayed depressed throughout the period of unloading. This depression was considered to be at monosynaptic latency as it corresponded in latency and time course with the excitatory monosynaptic response seen in the masseter when the chin was tapped with a reflex hammer or when the torque motor applied a downward pulse to the resting mandible. If the torque pulse in this unloading experiment was less than inertial load, second and third phases of depression of masseter activity were observed as well. The second phase began at a latency of 27 to 36 msec and the third between 60 and 70 msec after unloading. The third phase was curtailed after the jaw became stabilised at a new position.

In a second unloading experiment, subjects supporting the weight hanging from the lower bar were asked to close at the same speed as the spot triggered by the experimenter. In randomly selected trials, a torque pulse was applied in the closing direction. The EMG and displacement records for control and unloading trials were compared.

After unloading, there was an initial upward acceleration of the mandible and then the mandibular velocity fell to a value equal to, or less than the control. The silent period generally did not exceed 40 msec. An EMG response was recorded in the digastric with a mean latency of 24 to 34 msec (mean 27 msec) after the start of the torque pulse. In a third experiment, the head was rigidly held and the subject pressed down on the moveable lever with their chins. No mention is made as to whether a weight provided an inertial load to the bar in the closing direction. Loading of the jaw opening muscles during jaw opening movements by applying a torque pulse in the closing direction produced an EMG response of the same order of latency as in the previous experiments.

The first experiment, in which subjects maintained jaw position in the face of torque pulses, was repeated after local anaesthetic had been applied to upper and lower teeth. The unloading response of the jaw opening and closing muscles to a torque pulse in the closing direction was unchanged by local anaesthetic.

The possible mechanisms for the control of the events in the masseter and digastric muscles after unloading were discussed. It was considered that the lack of monosynaptic response in the digastric muscle was consistent with the paucity of spindles in these muscles. Their results from anaesthetising the teeth suggest that mechanoreceptors in the periodontal ligament are not responsible for the cessation of activity in the masseter. They felt that receptors in the temporo-mandibular joint may contribute to the reduction in EMG activity in the masseter and excitation of the digastric.

They discussed the work of Goodwin and Luschei (1974) who reported that destruction of the mesencephalic nucleus abolished the jaw jerk reflex but not elevator inhibition on unloading. If the second and third phases of elevator depression were mediated by receptors other than muscle spindles - perhaps those in the temporomandibular joint capsule - an unloading response would persist at a longer latency even though a monosynaptic spindle-mediated response was abolished. They considered that the type of movement transducer used by Goodwin and Luschei may not have been able to determine accurately the latency of elevator depression and that the response they reported may have been the second phase of depression, the monosynaptic spindle-induced response having been eliminated by the MES V lesion.

They considered that the longer response latency of the digastric compared with the masseter may indicate the function of a short-latency cortical-loop response as has been proposed for the limb muscles (Marsden *et al.*, 1972). These authors cited their earlier paper (Lund and Lamarre, 1974a) in which they suggested that the large majority of neurones in the cortical masticatory area are concerned with activation of jaw opening muscles and concurrent inhibition of the jaw closers. They suggest that in this case, these neurones may be responsible for the second phase of masseter inhibition and concurrent digastric excitation. They felt that this arrangement of neurones may reflect the important need to rapidly reduce jaw closing activity as soon as brittle food breaks.

Thus, a moderate amount of information has been accumulated on the subject of unloading reflexes in both the limb and jaw muscles. However, several important aspects of the nature of the signal which initiates the unloading responses are not clear. Angel *et al.* (1973) presented

evidence that, at the elbow joint, the signal apparently does not originate in the spindles of the stretched antagonist muscle. The paucity of muscle spindles in the digastric muscle (Cooper, 1960; Lennartsson, 1979) suggests that this is also likely to be true in the unloading of the jaw closing muscles. Other evidence refuting spindle involvement in jaw muscle unloading has been presented by Goodwin and Luschei (1974). Yet another potential trigger signal - from "unloading" of the periodontal mechanoreceptors - is excluded by the results of Lamarre and Lund (1975).

The other major questions left unanswered from the existing body of data are: (1) to what extent do the reflex events in the agonist and antagonist muscles which occur after unloading account for the rapid arrest of the upward mandibular movement when the resistance to muscle contraction is suddenly and unpredictably removed; and (2) what is the effect on the behaviour of the muscles of varying the force and joint angle at the time of unloading?

It is to these questions that the present study is addressed.

METHODS

A total of 19 healthy young adult volunteers, 14 males and 5 females, aged between 15 and 30 years were used in this study. None of the subjects had a history of neuromuscular disorders and none showed evidence of tenderness when the masticatory muscles were palpated.

The experimental protocol required that subjects should bite on bars between the upper and lower teeth with increasing force until the resistance to jaw closing was suddenly and unexpectedly withdrawn. This was achieved with the apparatus shown in Figure 1. The subjects bit on these two bars, the upper one being fixed and the lower one hinged.

The upper bar was fabricated from 10 mm thick stainless steel and could be fixed at various heights (A) to give a range of openings between the bars of between 4 and 35 mm. Jaw position relative to the bars was kept constant from trial to trial by means of an impression of the upper premolar occlusal surfaces in dental impression compound on the upper bar (E).

The lower bar was fabricated from stainless steel of similar thickness with a bearing (C) to provide an axle around which the bar pivoted. The resistance to closing was achieved by inserting a brittle 1.5 mm diameter rod through one of the holes (B) in the lower bar and backing plate. Different shearing forces were obtained by placing the rod in holes at different distances from the fulcrum. In preliminary experiments, milled brass and cast iron shear pins were used to provide the resistance. As these were difficult to obtain, alternative test pieces were sought. Soda glass rod (1.5 mm diameter) proved to be a

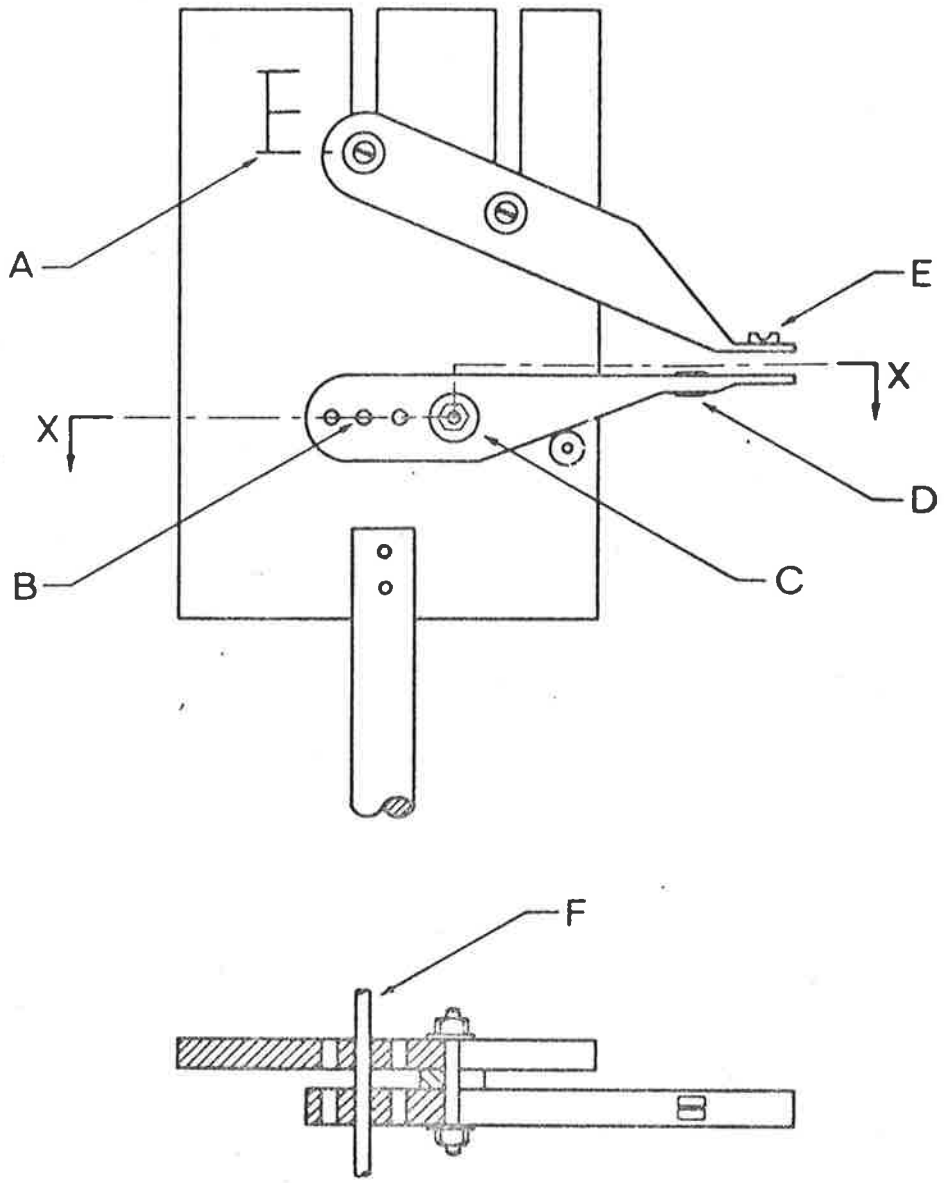

Figure 1. Apparatus used to unload jaw closing muscles. A, calibration of initial bar position; B, holes for glass rod; C, axle bearing; D, strain gauges; E, locator for upper teeth; F, glass rod; SECTION XX, horizontal section through lower bar and backing plate.

suitable substitute as it fractured cleanly and gave the subject no warning prior to the event.

The experimental procedures were consistent with N.H. & M.R.C. Guidelines for Human Experimentation. Subjects were seated in a comfortable position with their upper premolars located in the impression compound on the upper bar and their lower premolars biting on a Teflon® pad on the lower surface of the lower bar. Subjects were fully informed of the experimental procedure and instructed to "bite cleanly through" the resistance which prevented their teeth from closing together.

The force applied to the lower bar was measured by strain gauges (D). These gauges were cemented 20 mm from the end of the bar with two above and two below the bar. This system was linear over a +400 to -400 N range. Calibration records were taken before each experiment by loading the bar with weights of known values.

Mandibular position in the vertical plane was monitored by means of a small extra-oral "grain of wheat" light globe attached to an orthodontic bracket on the lower incisors by a stem made from two lengths of 4 mm thickness dead-soft solder. The stem was bent to suit each subject so that the globe could be placed extra-orally in order to minimise any mechanical interference to the lips and to keep the heat of the globe away from the tissues. The stem also provided mechanical damping for the light during the sudden mandibular movements. The globe was connected to a 12 volt DC power supply by very fine wires which did not interfere with mandibular movements.

A photo-sensitive position sensor (United Detector Technology PIN

SC/10) was placed 60 mm in front of the light to transduce the movement of the light in the vertical plane. This sensor was housed in a light-proof box with the light beam reaching it through a 2 mm "pin hole" opening 10 mm in front of the sensor. The opening was covered with a Kodak Wratten #12 filter to prevent interference from day-light or fluorescent light sources. The box was attached to a helmet placed on the subject's head by a hollow aluminium rod along which travelled the power leads to the position sensor and by which the output leads returned to the distant integrated-circuit instrumentation amplifier which amplified and conditioned the signal to maximise the linearity of the output. This system provided a continuous readout of vertical jaw position as a DC voltage (Milberg *et al.*, 1980).

Mandibular movement was calibrated at regular intervals during each experiment by recording the voltage read-out with the premolars in contact and then with a 10, 20 and 30 mm block interposed between the premolar teeth.

The EMG activity of the masticatory muscles on the biting side was recorded by silver/silver chloride surface electrodes placed 10 mm apart. Surface resistance of the skin was reduced by roughening it with fine sand-paper, cleansing with alcoholic ether and rubbing in a conductive paste. Cup surface electrodes were placed on the anterior border of the temporalis, just below the mid-point of the anterior border of the masseter and over the centre of the anterior belly of the digastric. The rims of these cups were held in place by double-sided adhesive rings with cut-out centres which allowed the space between the inner surface of the cup and the skin to be filled with conductive electrode paste. The activity of the middle fibres of the temporalis was recorded by

stainless steel subdermal clip electrodes placed 10 mm apart on the scalp. All electrode leads were taped to the skin in an effort to minimise movement during jaw movements. An earth electrode was attached to the lobe of the ear on the recording side. The EMG activity was amplified by Isleworth differential AC amplifiers with the gain set at X1000 and at a bandwidth of 200 Hz to 5 kHz.

To confirm that the surface electrodes over the digastric muscle were recording the electrical activity specifically from the muscle and not, for example from the platysma, digastric EMG activity in one subject was recorded simultaneously from intra-muscular wire electrodes (Basmajian, 1967) as well as from electrodes on the skin over the same muscle.

