

Monitoring of head injury by myotatic reflex evaluation

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Abstract

Objectives—(1) To establish the feasibility of myotatic reflex measurement in patients with head injury. (2) To test the hypothesis that cerebral dysfunction after head injury causes myotatic reflex abnormalities through disordered descending control. These objectives arise from a proposal to use reflex measurements in monitoring patients with head injury.

Methods—The phasic stretch reflex of biceps brachii was elicited by a servo-positioned tendon hammer. Antagonist inhibition was evoked by vibration to the triceps. Using surface EMG, the amplitude of the unconditioned biceps reflex and percentage antagonist inhibition were measured. After standardisation in 16 normal adult subjects, the technique was applied to 36 patients with head injury across the range of severity. Objective (1) was addressed by attempting a measurement on each patient without therapeutic paralysis; three patients were also measured under partial paralysis. Objective (2) was addressed by preceding each of the 36 unparalysed measurements with an assessment of cerebral function using the Glasgow coma scale (GCS); rank correlation was employed to test a null hypothesis that GCS and reflex indices are unrelated.

Results—In normal subjects, unconditioned reflex amplitude exhibited a positive skew requiring logarithmic transformation. Antagonist inhibition had a prolonged time course suggesting presynaptic mechanisms; subsequent measurements were standardised at 80 ms conditioning test interval (index termed “ TI_{80} ”).

Measurements were obtained on all patients, even under therapeutic paralysis (objective (1)). The unconditioned reflex was absent in most patients with GCS less than 5; otherwise it varied little across the patient group. TI_{80} fell progressively with lower GCS, although patients' individual GCS could not be inferred from single measurements. Both reflex indices correlated with GCS ($p < 0.01$), thereby dismissing the null hypothesis (objective (2)).

Conclusion—Cerebral dysfunction in head injury is reflected in myotatic reflex abnormalities which can be measured at the bedside. With greater reproducibility, reflex measurements may assist monitoring of patients with head injury.

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Acute management of the patient with head injury involves close monitoring of clinical condition to detect any deterioration which may presage the development of secondary brain damage. A cornerstone of head injury monitoring is regular assessment of global cerebral function in terms of the patient's level of consciousness. For this purpose, clinical findings are usually standardised according to the Glasgow coma scale (GCS).¹ However, the frequency of GCS findings is rarely more than once hourly because they are labour intensive and also disturb the patient. Because secondary brain damage can develop over a period of minutes, this may go unnoticed between GCS observations. Moreover, the use of neuromuscular blocking agents for elective ventilation may render the patient completely inaccessible to GCS assessment. To address these shortcomings of the GCS, EEG² and evoked potential³ techniques have been applied to monitoring of head injury, but these have not gained widespread acceptance, perhaps because their indices cannot easily be related to the GCS standard. There remains a need for an alternative means of monitoring the cerebral function of patients with head injury which is capable of providing measurements every few minutes, even if the patient is under moderate neuromuscular blockade.

This paper proposes a non-invasive myotatic reflex technique to supplement GCS observations. The technique exploits the strong descending control which spinal reflex circuitry normally receives from supraspinal centres.⁴ It is postulated that cerebral dysfunction after head injury may be reflected in myotatic reflex abnormalities as a consequence of disordered descending influences. Specifically, it is envisaged that inhibition of the phasic stretch reflex from antagonist muscles will be deranged to a degree which reflects the extent of cerebral dysfunction. The argument here is that the normal reciprocal relation between antagonist muscle groups, which clinically seems to be preserved in patients who retain the capacity for purposeful limb movement (obeying commands or localising a painful stimulus), seems to be disturbed in the deeper levels of coma (flexing or extending to pain). It is anticipated that an index of cerebral function based on myotatic reflex findings will correlate better with GCS observations than do EEG or evoked potential indices, as the myotatic reflex is an integral component of the motor system, and motor response is specifically examined in GCS assessment.

