ANTERIOR DIGASTRIC MUSCLE RESPONSES TO SUDDEN UNLOADING OF THE MANDIBULAR ELEVATOR MUSCLES

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Iraklis C. Karkazis

DDS, Dr Dent (Athens)

Submitted in partial fulfillment of the Degree of
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Abstract

It is well documented that when the mandible is suddenly unloaded,as happens when a nut-shell is cracked between the teeth,a sudden mandibular deceleration prevents the teeth snapping together.

This reaction of the muscles known as "**unloading reflex**" is generally associated with reflex inhibition of the masseter and temporalis muscles and a post-collapse activation of the anterior digastric muscle (digastric burst).

A pre-collapse digastric activation has also been reported.

The aims of this study were a) to investigate the occurrence of the precollapse digastric activity b) the implication of some factors (past experience-foreknowledge-immediate past performance) on the development of this motor response and c)to investigate the latency and the possible effect of foreknowledge on the digastric burst.

For that purpose 10 young adult volunteers participated in a series of three sets of experiments.A properly designed apparatus was also used to simulate the physiological situation of cracking a nutshell between the teeth.

Nearly consistent coactivation of the digastric muscle was found in half of the subjects.

Evidence also has been provided that this digastric coactivation,is subjected to some form of central control.

From the three factors studied in relation to the precollapse

digastric activity, immediate past performance seems to be the potentially responsible factor for the development of certain strategies of coactivation.

Digastric burst was occasionally recorded in the experiments (59%). The results of the study for the latency of the digastric burst (32.7±9.5 msec) indicated that a polysynaptic pathway may be involved. However no evidence has been provided to suggest some form of relationship between the foreknowledge and this latency.

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1.1.Introduction

It is well documented from clinical observations that when the mandible is suddenly unloaded,as it happens when a nut-shell is cracked between the teeth,a sudden deceleration of the mandible prevents the teeth of snapping together.
The reaction of the muscles to the sudden unloading is known as "unloading reflex" and was first demonstrated in the masticatory muscles by Hannam,Matthews and Yemm in 1968.
The unloading reflex is generally associated with reflex inhibition of the masseter and temporalis muscles and post collapse activation of the digastric muscle.
Miles and Wilkinson (1982) however were the first who pointed out the important role of the precollapse digastric activation in the rapid arrest of the upward movement of the mandible.
Although some evidence has been provided that the triggering signal which causes this digastric coactivation is of central origin much information is still required about:
a.the modifiable nature of this coactivation and
b.the potential factors which may contribute to the development of this activity.
It is also not clear whether the digastric burst with its remarkable latency has a central or peripheral origin.

1.2.Aims

Therefore the aims of this study were:

1.To investigate the occurrence of the precollapse digastric activity during the sudden unloading of the mandibular elevator muscles.

2.To investigate the possible influence of the previous experience,the foreknowledge and the nature of the current task on the development and modification of the digastric motor response.

3.To investigate the latency of the digastric burst and the possible effect of the foreknowledge of the task to be executed on this latency.

CHAPTER 2.REVIEW OF THE LITERATURE

JAW MUSCLE REFLEXES

2.1 Introduction

Masticatory movements involve exceedingly complex but highly coordinated neuromuscular events.

These masticatory movements require the coordinated activity of several groups of muscles attached primarily to the jaws,and are partly generated from brain stem centres.

On the other hand normal masticatory function is conditioned by a system of neuromuscular reflexes.These reflexes can be simply characterized as input-output phenomena,that are not under consious control.

The input of the reflex is the information given to the central nervous system from the periphery by various receptors(periodontal,orofacial,TMJ,muscular)while the output is the continuous adjustments of the functional muscular patterns by the central nervous system,according to this information.(Mongini 1984)

Although the possibility of modifying effects from higher brain centres can not be discounted,there is still little information about the exact influence of these centres on the various elements of the reflex pathways.(Sessle 1981,Lavelle 1988).The jaw reflexes have been extensively studied in both human subjects and experimental animals and the information from the relevant studies is considered to be potentially

important from the clinical view-point,in providing a clearer understanding of normal and abnormal jaw movement patterns and maxillomandibular relationships.(Taylor 1976,Matthews 1976)

Although the matter still remains controversial the existence of the jaw reflexes suggests that neural connections could provide:

a)length servomechanisms for jaw movement control

b)positive feedback mechanisms to reinforce elevator muscular contraction forces when the teeth contact a bolus and c)protective mechanisms which limit the maximum forces developed on the teeth and mucous membrane.(Lavelle 1988)

All these could also be proved potentially important information upon which to base TMJ treatment (De Laat 1985)

2.2 The jaw-closing reflex (stretch or jaw-jerk reflex)

The simple jaw-closing reflex is elicited by stretching the muscle spindles in the jaw closing muscles.

Extensive studies in this field (Harrison and Corbin 1942,Szentagothai 1948,McIntyre and Robinson 1959,Hugelin and Bonvallet 1956) have shown that the jaw closing reflex is a monosynaptic reflex mediated through the mesencephalic nucleus of the trigeminal nerve and the motor nucleus of the same nerve.(Fig. 1)

The term jaw-jerk reffers to the similarity of this reflex to the monosynaptic tendon jerk seen in the limp muscles(knee-jerk)

It is generally believed that one function of the stretch reflex seen in the body,is to compensate for an increase of load on the muscles(Angel and Lewitt 1978).

In the area of the stomatognathic system the jaw closing reflex is considered to be one of the main factorsresponsible for the postural position of the mandible(Dubner ,Sessle and Storey 1978),although tissue elasticity might be of similar importance(Feigenblum 1966,Yemm 1976).

Tapping on the chin is the most convenient way to produce the reflex and can be done under three experimental situations

a)with the subject relaxed

The principal response to the sudden mandibular depression is the contraction of the masseter and temporal muscles with 6-8 msec. latency period between the stimulus and the closing

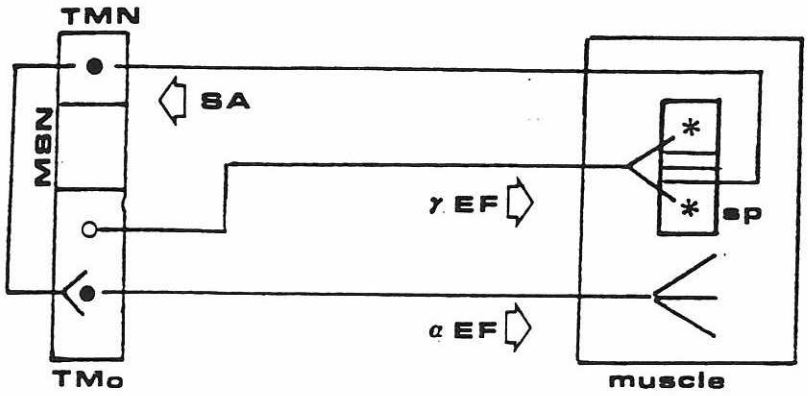

Fig. 1. Jaw-closing reflex pathway.

TMN Trigeminal mesencephalic nucleus.
MSN Trigeminal main sensory nucleus.
TMo Trigeminal motor nucleus.
sp Muscle spindle.
SA Spindle afferent.
γEF Gamma efferent.
αEF Alpha efferent.

movement.Neither digastric nor medial pterygoid muscles seem to react in a similar manner. (Lamarre and Lund 1975)

b)with the subject contracting his elevator muscles.

If the experiment is repeated with the subject biting on a hard object there is a greater increase in reflex masseter muscular activity.(Goldberg 1972,Hannam 1972).

This initial response is normally followed by a period of decreased activity,the "silent period" after which the muscle returns to its previous level of activity.

Records extending over longer periods quite often provide evidence of further cycles of increased and decreased muscular activity (double silent period) before the mandible stabilizes to the preexperimental level of activity.

The establishment however of the exact duration and latency of the double (or more) silent periods is still controversial.Although some authors state that the bite force exerted by the patient does not influence the reflex phenomenon(Palla,Bailey,Grassl and Ash 1981) others present an inverse relationship between the silent period duration and the bite force.(Dubner et al 1978,Bernstein et al 1981,Yu et al 1973,Grimm and Hunt 1982).

Moreover there is little disagreement on the possible influe nce of the strength of the tap on the silent period duration. Some studies report that the strength of the tap does not influence the silent period duration (Bessete et al 1973, Baily,McCall et al 1977

,Palla et al 1981)while others conclude that the silent period is longer for stronger taps and that the single type silent periods tend to be replaced by double and merged ones(Kroon and Naeije 1984).

It is obvious therefore that the jaw jerk reflex following mechanical chin tap exhibit highly variable characteristics. The lack of a well standardized system for delivering mechanical stimuli considers to be the main factor for this variability.On the other hand if reflex responses are to be used as possible diagnostic and prognostic tools in the managment of muscle joint problems (Bessete et al 1971,Widmalm 1976,McCall et al 1978,Skiba and Laskin 1981) a well standardized system is necessary in order to allow valid comparisons with other data.

In 1984 Murray and Klineberg developed a standardized reproducible system (a solenoid driven plunger system) especially for mechanically jaw reflex responses. According to their results a)the produced stimuli was standardized and reproducible during delivery and b)on the basis of this reproducibility the instrument could used as a more precise aid in the diagnosis and managment of muscle joint pain dysfunction.

With the subject clenching his teeth

A similar experimental situation is to produce the reflex while the subject is clenching his teeth without anything

between them.In this case although the silent period is still present there is a reduced jaw jerk reflex(Hufschmidt and Spuler 1962).Monosynaptic reflex inhibition due to periodontal mechanoreceptors is a possible explanation for this response(Goldberg 1972).

c)with the subject contracting his depressor muscles .

If the experiment is repeated with the subject contracting his depressor muscles against resistance (an object is placed under the mandible to prevent mouth opening) a reduction in the digastric activity and a smaller response of the masseter muscle can be observed(Matthews 1976).

As a matter of fact the jaw jerk reflex is often reinforced during voluntary contraction of other muscles groups as happens with the monosynaptic tendon jerk seen in the limb muscles.(Hannam 1972)

If the chin tap is applied from underneath,no response in either the elevator or depressor muscles can be detected(there is no reverse jaw closing reflex)(Sessle 1981) These findings coupled with results from histological studies suggest that the jaw depressor muscles do not contain spindles (Blom 1960,Bossy 1959,Voss 1956) .

Although the chin tap as mentioned above is one of the more popular experimental ways to elicit the jaw jerk it is believed (Sessle 1981,Desmedt 1975,Hagbarth et al 1976)that the produced brief phasic reflex rarely occurs in normal

function.On the contrary a tonic reflex activity is more likely to occur.On the basis of this idea a masseteric tonic vibration has also been used to induce a gradually developed tonic activity(the tonic activity reflex,TVR,Hagbarth,1973). TVR was also reported in jaw depressor muscles (Hellsing,1977).

Finally the electrical stimulation of the trigeminal mesencephalic nucleus,can also evoke the jaw closing reflex. From the clinical point of view the jaw jerk has long served as a neurological test for the integrity of the trigeminal nerve (Gilroy and Holiday,1982)

It has been used in patients with multiple sclerosis (Goodwill and O Tuama,1969,Yates and Brown,1981)trigeminal neuralgia (Ongerboer de Visser and Goor,1974) and midbrain or pontine lesions (Ongerboer de Visser and Goor,1976). Administration of L-Dopa in Parkinsonian patients increases the amplitude of the jaw-jerk by about 90% (Widmalm et al 1979).

2.3.The jaw opening reflex

The jaw opening reflex was first described by Sherrington (1917) when he noted that stimulation of the palate,gingiva,or teeth could initiate a reflex jaw opening in decerebrate cats.

The jaw opening reflex is a polysynaptic one (Takata and Kawamura 1969).The input is the information sent from receptors in structures innervated by the second and third branches of the trigeminal nerve(Hoffman and Tonnies 1948,Kawamura and Fujimoto 1958).

The output consists of inhibition of the elevator muscles and activation of the depressor muscles although the latter still remains controversial(Gillings 1974). (Fig. 2)

Two main types of stimuli can provoke the jaw opening reflex:

a)electrical oral stimulation and

b)mechanical stimulation of the teeth.

a)Electrical oral mucosa stimulation

1.With the subject relaxed.

Electrical stimulation of the oral mucosa when the subjects elevator and depressor muscles are relaxed normally results in no muscle activity(Goldberg 1971).Stimuli applied to the incisive papilla leads to similar results with stimuli applied over the root of a tooth(Goldberg 1971).

2.With the subject contracting his elevator muscles.

Electrical stimulation of the same areas of the mucous

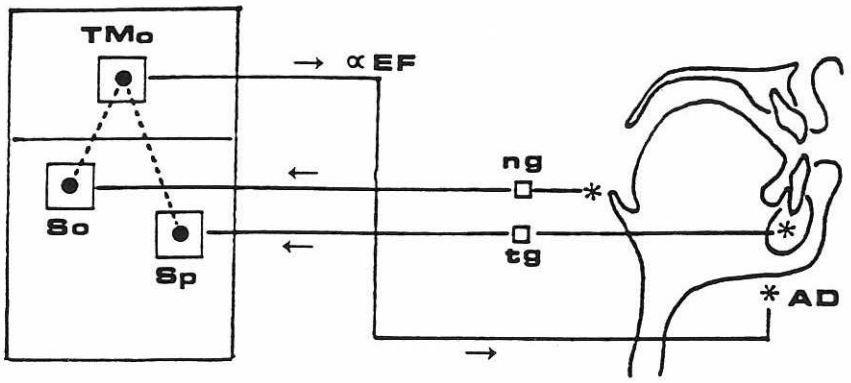

Fig. 2. Probable jaw-opening reflex pathway.

Sp Trigeminal spinal tract nucleus.
So Solitary tract nucleus.
TMo Trigeminal motor nucleus.
ng Nodose ganglion.
tg Trigeminal ganglion.
AD Anterior digastric muscle.
αEF alpha efferent.

From Sessle B.J.(1981) and Lavelle C.L.B. (1988) modified.

membrane but with the subject voluntary contracting his elevator muscles causes two periods of inhibition of the masseter muscle separated by a brief period of normal activity.

Latencies of about 13 to 15 and 45 msec. have been reported respectively(Matthews 1976).

The intensity of the stimulus also plays an important role in the muscular response.High intensity stimuli,up to the level of the maximum tolerance result in the already mentioned responses.A moderate reduction of the intensity of the stimulus causes only one silent period(Bratzlavasky 1972) while a radical reduction just above the sensory threshold does not provoke the jaw opening reflex(Yemm 1972).

3.with the subject contracting his depressor muscles.

If the electrical stimulation of the oral mucosa is repeated with the subject contracting his depressor muscles (forceful mouth opening against resistance)no change in the digastric muscle can be observed.

On the contrary similar experimental application in the animals produces obvious digastric activity(Matthews 1976).

b)Mechanical stimulation of the teeth

Mechanical stimulation of the teeth causes similar effects to those produced by electrical.

1.When the elevator and depressor muscles are relaxed tapping on the teeth does not elicit detectable response in any

muscles.

2.If the same stimulus is applied with the subject contracting his elevator muscles an inhibition in elevator muscle activity is produced but with no response in the digastric muscles(Beaudreau et al 1969,Hannam et al 1969 Sessle and Schmitt 1972).

Although the latency and duration of this response is variable depending on many factors ,a minimum latency of 13 msec has been reported(Lavelle 1988) as will be discussed later.

A high intensity stimulus often causes a double inhibition in the elevator muscles separated by an abrupt return of activity,while a smaller stimulus results in a single initial decrease in activity.

Just before the inhibition produced by tapping a tooth a transient activation of the elevator muscles can be observed with a latency of 7 msec.(Sessle and Schmitt 1972,Hannam et al 1970,Goldberg 1971).

It is apparent from the literature that the jaw opening reflex may play a protective role for the stomatognathic system,as it happens for instance when excessive forces are developed between teeth(a hard object in the food that causes a sudden and unexpected painful reaction).

As mentioned above however not only a painful stimulus but also a mild innocuous intraoral stimulation causes an

inhibition of the elevator muscles. This leads to the possibility that the jaw opening reflex is activated even during the compressive phase of the masticatory movement and before the occlusal contact thus contributing to the control of the mastication process(Hannam 1979).