An eight-channel tape recorder (Hewlett Packard 3968A) was used to record the amplified EMG activity, mandibular movement and strain. A vocal commentary was made on a 7th channel. An illustration of the raw data obtained in a typical recording is shown in Figure 2 in which the subject opens, closes to take up his position on the bars then bites together until the glass breaks (arrow).

In the first 10 experiments, the unloading response was studied at initial openings of 10, 20 and 30 mm. For any given jaw opening, subjects were required to overcome three resistance levels in the range of 20 to 400 N. An attempt was made to relate mandibular movement to the EMG activity and the strain on the basis of visual inspection of single trials on the screen. Interpretation of these results proved difficult and it was realised at that time that there was a need for quantitative analysis.

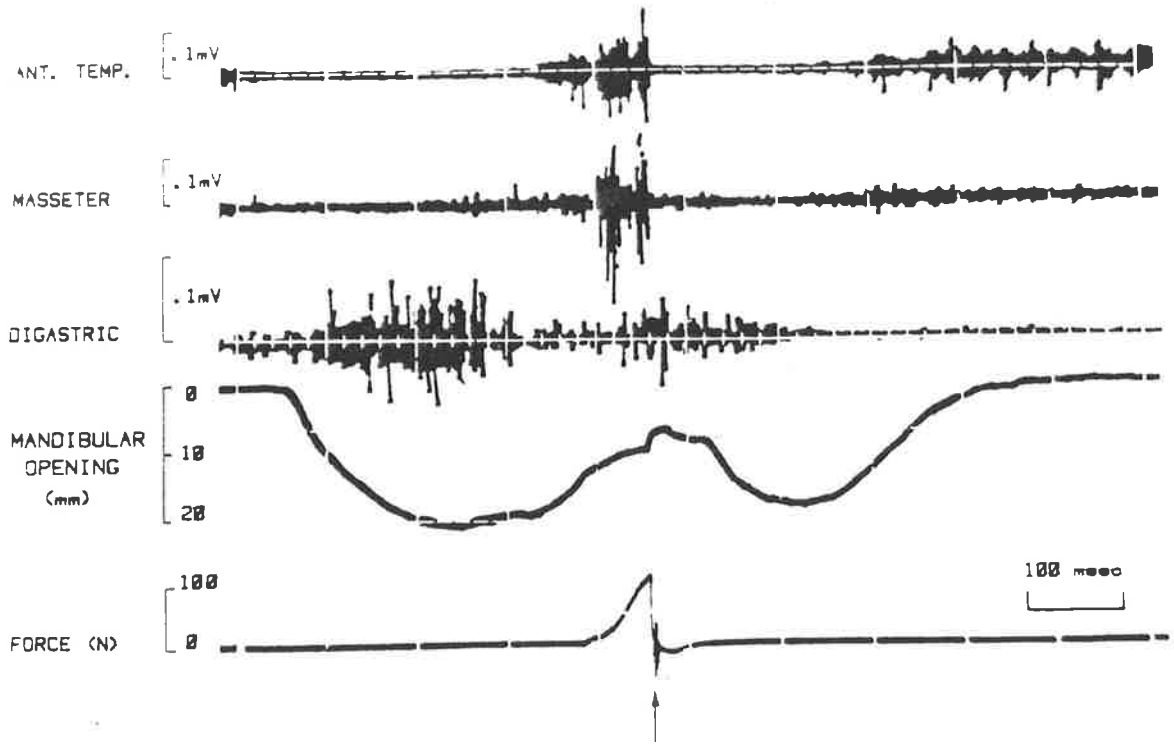

Figure 2. Oscilloscope traces from a single biting trial showing the EMG activity of the anterior temporalis (ANT. TEMP.), masseter and anterior digastric muscles. The mandibular opening trace shows the vertical position of the light attached to the lower anterior teeth as the subject starts with the teeth together, opens widely to come onto the biting bars and applies force to the bars up to the break point (arrow). After fracture of the glass rod, the initial closing movement is arrested and the subject voluntarily opens to move away from the bars and returns to the resting position with the teeth together. The force trace shows the output of the strain gauges attached to the lower bar.

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For the last 9 subjects, a standard protocol was followed to ensure that a large enough sample of biting trials was recorded at each combination of opening and force to allow analysis with signal averaging procedures. Up to 30 trials were recorded at each of 9 combinations of force and position for each subject.

Using programmes modified for this particular application, 6 channels of data were sampled at 2 kHz/channel and analysed off-line on a DEC PDP 11/40. Full-wave rectification was carried out by computer software after sampling. The sudden decrease of the strain signal when the glass rod sheared was used as the time reference point for the "break". An amplitude discriminator gave a TTL pulse at the start of the negative slope of the strain signal which caused the computer to record the data recorded for 100 msec before and after the break.

In each subject, each 200 msec trial was reviewed individually and any trials in which the glass did not break cleanly were not used in the signal-averaging procedure. Hence a group of trials was derived for 10 mm/LOW FORCE, 20 mm/LOW FORCE and 30 mm/LOW FORCE. Groups of trials were then found for these three openings at nominal "medium" and "high" forces. The EMG, strain and position records in each of these nine groups were then averaged.

Calibration trials of strain and mandibular position were also stored by the computer and used to calibrate the movement and strain signals. The velocity and acceleration of mandibular movement were calculated from the position record for each of these nine groups. A digital plotter (Hewlett Packard 9872A) was used to print out the averaged EMG records, the mandibular movement, velocity and acceleration

and the strain. A typical record is shown in Figure 3.

The cumulative integral of each averaged EMG record for each of the nine groups was also calculated and plotted for quantitative comparison of the averaged muscle activity during different conditions of strain and position. A typical plot is seen in Figure 4.

The averaged data for each subject were used to find the time taken ("time to level") and the vertical distance travelled ("distance travelled before levelling") by the mandible to reach zero velocity after the break. The "peak mandibular velocity" immediately after the break was measured from the computed velocity trace.

A burst of electrical activity of up to 10 msec duration, starting 2 to 3 msec after the break point, was found in 25% of the averaged records of most subjects. This was more likely to occur when unloading occurred at small initial jaw separation and where large forces were required to break the resistance between the bars. In an effort to determine whether this activity was a movement artefact, the experimental protocol was carried out with digastric activity being recorded by 2 sets of surface electrodes. One pair was placed over the anterior belly of the digastric muscle on the working side while a second pair, insulated from the skin by adhesive polyvinyl chloride tape, was placed over the contra-lateral muscle. A resistor was placed across the insulated electrodes to match the AC resistance between the electrodes on the biting side. The data obtained from these 2 pairs of electrodes was then averaged over 20 break trials, at each of 9 combinations of force and position, in the normal manner.

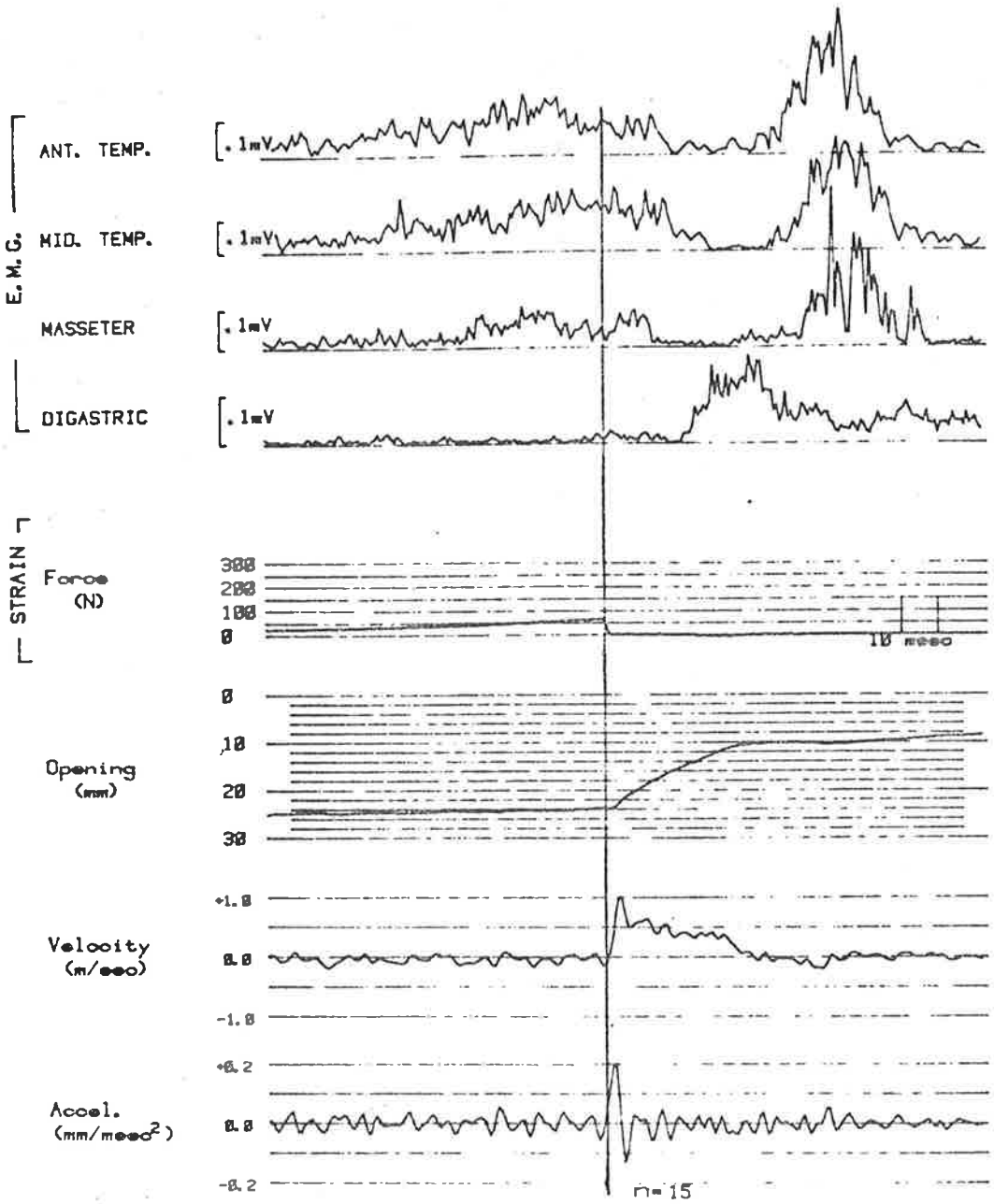

Figure 3. A representative record of analysed data. The traces shown are the average of 15 nominal "low force" biting trials in which the initial opening of the bars was 30 mm. The upper 4 traces show the rectified and averaged EMG records of the anterior temporalis (ANT. TEMP.), middle temporalis (MID TEMP.), masseter and anterior digastric muscles. The fifth trace is the average of the strain exerted onto the lower jaw bar. The sixth trace shows averaged mandibular movement from which the remaining two traces were computed. The vertical line through all traces indicates the instant of "unloading".