To test the above hypothesis, a robust method is required for eliciting the phasic

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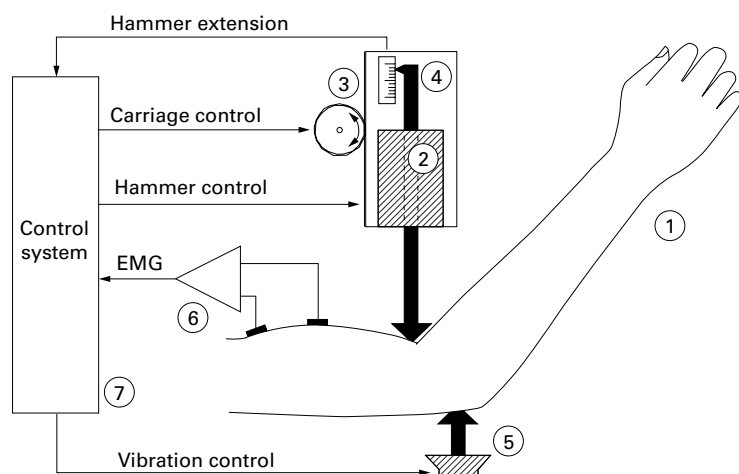


Figure 1 Experimental arrangement. 1=Right upper limb, supported on a gantry; 2=solenoid tendon hammer; 3=motorised carriage which deployed the hammer over the distal tendon of biceps; 4=an infrared transducer to measure hammer extension; 5=vibration generator applied over the distal tendon of triceps; 6=surface electrodes and a differential amplifier to measure biceps EMG activity; 7=microcontroller to drive the hammer, carriage motor and vibration generator, and to record transducer and EMG signals.

stretch reflex at the bedside of patients with head injury and quantifying antagonist inhibition. The method should not depend on patient cooperation and should even be applicable to restless patients. The apparatus should operate automatically if it is to offer the possibility of frequent measurements. As no existing method fulfils these criteria, a novel technique has been developed. This has been standardised and evaluated in a preliminary study of normal subjects. The technique has then been employed in a study of patients with head injury. The first objective of the patient study was to establish the feasibility of bedside measurement of myotatic reflexes in patients with head injury; this includes the ability to undertake measurements in restless patients and in patients under partial neuromuscular blockade for elective ventilation. The second objective of the patient study was to test the hypothesis that cerebral dysfunction after head injury causes myotatic reflex abnormalities through disordered descending control.

Methods

The phasic stretch reflex in biceps brachii has been studied, with antagonist inhibition from the triceps. Test reflexes were evoked in biceps by percussion of the distal tendon with an electromagnetic hammer, the position of which could be automatically adjusted to maintain tendon tap amplitude despite limb movements in restless patients. The amplitude of each test reflex was measured by surface EMG. Antagonist inhibition was evoked by conditioning the biceps reflex with 200 Hz vibration to the triceps (muscle vibration in this frequency range is known to elicit a group Ia dominated afferent discharge).⁵ Using this approach, two myotatic reflex variables may be generated: the absolute amplitude of the unconditioned biceps phasic stretch reflex and the relative conditioning effect of triceps vibration.

APPARATUS

The experimental apparatus comprised seven elements (fig 1):

(1) A bedside gantry on which the patient's upper limb rested. In this study, all measurements were taken from the right upper limb.

(2) A solenoid tendon hammer with a 10 mm travel. A coil spring kept the hammer extended to 10 mm before deployment on the limb, but allowed it to telescope when applied against the skin.

(3) A motorised carriage which deployed the hammer perpendicularly over the tendon of the biceps in the antecubital fossa. As the hammer telescoped as it was applied against the skin, the carriage defined the starting extension from which the hammer would be fired towards its 10 mm limit, and hence defined the amplitude of the tendon tap.

(4) An infrared transducer to measure hammer extension in the range 0 to 10 mm.

(5) A vibration generator applied over the distal tendon of the triceps. This delivered 200 Hz vibration of about 0.5 mm peak-peak amplitude.

(6) Surface electrodes and a differential amplifier to measure the EMG response of biceps to percussion.

(7) A Z80 based microcontroller to drive the hammer, carriage motor, and vibration generator, and to record transducer and EMG signals.