Jerge(1964) and Kawamura(1964,1967) have postulated that reflex excitation of jaw opening muscles and reflex inhibition of jaw closing muscles elicited by mechanical stimulation of the teeth constitute the underlying neural basis of cyclic jaw movements and mastication.

The neurophysiological basis of this concept is that the stimulated periodontal mechanoreceptors by virtu of central connections may result in the excitation of the jaw opening muscles and inhibition of the jaw closing muscles.

Although several groups of investigators have shown that the periodontal mechanoreceptors are mainly involved in the excitation and inhibition of the masticatory muscles (Ahlgren 1967,1969,Shaerer et al 1967,Brenman et al 1968,Beaudreau et al 1969,Griffin and Munro 1969,Munro and Griffin 1970,Goldberg 1971),Hannam,Matthews and Yemm (1969,1970)indicated that neutralization of the afferent activity by local anesthesia does not abolish the results from tapping a tooth.

They concluded therefore that the periodontal receptors can not solely be involved in the inhibition reflex and implicated other

receptors, particularly the muscle spindles.

In the resulting controversy, Sessle and Shmitt (1972) suggested that this residual activity even after the infiltration of a local anesthetic was the result of the uncontrolled and unreproducible means of tooth stimulation . Using an electronically controlled mechanical stimulator capable of delivering accurate and reproducible stimuli ,they showed that local anesthesia of a tooth almost completely abolished the inhibition elicited by the tooth stimulation.

They thus provided conclusive evidence that the inhibition can be completely attributed to activation of receptors in, or around the stimulated tooth.

2.4 The tooth contact reflex

When the teeth are snapped together,the reflex changes that take place are similar to those produced by mechanical single upper tooth stimulation.

An initial transient activation of the elevator muscles, which is followed by a silent period associated with later phases of increased and decreased activity,characterizes the muscular response .

No significant effect however on the depressor muscles can be detected.

A series of silent periods in the elevator muscles can also be observed during mastication (Brenman et al 1968,Hannam et al 1969,Munro and Griffin 1970,Ahlgren 1967,1969).

This periodical muscular inhibition is probably due to periodontal mechanoreceptors activation,following tooth contacts during mastication(Hannam et al 1969,1970)

Muscle spindles and joint receptors may also be involved.

In 1970 Matthews and Yemm investigated the masseter EMG of edentulous subjects wearing full dentures and showed that the changes in muscle activity that follow tooth contact are similar to those of subjects with natural teeth.

In 1976 Nagasawa,Sasaki and Tsuru conducted an experiment to study the silent period of the masseter EMG in participants wearing complete dentures .In four of them the effect of a topically applied anesthetic was also studied.

According to their results a silent period was evoked under all the experimental situations with a considerably prolonged duration in the subjects with the anesthesized mucosa. The results of these studies provide evidence that the origin of the silent period after the tapping of the teeth,is not necessarily due to periodontal mechanoreceptors but superficial mucosa receptors,muscle spindles and joint receptors could also be involved.

2.5.The horizontal jaw reflex

All the above mentioned excicatory and inhibitory reflex events in the elevator and depressor muscles are clinically manifested as mandibular movements in the vertical direction. It is evident however from the literature that particular reflexes can also cause horizontal mandibular displacement. Lateral,protrusive and retrusive mandibular reflexes seem to be important in controlling the masticatory movements(Lavelle 1988).

The implication of these reflexes in some kinesiological characteristics of patients suffering from TMJ disturbances is also of particular interest but the whole subject needs further elucidation.

The little experimental evidence on these reflexes is mainly due to the limitations of the neurologists "model" animal the cat which presents an occlusion that severely limits the lateral jaw movements.

On the other hand the primate models suffer from a relative inaccessibility of the lateral pterygoid muscles for satisfactory EMG recordings(Sessle 1981)

Studies in rabbits(Lund et al 1971) and monkeys(Sessle and Curza 1981) however provide some evidence that a real reflex reaction can be evoked in the lateral pterygoid muscle from tactile tooth or oral mucosa stimulation.

2.6.The unloading reflex

It is well documented from clinical observations that if a person is biting down on a hard and brittle object and it suddenly breaks it is rare for the teeth to come together with sufficient force to produce pain or tooth damage.
The limitation of further jaw closing muscular activity after a sudden unloading of the isometrically contracting muscles is associated with a reflex of protective nature which is called "jaw unloading reflex".
The response of the muscles (other than masticatory)to sudden unloading has been previously investigated by Hansen and Hoffmann,(Entlastungsreflex 1922),Angel,Eppler and Iannon (1965),and Alston,Angel,Fink and Hoffmann(1967).
In order to avoid the objections that had been raised since that time against the use of a)electrical stimulation and b)tendon jerk in the study of the silent period,and also to investigate the reflex events in the antagonist muscles, Angel et al (1965) used as experimental situation the sudden unloading during a muscular voluntary contraction.
After application of this technique in the elbow joint muscles of fifteen human subjects they concluded that the sudden release of the external force was followed by a predictable series of events as follows :
a)The hand immediately showed a rapid acceleration with a maximum velocity of 76-152 m/sec always depending upon the

size of the weight used.

b)With a latency of about 40 msec. a silent period begun in the agonist muscles

c)A burst of activity in the antagonist muscles was followed in about 50 msec. by deceleration of the limb.

d)A second burst of activity was again seen in the agonist 70-150 msec. after release of the weight.

Hannam,Matthews and Yemm (1968) were however the first who demonstrated the unloading reflex in the jaw muscles.

The apparatus used to unload the mandibular elevator muscles during voluntary contraction consisted of a plastic tube clamped at a variable distance from a metal plate.

Each subject was asked to fracture the tube by biting the device with his premolar teeth.

The force needed to break the tube,the force necessary to cause its fracture , the initial separation of the jaws and the vertical movement of the mandible relative to the maxilla were all recorded and under control.

According to their results of that study :

a)The upward movement of the mandible was always arrested at least 5 mm from the tooth contact,irrespective of the initial separation of the jaws and the force necessary to fracture the tube.

b)The masseter and temporalis muscles became electrically inactive approximately 20 msec after the moment of fracture

while the digastric activated at about the same time.

c)The silent period of the elevator muscles was followed after approximately 50 msec by a sudden return of activity a phenomenon also reported by Alston et al (1967).

In 1969 Beaudreau and associates in an effort to investigate the possible effect of the activation of the periodontal ligament organs on the tonic activity of the masticatory muscles,reported among others that the abrupt release of tension from the mandible can cause a silent period in the temporalis and masseter muscles,with a latency of onset at about 30-40 msec.

The digastric muscle however behaved in two different ways. In 5 instances it behaved as an agonist following the responses of masseter and temporalis i.e.contraction and silent period while in 13 instances it became active only after the unloading with a latency significantly longer than the latencies for temporalis and masseter motor pauses.

The differences in the digastric muscle behaviour and in the values of the latency and duration reported by Beaudreau and associates can be attributed to the different methodology used.

To obtain their records the authors suspended from the mandible different weights ranging from 1 to 5 kgm by a piece of cord looped over the mandibular cuspid teeth. The subject was instructed to close his eyes and the weight

was released by cutting a piece of wire which formed a make break circuit,registering the moment of release on one channel of the oscilloscope.

In a study of the load compensation in human masseter muscles Lammare and Lund (1975) also used an unloading apparatus . They also reported that unloading the masseter muscle during contraction is followed by a fall in EMG activity, beginning after a latency of 6.5-11 msec, with later phases of depression beginning 27-36 and 60-70 msec after unloading.

In 1982 Miles and Wilkinson published a very interesting paper in which they showed that the timing of the reflex events in the agonist and antagonist muscles well described previously by Hannam,Matthews and Yemm (1968),could not account for the rapid arrest of the upward jaw movement when the resistance to jaw closing is suddenly and unpredictably removed.

They concluded that the mechanism which is principally responsible for preventing the teeth snapping together is the resulting anticipatory stiffness of the digastric muscle.

The apparatus used to simulate the physiological situation of cracking a nutshell between the teeth was a similar one with that proposed by Hannam and associates (1968).

A year later Miles and Madigan (1983) in a further investigation of the characteristics of the jaw unloading reflex and using the same instrumentation they concluded that

a)the motor program executed by the digastric muscles during forceful isometric bites is modified according to whether or not the subject expects the resistance to jaw closing to yield.

b) during bites in which the resistance to jaw closing is expected to yield,different subjects use different strategies of digastric coactivation to oppose the anticipated overshoot In some the coactivation is tonic throughout the bite whereas in others,the onset of coactivation is sudden and tends to occur at a specific threshold of net closing force.

Although there is a general agreement about the neuromuscular events associated with the jaw unloading reflex little evidence is yet available about the initiation and regulation of that particular muscular reaction.

The remarkably long latency of the digastric activation in comparison with the latency for a monosynaptic jaw jerk reflex(6-8 msec) indicates that a polysynaptic pathway is involved (Lamare and Lund 1975).

Angel et al (1965) suggested that muscle spindles play an important role although joint,tendon and periodontal

receptors may provide additional sensory information (Hannam,Matthews and Yemm 1968,Sessle 1981)

It is also not known whether the triggering signal which causes the anticipatory digastric stiffening originates centrally or just in the peripheral sensory receptors, but logically it may be assumed that suprabulbar loops contribute to the total final response.(Miles and Wilkinson 1982,Sessle 1981).

2.7.The unloading reflex in abnormal situations.

As mentioned above the various neuromuscular events following the sudden unloading of the isometrically contracted muscles had been first described by Hansen and Hoffmann in 1922. Soon after that initial observation which was explained only on the basis of a spinal reflex activity the unloading reflex was applied to patients with neurological diseases(Hansen and Rech 1925).

In the light however of the more recent knowledge about the neurophysiological basis of the unloading reflex,it seems to provide a quite useful technique for the study of the human motor function,in both normal and abnormal situations.

The unloading reflex has been previously documented in the following abnormal conditions:

a) In Parkinsons disease

b) In patient with infarction of the medial lemniscus

c) In patients with hemiparesis

d) During blockade of the antagonist muscle nerves.

a)The unloading reflex in Parkinson's disease

In 1978 Angel and Lewitt based upon a previous observation that the shortening reaction* is exaggerated in Parkinsonism (Broman 1949) conducted an experiment to test the hypothesis that the contraction after unloading and the shortening reaction* are both produced by the same neural mechanism. For the needs of the experiment they studied a patient with asymmetrical Parkinsonism,comparing responses between the sides with and without clinically evident extrapyramidal disease.

According to their results:

a)the contraction after unloading was greater on the side that was affected .

b)the contraction began earlier ,after a shorter silent period in the more affected side.

These findings supported the initial hypothesis that the response to unloading and the shortening reaction share a common control neural mechanism which is exaggerated in Parkinsonism (cog wheel rigidity)

*shortening reaction or "paradoxical muscle reaction"
According to Broman (1949) when a passive movement is performed the passively shortened muscles exhibit a contraction which tends to maintain the passively induced position.Rondot and Metral (1973) suggested that the shortening reaction may be important in the pathophysiology of Parkinsons disease,athetosis,and dystonia. An implication in the control of normal posture and movement is also possible.

b)The unloading reflex in patient with infarction of the medial lemniscus

Several experiments have suggested that long loop reflexes involving supraspinal structures may play a role in human motor control.

In 1983 Angel and Goldstein conducted an investigation to study whether the muscular response to unloading may also have a supraspinal component.

In that investigation the unloading reflex was elicited by partially removing the load on voluntary contracted muscles. The removing of fractions of a load rather than the complete removal of the force, was considered to be a superior technique for the detection of the response to very small perturbations.

The method was applied to a patient with a pontine lesion who had impaired position sense in the left hand.

The unloading reflex was found to be abnormally weak in the affected (left)hand as compared with the right one while such an asymmetry was not found in the control subjects.

Although the authors could not assume that the abnormality tested was related specificaly to the lemniscal damage they suggested that a "long loop" supraspinal mechanism contributes in the mediation of the unloading reflex.

c) Unloading reflex in patients with hemiparesis.

In 1968 Angel conducted experiments to study the unloading reflex in 5 patients with hemiparesis due to cerebrovascular disease.

On the basis of the fact that the neuromuscular events following a sudden muscular unloading provide information about different aspects of the motor control system, each of the two main phases of the reflex i.e. the silent period and the renewal of the motor activity, investigated separataly. According to the results of that study a silent period was demonstrated in all of the limbs tested (including the paretic ones) while the renewal of the motor activity was found to be absent or greatly reduced in all of the paretic limbs.

Some possible explanations for these findings have as follows:

Phase 1 (silent period)

This phase of the unloading response is believed to result from a pause in spindle afferent discharge due to muscle shortening and provides indirect evidence that the motor discharge is maintained at least in part by proprioceptive feedback.

On the other hand the occurence of the silent period even on the paretic limbs, indicates that a lesion of the corticospinal pathway does not necesarily abolish the role of

the proprioceptive feedback in sustaining a voluntary contraction and confirms to some extent a previous hypothesis (Granit 1966) according to which the "gamma loop" is a motor route in its own right.

Phase 2 (renewal of the motor activity)

Although the origin and the functional significance of that phase are not still clear the depression of this motor discharge in the paretic limbs suggests that a corticospinal tract lesion,tends to damage the neural mechanism which underlies this response.

In that case it is reasonable to assume that afferent impulses from the moving extremity may be routed through the cerebral cortex and then returned to the spinal cord by way of the corticospinal tract.

From the clinical point of view the results of this study also suggest that the depression of the motor discharge , that normally follows the silent period , could be added to the list of the "upper motor neuron signs" seen in the stroke patients.

d) Unloading reflex during blockade of antagonist muscle nerves.

As discussed previously,the unloading reflex is a quite complex muscular response.

Two of the main neuromuscular events that take place in the area of the agonist muscles are:

a)the silent period and

b)the burst of activity following that electrical inhibition.

Several authors have suggested that the silent period results entirely (or largely) from reciprocal inhibition due to the stretch of the antagonist(Struppler et al 1964,Angel et al 1965,Hagbarth and Eklund 1966,Matthews 1970).

On the other hand the motor activity following the silent period has been explained as a result of a renewal of the spindle afferent discharge(Alston et al 1967).

Since however a passive stretch reflex can not be regarded as the initiation factor (because the burst occurs while the agonist is still shortening) the "fusimotor reflex" i.e. activation of the fusimotor neurons of the agonist through the gamma loop by stretching of the antagonist,seems to be the more likely explanation.((Barrios et al.1967,Haase and Vogel 1969,Schlegel and Sontag 1969,1970).

In order to test these two hypotheses Angel,Garland and Moore (1973) conducted experiments on 7 subjects.

Unloading responses from the brachial triceps were studied

before and after the application of a local anesthetic to the antagonist muscle nerves, with the expectation that the nerve blocking would abolish both the silent period and the following motor volley.

The results of this study did not support the views

a)that reciprocal inhibition is necessary to produce a silent period and

b)that the "fusimotor hypothesis",can sufficiently explain the motor activity following the silent period.

Table 1

UNLOADING REFLEX IN HUMAN MUSCLES

AUTHOR	* MUSCLES STUDIED	METHOD
ANGEL et al 1965	Elbow joint muscles	Sudden release of an external force
HANNAM et al 1968	Jaw muscles	Unloading apparatus
BEAUDREAU et al 1969	Jaw muscles	Suspension and release of various weights from the jaw
LAMMARE and LUND 1975	Jaw muscles	Unloading apparatus
MILES and WILKINSON 1982	Jaw muscles	Unloading apparatus
MILES and MADIGAN 1983	Jaw muscles	Unloading apparatus

* Elbow joint muscles
Biceps and triceps brachii, pectoralis major and infraspinatus, posterior fibres of deltoideus
Jaw muscles
Masseter, temporalis, anterior digastric.