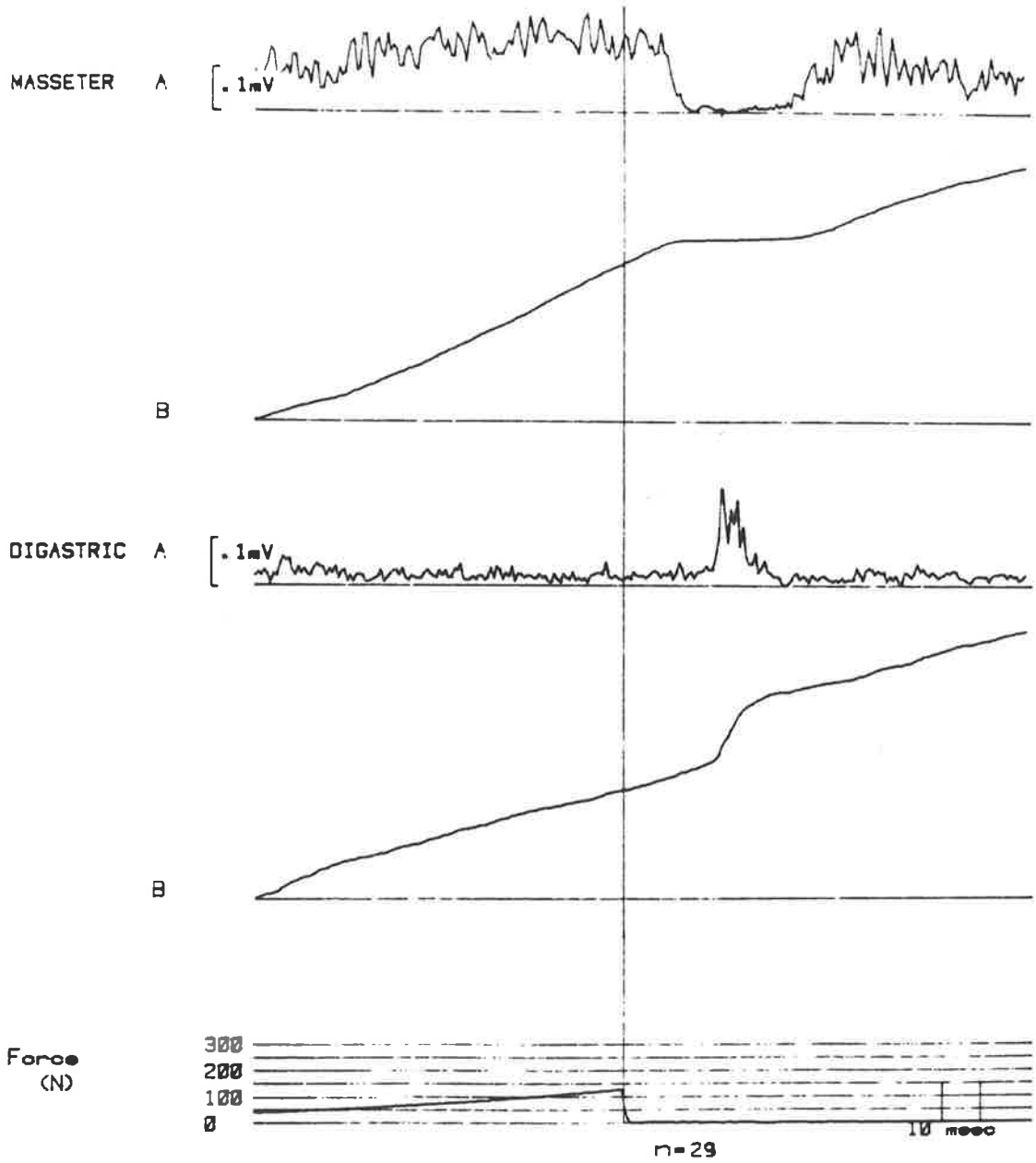

Figure 4. Computer-generated plot of the cumulative integrated EMG. The averages of rectified EMG activity (A) of the masseter and anterior digastric muscles was used to compute a cumulative integral of EMG activity (B) over the sampling period. The "force" trace is the output of the strain gauge on the lower bar as the subject bites through the resistance. The vertical line through all traces indicates the instant of "unloading".

RESULTS

1. VERIFICATION OF THE ACCURACY OF THE TECHNIQUE OF SURFACE ELECTRODE RECORDING OF THE DIGASTRIC MUSCLE

When the intra-muscular wire electrodes were placed so that they recorded from several digastric motor units, the burst of EMG activity which started approximately 20 to 22 msec after the break was similar to that seen in the signal recorded from surface electrodes (Figure 5). In instances when the wire electrodes were recording from single muscle units, the burst of activity occurred in the same time period as the activity recorded in the surface electrodes but was often of shorter duration.

2. IDENTIFICATION OF MOVEMENT ARTEFACTS

The possibility that the early burst of electrical activity which occurred at short latency might be a movement artefact was investigated in 2 subjects using insulated surface electrodes.

A burst of digastric activity was always seen in the electrodes in contact with the skin at a latency of 20 to 22 msec after unloading. In some trials, where higher forces were required to overcome the resistance between the teeth, an earlier burst of electrical activity was recorded in these electrodes at a latency of less than 5 msec after unloading (Figure 6). This "early" burst of activity then decayed over the next 10 to 15 msec.

The form of the rectified, averaged potentials recorded from the

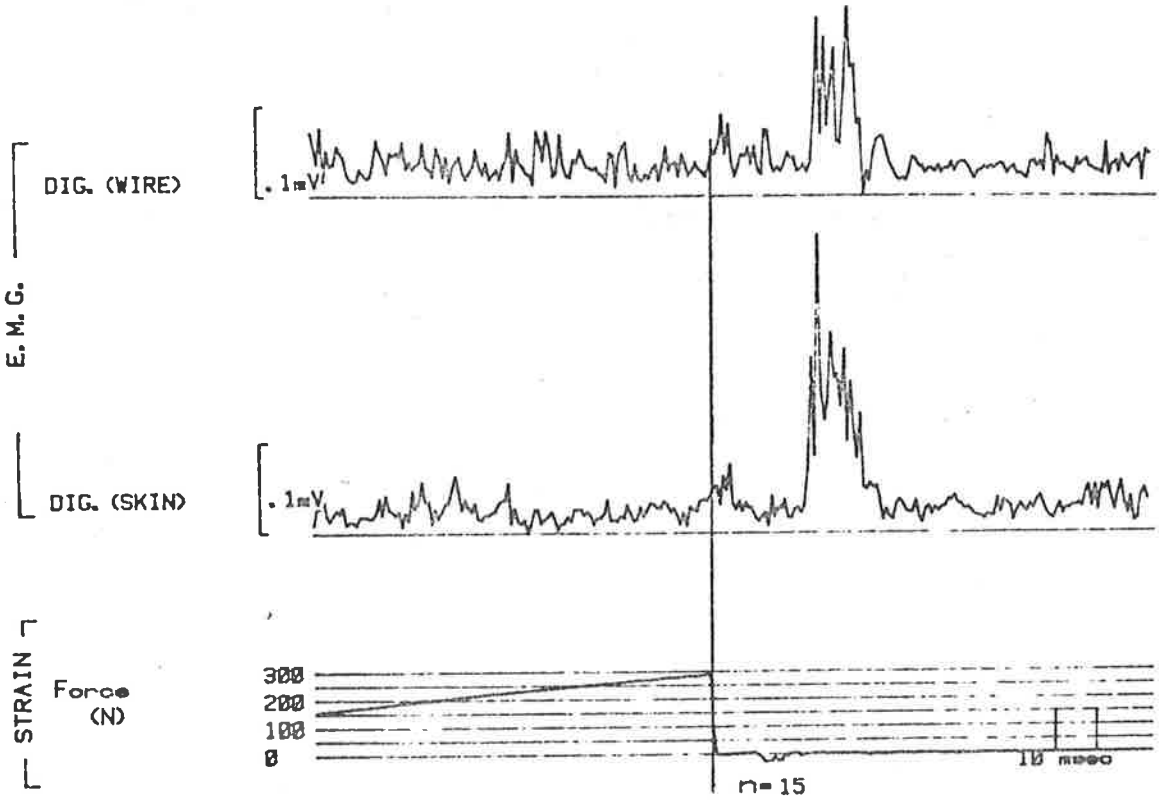

Figure 5. The averages of the rectified EMG activity of the anterior digastric muscles (N=15 trials) obtained simultaneously from wire electrodes placed in the anterior belly of the digastric muscle (DIG. WIRE) and from surface electrodes placed on the skin over the same muscle (DIG. SKIN). The force trace shows the increase in the output of the strain gauge on the lower bar as the subject overcomes the resistance to closing. The vertical line through all traces shows the instant of "unloading".

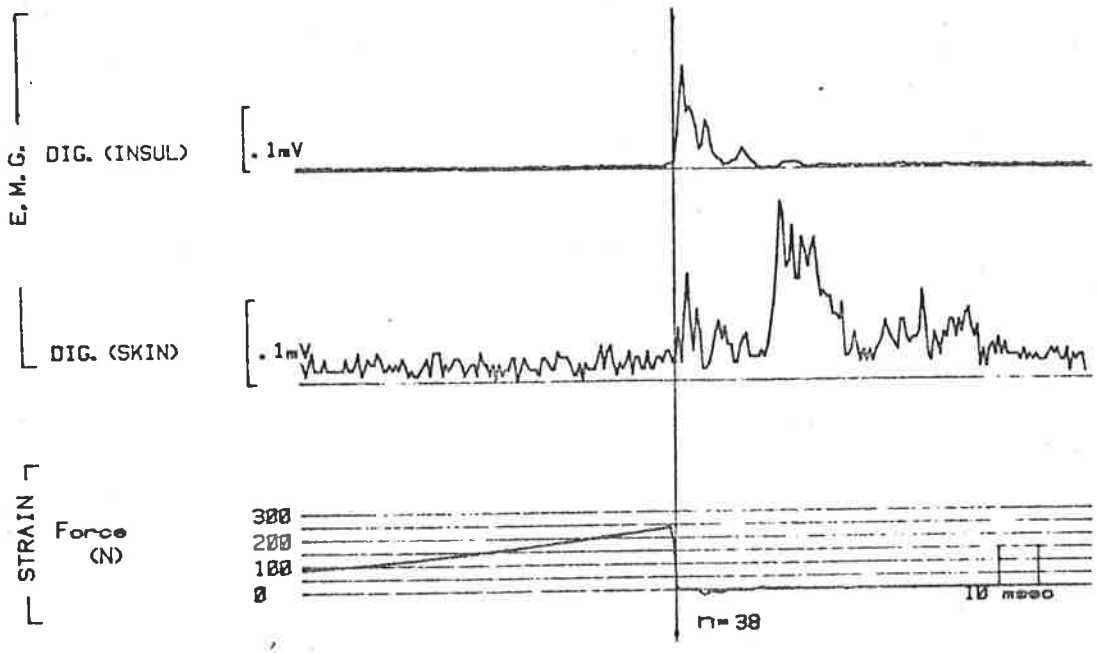

Figure 6. The electrical activity recorded from surface electrodes placed on the skin over the anterior belly of the digastric muscles rectified and averaged for 38 biting trials. The upper trace [DIG. (INSUL.)] shows the activity from digastric surface electrodes which were electrically insulated from the skin by adhesive polyvinyl chloride tape and joined by a resistor of a value approximated equal to the AC resistance between the two contralateral electrodes that were in contact with the skin. The second trace [DIG. (SKIN)] shows the electrical activity from these contralateral electrodes. The force trace shows the output of the strain gauge on the lower bar as the subject overcomes the resistance to closing. The vertical line through all traces indicates the unloading of the bar.

insulated electrodes pair was, roughly, that of a recified sinusoid which decayed over 10-15 msec (Figure 6). Its latency of onset and duration was similar to the "early" burst of activity in the conventionally attached electrodes on the working side.

This "early" burst of electrical activity was also seen when recordings were made from intra-muscular wire electrodes (Figure 5).

3. MANDIBULAR MOVEMENT

3a. The pattern of mandibular movement:

Although the opening of the bars was nominally set at 10, 20 and 30 mm at the start of each trial, the distance between the bar at the time of break depended on how much the bars had bent under load before the break occurred. This resulted in a range of openings at the time of break ("opening at break") of between 7 and 30 mm.

The upward mandibular movement commenced within 1 msec of the output of the strain gauge reaching zero (Figure 3) and the mandible always reached its maximum upward velocity within 5 msec of unloading. The movement record traced an upward curve until it reached a point where the closing movement had been arrested and the velocity fell to zero. The separation of the teeth at this point was never less than 5 mm. When unloading occurred at low jaw openings, there was a tendency for the upward curve of the movement record to be rapidly levelled and the time for mandibular velocity to reach zero ("time to level") was shortened (Figure 11). In some averaged records, a small

upward deflection of the movement trace was seen in relation to the return of elevator activity after 50 msec or so.

3b. The vertical distance travelled by the mandible after the break:

The mean opening of the premolar teeth (i.e., on the loaded and deformed bar) at the time of unloading was plotted against the mean distance that these teeth travelled before the upward movement of the mandible was arrested for the 9 subjects. The correlation coefficient was calculated using the statistical programmes of the Hewlett Packard 9815 A/S calculator. It was found that there was a close relationship between these two variables with a high correlation coefficient (r=-.87). The scatter diagram (Figure 7) shows that the relationship between the initial opening and the distance travelled before levelling is essentially linear.

When the distance travelled after the break was related to the force at the time of the break (Figure 8), there appeared to be no correlation between these two variables (r=zero). A very low correlation was also seen when the averaged results were categorised into 3 groups on the basis of the separation of the teeth at the time of the break. When the initial opening was equal to or less than 10 mm, it was found that, irrespective of force at the time of the break, the distance travelled was between 2 and 6 mm. When the initial opening was between 10 and 20 mm, the distance travelled was between 4 and 12 mm and above an initial opening of 20 mm the distance travelled was between 8 and 18 mm.