The process of collecting a pair of myotatic reflex measurements began with positioning the hammer. The microcontroller acted as a servo, driving the motorised carriage to advance the hammer against the biceps tendon until the transducer signalled that the hammer had telescoped from its 10 mm fully extended position down to $1 (\pm 0.5)$ mm; firing the hammer from this starting extension would generate a tendon tap of amplitude $9 (\pm 0.5)$ mm.⁶ Without operator intervention, the hammer was then fired repeatedly. Between each tendon tap, any change in starting hammer position due to patient movement was detected by the transducer and servo-corrected by adjustment of the carriage position; a standardised tap amplitude could thereby be maintained even for a restless patient. In pseudorandom sequence, half of all tendon taps were accompanied by a conditioning burst of vibration to the triceps. The biceps EMG responses to 64 unconditioned and 64 conditioned taps were averaged separately, accompanied by records of hammer trajectory from the transducer. Once 128 biceps responses had been recorded, the hammer was automatically withdrawn to avoid pressure necrosis of the skin underlying the hammer head. After data collection, the peak to peak amplitudes of the averaged unconditioned and conditioned EMG records were automatically read and the percentage conditioning effect calculated.

PRELIMINARY STUDY IN NORMAL SUBJECTS

With informed consent and the approval of the local ethics committee, the technique was applied to 16 healthy volunteers aged 19 to 68 (median 30) years; one volunteer was a woman. This preliminary study allowed the rate of

biceps percussion to be standardised and a single interval between triceps vibration and biceps percussion to be selected for conditioned cycles. The reproducibility of the standardised reflex measurements was evaluated. All measurements were made with subjects resting supine on a hospital bed.

Standardising the rate of biceps percussion

If the reflex measurement technique is to have any potential for monitoring head injury it must be capable of providing frequent measurements. However, repeated stimulation of the phasic stretch reflex carries the risk of postactivation depression.⁷ Comparison was therefore made of the amplitudes of unconditioned biceps reflex responses at percussion rates of one tap per 1, 2, 5, or 10 seconds. As the collection of 64 repetitions at the lower percussion rates would be lengthy (raising the possibility of systematic changes in subject arousal), measurements at each rate were divided into four 16 tap subtrials. In each of three subjects, a total of 16 subtrials covering the four percussion rates was made in irregular order. For each percussion rate, data from the four subtrials were then pooled to calculate an average biceps reflex response comparable with a standard 64 repetition study.

Standardising the conditioning-test interval for triceps vibration

To select a standard conditioning-test interval, the time course of inhibition from the triceps was determined in seven subjects. Using a 25 ms (five cycle) burst of vibration, inhibition was assessed for each subject at conditioning-test intervals of -20 ms to +300 ms (corresponding to the hammer being fired 20 ms before and 300 ms after the onset of vibration, respectively). The mean time course for all subjects was constructed, from which the interval associated with greatest mean inhibition was adopted as the standard for all subsequent measurements in normal subjects and patients.

Reproducibility of standardised measurements

Standardised measurements of unconditioned biceps reflex amplitude and antagonist inhibition were assessed in terms of immediate and daily reproducibility. Immediate reproducibility was assessed in eight subjects by making a total of four measurements per individual with an interval of less than 2 minutes between each measurement. Daily reproducibility was assessed in four subjects by making a measurement in each subject at the same time of day on three consecutive days. Both immediate and daily reproducibility were calculated as coefficients of variation (SD/mean), averaged over the subject groups.

STUDY IN PATIENTS WITH HEAD INJURY

The patient study was approved by the local ethics committee. Thirty six patients aged from 17 to 75 years (median 31 years) were studied with informed consent (from relatives if appropriate). They were recruited without exclusion from admissions of adults with head injury to the Neurosurgical Department of Newcastle

General Hospital. Five patients were women. The sample spanned the entire range of severity from mild concussion to brain death. Some patients had undergone craniotomy and some were intubated and ventilated. Measurements were made regardless of concurrent medication, except to distinguish records taken when therapeutic neuromuscular blockade prevented clinical assessment of the level of consciousness.

Applicability of the reflex measuring technique to patients

The first objective of the patient study was addressed by attempting a reflex measurement on every recruited patient at a time when he or she was not receiving therapeutic paralysis. The ability to measure reflex responses under therapeutic paralysis was also tested in three patients receiving intermittent pancuronium for elective ventilation. The depth of neuromuscular blockade in these cases was only moderate (usually two-three twitches in the "train of four" estimation),⁸ but it was sufficient to abolish any visible motor response to pain.