UNLOADING REFLEX IN HUMAN MUSCLES... continue

AGONIST MUSCLES SILENT PERIOD Latency	Duration	Return of activity	ANTAGONIST MUSCLES
40 msec	69-14 msec	Yes	---
20 msec	50 msec	Yes	Activation after approx. 20 msec
Masseter 36±16 msec Temporalis 34±9 msec	---	---	ANTAGONIST BEHAV. Activation after 94±30 msec AGONIST BEHAVIOUR Silent period Latency 38±14 ms
Masseter Three phases 6.5-11 msec 27-36 msec 60-70 msec	---	Yes	---
10-20 msec	50-70 msec (not seen in all subjects)	Yes	Activation after 25-30 msec Anticipatory stiffness
---	---	---	Different strategies of digastric activation

CHAPTER 3.MATERIAL AND METHOD

3.1 Criteria for sample selection

A total of 10 young adult volunteers 6 male and 4 female aged between 20 and 35 years was selected according to the following criteria:

1. Natural dentition with at least all the incisor and canine teeth present.

2.No signs and symptoms of Temporomandibular Joint Disorders.

3.No history of neuromuscular disease.

All subjects were fully informed of the procedures before their consent was obtained,although the aims of the experiment were not revealed in advance(Appendix 1)

3.2 Unloading apparatus* or "collapse machine"

The apparatus used to unload the mandibular elevator muscles during voluntary contraction consisted of two bars,the upper one being fixed and the lower one hinged arround a pin. The distance between the two bars was 2mm and the initial resistance to closing was achieved by the ratio of the moments **B d1/M d2** where B=biting force,M=magnet force,d1=bite forke-hinge pin distance,d2=magnet-hinge pin distance(Fig.3) Different unloading resistance levels could thus be obtained by inserting the hinge pin at different distances from the magnet.

The force applied was measured by strain gauges mounted to the upper bar.

The output of the strain gauges was displayed as a spot moving in the horizontal axis on an oscilloscope** screen placed in front of the subject.

The vertical movement of the lower bar relative to the upper one was recorded by means of an axial accelerometer *** cemented with insulating mounting to its lower surface.

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* On loan from Univ. of Groningen Dept.of Neurobiology and Oral Physiology
** Oscilloscope Tektronix 2445
*** Single axis piezoelectric accelerometer 4374 Bruer and Kjaer.

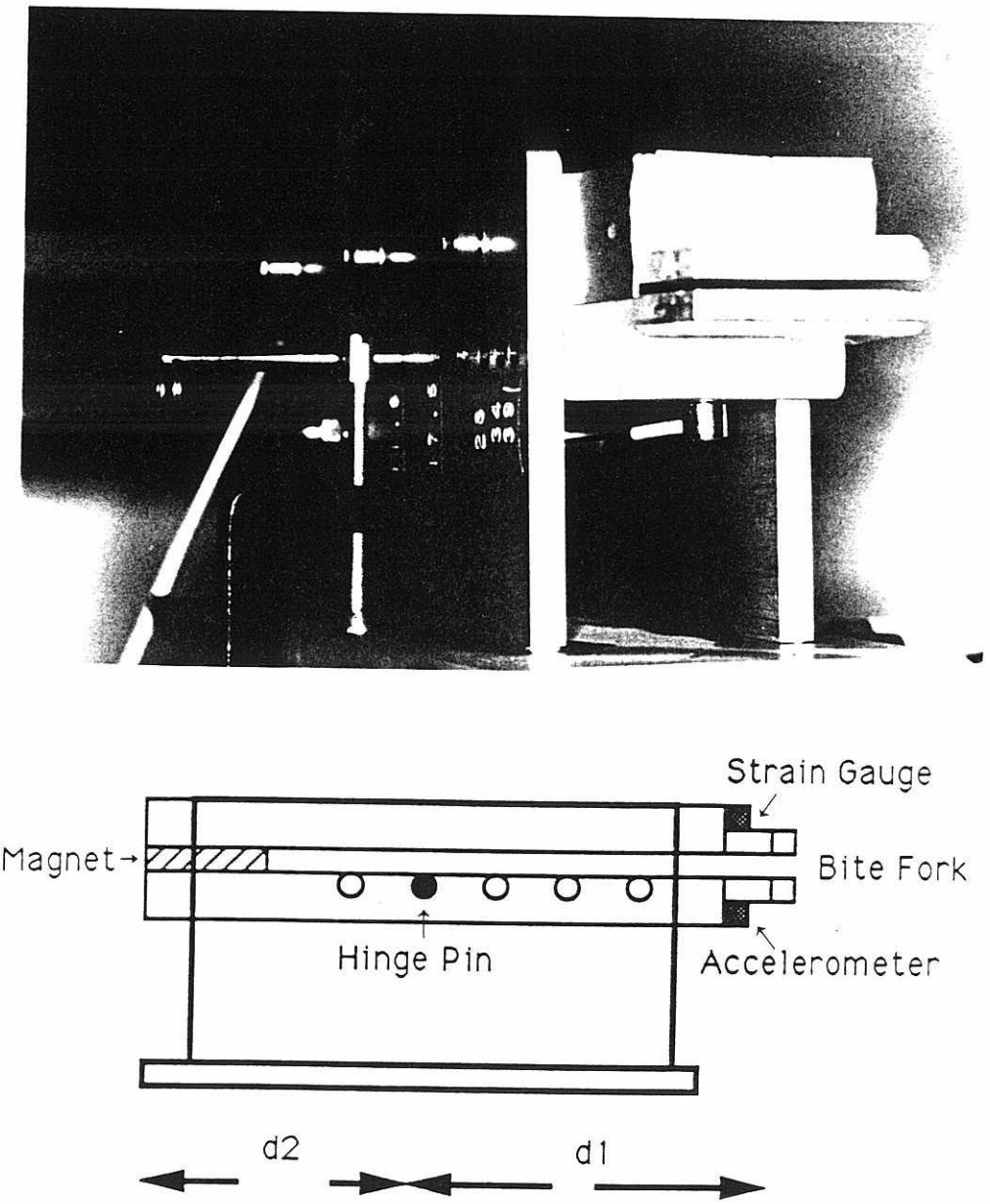

Fig.3. Unloading apparatus
d1=hinge pin-bite fork distance
d2=hinge pin-magnet distance

The bite fork was covered in plastic to prevent damage of the teeth while disposable plastic covers were used to prevent cross infection.

Calibration

The electrical output of the strain gauges was calibrated by static loading.According to the results a linear relationship was found to be between the voltage output and these loads. This linearity was checked three times during the experimental period and remained unchanged.

The calibration plot for force and electrical output is shown in Fig 4.

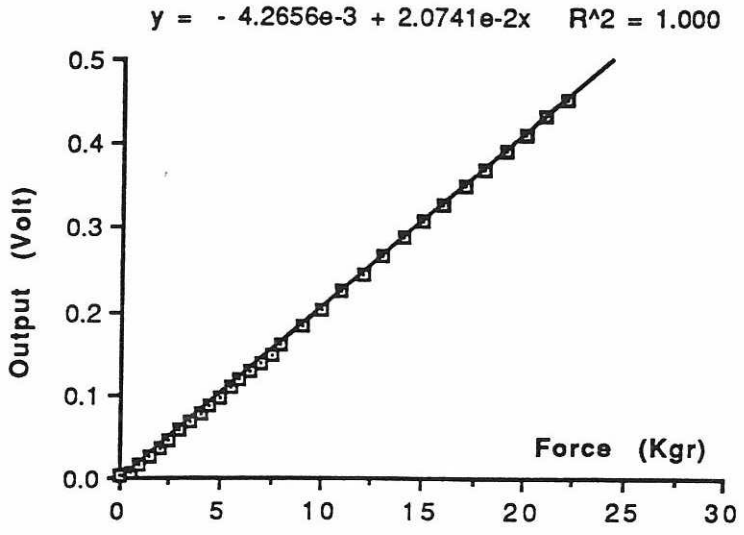

fig 4

3.3 Recording system (Fig.5)

Electromyographs were recorded from both masseter and anterior digastric muscles with surface electrodes. The activity from masseter muscle was amplified with an isolated preamplifier* and a differential main amplifier** while the digastric muscle signal was amplified with a biofeedback appliance ***

To avoid clipping of large signals the gain of the amplifier was adjusted for each individual according to the signal amplitude during maximum mouth opening and clenching.

The strain gauges and the accelerometer signals were amplified with a strain gauge amplifier with digital readout**** and a charge amplifier respectively*****

.....

* EMG ISOLATED PREAMPLIFIER NL850
Neurolog system Digitimer LTD England
** Neurolog. A.C.PREAMP NL 104
***BIOFEEDBACK EMG 200 (Preampl. and Main ampl.together)
**** A.M.Ferman Biometrics Lab. LHMC
***** Charge amplifier.2634.Bruel and Kjaer.

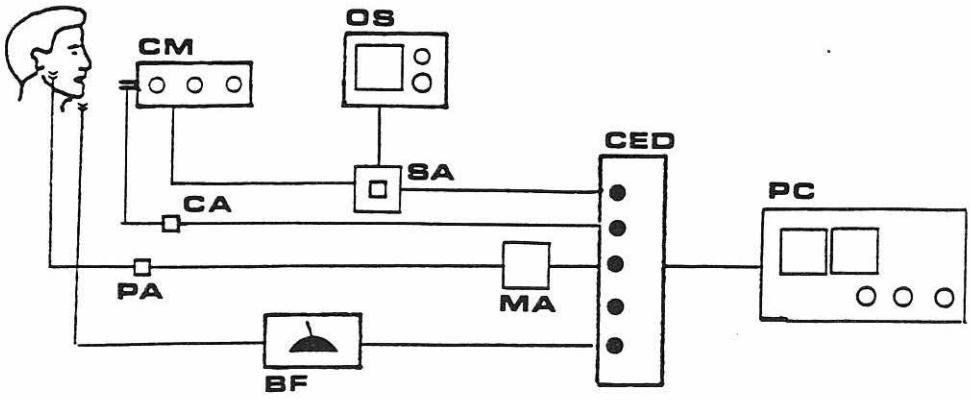

Fig.5 Recording system
CM=collapse machine
OS=oscilloscope
SA=strain gauges amplifier
CA=charge amplifier
PA=preamplifier
MA=main amplifier
BF=biofeedback
CED=AD converter
PC=personal computer

Electromyographic signals from both masseter and anterior digastric,as well as the strain gauge and the accelerometer outputs were finally led to an AD converter* which was driven by a commercial software package** running on a NES PC 286 S computer.

Data was stored on a 30 MB hard disc and backed up on to a high density floppy disc for later analysis .

.....

* Multichanel Intelligent Analogue Interface type CED 1401

** Spike2 Version 2
Cambridge Electronic Design LTD

3.4 Electromyography

During this study electrical activity was recorded from the right masseter and left anterior digastric muscles using bipolar surface electrodes.*

Skin impedance was reduced by alcohol scrub (Møller 1966) Electrodes were filled with electrolyte jelly** and secured over the recorded muscles as follows :

Masseter muscle

The subject was asked to clench her teeth and surface electrodes were placed over the centre of the fleshy body of the superficial portion of the muscle 15mm apart and in line with the muscle fibres (ad modem: Muray and Klineberg 1984) The refer ence (ground) electrode was placed on the back of the neck (Fig 6)

Digastric muscle

The mandible was forcibly lowered against manual resistance the muscle palpated and the electrodes were placed 10 and 25 mm behind the mandibular insertion of the muscle.

Electrodes were always placed in line with the main muscle direction (Ahlgren and Lipke 1977)

The reference (ground) electrode was secured on the back of the neck.(Fig 7)

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*Electrodes Dantec 13L20 .Silver/silver chloride ECG electrodes,contact=0.7mm pin.Denmark.

** Dantec type 15B 411 electrode paste

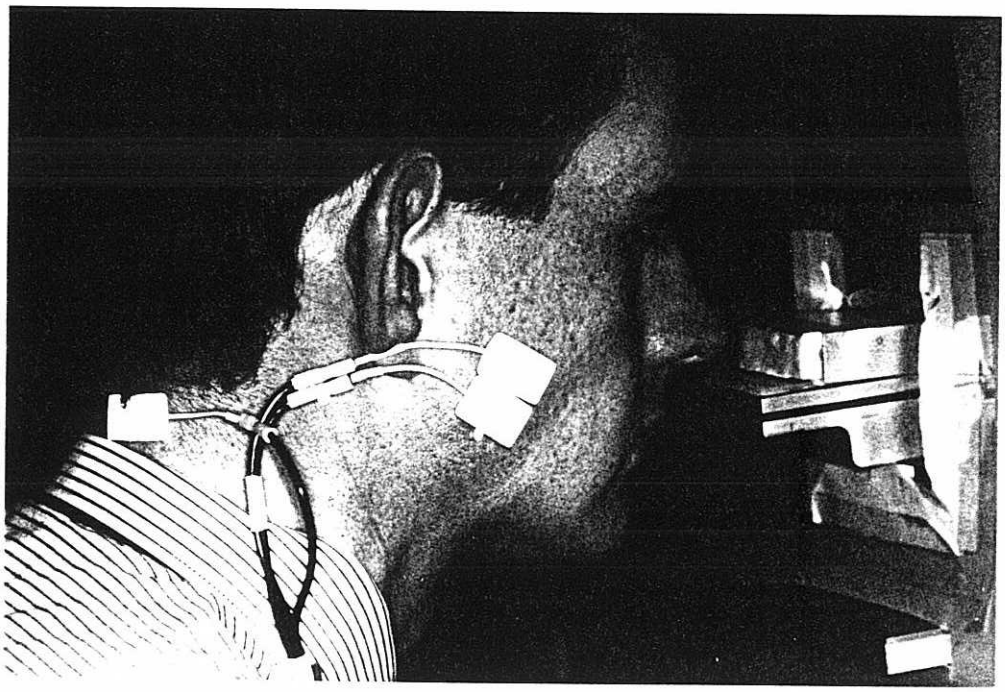

Fig. 6 Position of the surface electrodes.
Masseter muscle.

Data from digastric muscle dissections were also used to confirm the correct electrode placement.

3.5 Digastric muscle dissection

A small number of digastric dissections* on fresh cadavers offered valuable information about the anatomy of the muscle and the position for the placement of the surface electrodes in order to minimize cross-talk from the adjacent muscles.
As it has been described previously(Schumacher 1961,Sicher 1965,Larsson and Lufkin 1987,Zuckerman 1986) each of the digastric muscles consists of two bellies a)the posterior and b)the anterior belly.
The posterior belly arises from the medial side of the mastoid process running down and forward to the hyoid bone.
The anterior belly takes its origin from the digastric fovea of the mandible close to the midline and its fibres converge postero-laterally toward a strong tendon which is anchored to the body of the hyoid bone by a fibrous sling.
According to our findings,the fleshy body of the anterior belly,lies approximately 1cm laterally to the midline and on a line drawn perpendicular to the later one,2cm behind the mandibular symphysis (Fig 8)

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* The series of dissections were carried out by Mr M.R.Norton in the Institute of Pathology at the LHMC.