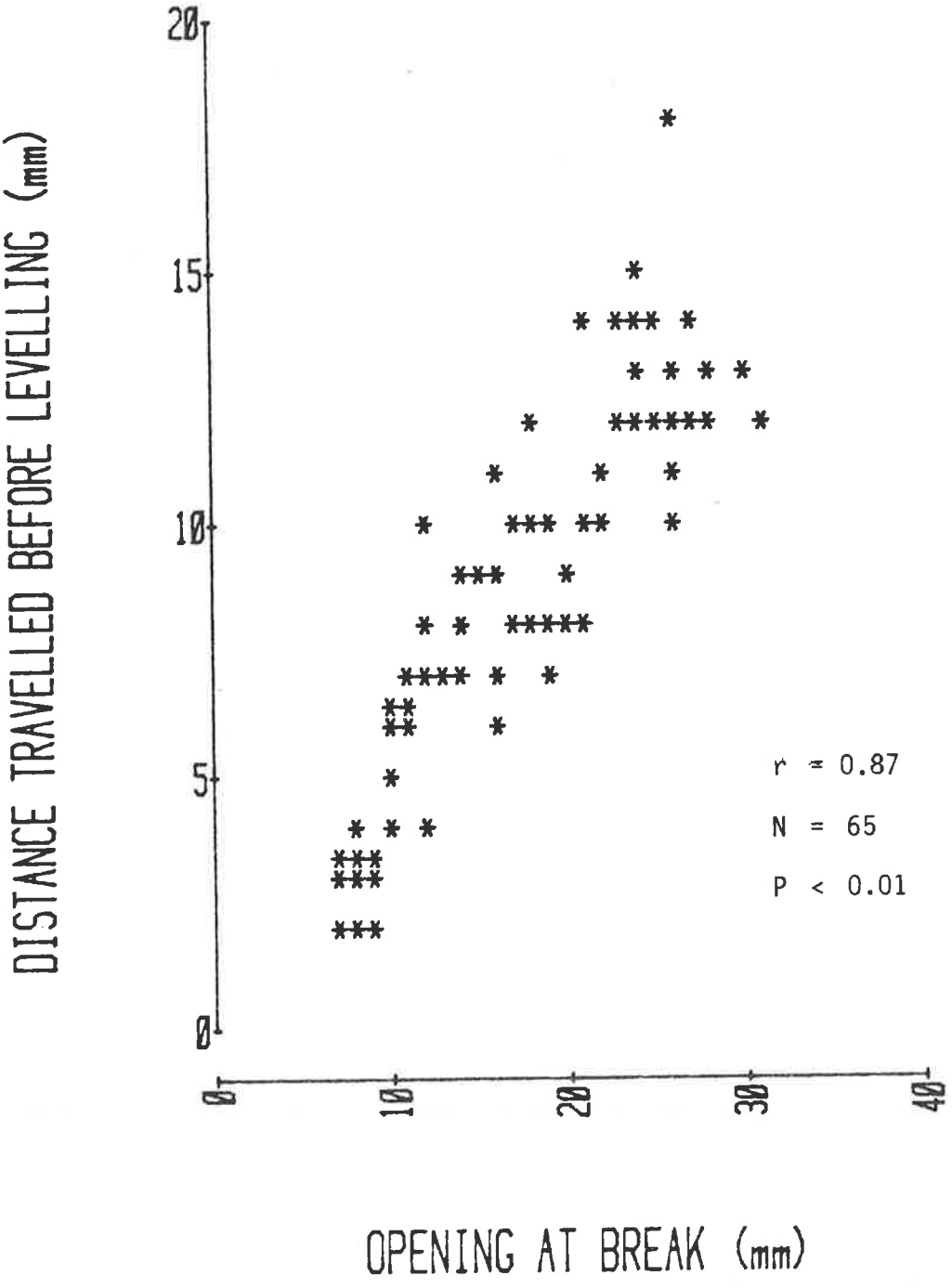

Figure 7. The mean opening of the premolar teeth on the loaded and deformed lower jaw bar at the instant of unloading (horizontal axis) is plotted against the mean distance travelled by these teeth before the upward movement of the mandible was arrested (vertical axis) for 9 subjects. In each subject averaged values of this movement were obtained when low, medium and high resistances were overcome at 10, 20 and 30 mm initial openings of the bars. Note that the plotter has superimposed many points with the same coordinates.

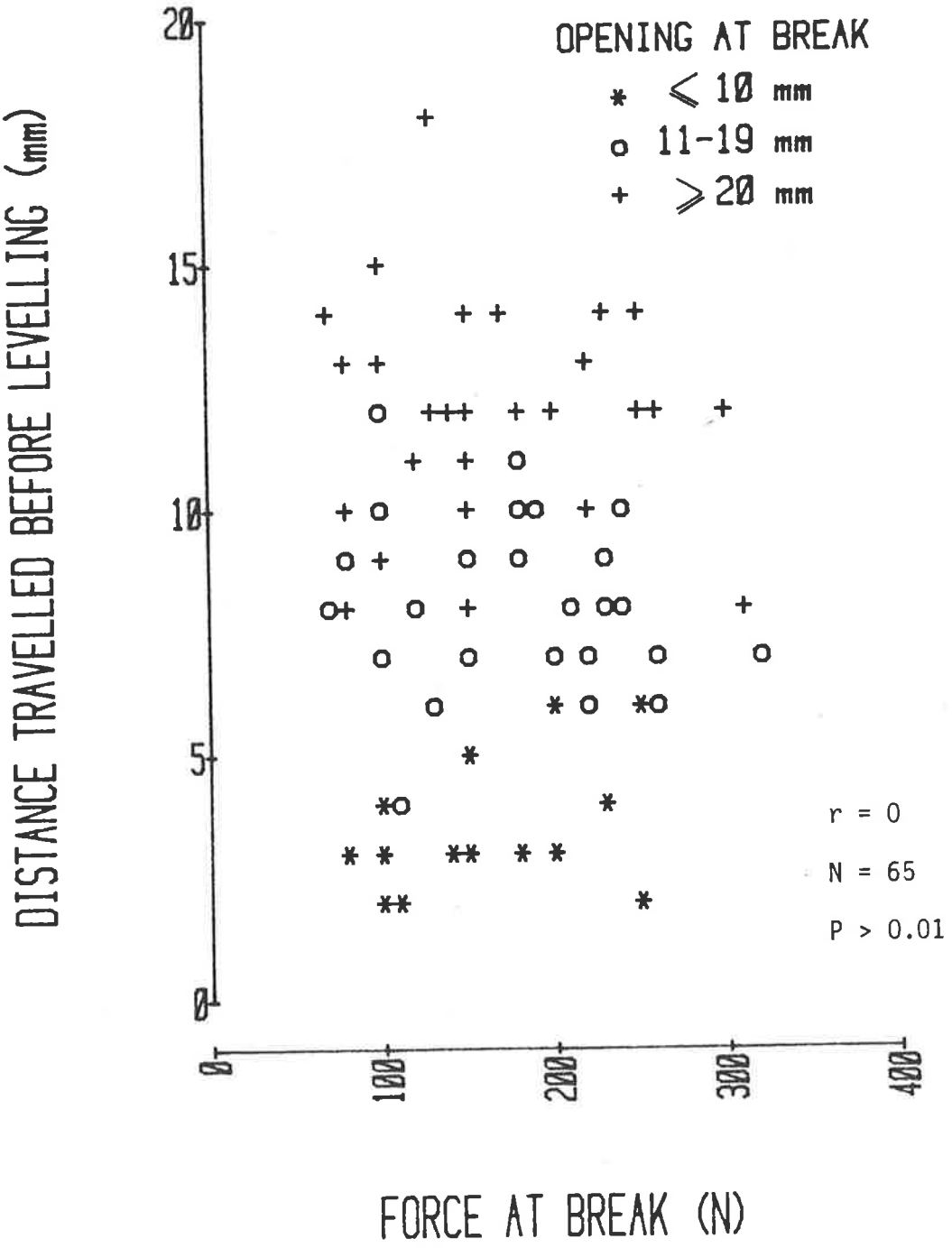

Figure 8. Relationship of force at the time of the break to the distance travelled before the upward movement of the mandible was arrested after unloading, for the same 9 subjects in Figure 7. Note that the plotter has superimposed many points with the same coordinates.

3c. The peak mandibular velocity after the break:

In all subjects, examination of the averaged position record revealed that the peak closing velocity was reached within the first 5 msec after the break.

When the peak mandibular velocity was related to the opening at the time of the break, it was seen that there was a low correlation between these two variables (r=0.42). The scatter diagram (Figure 9) shows that at initial openings below 10 mm, the peak velocity always lay in the narrow range between 0.8 and 1.0 m/sec. When the opening at the time of the break was greater than 10 mm, the velocity reached varied between 0.9 and 1.5 m/sec.

The correlation between the peak mandibular velocity and the force at the time of the break was low (r=0.37) which suggests that these two factors are essentially unrelated (Figure 10).

3d. The time taken for the mandibular velocity to reach zero:

The relationship between the time taken to arrest the closing movement of the mandible after the break and the initial opening at the time of the break is shown in Figure 11. The correlation (r=0.1) between these two variables was low. In most subjects, the closing movement was arrested between 35 and 65 msec after the break. There was a tendency at initial openings of less than 10 mm for this time to be reduced to less than 20 msec in some subjects.

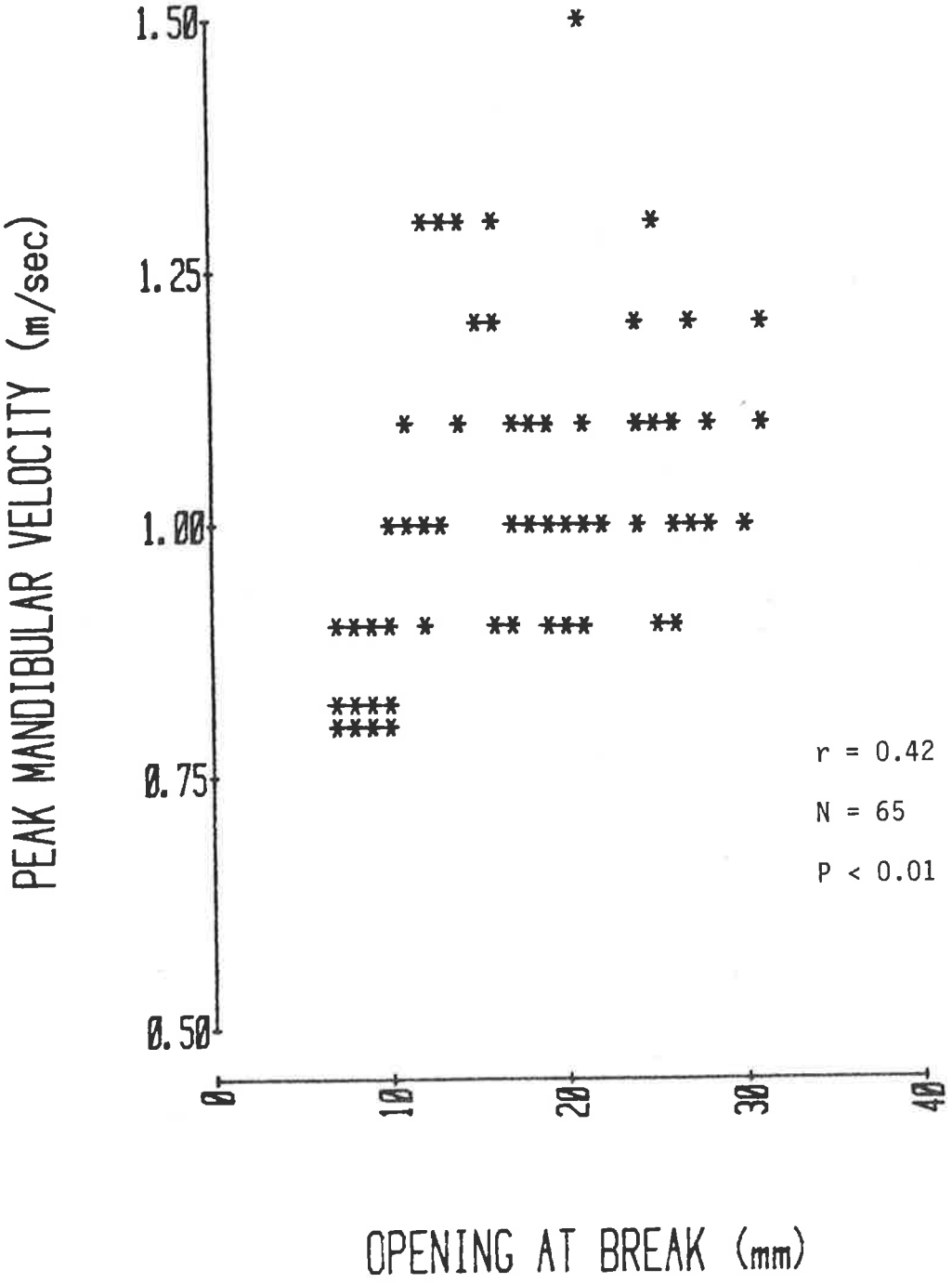

Figure 9. Relationship of the opening at the time of the break to the peak closing velocity reached by the mandible after "unloading", for the same 9 subjects as in Figure 7. Note that the plotter has superimposed many points with the same coordinates.