Hypothesis test

The second objective of the patient study was addressed using a cross sectional experimental design in which a reflex measurement in each patient was related to a clinical assessment of cerebral function. The reflex measurements in question were those made on the patients in the absence of therapeutic paralysis (as described above). In each patient, the measurement was immediately preceded by a clinical assessment by the same examiner (JAC) using the 14 point GCS. In accordance with the standard method of assessing GCS, the best motor response of all limbs was recorded, even if the motor response in the right upper limb (upon which reflex measurements were being made) was poorer. Most assessment-measurement pairs were undertaken within the first 5 days of brain injury; all were within 21 days. A null hypothesis was formulated to the effect that neither unconditioned biceps reflex amplitude nor antagonist inhibition measurements bear any relation to GCS score across the patient sample. The null hypothesis was tested by Kendall rank correlation.

Results

PRELIMINARY STUDY IN NORMAL SUBJECTS

In each of the 16 normal subjects, biceps percussion elicited a biphasic EMG response similar to that illustrated in fig 2. The latency of the response ranged from 16.8 ms to 23.4 ms among the subjects. On the basis of its latency and waveform, this EMG response is assumed to represent the biceps phasic stretch reflex (see Discussion); it is henceforth termed the "biceps reflex". Over the sample of 16 subjects, the peak to peak amplitude of the unconditioned biceps reflex exhibited a positively skewed frequency distribution (kurtosis=2.27; skew=1.62; fig 3 A). The skew was ameliorated by logarithmic transformation (kurtosis=0.75;

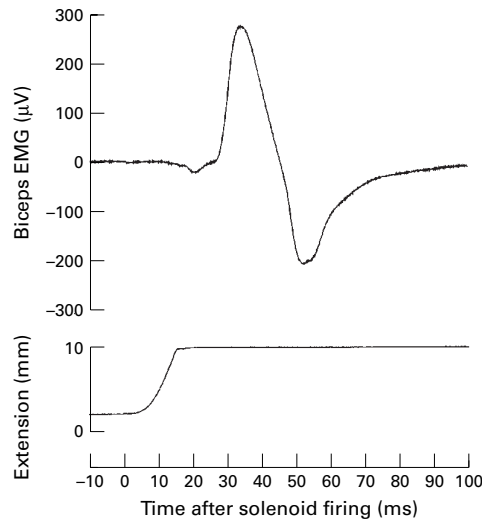


Figure 2 Biceps EMG response to percussion in a normal subject. Upper trace=EMG activity recorded over biceps; lower trace=hammer trajectory as simultaneously recorded by the transducer. Both traces represent records averaged over 64 trials.

skew=-0.54; fig 3 B). For this reason, logarithmic transformation was applied to all biceps reflex amplitude measurements reported.

Standardising the rate of biceps percussion

Figure 4 shows that the log amplitude of the unconditioned biceps reflex is scarcely influenced by changes in percussion rate from one tap per second to one per 10 seconds. To minimise data collection time, the rate of biceps percussion was therefore standardised at one

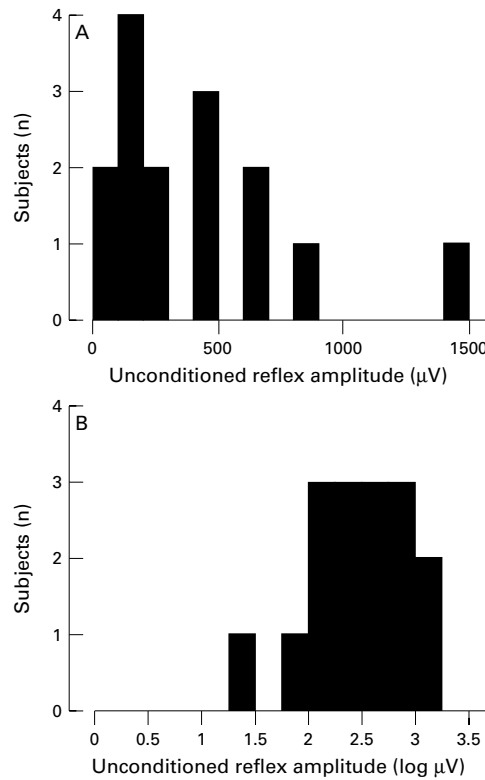


Figure 3 Frequency distribution of unconditioned biceps reflex amplitudes in 16 normal subjects. (A) Distribution of the untransformed amplitudes. (B) Effect of logarithmic transformation.