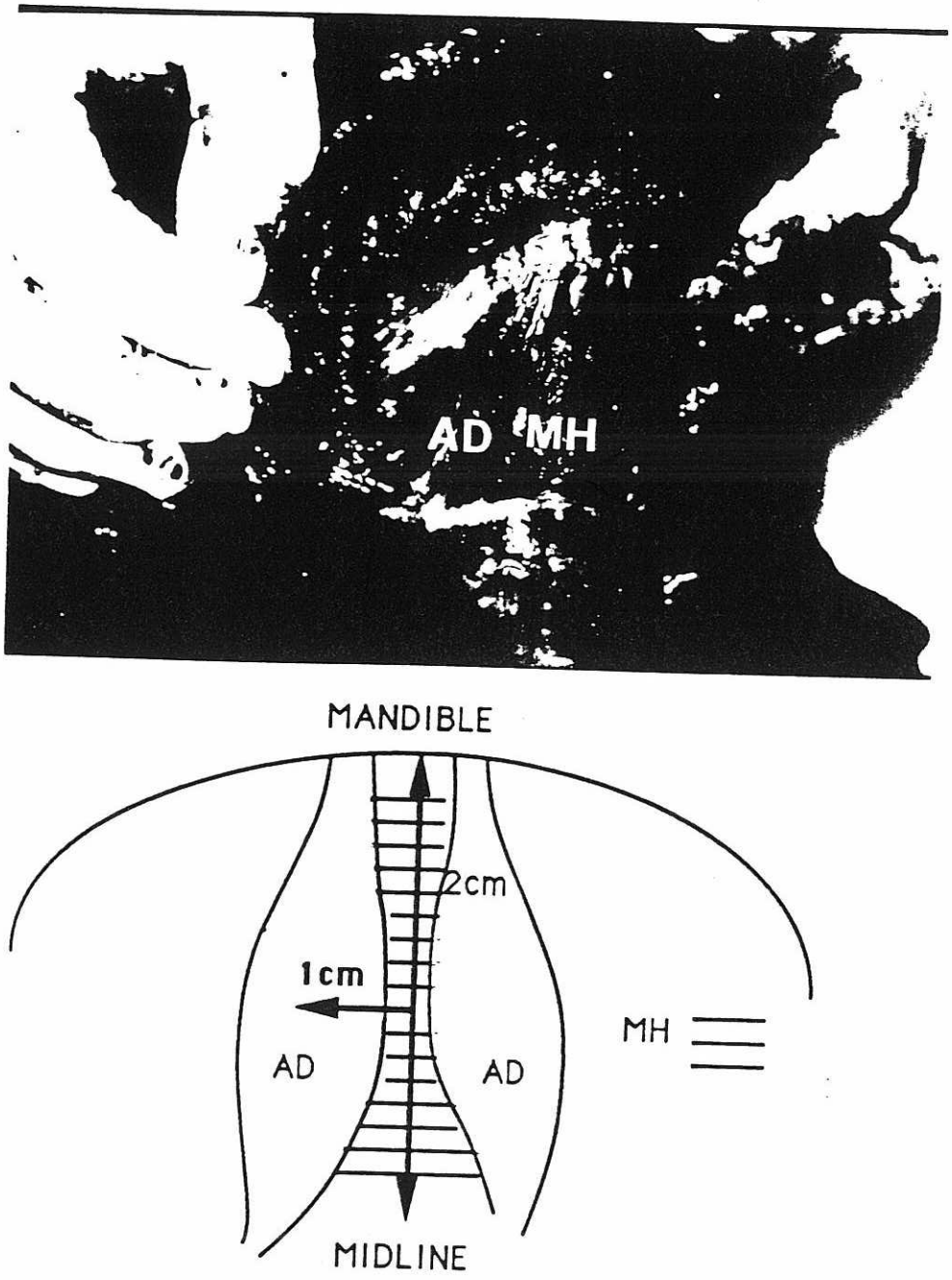

Fig. 8a.Dissection of the anterior digastric muscle .
8b.Diagram illustrating position for the placement of the surface electrodes.
AD=Anterior digastric.
MH=Mylohyoid muscles.

3.6 Experimental protocol

The subjects were comfortably seated throughout the experiment in front of the unloading apparatus described above and were asked to performe a task while they bit with their upper and lower anterior teeth on the bite fork. They were able to control their biting force level with visual feedback from an oscilloscope screen displaying the strain gauge output against time, 1.2sec/division(10mm) . Three oblique lines (ramps) were drawn on the oscilloscope screen at equal distances. (Fig.9)

The experimental protocol comprised four stages as follows:

Control experiments

Initially each subject received a description of the unloading apparatus and the general aims of the experiment. He was also given experience about the machine by asking him to bite on the bite fork and make the moving spot follow as closely as possible each of the ramps as drawn on the screen. The machine was in the locked mode with two hinge pins placed at different positions.

The maximum biting force corresponding to the top of each of the ramp was 110 N. (Fig 9)

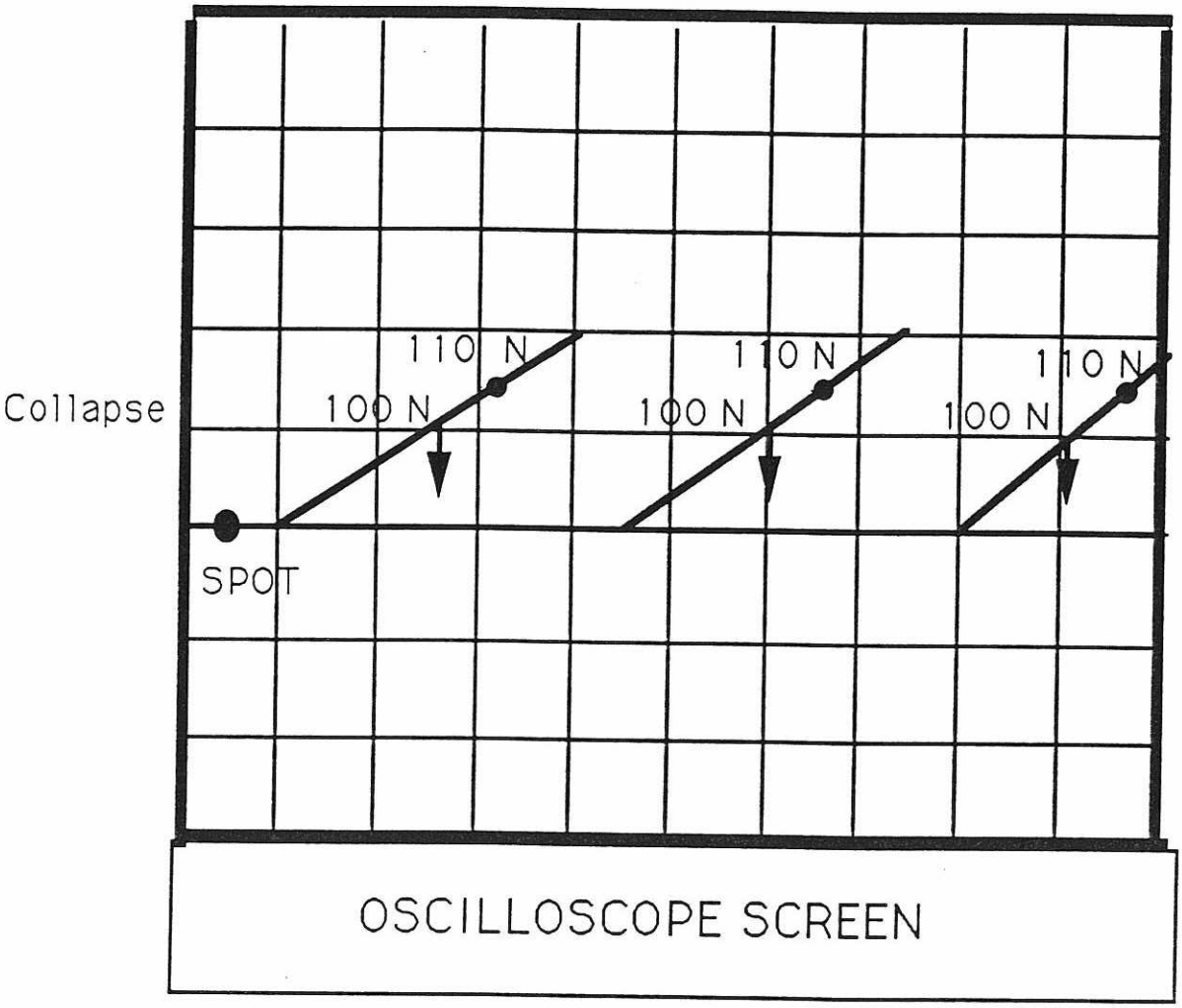
Collapse
110 N
100 N
110 N
100 N
110 N
100 N
SPOT
OSCILLOSCOPE SCREEN

Figure 9

During control experiments subjects became familiar with the machine and also reduced "**irrelevant**" activity due the experimental environment.

Study 1. "Naive"

The subject was asked to bite on the bite fork and follow the ramps as in the control experiments.

Study 1 comprised 6 experiments.

Between the fifth and the sixth experiment,one of the two hinge pins was removed and the naive subject underwent a sudden and unexpected unloading at 100 N.

Study 2."Informed"

The subject was informed about the experimental situation.

Study 2 comprised 6 experiments (collapses)

The machine was at the unlocked mode .

The force level for the" collapse experiments" was calibrated at 100 N.

Study 3."Uncertain"

The real experimental situation was left uncertain.

The machine was behind a screen so that the subject could not see wether it was locked or not.

In a random order the machine was unlocked 2 times in a round of 6 experiments.

In order to enable intra-subject comparisons,the conditions of the experiment remained constant in regard to the force level,the position of the surface electrodes,and the amplification gain.

3.7 Analysis of the data

Data analysis was carried out off-line using two commercial software packages* both with graphic display capacity. The precollapse pattern of digastric activity and the digastric reflex events following the mandibular unloading were studied separately.

A.Precollapse period.

Patterns of digastric activity.

The occurence(Yes\No) of precollapse digastric activity during the tracking task,was evaluated from the recorded EMG in comparison with the level of the digastric activity at rest .For the EMGs presenting precollapse activity,a section corresponding to the total duration of the tracking task for each collapse experiment was extracted,full-wave rectified and integrated.(Figs 11 and 12) The initiation of the isometric contraction was determined from both the strain gauge and the masseter muscle recordings (Fig 10).Integrated EMGs from different experiments were superimposed where necessary for direct comparisons.

For the further study of the potential changes in the patterns of digastric activity,recorded (raw) EMGs from

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* Spike2 Version 2 Cambridge Electronic Design LTD

DADiSP DSP Development Corporation Cambridge UK

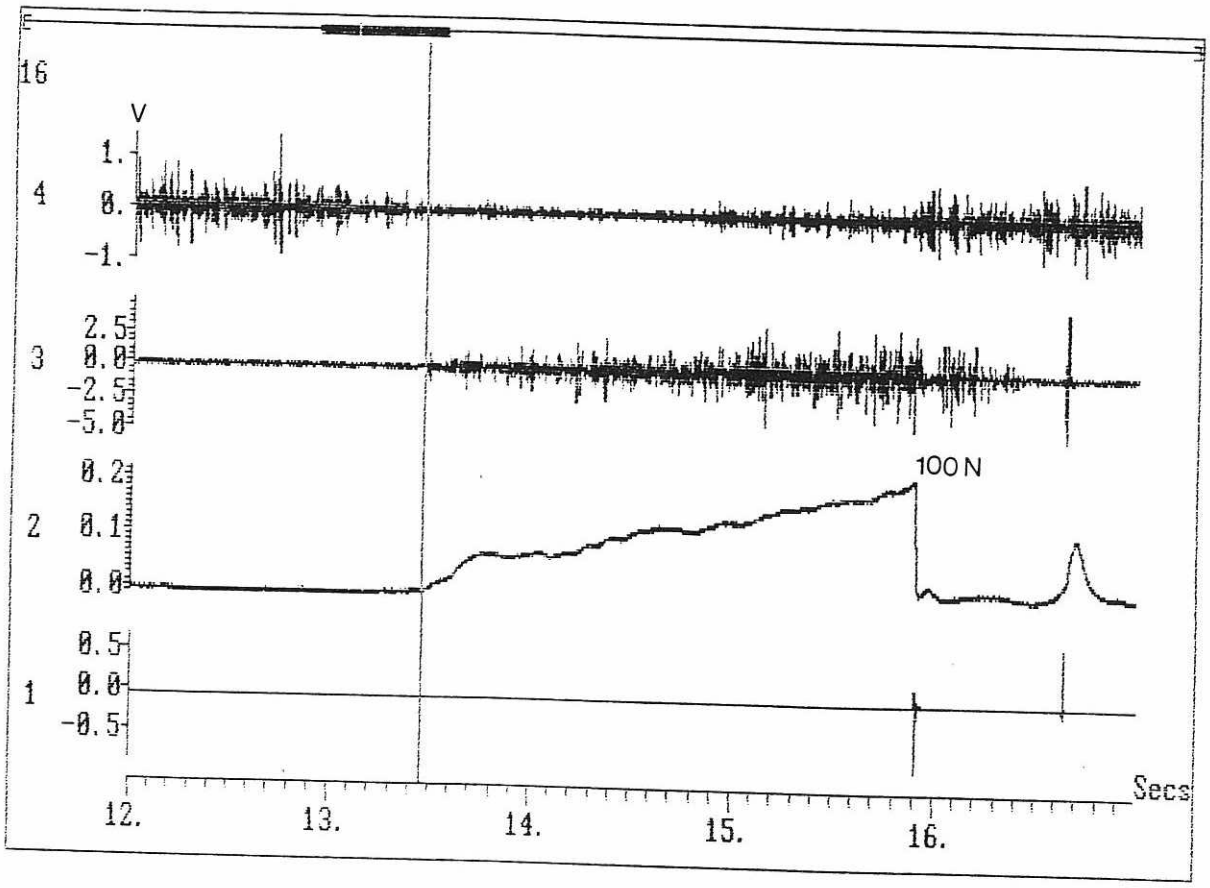
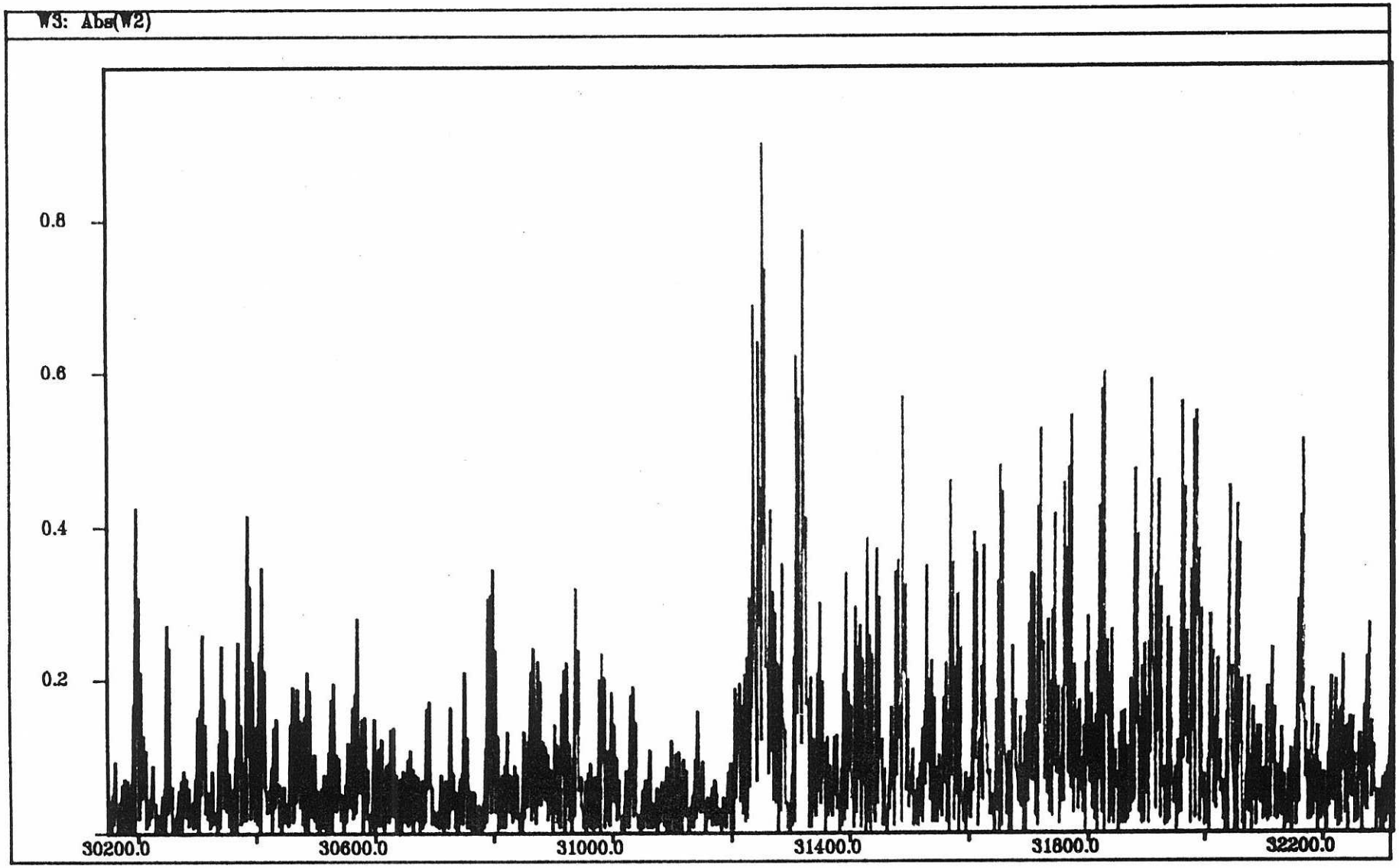


Fig.10. Recorded data from a collapse experiment.The cursor is placed at the beginning of the isometric contraction of the masseter muscle.
Channels:
1.Accelerometer
2.Strain gauge
3.Masseter muscle
4.Anterior digastric muscle

Fig.11.A full wave rectified EMG of the precollapse digastric activity.Subject AM. Study 2.Experiment 5.



the non-collapse experiments of studies 1 and 3 were also examined.