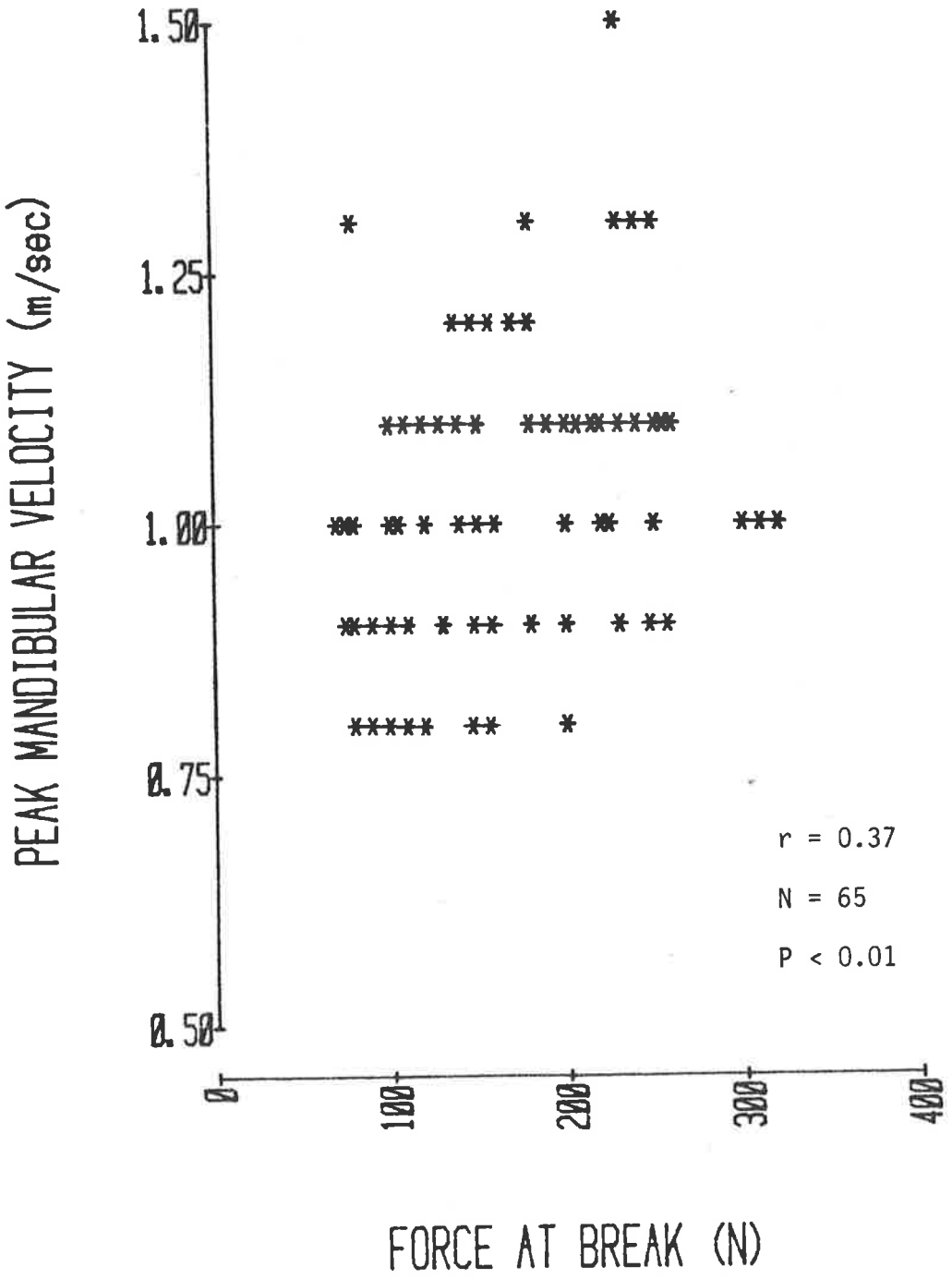

Figure 10. Relationship of the force at the time of break to the peak closing velocity reached by the mandible after unloading, for the same 9 subjects as in Figure 7. Note that the plotter has superimposed many points with the same coordinates.

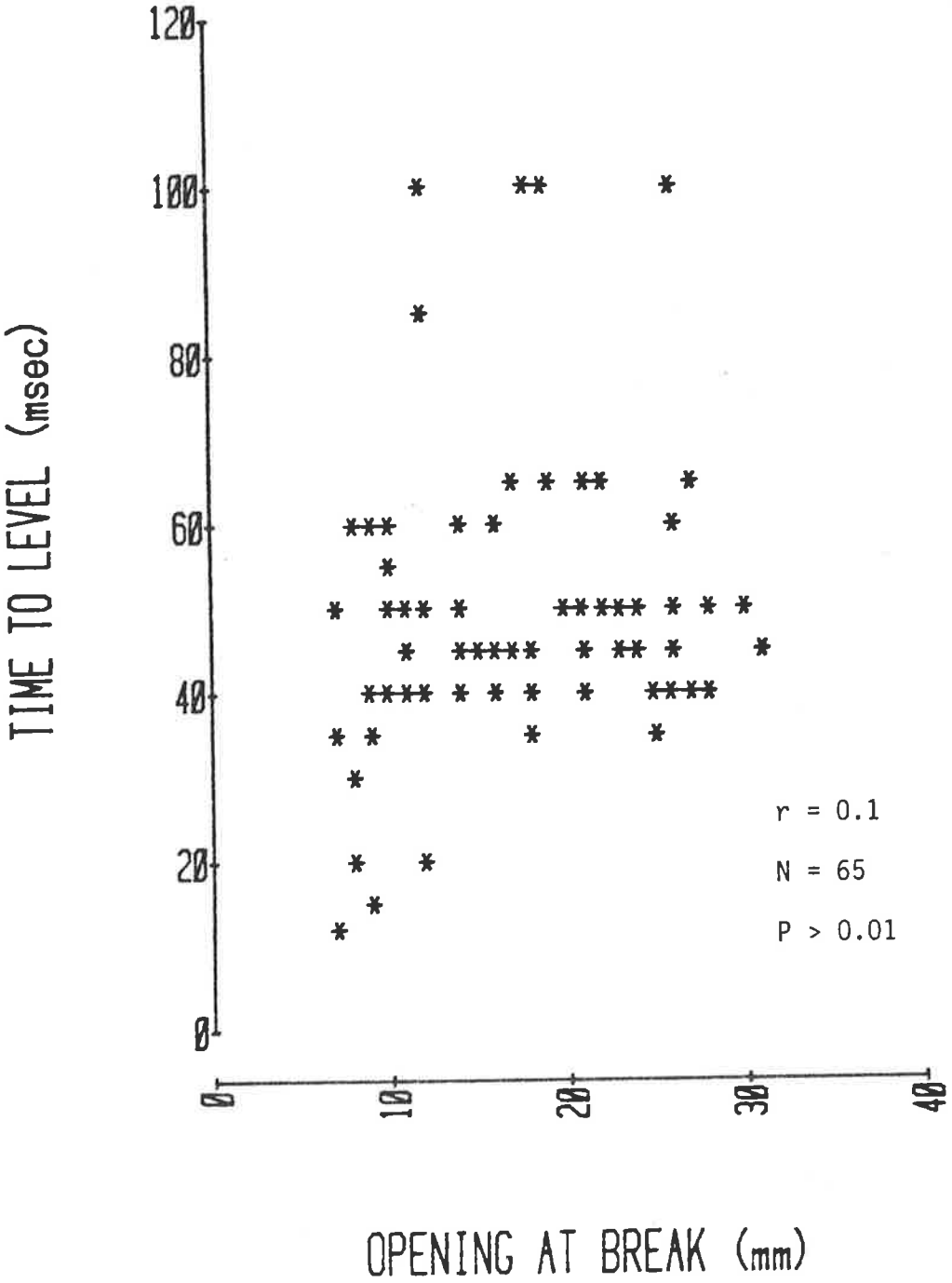

Figure 11. Relationship of the opening at the time of the break to the deceleration time (time to level) of the mandible, for the same 9 subjects as in Figure 7. Note that the plotter has superimposed many points with the same coordinates.

When the time for the movement trace to level and the force at the time of break are compared (Figure 12), no correlation (r = zero) was found between these factors.

4. THE ACTIVITY OF THE ELEVATOR MUSCLES

Over the 200 msec sampling period, the anterior and middle temporalis and masseter muscles showed similar patterns of amplitude and timing of EMG activity (Figure 3).

Before the break, as the level of EMG activity increased, the force applied to the bar increased. Some subjects bit strongly and rapidly on the bar causing a rapid fracture of the glass rod. Other less confident subjects increased their biting force more slowly.

The elevator EMG activity continued for 10 to 15 msec after the break, after which a motor pause followed. In some traces, elevator activity continued for as long as 20 msec. In 4 out of the 9 subjects, there was no return of elevator activity for the remainder of the sampling period.

In the other 5 subjects, there was a re-excitation of the elevator muscles starting between 40 and 85 msec after unloading. This always occurred after the upward mandibular movement had ceased. Each subject had a characteristic latency for the return of this activity and there was less than 20 msec variation in this timing in the 9 averaged groups of results for any particular subject. In 3 subjects, this return of

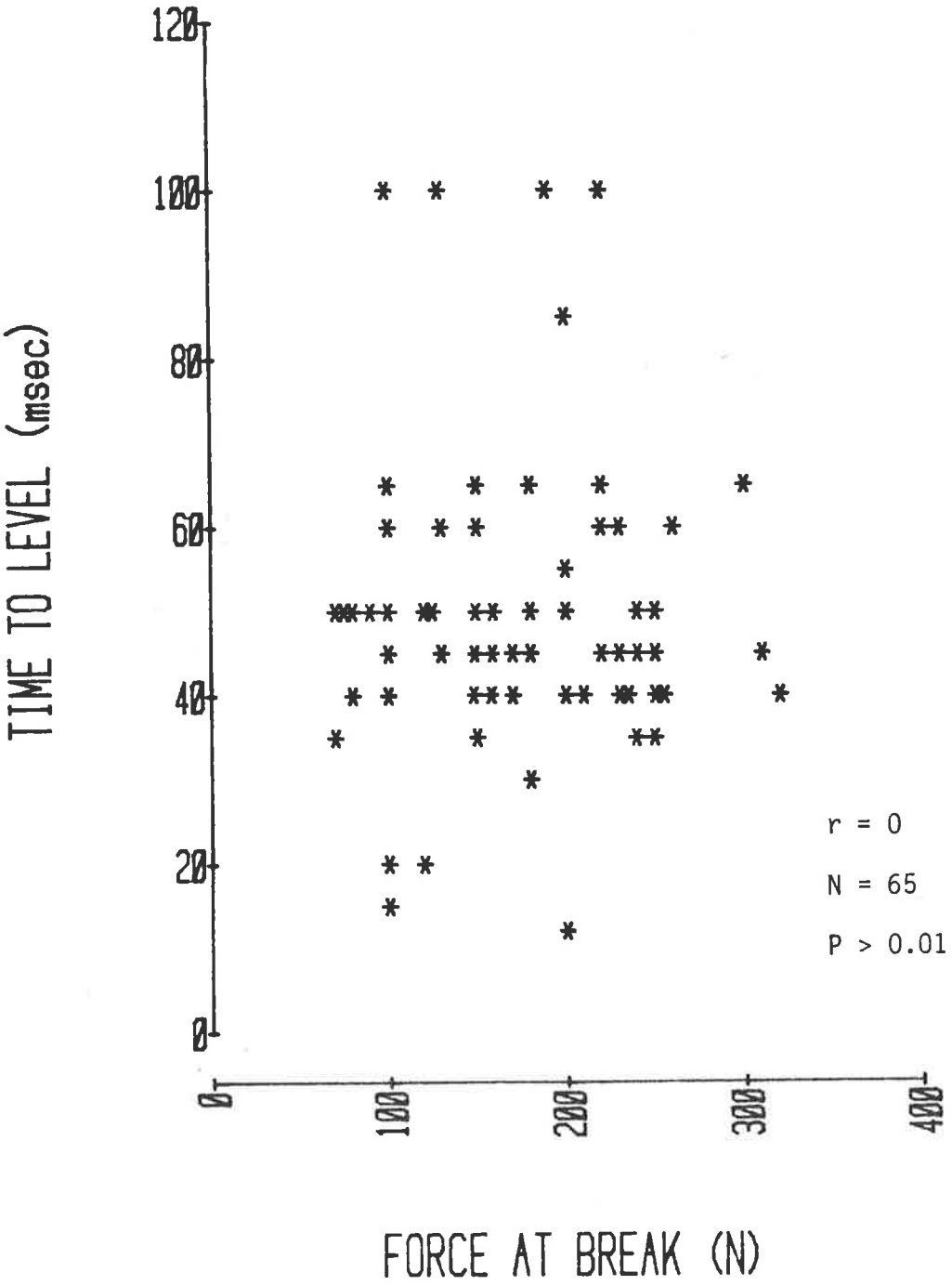

Figure 12. Relationship of the force at the time of break to the deceleration time (time to level) of the mandible, for the same 9 subjects as in Figure 7. Note that the plotter has superimposed many points with the same coordinates.

activity closely followed cessation of the digastric response (Figure 4), whereas in the other 2 subjects, there was a delay between the end of the digastric response and the return of elevator activity. In some of these subjects in whom a return of elevator activity occurred, a second motor pause was seen in some of the averaged records at a latency of about 90 msec after unloading (Figure 3).

The "second phase of elevator depression" as reported by Lamarre and Lund (1975) was rarely seen in this present study. In 4 out of a total of 81 averages there was a return of elevator activity at a latency of about 30 to 40 msec after unloading which was followed by a second motor pause.

5. THE ACTIVITY OF THE DIGASTRIC MUSCLE BEFORE UNLOADING

The lowest level of digastric EMG activity seen in these experiments occurred when subjects made normal unopposed closing movements without the bars being between their teeth. This is illustrated, for example, in Figure 2 after the subject had moved away from the bars and was returning his mandible to the rest position with the teeth together. The level of EMG activity in the 100 msec sampling period before the break was always at least twice the amplitude seen during the period of unopposed closure after the break.

The area enclosed by a rectified EMG record is related to the force exerted by the muscle (Lippold, 1953). This area was obtained in the present experiments by integration of the average rectified EMG signal. The total integral or cumulative integral calculated by the computer

revealed that when the separation of the bars at the start of each trial was 10 mm, 8 out of 9 subjects showed that the cumulative integral of digastric activity increased at least two fold as the force doubled between the nominal "low" and "high" force trials. That is, the mean level of digastric activity increased markedly when higher forces were required to overcome the resistance between the teeth.

In averaged records obtained with an initial bar separation of 20 mm, there was no clear pattern of change in the cumulative integral of digastric activity between the nominal "low" and "high" force averaged groups. For 5 subjects, the average digastric activity was greater in the trials in which larger biting forces were used then when smaller forces were used. The other 4 subjects showed no change or a reduced level of activity at the higher force levels.

In averaged groups in which the initial separation of the bars was 30 mm, only 2 subjects showed greater average digastric activity in trials in which larger biting forces were used than when smaller forces were used. Seven subjects showed no change or a reduction in activity at the higher force levels.

In the averaged records of 7 out of 9 subjects recorded at the nominal "high" force level, the average digastric activity was unchanged or reduced in trials where the initial opening was 30 mm compared with trials where initial opening was 10 mm. Averaged results from the nominal "low" or "high" force groups showed no clear trend of change in digastric activity between trials recorded at different initial opening.

6. THE ACTIVITY OF THE DIGASTRIC MUSCLE AFTER UNLOADING

The burst of electrical activity which was frequently seen in the first 10 msec after the "break" was described in Section 1 of the Results and was considered to be a movement artefact. A burst of digastric activity, starting between 20 and 22 msec after the break and continuing for 15 to 70 msec, was seen in all trials. Calculation of the correlation coefficients between onset and duration of this response and the jaw opening and force at the time of the break revealed that these parameters were all independent of one another ($P>0.01$).

The cumulative integral of the digastric activity in this response increased as the breaking force increased in all averaged groups of trials in which the initial separation of the bars was 10 mm. When the bars were initially set at 20 mm, the digastric response after unloading increased with force in 8 out of the 9 subjects. When the bars were originally set at 30 mm, however, this relationship between breaking force and reflex digastric EMG activity was found in only 5 out of the 9 subjects. In the other 4 subjects, the reflex digastric activity was unchanged or reduced as the force increased.

DISCUSSION

1. VERIFICATION OF THE ACCURACY OF THE TECHNIQUE OF SURFACE ELECTRODE RECORDINGS OF THE DIGASTRIC EMG

It was found in this study, as in the study by Hannam *et al.* (1968), that the records obtained through properly-placed surface electrodes over the anterior belly of the digastric were qualitatively similar to those obtained when intra-muscular electrodes were used. Surface electrodes were convenient for recording the EMG activity of this muscle as this technique avoided the discomfort and irritation to motor units of placing wire electrodes into the muscle and gave a more representative record of the overall activity of the muscle. Flexible intra-muscular wire electrodes, on the other hand, were frequently too selective and recorded only from a relatively small population of motor units which may not have been representative of the overall digastric response.

2. IDENTIFICATION OF MOVEMENT ARTEFACTS

Movement artefacts are a common problem when EMG activity is recorded from muscles during rapid movements. This was the case in this experimental procedure where peak mandibular velocities as high as 1.5 m/sec were reached within 5 msec of unloading. With accelerations as high as 0.8 $m.sec^{-2}$, such artefacts may be indistinguishable from EMG signal by the usual criteria of amplitude, timing and bandwidth.

Considerable care was taken to minimise these artefacts with little success in the initial experiments. Recordings from the anterior belly

of the digastric proved to be paricularly susceptible to artefacts. These may have been caused by rapid acceleration and deceleration of the mandible during unloading whipping the electrode wires. Careful taping of these leads to the skin did little to reduce this effect. The strategy of using a skin mounted pre-amplifier was also ineffective in reducing movement arefacts, probably because the small but significant mass of the instrument resulted in its movement in relation to the skin and this generated EMG-like potentials during jaw movements.

In several experiments, flexible wire electrodes were placed into the anterior belly of the digastric in the expectation that these would give records with smaller artefacts, but records from these were in fact no better than those obtained with surface electrodes (Figure 5).

In the experiments of Beaudreau *et al.* (1969), a piezo-electric transducer was used to signal the instant of unloading. The output of this transducer, as shown in their Figure 6, was similar in pattern, onset and duration to the potential generated in the insulated digastric electrodes in this experiment (Figure 6). This may suggest that jarring of the head on unloading may generate a potential in surface and intra-muscular electrodes.

The strategy of recording the artefact-contaminated EMG on one channel and the artefact uncontaminated by EMG on the other proved to be successful in delineating the time course of the movement artefact. The signal from the electrode pair that was electrically insulated from the skin must have occurred as a result of the movement. It was decided that the electrical activity seen in the other pair of normal surface electrodes immediately after unloading and which corresponded in time course and

latency to this movement artefact (Figure 6), was also presumably due to movement. If this movement artefact were present in all groups of averaged records, it is possible that it may have been superimposed on true EMG activity and have masked an early digastric response. As the artefact was present on less than 25% of the groups of averaged records, it was concluded that no digastric activation occurred at a latency of less than 20 msec and that the apparent early "response" was entirely due to movement artefact.

This technique may be useful in detailing the time course of movement artefacts in other muscles during function. For large superficial muscles, two pairs of surface electrodes could be used with one pair electrically shielded from the skin. For deep muscles (e.g. lateral pterygoid) a similar result could be obtained by implanting two pairs of wire electrodes into the muscle, the tips of one pair exposed and the other pair electrically insulated from the surrounding muscle.

3. THE UNLOADING REFLEX

3a. A comparison with studies of unloading of limb muscles:

In the present study there is a similar general pattern of initial inhibition of agonists, reflex excitation of antagonists, then a rebound activity in the agonists to that described in limb muscles (Angel *et al.*, 1965; Alston *et al.*, 1967; Angel *et al.*, 1973). These workers reported that when a forearm is unloaded, the peak velocity of the arm coincides with the start of the silent period and the burst of

activity in the antagonist. It was suggested that this sequence of events decelerates the limb. In the present study, the peak mandibular velocity occurred within 5 msec of unloading which was well before both the usual onset of the agonist silent period at 10 to 15 msec, and the antagonist rebound activation at 20 msec. However, there is a major difference between flexor movements around the elbow joint compared to closing the temporomandibular joint in relation to the consequences of the movement which ensues after unloading of the contracting antagonist. When the forearm is unloaded during forceful flexion, the endpoint of an unrestrained movement is a soft tissue cushion. In the case of the mandible, the endpoint of unrestrained high-speed closing movements is the forceful occlusion of the teeth, with the potential for damage to hard and soft tissues. The pattern of reflex elevator inhibition and activation of depressors occurring after unloading is inadequate to explain the rapid deceleration of the mandible and suggests that some other mechanism is acting.

The presence of two or more silent periods, as reported by Alston *et al.* (1967) in at least 50% of unloading trials, was seen in only a few trials in this experiment. This may be due to the fact that the multiple silent periods that they reported were usually associated with the movement of the arm being mechanically blocked after unloading.

3b. A comparison with other previous studies of unloading of the mandibular elevator muscles:

The results obtained in the present study are generally consistent with those of previous authors (Hannam *et al.*, 1968; Beaudreau *et al.*, 1969; Gill, 1970; Luschei and Goodwin, 1974; Lamarre and Lund, 1975),

in that the elevator EMG activity showed a silent period after unloading with a return of activity at a varying latency. A burst of activity was also seen in the digastric muscle after the start of the elevator silent period. In this study, the latency of elevator inhibition was usually 10 to 15 msec, being as long as 20 msec in some averaged groups, whereas that reported by Lamarre and Lund (1975) was 6.5 to 11 msec and by Hannam *et al.* (1968) at 20 msec. The latency of the burst of activity in the digastric muscles in this study was 20 to 22 msec whereas the previous authors reported mean latencies of 27 and 20 msec respectively. Although a similar sequence of elevator and depressor muscle activity is reported by Beaudreau *et al.* (1969), the latencies of the muscle activities set out in their table are five times as great as those reported in this and the previously-mentioned experiments. The fact that their latency values are exactly five times those of other investigators suggests that they may have inadvertently misread their time calibrations.

None of the subjects in this study showed the digastric acting as an "agonist" before unloading as Beaudreau *et al.* (1969) reported in 5 out of their 13 subjects. This may be explained by the fact that in their experimental procedure, the subject had to brace the head and neck muscles to support the weight prior to unloading. In this present experiment, subjects were applying force to bars that were fixed in space.

The coactivation of digastric and elevator muscles seen in the present experiment before the break, especially when the initial opening was small and the applied force was large, is also evident in the records presented by Hannam *et al.* (1968).

The differences in the latencies of elevator inhibition and digastric activation between this study and that of Lamarre and Lund (1975) may be due to the fact that the extremely rapid reduction of force exerted on the bar when the glass breaks gives a very accurate point from which to measure these latencies. If the system used in the present experiment is able to signal the moment of unloading more rapidly than that in the experiment of Lamarre and Lund, the latency of the elevator inhibition and digastric activation in the present experiment would be greater than that determined by Lamarre and Lund. However, the latency of digastric activation was shorter in this experiment than that determined by Lamarre and Lund. The timing differences between these experiments may be related to differences in techniques of determining the onset or cessation of muscular activity.

The difference between the reported latency of elevator inhibition of 20 msec by Hannam *et al.* (1968) and the usual latency of 10 to 15 msec seen in this study may be explained by the fact that Hannam *et al.* determined their latencies from raw data and stated that they found difficulty in determining accurately the points on the EMG records at which a change in activity occurred. The technique of rectifying and averaging data, as used by Lamarre and Lund and in this experiment, would be expected to give a more reliable means of determining the mean latencies of changes in EMG patterns.

The duration of the elevator silent period following unloading in the present study varied from 30 msec to over 100 msec, continuing past the end of the sampling period. The "second" phase of depression of elevator activity at a latency of between 27 and 36 msec after unloading,

as found by Lamarre and Lund (1975), was only seen in 4 out of a total of 81 groups of averaged records. The "third" phase of elevator depression at a latency of 60 and 70 msec after unloading was not seen in this study but, on occasion, there was a period of depression of elevator EMG activity at about 100 msec after unloading. These differences in elevator activity are presumably due to differences in experimental protocol. Subjects in this study expected rapid and complete unloading once the resistance between their teeth was overcome. However, in the study of Lamarre and Lund, the subjects did not expect that complete closure would be allowed. When the torque pulse was larger than the inertial load and complete unloading did occur, they also reported that the elevator EMG activity remained depressed throughout the period of unloading.