Finally a description of the patterns of the digastric activity was given for each subject.

B.Postcollapse reflex events.

Digastric burst

The occurence(Yes\No),and the latency(msec) of the digastric burst were both studied from the recorded EMG using the various display facilities of the Spike2 software.

The latency of the digastric burst was defined as the interval between the collapse(determined from the accelerometer recording) and the beginning of the first distinct major increase from the preceding activity,or baseline when no activity was recorded (Widmalm and Hedegard 1976) (Fig 13)

Records were discarded where accurate measurement of the latency was impossible.

3.8 Statistical analysis

Two data collection forms were made to collect all the measurements and observations(Appendices 2 and 3). One for the precollapse activity(observations) and one for the latency of the digastric burst (measurements)

Observed frequencies and percentages were calculated for the occurence of the precollapse activity and a histogram of

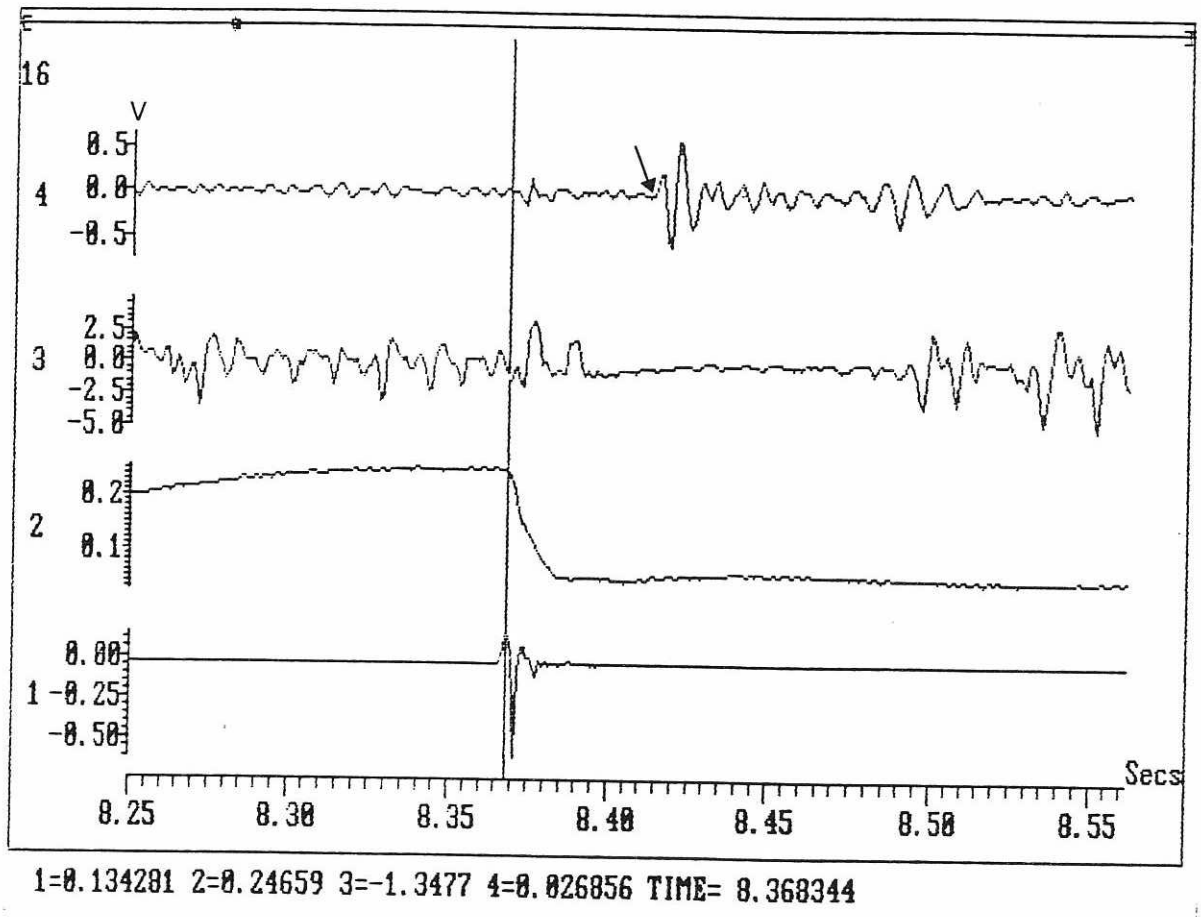


Fig.13. Latency of the digastric burst.The latency is measured between the cursor(collapse)and the arrow.
Channels:
1.accelerometer
2.Strain gauge
3.Masseter muscle
4.Anterior digastric muscle

those data was drawn.

Conventional statistical methods were used to calculate means,standard errors and standard deviations for the digastric burst latencies of each of the three studies. (Naive,Informed,Uncertain)

The differences between the mean values, were then statistically evaluated using ANOVA

CHAPTER 4. RESULTS

4.1.Occurrence of precollapse digastric activity

In five of the ten subjects studied ,the digastric muscle remained inactive during all the experiments.

Two subjects,presented activity throughout all studies while three subjects only during studies 2(informed) and 3(uncertain) .

Observed frequencies and percentages for all the collapse experiments are demonstrated in Appendix 2 and Fig 14.

4.2.Patterns of precollapse activity

General notes

- The accuracy with which the subjects managed to follow the oblique lines (ramps) presented relevant variability from subject to subject and from experiment to experiment.

- The patterns of digastric activity during the tracking task of the collapse experiments differed from subject to subject and from one experiment to another.

- To facilitate the study and enable the direct comparisons of the various levels,integrated EMGs from different experiments were superimposed for each of the five individuals.The superimpositions for the subjects:TK,GK,JT,AM and HK are shown in Figs 18a,b,c,19a,b,20,21,22a,b.

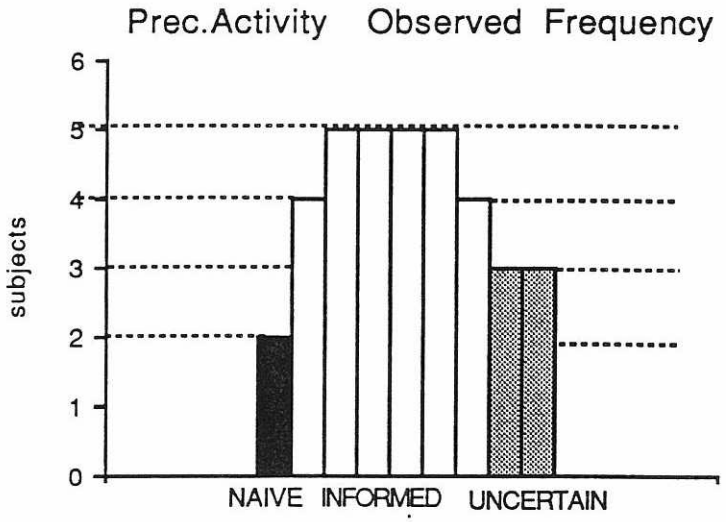


Fig.14. Occurence of precollapse digastric activity.Observed frequency.Each column represents one collapse experiment.

- The obvious differences in the length of the integrated lines, reflect the variability in the accuracy with which each subject managed to follow the ramps from the one experiment to another.

- The study of the recorded, full wave rectified and integrated EMGs showed that despite the variability of the digastric activation three main patterns could be described :

  1. **A continuous tonic activity** throughout the period of the tracking task (Fig. 15)
  2. **A progressively increasing tonic activity** (co-contraction in line with the masseter muscle) (Fig. 16)
  3. **A sudden increase in the amplitude of the digastric EMG**, at either the **middle** or the **end** (and rarely beginning) of the tracking task. (Fig. 17)

  These three models of digastric activity will be used in the following descriptions.

Description of the cases.

To facilitate description the following code was used:

n6. The collapse experiment of the study 1(naive)

info1,2,3,4,5,6. The six collapse experiments of study 2(info)

unc 1,2. The two collapse experiments of study 3(uncertain).

PA. Precollapse activity of the digastric muscle.

The patterns and levels of the digastric activity for each particular subject were:

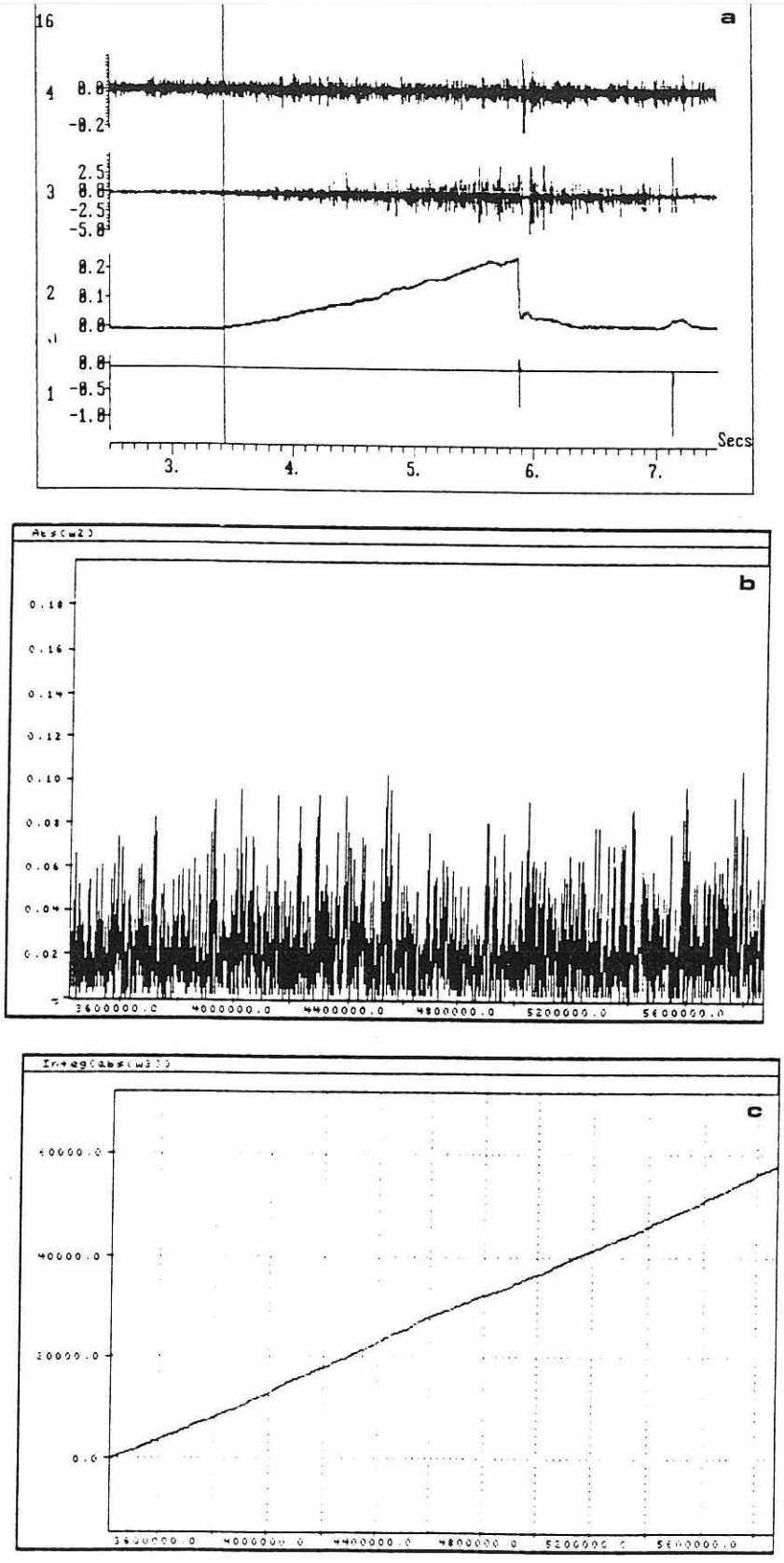


Fig.15a,b,c. An example of continuous tonic digastric activity
a.Recorded EMG.Digastric muscle channel 4.
b.Full-wave rectified EMG of the precollapse activity.
c.Integrated EMG of the precollapse activity.

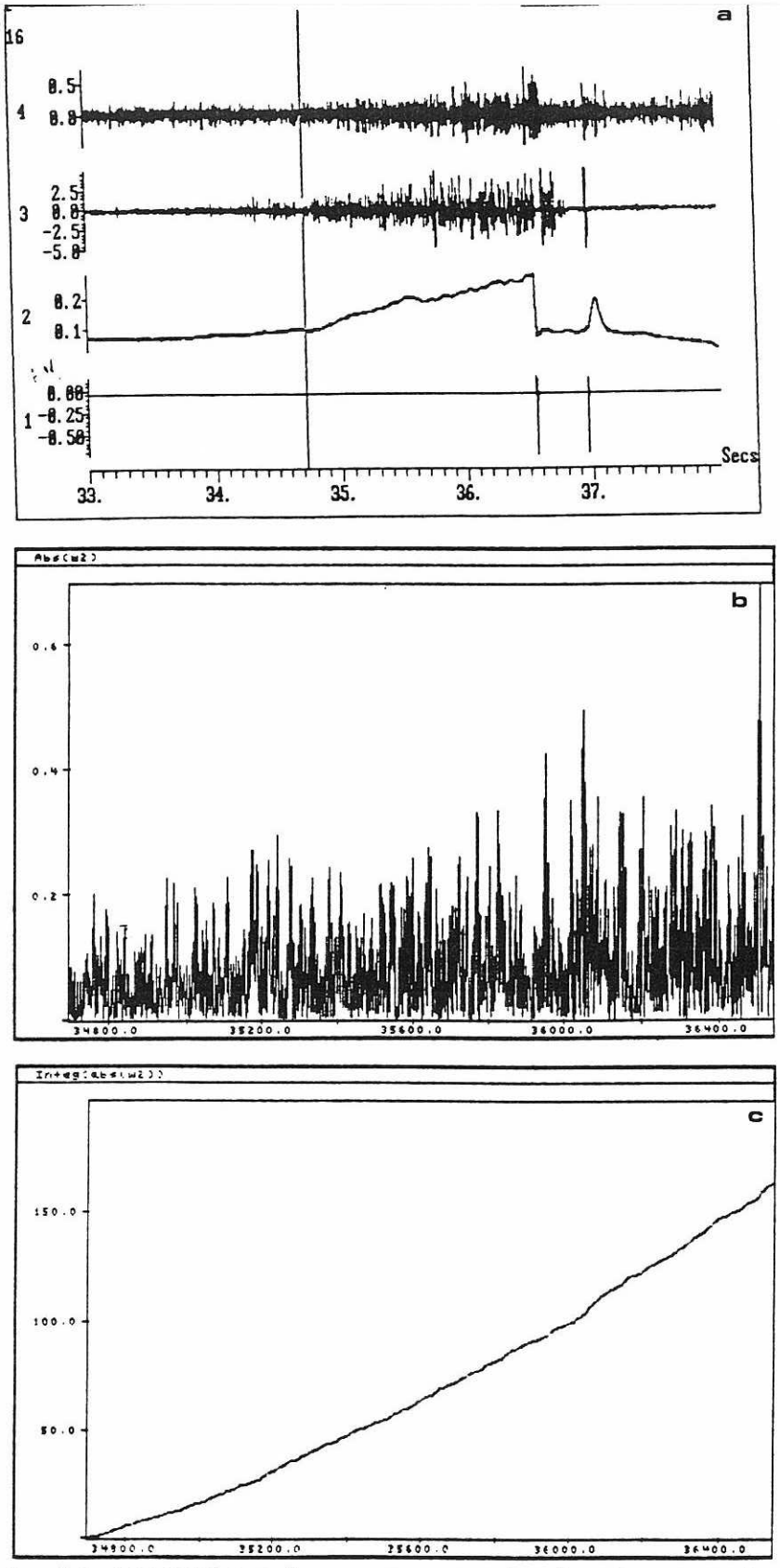


Fig.16 a,b,c. An example of a progressively increasing digastric activity.Subject AM.Study 2.Experiment 6.
a.Recorded EMG.Digastric muscle channel 4.
b.Full wave rectified EMG of the precollapse activity.
c.Integrated EMG of the precollapse activity.