3c. Neural control of agonist inhibition during the unloading response of the jaw muscles:

The latency of this agonist inhibition has been reported by Lamarre and Lund (1975) to be as short as 6.5 msec which indicates that this must be a segmental response. If muscle spindles are excluded from triggering this response, consideration must be given to other possible sources of the trigger signal e.g., receptors in the antagonist muscle, periodontal ligament, temporomandibular joint, the skin of the face or unknown vibration-sensitive receptors (e.g. Pacinian corpuscles) which are activated by the jarring of the skull that is known to occur at the instant of unloading (Beaudreau *et al.*, 1969).

Goodwin and Luschei (1974) suggest that some mechanism other than unloading of agonist spindles is responsible for this phenomenon since

it persisted after destruction of cell bodies of these spindle afferents in the mesencephalic nuclei. Repetition of their experiment with a more accurate measurement of the exact moment of unloading may resolve the doubt expressed by Lamarre and Lund (1975) that the inhibition of the elevators that remained after this lesion may not have been at a monosynaptic latency and may have been the later, second phase of inhibition that Lamarre and Lund reported. This suggests that destruction of spindle afferents may have eliminated the usual monosynaptic inhibition and left intact the later inhibitory period which is possibly mediated by other receptors.

The work of Angel *et al.* (1973) on forearm muscles suggests that antagonist stretch does not initiate agonist inhibition as the agonist silent period persisted after anaesthesia of the antagonist nerves. In the jaw opening muscles, the apparent absence or scarcity of stretch receptors also argues against an antagonist-mediated reflex (Cooper, 1960; Lennartsson, 1979). The role of mechanoreceptors in the periodontal ligament appears to be unimportant in mediating this response as it was present in edentulous patients (Gill, 1970) and it persisted after the teeth were anaesthetised (Lamarre and Lund, 1975).

Afferents from the temporo-mandibular joint providing proprioceptive information regarding the relative position of the joint (Thilander, 1961) and the speed and direction of mandibular movement are candidates for the signal which triggers this response. One piece of evidence which supports this possibility has been adduced by Kawamura and Majima (1964), who reported that, in anaesthetized cats, condylar rotation in the jaw-closing direction causes a rapidly-acting response leading to ipsilateral inhibition of jaw elevator muscles.

Appenteng *et al.* (1981) have presented evidence that circumoral hair follicle afferents in rabbits can signal velocity during all phases of chewing. This information may be especially important during jaw closure as the antagonist jaw opening muscles contain few, if any, muscle spindles (Cooper, 1960; Lennartsson, 1979). Hence, it is also possible that this type of receptor may have a role in inhibiting elevator activity on unloading, although there is no information concerning their influence on jaw-closing motor neurones.

It has been shown that a silent period may be generated in the contracting elevator muscles by tapping the skull (Matthews, 1975). The silent period on unloading may be due to a "jar" reflex mediated through elevator muscle spindles, which are known to be sensitive to vibration, but it would be expected that this response would cease if spindle afferent neurones were destroyed. If this response were due to "jarring" of unknown vibration sensitive receptors, it would presumably be unchanged by destruction of spindle afferent cell bodies.

3d. Neural control of the re-excitation of agonist muscles after the silent period:

In 5 out of 9 subjects, the inhibition of the jaw elevators after unloading was followed by a rebound excitation of these muscles before the end of the sampling period. Several mechanisms have been proposed to control this return of agonist activity. Alston *et al.* (1967) discussed the hypothesis that alpha motor neurone silence on unloading would be followed at a set latency by Renshaw cell silence, allowing re-excitation of the alpha motor neurones. Their results did not support this hypothesis since the return of agonist activity did not

occur at a set latency and could be changed by altering the amount of limb movement permitted after unloading. They also considered that unloading of the agonist tendon organs was not important in causing re-excitation as the volley persisted when these receptors were "re-loaded" by a wire restraining the movement of the arm after unloading. Angel and Lewitt (1978) suggested that there is a common mechanism controlling the contraction on passive shortening of a muscle and the contraction ending the silent period after unloading. They suggested that this activity may be taking up the slack in the muscle to prepare it for efficient contraction at the new shorter length.

A mechanism for this type of response has been considered by Angel *et al.* (1973). Their fusimotor reflex theory proposes that there is gamma motor activity during the silent period setting the fusimotor system to the shortened muscle length and that there is an after volley through the 1a reflex loop once the intrafusal fibres become shorter than the extrafusal fibres. They found support for this hypothesis in that re-excitation occurred at the latency of monosynaptic stretch reflex when a wire restrained the arm after unloading. This hypothesis would also be consistent with the fact that in the present study, the return of elevator activity always occurred after the closing movement of the mandible had been arrested and shortening of the elevator muscles had ceased.

The work of Angel *et al.* (1973) in anaesthetising the antagonist nerve supply during unloading, suggests that antagonist stretch does not play a part in the return of activity. They discussed the possibility that the gamma activity which is inhibited by recurrent impulses from the alphas during voluntary movement may return when alpha silence removes

this inhibition. They also considered the possibility that, if stretching of a muscle causes inhibition of gamma motor neurones, shortening may cause disinhibition. These motor neurones may increase their firing as the muscle shortens, leading to an after-volley through the reflex loop. This would bring the muscle to a resting level of activity at the new shortened length.

It is not known whether there is a set latency at which this volley occurs if jaw movement is arrested soon after unloading, as is the case in the forearm (Angel *et al.*, 1965). The hypothesis of the fusimotor reflex would be strengthened if this volley occurred at a monosynaptic latency for the jaw elevator muscles but this would not confirm that the spindle 1a afferents were controlling this reflex.

The work of Goodwin and Luschei (1974) suggests that spindle afferents are not important in signalling the start of the silent period but present no data pertinent to the question of whether they affect the latency of the return of agonist activity.

The pattern of the return of the elevator activity was characteristic for each subject in the present study, even when the size of the force overcome or the initial jaw position was altered. This may be due to the fact that each individual may have a characteristic way of positioning his teeth and applying force to the bar, depending on his previous experience and the amount of confidence with which he approached the task. There may be a segmental reflex common to all individuals that initiates the return of elevator activity after unloading. The latency at which this is activated may depend on an individuals biting strategy and therefore, the movement which ensues after unloading. The effect

of higher centres in controlling or modifying this response must also be considered.

3e. The neural control of the burst of activity in the antagonist muscle following unloading:

The independence of the latency of onset of this burst of activity in the digastric to changes in initial opening and force at the time of the break suggests that the response is reflex in nature. Its long latency in comparison to the monosynaptic response in the elevator muscles suggests the possibility that a polysynaptic, possibly suprasegmental, pathway is involved. Many receptors must be considered as being possible triggers for this reflex.

The reported scarcity of muscles spindles in the anterior belly of the digastric (Cooper, 1960; Lennartsson, 1979) and the absence of a monosynaptic stretch reflex in cats (Blom, 1960) make it unlikely that this is a digastric stretch response. It is not known whether other infra- and suprahyoid muscle groups have spindles and whether they would be capable of mediating a stretch reflex. Lamarre and Lund (1975) report that anaesthesia of the periodontal ligament does not change the digastric reflex following unloading.

Removal of reciprocal inhibition of the digastric as the elevators are silenced, or the fact that the agonists are being shortened, could be responsible for this antagonist activity. It has been reported by Kuwamura and Majima (1964) that temporomandibular joint receptors are involved in digastric activation during condylar rotation in the closing direction. Appenteng *et al.* (1981) reported that facial hair follicle

afferents have the ability to signal velocity during jaw opening and closing. All these receptors must therefore be considered as possible triggers for this digastric reflex but the latency of digastric activation is longer than would be expected by a mono- or disynaptic segmental reflex.

Lund and Lamarre (1974a) suggested that the large majority of neurones in the cortical masticatory area are concerned with activation of jaw opening muscles with the concurrent inhibition of jaw closers. These neurones may participate in a short-latency cortical-loop response, functional only during unloading of jaw closing muscles, and responsible for the "second" phase of masseteric inhibition and concurrent digastric excitation.

4. THE MECHANISM RESPONSIBLE FOR DECELERATION OF THE MANDIBLE FOLLOWING UNLOADING

The fact that the onset of deceleration of the closing movement of the mandible begins within 5 msec of unloading of the jaw closing muscles, while indeed the elevator muscles are still active, suggests that a braking system must be acting with a shorter latency of onset than would be possible with a reflexly-induced load compensation mechanism.

It would be expected that as the amount of force required to overcome a resistance between the teeth increased, the amount of acceleration given to the mandible after the "break" would also be increased, as would the distance travelled before the closing movement was arrested. No correlation was found between the distance travelled

after the break and the force at the time of the break, whereas there was a high correlation ($r = 0.87$) between the distance travelled and the initial opening (Figure 7). This suggests that some protective mechanism which opposed the forthcoming closing movement was operating before the time of the break. As the timing of the breakage of the glass rod was unpredictable, this system must have been ready at all times during the build up of force to limit the acceleration of the mandible so that its movement was arrested before damage to the teeth and soft tissues occurred. For this mechanism to be so precise in opposing a varying range of forces and initial openings, there would be a need for receptors to be accurately signalling the relationship of the mandible to the maxilla and the amount and rate of increase of force being applied to the bars.

Grillner (1972) reported that skeletal muscles are more resistent to elongation, i.e., they are stiffer, when they are contracting than when relaxed, probably because the cross bridges between myofilaments must be distorted if a contracting muscle is to be stretched (Rack and Westbury, 1974). Grillner showed that muscle stiffness is important where a load compensation mechanism is needed in a situation where there is not enough time for such compensation to be provided by a proprioceptive reflex route.

The possibility must be therefore considered that the principal mechanism which limited the closing movement of the mandible after unloading is a pre-set increase in the stiffness of the digastric muscle which anticipated the onset of the closing movement. Contraction of the digastric muscle began early in the phase of isometric contraction of the jaw closing muscles and continued at least until the reflex

burst of digastric activity. The short sampling period of the averaged data before the break does not show the start of this increase in digastric activity. However, in this experimental paradigm, examination of raw data reveals that the amplitude of digastric activation during the period of isometric elevator contraction was always at least 200% of its amplitude during normal, unopposed closure (e.g., Figure 2).

Thus, by being activated before the movement began, the stiffened digastric muscle was in a state of preparedness at all times during elevator contraction to resist the forthcoming jaw movement. It could be argued that co-contraction of opposing muscles in a movement of this type is inefficient in that the tension developed by the antagonist must reduce the net force exerted by the agonist. *However, the important point is not the amount tension which is developed by the antagonist muscle, but the stiffness with which it will oppose elongation.* In fact, the amplitude of the digastric EMG during the phase of co-contraction was relatively low in comparison to its amplitude during a normal opening movement (Figure 2), which suggests that the active force which it exerted against the jaw closing muscles was small. Nevertheless, it is known that the stiffness of contracting muscles is increased significantly even at low stimulation rates (Rack and Westbury, 1969; Grillner, 1972), so it is probably feasible that even a small increase in digastric activity could increase its stiffness sufficiently to account for the rapid deceleration of the mandibular closing movement which occurred in the first 5 msec after the break.

Further evidence that a pre-programmed mechanism was operating comes from the fact that in 8 out of 9 subjects, when the initial opening of the bars was 10 mm, the cumulative integral of averaged digastric EMG

activity over the pre-break period increased as larger resistances between the teeth were overcome. In trials where the need to rapidly decelerate the mandible is reduced because of wider initial separation of the teeth at the time of the break, this protective mechanism seems to be less constant. When the amount of digastric activity prior to the break was studied in the nominal "high" force group, the digastric activity was unchanged or reduced where the separation of the teeth at the time of the break was greater. No consistent pattern of change in digastric activity was seen with changes in initial openings in the nominal "medium" or "low" force groups.

The nature of the signal which causes the anticipatory stiffness in the digastric is not known, but it could be expected that learning from past experience would be important in reinforcing the mechanism. It may be possible that an individual may only show this co-activation of digastric and elevator muscles when he anticipates that he is biting on a brittle object, and not when his experience tells him that the object between the teeth is unlikely to shatter. Digastric activity while biting on an ice block, for example, may be much greater than that seen when biting on a wooden peg. This could be tested experimentally by directing subjects to steadily increase the force on the bars of an apparatus similar to that used in this study. The digastric activity in trials where the breakage of a glass rod caused unloading of the elevator muscles could be compared with trials where the subject was aware that the glass rod had been replaced by a metal one that would not fracture.