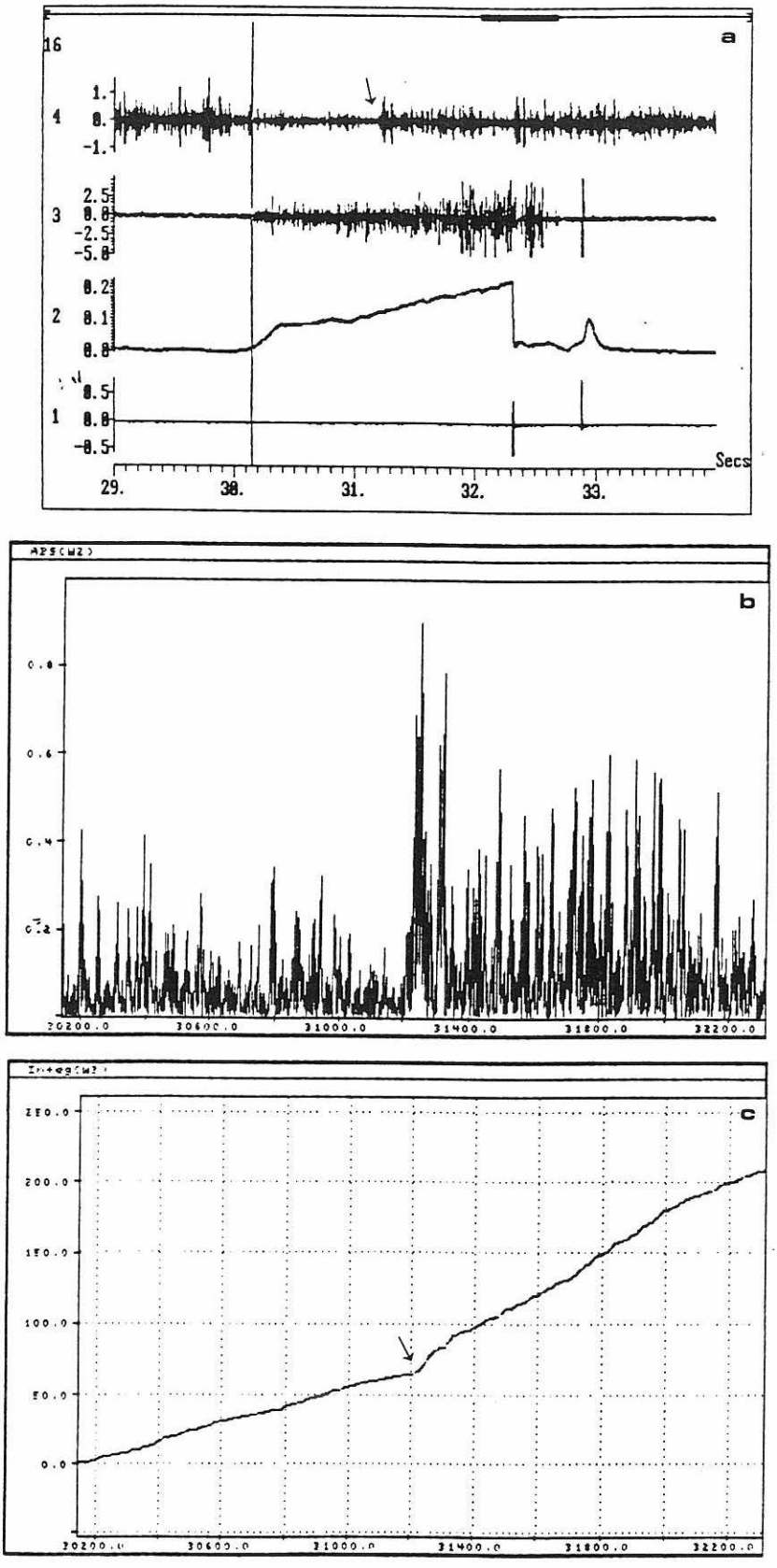


Fig.17 a,b,c. An example of a sudden increase in the amplitude of the digastric EMG at the middle of the tracking task(arrows).Subject AM.Study 2.Experiment 5.
a.Recorded EMG.Digastric muscle channel 4.
b.Full wave rectified EMG of the precollapse activity.
c.Integrated EMG of the precollapse activity.

Subject TK

In subject TK the anterior digastric was active throughout all studies(Appendix 2).

Study 1

In the collapse experiment of the first study **(n6)** the digastric presented a rather low and progressively increasing **PA**. The pattern and the amplitude of the digastric activity was almost similar for all the preceding non-collapse experiments. (Fig 24)

Study 2

In all of the experiments of study 2,a progressively increasing **PA** (co-contraction) was obvious,while the muscle was clearly more active during the last **(info 6)** experiment (Fig 18a).

Study 3

In the third study and during the first collapse experiment **(unc 1)** the muscle presented a similar pattern and level of activity with that of the naive situation (Fig. 18 b).

On the contrary during the second collapse experiment of this study **(unc 2)**,the level of the **PA** was suddenly increased even over the level of the sixth collapse experiment of the previous study (Fig.18 c)

The overall assessment of the digastric behavior during study

3 (non-collapse and collapse experiments together) revealed the development of a certain pattern.

All the recorded data (raw EMGs)from study 3 are demonstrated in Fig.23a-f and special reference is made in the discussion.

Subject GK

In subject GK the anterior digastric muscle was active during studies 2 and 3 (Appendix 2)

Study 2

In the first experiment of the study 2 **(info 1)** there was a sudden increase in the level of the **PA** about 1000 msec after the initiation of the tracking task (Fig. 19a,arrows)

A similar sudden but earlier(750 msec) onset of **PA** was seen in the fourth experiment **(info 4)** .

In contrast the sixth **(info 6)** experiment of this study was characterized by a continuous tonic pattern but with dramatic reduction in the level of the **PA** (Fig.19a)

Study 3

In the first experiment of study 3 **(unc 1)** the anterior digastric presented an initialy increased **PA** which was followed by a progressive reduction by the end of the task.

In the second collapse experiment of this study **(unc 2)**,the muscle presented a similar pattern and level of activity with that of the first informed experiment . (Fig. 19 b)

However the overall assess ment of the digastric behavior during this study (non-collapse and collapse experiments

together) could not reveal the development of a certain pattern.

Subject JT

In subject JT the anterior digastric was active only during the first five experiments of study 2 (Appendix 2).
All of these five collapse experiments were characterized by a continuous tonic activity of the digastric muscle throughout the isometric biting task.
The level of activity was almost the same for all the experiments apart from the fifth one (Fig. 20) which was rather lower.A more careful examination however of the fifth collapse experiment (**info 5**) revealed that the continuous tonic activity was replaced by a rather progressively increasing tonic activity .

Subject AM

In subject AM the anterior digastric muscle was active during the 2,3,4,5 and 6th experiments of the study 2 (**info 2,3,4,5,6**) . In the first four collapse experiments,the anterior digastric presented a striking sudden increase in the level of **PA** in the middle of the task (Fig. 17) with a trend for rising in activity from experiment to experiment.In the sixth (**info 6**) experiment however,the level of activity was dropped (Fig. 21) and the previous pattern was clearly replaced by a steadily increasing tonic **PA** (Fig.16)

Subject HK

In subject HK the anterior digastric muscle was active throughout all studies.(Appendix 2).

In all the collapse experiments,the anterior digastric presented a continuous tonic **PA** but of various levels.

Study 1

The patterns and the levels of the **PA** were almost similar during both the non-collapse and collapse experiments of this study.(Fig. 25).

Study 2

In the **info 1,info2 and info 3** experiments the anterior digastric presented almost the same level of activity,while a marked increase was seen in experiments **info 5 and info 6** (Fig. 22a.)

Study 3

The level of the the **PA** during the experiment **(unc 2)** was higher compaired to the experiment **(unc 1)** (Fig. 22 b)

However the overall assessment of the digastric behavior during this study (non-collapse and collapse experiments together) did not reveal the development of an identifiable pattern.

In summary it can be seen that:

Study 1

a) In the two subjects whose the digastric muscle was active during the naive experiments it was also active during all

the experiments of the other two studies.

b)In both these cases the pattern and the level of the digastric activity were similar for all the non-collapse and the collapse (n6) experiments.

c)The level of the digastric activity during this study was generally lower than the level of the **PA** during the informed experiments.

Study 2

From the five subjects whose the digastric was usually active during the collapse experiments,it can be seen that:

a.In three cases (GK,JT,AM) **PA** was recorded for the first time having been absent in study 1 .

b.In two cases (Fig. 18a and 22a) the last experiment presented the highest level of **PA** but following the same patterns as the previous ones.

c.In the other three cases (Figs. 19a,20a,21)the **PA** showed a tendency to drop but with a modified pattern of activity.

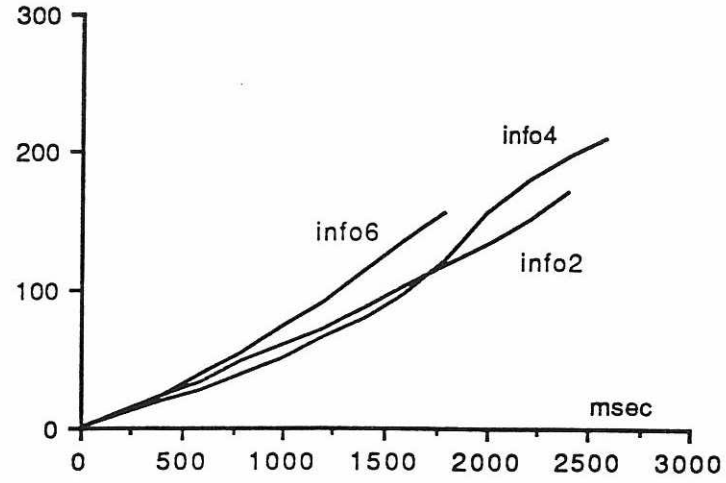
Study 3

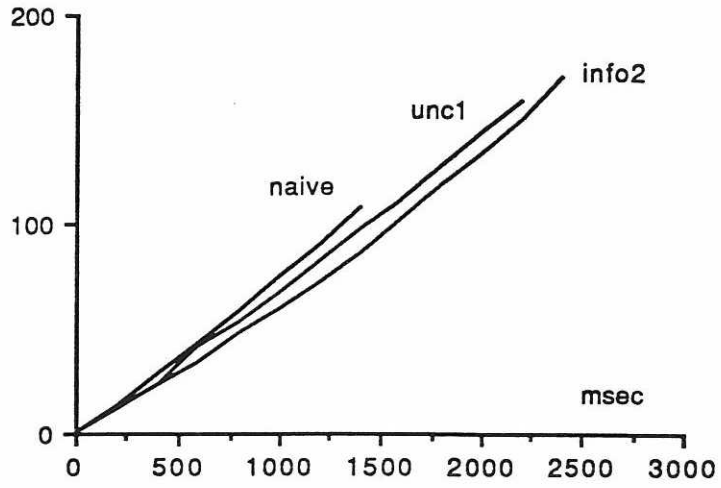
a.In two of the three subjects whose the digastric was active during study 3 (Fig.18c,22b) the continuous tonic rather low level of **PA** in the experiment **(unc1)** was replaced by a higher level of **PA** in the experiment **(unc2)**.

In the other case an opposite situation was recorded.

b.The overall assessment of the digastric behavior during this study (non-collapse and collapse experiments together)

revealed that despite the observed variability at least in one case (TK) a definite pattern was developed.
Neither of the other two subjects showed a consistent pattern
It should be noted that the overall assessment was mainly based upon the potential changes in the digastric activity after the onset of each of the two collapse experiments of this study i.e.the instances at which the nature of the current task was changed.





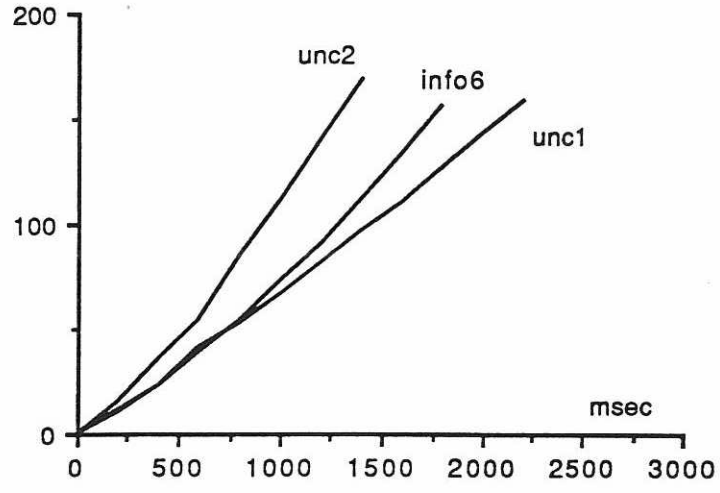
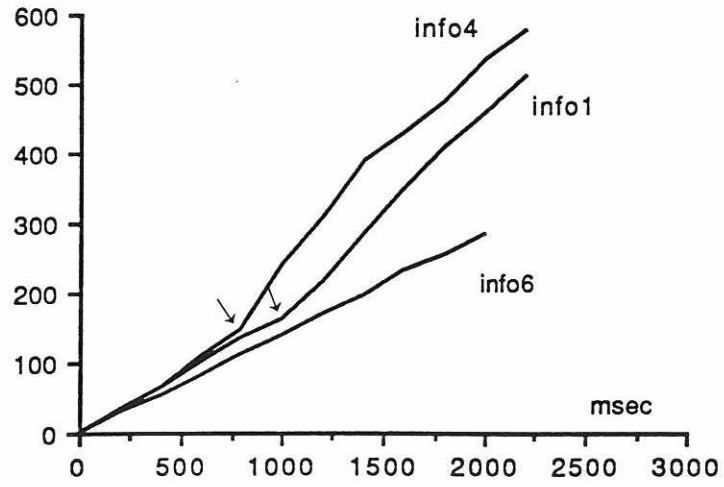


Fig. 18a,b and c. Subject TK. Superimposition of integrated EMGs for direct comparisons.
For further description see text.



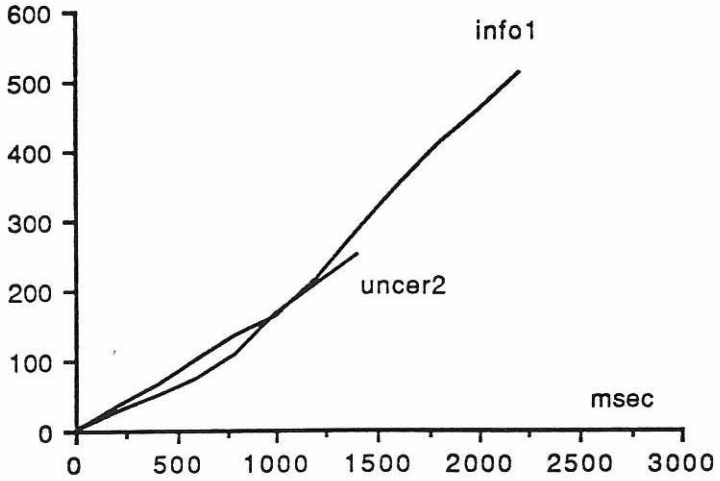


Fig. 19a and b. Subject GK. Superimposition of integrated EMGs for direct comparisons.

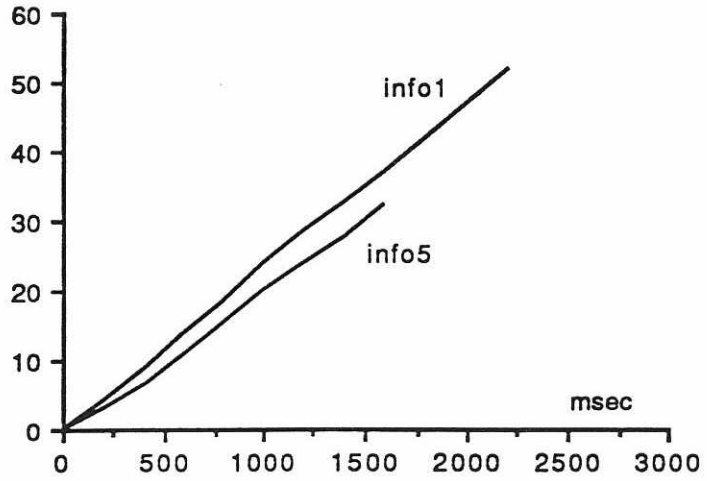


Fig.20 Subject JT. Superimposition of integrated EMGs for direct comparisons.

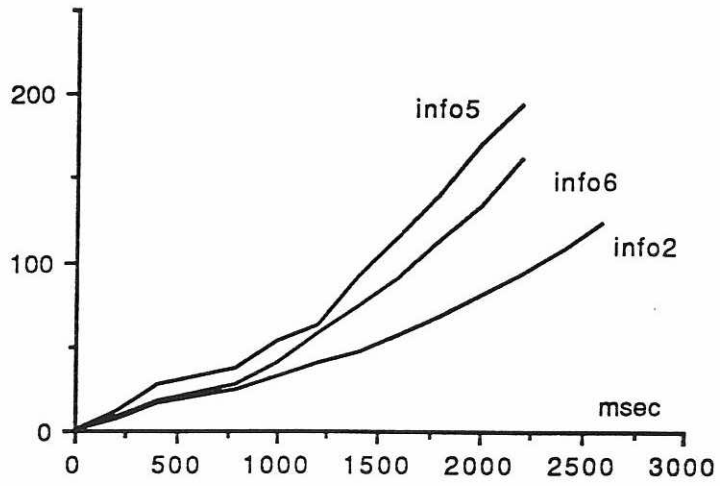
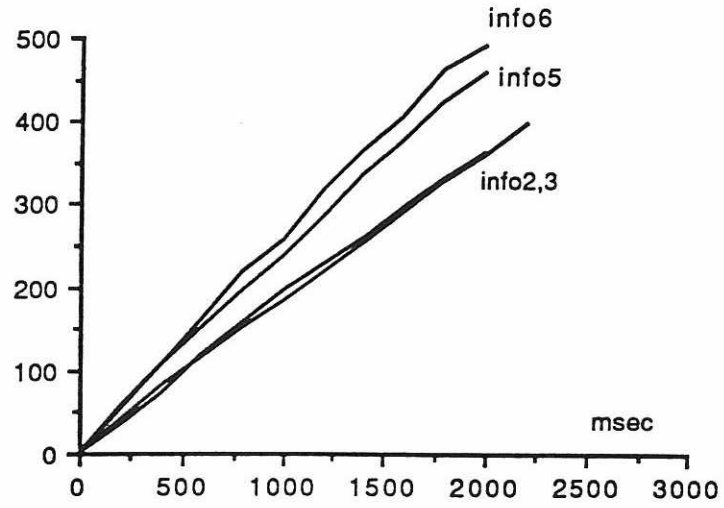


Fig.21 Subject AM. Superimposition of integrated EMGs for direct comparisons.



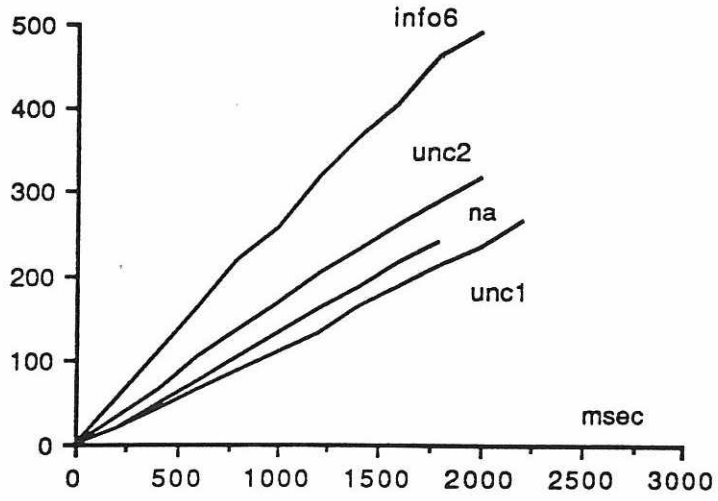


Fig.22 a,b. Subject HK. Superimposition of integrated EMGs for direct comparisons.

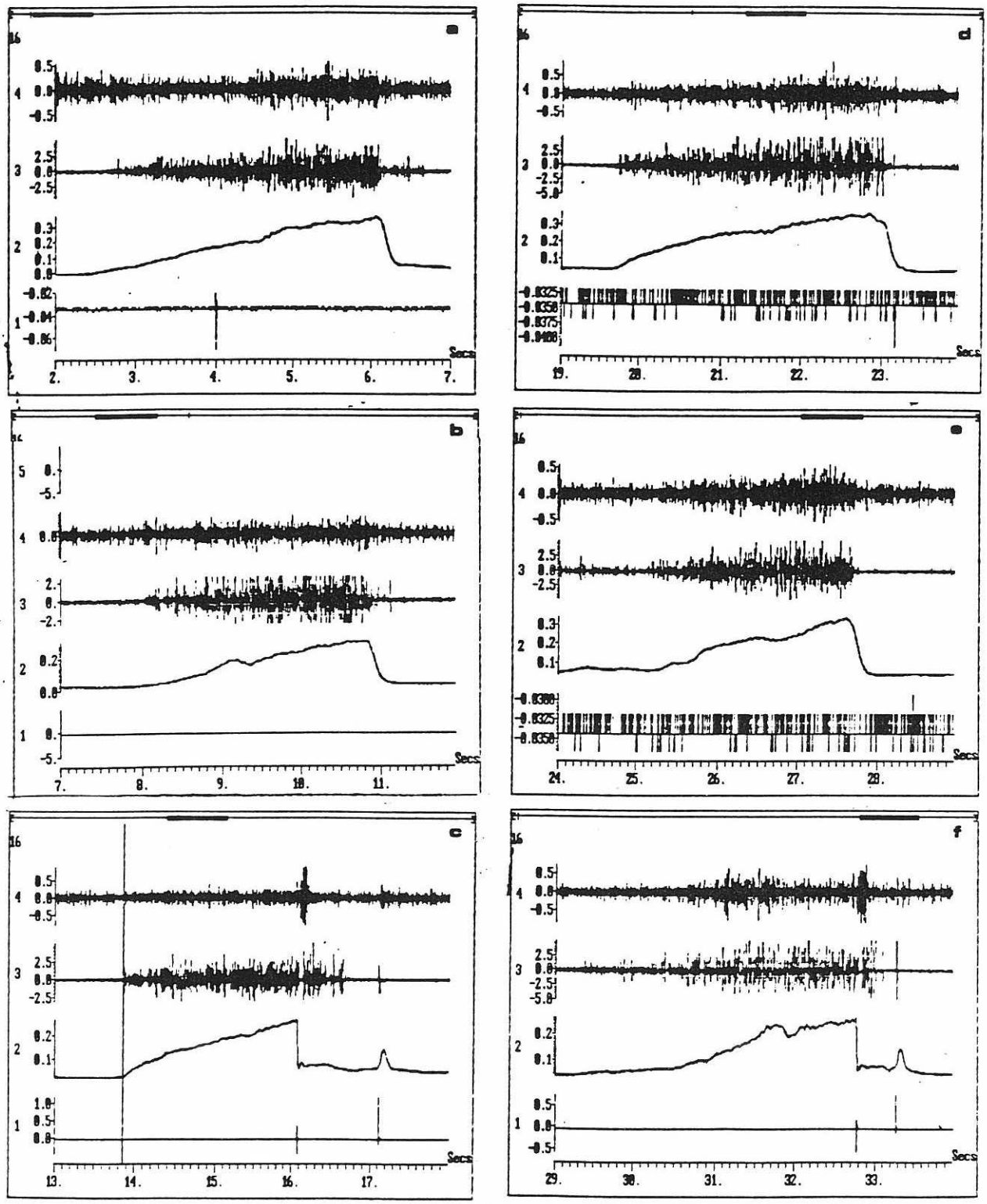


Fig.23 a-f. Subject TK.Study 3.Experiments 1-6.
c=collapse experiment (unc 1)
f=collapse experiment (unc 2)

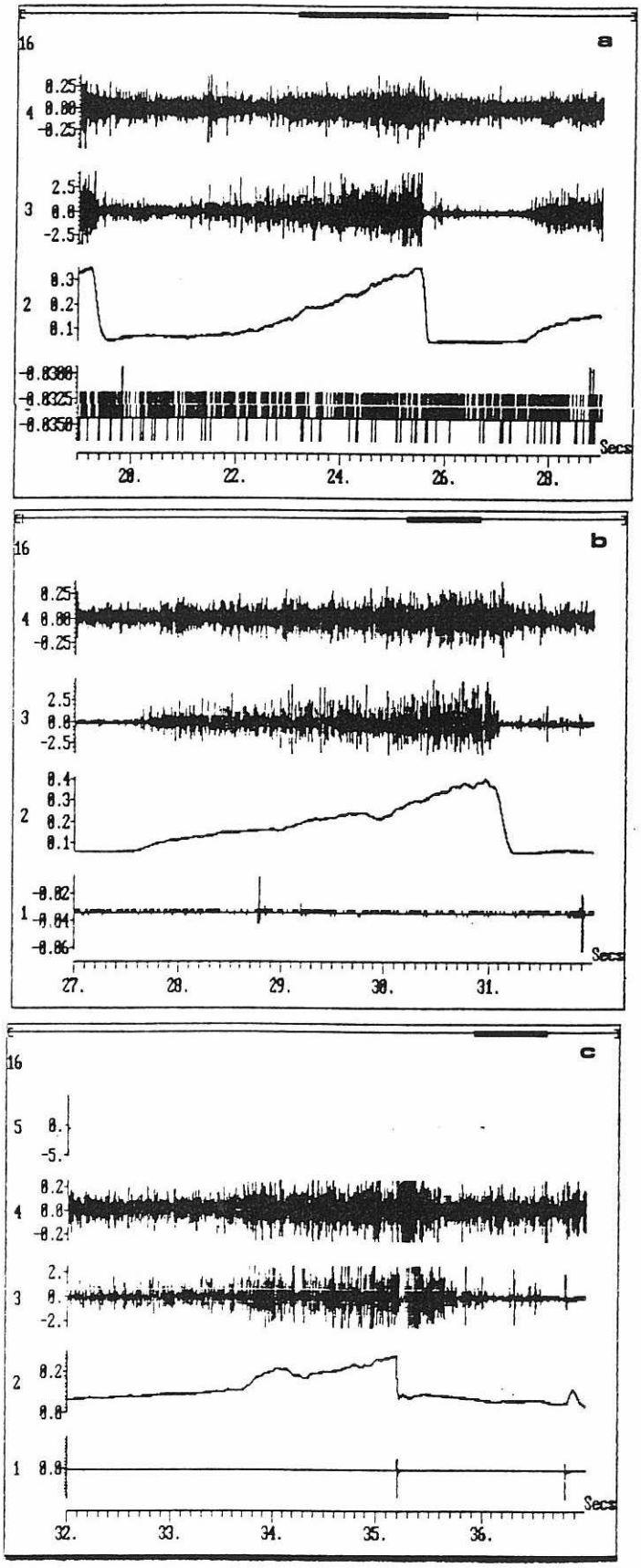


Fig.24 a-c.Recorded data from subject TK. Study 1.
Experiments 4,5,6.
c=collapse experiment (n6)

4.3.Post-collapse reflex events.

Digastric burst

Postcollapse digastric bursts were clearly identified in 53 out of 90 collapse experiments (59%)

It can be seen that only one from the ten subjects who participated in the experiments did not present digastric burst in any of the three studies.

Latencies of the digastric burst for all subjects and experiments are shown in Appendix 3.

Fig 25 (histogram) shows the frequency distribution of the digastric burst latencies for the total experiment.

Similar mean latencies were found in the three studies.

The mean latencies were 34.0 msec for the 1st study ,32.4 msec for the 2nd study,32.3 msec for the 3d study and 32.7 msec for the total experiment.

No significant differences($p>0.1$) were found between the three studies (Table 6)

Descriptive statistics for each particular study (naive,informed,uncertain) as well as for the total experiment are demonstrated in Tables 2,3,4 and 5 respectively.

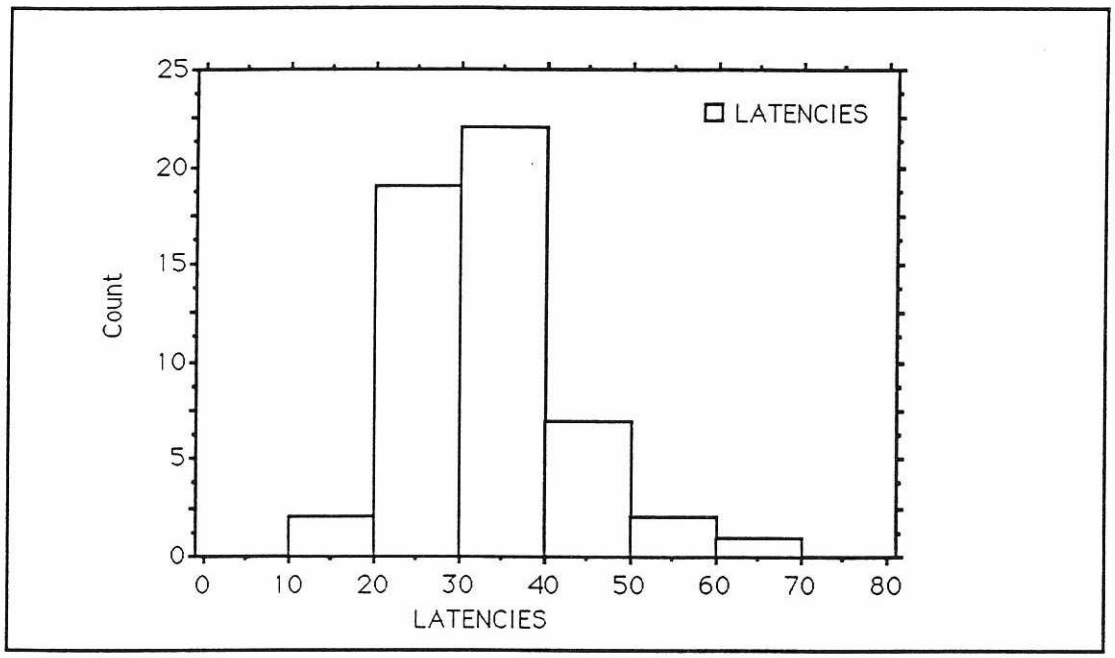


Fig.25. Histogram displaying the distribution of the digastric burst latencies.Interval width 10 msec.