Mechanoreceptors that can signal rapid changes in force may be involved in this protective response, as subjects in attempting to overcome the resistance between their teeth often built up forces of

over 300 N on the bars in less than 20 msec. Periodontal mechanoreceptors may be involved in signalling the force being applied to the bars. Lamarre and Lund (1975) reported that anaesthesia of the teeth did not change the muscle reflex patterns after unloading but it is not known if it changed the muscle activity before unloading, i.e., the anticipatory stiffness and hence mandibular movement where complete unloading occurred. Many other receptors in the masticatory system for example, joint receptors, tendon organs and muscle receptors may contribute to the final signal of the amount and rate of increase in force. As grimacing was often seen as subjects attempted to bite through the resistance between their teeth, skin and hair follicle receptors may also be important in signalling the increasing elevator activity.

CONCLUSIONS

1. The distance travelled by the mandible before its upward velocity was arrested was closely related to the initial separation of the teeth and independent of the force at the time of unloading.

2. The general pattern of activity seen in the jaw muscle EMGs after unloading in this study was similar to that described in earlier reports, i.e. inhibition of agonist muscles closely followed by excitation of antagonist muscles. In some subjects, the agonist muscles were re-activated after a silent period of characteristic latency for that subject.

3. The inhibition of elevator muscles and the activation of the digastric muscle was unaffected by changes in the force and separation of the teeth at the time of unloading and was as a result of a reflex mechanism of which the neural pathways are not known.

4. This reflex activation of antagonist muscles and inhibition of agonist muscles occurred too slowly to account for the very rapid initial deceleration of the mandibular closing movement.

5. The digastric muscle was always active before the onset of the closing movement i.e. co-contraction of agonist and antagonist always occurred during the phase of isometric agonist contraction.

6. It was concluded that this pre-programmed activation of the digastric muscle resulted in an increased stiffness of the muscle before the onset of the closing movements, and furthermore that it was this

increased stiffness in the antagonist muscle which was principally responsible for the initial restraint of the upward movement of the jaw.

7. It is concluded that there are two types of mechanisms limiting movement around a joint after unloading. Firstly there is reflexly induced load compensation as a result of agonist inhibition and antagonist activation after unloading; a second more rapid mechanism results from increased antagonist stiffness in anticipation of unloading. Learning from past experience of the end result of uncontrolled movement in a given unloading event would determine whether this second mechanism would be activated to rapidly limit movement.

BIBLIOGRAPHY

ALSTON, W., ANGEL, R. W., FINK, F. S. and HOFMANN, W. W. (1967). Motor activity following the silent period in human muscle. *J. Physiol. (Lond.)*, 190, 189-202.

ANDREWS, C. J. and BURKE, D. (1972). A quantitative study of the effect of L-dopa and phenxoybenzamine on the rigidity of Parkinson's disease. *J. Neurol. Neurosurg. Psychiat.*, 36, 321-328.

ANDREWS, C. J., BURKE, D. and LANCE, J. W. (1972). The response to muscle stretch and shortening in Parkinsonism ridigity. *Brain*, 95, 795-812.

ANDREWS, C. J., NEILSON, P. D. and LANCE, J. W. (1973). Comparison of stretch reflexes and shortening reactions in activated normal subjects with those in Parkinson's disease. *J. Neurol. Neurosurg. Psychiat.*, 36, 329-333.

ANGEL, R. W., EPPLER, W. and IANNONE, A. (1965). Silent period produced by unloading of muscle during voluntary contraction. *J. Physiol. (Lond.)*, 180, 864-870.

ANGEL, R. W., GARLAND, H. and MOORE, W. (1973). Unloading reflex during blockade of antagonist muscle nerves. *Electroenceph. clin. Neurophysiol.*, 34, 303-307.

ANGEL, R. W. and LEWITT, P. A. (1978). Unloading and shortening reactions in Parkinson's disease. *J. Neurol. Neurosurg. Psychiat.*, 41, 919-923.

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APPENTENG, K., LUND, J. P. and SEGUIN, J. J. (1981). The behavior of cutaneous afferent neurons recorded in the mandibular division of the Gasserian ganglion of the rabbit during movements of the lower jaw. *Personal Communication.*

BASMAJIAN, J. V. (1967). Muscles Alive: Their Functions Revealed by Electromyography. 2nd Edition, Chapter 4, Baltimore, Williams and Wilkins.

BEAUDREAU, D. E., DAUGHERTY, W. F., Jr. and MASLAND, W. S. (1969). Two types of motor pause in masticatory muscles. *Amer. J. Physiol.*, 216, 16-21.

BLOM, S. (1960). Afferent influences on tongue muscle activity. A morphological and physiological study in the cat. *Acta physiol. scand.*, 49, Suppl. 170, 1-97.

BURKE, D., ANDREWS, C. and GILLIES, J. D. (1971). The reflex response to sinusoidal stretching in spastic man. *Brain*, 94, 455-470.

COOPER, S. (1960). Muscle spindles and other muscle receptors. In: The Structure and Function of Muscle. (G. H. Bourne, Ed.). Vol. 1, Academic, New York, 381-420.

GILL, H. I. (1970). Unloading reflex of the jaw muscles. *J. Dent. Res.*, 49, 649.

GOODWIN, G. M. and LUSCHEI, E. S. (1974). Effects of destroying spindle afferents from jaw muscles on mastication in monkeys. *J. Neurophysiol.*, 37, 967-981.

GRILLNER, S. (1972). The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors. *Acta physiol. scand.*, 86, 92-108.

HANNAM, A. G., MATTHEWS, B. and YEMM, R. (1968). The unloading reflex in masticatory muscles of man. *Archs oral Biol.*, 13, 361-364.

HANSEN, K. and HOFFMAN, P. (1922). Weitere Untersuchungen über die Bedeutung der Eigen-reflexe für unsere Bewegungen. I. Anspannungs- und Entspannungsreflex. *Z. Biol.*, 75, 293-304.

KAWAMURA, Y. and MAJIMA, T. (1964). Temporomandibular - joint's sensory mechanisms controlling activities of the jaw muscles. *J. Dent. Res.*, 43, 150.

LAMARRE, Y. and LUND, J. P. (1975). Load compensation in human masseter muscles. *J. Physiol. (Lond.)*, 253, 21-25.

LENNARTSSON, B. (1979). Muscle spindles in the human anterior digastric muscle. *Acta odontol. scand.*, 37, 329-333.

LIPPOLD, O. C. J. (1953). The relation between integrated action potentials in a human muscle and its isometric tension. *J. Physiol. (Lond.)*, 177, 492-499.

LUND, J. P. and LAMARRE, Y. (1974a). Activity of neurones in the lower precentral cortex during voluntary and rythmical jaw movements in the monkey. *Expl. Brain Res.*, 19, 282-299.

LUSCHEI, E. S. and GOODWIN, G. M. (1974). Patterns of mandibular movement and jaw muscle activity during mastication in monkeys. *J. Neurophysiol.*, 37, 954-966.

MARSDEN, C. D., MERTON, P. A. and MORTON, H. B. (1972). Servo action in human voluntary movements. *Nature*, 238, 140-143.

MATTHEWS, B. (1975). Mastication. In: Applied Physiology of the Mouth. (C. Lavelle, Ed.). Bristol, Wright, Chapter 10, 199-242.

MATTHEWS, P. B. C. (1964). Muscle spindles and their control. *Physiol. Rev.*, 44, 219-288.

MERTON, P. A. (1953). Speculations on the servo control of movement. In: The Spinal Cord. (G. E. W. Wolstenholme, Ed.). London, Churchill, 247-255.

MILBERG, D. J., GARTHWAITE, C. R. and LUSCHEI, E. S. (1980). Instrumentation and procedures for quantitative study of human mandibular movement. *Personal Communication.*

PRUIM, G. J., TEN BOSCH, J. J. and DE JONGH, H. J. (1978). Jaw muscles EMG-activity and static loading of the mandible. *J. Biomechanics*, 11, 389-395.

RACK, P. M. H. and WESTBURY, D. R. (1969). The effects of length and stimulus rate on tension in the isometric cat soleus muscle. *J. Physiol. (Lond.)*, 204, 443-460.

RACK, P. M. H. and WESTBURY, D. R. (1974). The short range stiffness of active mammalian muscle and its effect on mechanical properties. *J. Physiol. (Lond.)*, 240, 331-350.

THILANDER, B. (1961). Innervation of the temporo-mandibular joint capsule in man. *Trans. Roy. Sch. Dent. Stockholm and Umea*, No. 7, 9-67.

APPENDIX

1. FRACTURE OF GLASS ROD

In preliminary experiments, it was found that the force required to fracture the glass rod placed in any given hole varied unpredictably by up to a factor of three from one trial to another. This was presumably due to the non-uniform properties of glass. Accordingly, the force at the time of the break was effectively random and it was not possible for a subject to predict the breaking force from one trial to another.

It was also noted by subjects and experimenter that the rod occasionally fractured in two stages, causing two steps in the descending limb of the strain record. These two-stage breaks were readily identified by examination of the strain records of each trial, and were not used in any subsequent analysis.

2. TAPE RECORDER

The tape speed was 7½ inches per second. The anterior and middle temporalis muscles were recorded on direct channels (bandwidth 50 Hz to 32 kHz). The masseter and digastric muscles, force and movement were recorded on frequency modulated channels (bandwidth 0 to 2.5 kHz).

3. FILTERING AND ALIASING

The high pass setting of the EMG filter was empirically set at

200 Hz since this was the level which best reduced the amplitude of the troublesome movement artifacts during unloading. It is acknowledged that this setting excluded a significant part of the EMG power spectrum. However, this unfavourable bandwidth would have had a minimal effect on the accuracy of measurement of the timing of the EMG events recorded after the break, which was an important objective of the study.

I accept Dr Krantz's point regarding the low pass filter setting. It is acknowledged that, with a computer sampling rate of 2 kHz per channel, there is a possibility that "aliasing" can occur. However, any adverse effects introduced by aliasing would have been minimised by the subsequent averaging procedure. It would not, however, have effected the accuracy of the measurements of the timing of the EMG signals, but may have affected their amplitudes. Accordingly, amplitude changes were reported only as trends and not as quantitative changes.

4. CORRELATION COEFFICIENTS

Correlation coefficients and levels of significance were calculated to determine the relationship between mandibular movement and force and initial openings. These relationships are presented visually as scatter diagrams. Because of the low correlation coefficients found for all relationships other than that shown in Figure 7, the regression lines were not included.

5. THE DECELERATION OF THE MANDIBLE

It is known that the tension in muscles decreases rapidly when they

shorten. The question is, can this rapid decrease in tension in the jaw muscles as they shorten after the break account for the deceleration of the upward movement of the mandible? The extreme hypothetical case would be the situation in which the jaw was given a short pulse of upward acceleration after the break by the tension in the jaw closing muscles, after which the tension in these muscles would fall instantaneously to zero. In this circumstance, the upwardly-moving jaw would be decelerated at a constant rate by gravity. However, it is clear from Figure 3 that the deceleration was highly non-linear. Indeed, 50% of the deceleration occurred within the first 3 msec after the break, after which the velocity fell rather slowly to zero. This pattern is far more consistent with the proposal that the mandible moved upwards after the break until its progress was checked by a relatively inelastic, soft tissue restraint (the "short-range stiffness" of the contracting digastric muscle) which abruptly decelerated it.

6. DETERMINATION OF THE TIMING OF MUSCULAR EVENTS

The timing of muscular events was determined by extrapolation of the rising or falling edge of the averaged EMG signal to the baseline level. It is accepted that this measurement contains two sources of error. Firstly, averaged records tend to give falsely high values for latencies since the most accurate value for latency of, e.g. masseter inhibition, would be the minimum latency determined from a series of single sweeps rather than the mean latency determined with the present procedure. However, measurements of latencies and durations from single EMG records is also an imprecise procedure.

The second source of error relates to the empirical criteria used

to determine at which point the averaged signals exceeded or returned to their baseline levels. Statistical methods for assigning limits of error for this procedure have been described but were not available to the author for use in these analyses. The maximum error of the latency measurements made is estimated to be ± 3 msec.

7. PUBLICATIONS ARISING FROM WORK DONE FOR THIS THESIS

1. MILES, T. S. and WILKINSON, T. M. (1982). Limitation of jaw movement by antagonist muscle stiffness during unloading of human jaw closing muscles. *Exp. Brain Res.*, 46, 305-310.

2. MILES, T. S., BROWNE, E. and WILKINSON, T. M. (1982). Identification of movement artefacts in electromyograph recordings. *EMG clin. Neurophysiol.*, 22, 245-249.

3. MILES, T.S., WILKINSON, T. M. and BROWNE, E. (1981). The response in the human masticatory system to the sudden unloading of the jaw closing muscles. *Proc. Aust. Physiol. Pharmacol. Soc.*, 12, 198P.

4. MILES, T. S., WILKINSON, T. M. and BROWNE, E. (1982). The response to unloading in the human masticatory muscles. *J. Dent. Res.*, 61, 849P.