X_1 : NAIVE STUDY 1

Mean:	Std. Dev.:	Std. Error:	Variance:	Coef. Var.:	Count:
34	10.6	3.8	112.9	31.2	8
Minimum:	**Maximum:**	**Range:**	**Sum:**	**Sum of Sqr.:**	**# Missing:**
26	55	29	272	10038	28

X_2 : INFOR. STUDY 2

Mean:	Std. Dev.:	Std. Error:	Variance:	Coef. Var.:	Count:
32.4	9.5	1.6	90.9	29.4	36
Minimum:	**Maximum:**	**Range:**	**Sum:**	**Sum of Sqr.:**	**# Missing:**
18	68	50	1168	41076	0

X_3 : UNCERTAIN STUDY 3

Mean:	Std. Dev.:	Std. Error:	Variance:	Coef. Var.:	Count:
32.3	9.6	3.2	91.8	29.6	9
Minimum:	**Maximum:**	**Range:**	**Sum:**	**Sum of Sqr.:**	**# Missing:**
21	50	29	291	10143	27

X_1 : TOTAL

Mean:	Std. Dev.:	Std. Error:	Variance:	Coef. Var.:	Count:
32.7	9.5	1.3	90.8	29.2	53
Minimum:	**Maximum:**	**Range:**	**Sum:**	**Sum of Sqr.:**	**# Missing:**
18	68	50	1731	61257	0

Tables 2,3,4 and 5. Latency of the digastric burst. Descriptive statistics for studies 1,2,3 and the total experiment

One Factor ANOVA X_1 : Column 4 Y_1 : N

Analysis of Variance Table

Source:	DF:	Sum Squares:	Mean Square:	F-test:
Between groups	2	16.998	8.499	.09
Within groups	50	4704.889	94.098	p = .9138
Total	52	4721.887		

Model II estimate of between component variance = -6.633

One Factor ANOVA X_1 : Column 4 Y_1 : N

Group:	Count:	Mean:	Std. Dev.:	Std. Error:
N	8	34	10.623	3.756
I	36	32.444	9.533	1.589
U	9	32.333	9.579	3.193

One Factor ANOVA X_1 : Column 4 Y_1 : N

Comparison:	Mean Diff.:	Fisher PLSD:	Scheffe F-test:	Dunnett t:
N vs. I	1.556	7.616	.084	.41
N vs. U	1.667	9.467	.063	.354
I vs. U	.111	7.261	4.723E-4	.031

Table 6. Latency of the digastric burst.Statistical evaluation between the three studies.
p:Probability of the observed difference on null hypothesis.

CHAPTER 5.DISCUSSION

5.1.Occurrence of precollapse digastric activity.

The broad field of the dental science has been faced from the earliest days with the problem of understanding the structure and function of the various components of the Stomatognathic System.

The introduction of the Electromyography,offered an additional tool in the particular study of the function of the masticatory muscles.

The digastric muscles whose primary action is the opening of the mouth,are also involved in a number of other functional movements.

Electromyographic studies have revealed a marked activity during coughing,breathing,swallowing and forceful opening of the mouth against resistance(Ahlgren 1977,Koning et al 1978).

A moderate activity was also shown during unilateral molar chewing (in antagonism with elevator muscles) lateral movements and protraction of the jaw with or without tooth contact (Moller 1966,Basmajian and De Luca 1985).

It is also well documented,that the digastric muscle is practically inactive at rest and during unopposed closing movement(except the initial phase,Yemm 1976)

The reflex responses of the anterior digastric muscle during a sudden unloading of the mandibular elevator muscles,were first demonstrated by Hannam,Matthews and Yemm (1968).

Miles and Wilkinson (1982) however,were the first who called attention to the importance of the precollapse digastric activation,in the limitation of the sudden mandibular closing movement.

The development of the digastric activity during the period of the precollapse isometric contraction of the agonist muscles,has been attributed to either a peripheral sensory input,or an "open loop strategy"(Fenyes et al 1960,Miles and Wilkinson 1982,Van Willigen et al 1989)

In general the initial phase of learning a new motor skill appears to favour antagonist co-contraction.(Smith 1981).

On the basis of this concept it is logical to assume that agonist-antagonist co-ordination (as an insurance mechanism) might be a function of foreknowledge and time.

Van Willigen et al (1989) tested this hypothesis with a carefully designed pilot experiment on dental students.

According to their results,jaw - closing and jaw-opening muscles always co-contracted during the tracking task of the experiments.

Anticipatory strategies in terms of changes in the contraction patterns were also recorded.

These strategies however were mainly based on the immediate past performance (current situation) rather than the foreknowledge of the task to be excecuted.

On the assumption that the precollapse digastric activation

is irrelevant to the foreknowledge one might have expected a similar number of subjects with digastric co-contraction in all the three studies.

In the present work however the number of subjects with precollapse digastric activity was slightly bigger in study 2 than studies 1 and 3.

It is also interesting to note,that one of the two subjects with precollapse digastric activity throughout all studies, reported a recent painful experience of tooth cracking during a forceful isometric bite (in chewing).

According to Miles and Madigan (1983) past experience is a potential factor capable of modifying the motor performance. Our results suggest that some correlation might exist between the previous experience,the foreknowledge of the task to be executed and the digastric motor response.

5.2.Patterns of precollapse digastric activity

Considering now the development of the precollapse digastric activity as a time-based function, the data proved to be more interesting.

The striking feature of this time-based analysis was the close association between the patterns (and activity levels) and the nature of the preceding tasks.

Study 1

In both subjects with PA during the naive collapse

experiment (where neither foreknowledge nor immediate past experience of the unloading situation was present) the level and the pattern of the activity were similar for all the experiments.(Fig. 24)

In other words the digastric motor reaction adjusted to the nature of the current task.(sustain isometric contraction)

Study 2

Similarly from the five subjects where the digastric was active during the collapse experiments of study 2 it could be seen that: firstly in two subjects the last experiment of the sequence presented the highest level of PA but keeping the same pattern with the previous ones (Fig.18a,22a) and secondly in the other three subjects the PA during the last experiment of the sequence showed a tendency to drop but with a modified pattern of activity (Fig. 19a,20a,21).

In the first case an obvious shift from lower to higher levels of activity occured.

Apparently the subjects realized from the very beginning the role of co-contraction and adjusted to the current situation by progressively increasing the level of activity from experiment to experiment.

Surprisingly an opposite behavior recorded in the other three subjects. Currently this shift from higher to lower levels of activity is thought to be a function of training.

It appears that learned supraspinal control mechanisms

eliminate the "undesirable" or "useless" coactivation. Nevertheless even a small level of coactivation may decelerate the mandible on the basis of the "stiffness " theory (see below).

Study 3

Study 3 proved to be particularly instructive on the role of **foreknowledge** and **immediate past performance** in the development of the precollapse digastric activity. Superimposition of the integrated EMGs revealed a trend for higher levels of digastric activity in the second collapse experiment (unc 2) of this study.(Fig. 18c and 22b) A further examination of the recorded (raw)EMGs of this study offered additional information about the role of the immediate past performance in the development of the digastric strategies. As mentioned before this examination was mainly focused on the potential changes in digastric activity after the onset of each of the two collapse experiments of this study.

Subject TK

As it can be seen in Fig. 23a and 23b,the digastric muscle co-activated during the tracking task of the first and second **non-collapse** experiments of this study as also happened during the experiments of the previous study 2.(informed) In contrast and despite the subjects foreknowledge of a possible collapse the digastric activity showed an obvious reduction during the next collapse experiment(unc1) Fig 23c.

In the following **non-collapse** and collapse(unc 2) experiments however the digastric immediately regained its activity (Fig.23d,23e-f)
The observed variability in the other two subjects (HK,GM) is presumably inherent to both the complex nature of this study and the relatively small number of experiments involved (six bites).

Based on these findings it is logical to assume that at least in some subjects,the motor program executed by the digastrics during the isometric task of the collapse experiments,is centrally originated and designed on the basis of the immediate past performance (current situation) rather than the foreknowledge of the task to be executed.
This approach seems to fit better with the observed variability in the motor strategies (patterns and levels) from experiment to experiment.
As Van Willigen(1989) suggested,"subjects throw a motor program into activity on the basis of the current task" .
If however the outcome is not the expected a new motor program is used and so on until the ultimate goal is reached. (the mandibular deceleration in this case).
The possibility also that traumatic past experiences (tooth cracking,tongue and cheek biting etc.) may sometimes override the above motor model,can not be excluded.

It is therefore obvious that this digastric co-activation follows an individualized rather than a common pattern for all subjects.

5.3.Clinical significance of the precollapse digastric activity.

The importance of the precollapse digastric activity came into prominence,when Miles and Wilkinson (1982) realized that the timing of the reflex events in the agonist and antagonist muscles(masseteric inhibition- digastric burst) could not account for the rapid arrest of the upward jaw movement as it had been previously thought. The mean latencies for the postcollapse digastric burst,25-30 msec (Miles and Wilkinson 1982) 32.7±9.5 msec (in this study) and for the agonist inhibition 10-20 msec (Hannam,Matthews,Yemm 1968,Lamarre and Lund 1975,Miles and Wilkinson 1982) suggest that these reflexes are not involved in the mandibular deceleration, which normally is completed whithin 30-40 msec (Miles and Wilkinson 1982).

The "**short range stiffness**" of the active muscles,that offer considerable increase in the resistance to passive stretch, even at low levels of activation,may account for the rapid mandibular arrest (Joyce,Rack and Westbury 1969,Grillner 1972,Rack and Westbury 1974).

Rack and Westbury 1974,reported after studies in the cut

soleus muscles,that the response to the first part of unexpected movement ,must largely be dependent upon the physical propertiesof the muscles,since some time is required before any form of reflex response can develop.
Passive tension mainly due to the connective tissue within and around the muscle as well as the tension produced by the contractile part of the muscle it-self (due to formation of cross-bridges between thick and thin filaments in the myofibrils) contribute to the short range stiffness phenomenon.(Grillner 1972).

5.4.Mandibular deceleration in the subjects with no recorded precollapse digastric activity.

So far we discussed the strategies of the control of the mandible after unloading,in the five subjects who presented precollapse digastric activity at least in some experiments.
The question now arises,why some subjects do not develop anticipatory digastric strategies, and how the mandibular deceleration is achieved in those cases ?
A possible explanation would be the well established concept of reciprocal inhibition.
According to this concept an antagonist pause is normaly expected when agonist voluntary contraction is initiated. (Sherrington 1909,Basmajian,De Luca 1985)
This reciprocal inhibition,well demonstrated in rhythmical

motor processes such as mastication,was also recorded at least in the beginning of some of our collapse experiments (Figs.10,17a)

Tilney and Pike (1925) however were the first who offered serious objections to Sherringtons concept as a general reflex model for voluntary movements.

They suggested that under certain circumstances the cerebellum may play an important role in switching between coactivation and reciprocal inhibition of the antagonist muscles.Smith (1981) supported these suggestions by his findings.

Preliminary observations in the area of the Stomatognathic System,suggest that the reciprocal inhibition does not seem to apply in tasks involving sustain isometric contraction especially when the tasks are part of a learning process.(Smith 1981) In these cases some coactivation of the jaw opening muscles may be a more frequent occurence (Pruim et al 1978).

If that were the case a residual and not detectable (within the limitations of our experiment) precollapse digastric activity ,might have accounted for the mandibular deceleration.

Yemm's (1975) suggestion,that because of the introduction of electronic noise,due to the necessary amplification,surface EMGs may not be the appropriate method of detecting low

levels of muscle activity,is compatible with the above idea. Miles and Madigan (1983) based upon observational assessment of EMG recordings,stressed that the amplitude of the digastric EMG although minimal to prevent the achievement of the principal goal (tracking of the ramp) was enough to oppose a sudden elongation on the basis of the stiffness concept.

Finally in the light of the possible implication of higher centers in the regulation of the unloading reflex,multiple parallel mechanisms (and not always identifiable) are expected to contribute to the accomplishment of the mandibular deceleration.

It is also important to note here,that the present study did not enable the direct confirmation of the previously described mandibular deceleration hypothesis.

Although none of the subjects reported that their teeth struck the bars during closure,the lack of a simultaneous recording of the jaw position,precludes any conclusions on this.

To meet this problem,Hannam,Matthews and Yemm (1968) in their original study of the unloading reflex in masticatory muscles developed a recording system (transducer) working on the principle of a variable transformer. Their results established that the mandible was always arrested before any

tooth contact occured.

In 1982 Miles and Wilkinson reached the same conclusion,using a more advanced recording system involving a small light cemented on the lower incisors which was activating a photosensitive position detector giving a continuous readout of the vertical jaw position.

5.5.Post-collapse reflex events .

Digastric burst

An occasional recording of digastric bursts with an average latency of 20 msec was firstly reported by Hannam,Matthews and Yemm (1968).

This was in close agreement with the findings of Miles and Wilkinson (1982) who reported a mean latency of 25±30 msec.

Post collapse digastric bursts were clearly identified in 53 out of the 90 collapse experiments of this study.

However mean latencies were slightly higher than the previously reported (32.7±9.5 msec)

In contrast Beaudreau and associates (1969) reported a mean latency of 90±30 msec on the basis of 13 measurements.

Although no sufficient explanation has been offered for this discrepancy differences may be attributed to the methodology used.

However even a latency of 30-40 msec is remarkably long in comparison to the mean latencies (6-8 msec)for the

monosynaptic jaw closing reflex .(Lamarre and Lund 1975). This led Miles and Wilkinson (1982)

a.to suggest the polysynaptic nature of this reflex with a possible involvement of a suprasegmental pathway and

b.to put forward the theory of the limited (or absent) involvement of this reflex in the mandibular deceleration.

In our study no statistical significant differences were calculated between the mean latencies of the three series of experiments.($p>0.1$) Thus these experiments fail to show that the foreknowledge of the experimental situation influences the latency of the digastric burst.

CHAPTER 6. CONCLUSIONS

On the basis of the results and within the limitations of our experiments,the following conclusions can be drawn .

1.The co-activation of the digastric muscle during the isometric phase of the collapse experiments was nearly consistent in half of the subjects.

2.Evidence has been provided that at least in some subjects the digastric co-activation is subjected to some form of central control.

2.a.Previous experience,foreknowledge of the task to be executed and immediate past performance all contribute to the development of a modifiable digastric co-activation.

2.b.Although the evidence presented in this study is incomplete it seems likely that the immediate past performance is the potentially responsible factor for the development of certain strategies of co-activation.

3.The results for the latency of the digastric burst indicate that a polysynaptic pathway may be involved.

4.No evidence has been provided to suggest some form of relationship between the foreknowledge and the latency of the digastric burst.

PROPOSALS FOR FURTHER RESEARCH

Further studies are needed to improve understanding of the nature of the digastric coactivation,the development of various motor strategies and finally the specific role of the higher centers in the learned behavior processes.
Some suggestions for further research towards this end are:

a.to clarify the role of the immediate past performance on the development of certain motor strategies,larger number of experiments (bites) is needed especially in studies involving more than one task (collapse-noncollapse)
b.the investigation of the role of the biting force and the initial separation of the jaws in the development of the motor strategies will give an insight to the protective nature of this reflex.
c.investigation of the digastric and masseteric motor responses to unloading in both sides of stroke patients might give information about probable suprasegmental involvement.

Technical improvements

Some technical improvements are most desirable:
The recording of the jaw movements and the more accurate determination of the beginning of the biting force might facilitate analysis.

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APPENDICES 1,2,3.

INSTRUCTIONS*

STUDY 1

1.BITE ONTO GAUGE

2.FOLLOW EACH OF THE RAMPS SEEN ON THE SCREEN

3.RELEASE BITE BETWEEN THE RAMPS

4.REPEAT SIX TIMES

STUDY 2

1.BITE ONTO GAUGE

2.FOLLOW EACH OF THE RAMPS SEEN ON THE SCREEN

3.THE MACHINE NOW WILL COLLAPSE !

4.REPEAT SIX TIMES

STUDY 3

1.BITE ONTO GAUGE

2.FOLLOW EACH OF THE RAMPS SEEN ON THE SCREEN

3.THE MACHINE NOW MAY COLLAPSE OR NOT !

4.REPEAT SIX TIMES

PLEASE STOP AT ANY TIME IF YOU FEEL UNCOMFORTABLE !

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* Instructions were given separately before each of the studies.

LATENCY OF DIGASTRIC BURST (msec) COLLECTIVE DATA

SUBJECT	NAIVE	INFORMED						UNCERTAIN	
M.T.	55	43	33						
E.K			30		34				
J.KN	26		25		22		18		
C.M	41	38	22	32	19		21	30	21
T.K.	27	22	26		24	40	30	26	
G.K.	27		38	39	68				
J.T.	29	49	34	30	35	29	44	50	31
A.M.	26	27		26		28	32	46	25
H.K.	41	36	36	39	36	30	33	32	30
A.K.									

Appendix 3. Data collection form, for the latencies of the digastric burst.