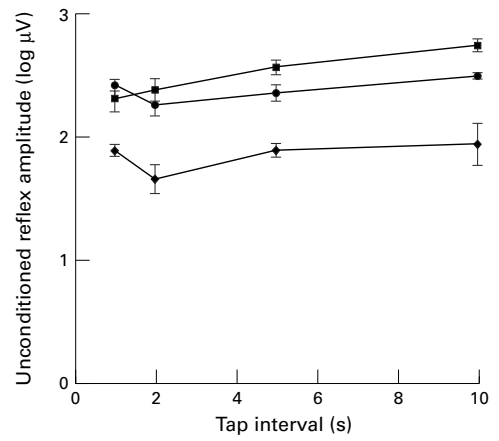


Figure 4 Dependence of the normal biceps reflex on rate of percussion; log amplitudes of the unconditioned biceps reflex elicited by percussion at rates of one tap per 1, 2, 5, or 10 seconds. Each set of data points represents findings in one normal subject (mean (SE)).

tap per second for all subsequent measurements in normal subjects and patients. This allows collection of 128 reflex responses in less than 3 minutes (including data storage and analysis). In restless patients, the occasional need for a large adjustment of carriage position sometimes introduced an additional delay of a

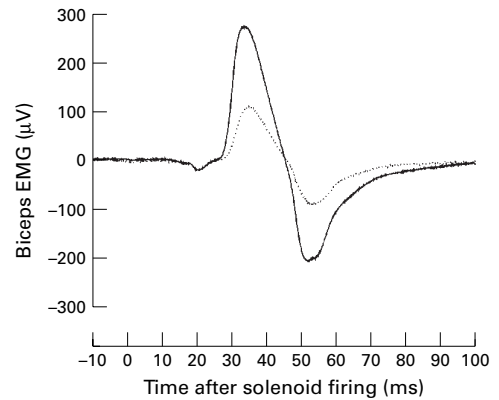


Figure 5 Antagonist inhibition of the biceps reflex in a normal subject. Solid trace=unconditioned biceps reflex response to percussion alone. Broken trace=biceps response conditioned by triceps vibration for 70 ms before hammer firing.

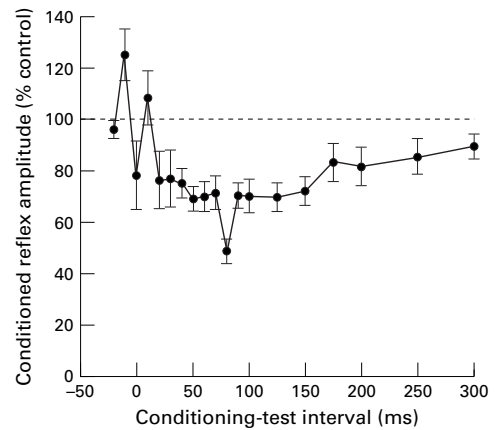


Figure 6 Time course of antagonist inhibition. Conditioning of the biceps reflex by five cycles of vibration to triceps. For each conditioning test interval, the mean conditioning effect (SE) is shown for seven normal subjects.

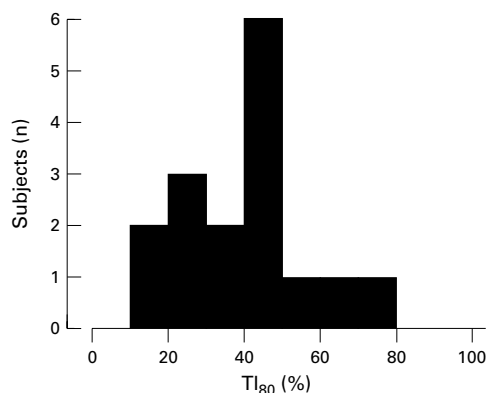


Figure 7 Frequency distribution of TI_{80} in 16 normal subjects.

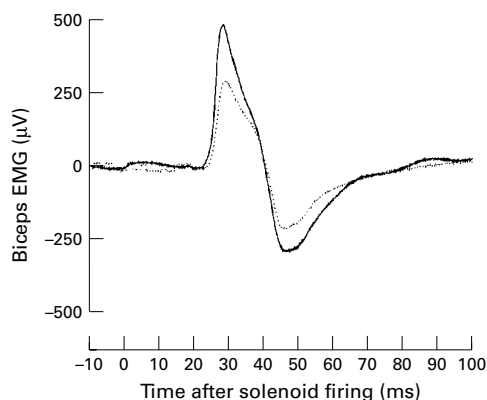


Figure 8 Biceps reflex and antagonist inhibition recorded under therapeutic paralysis. Solid trace=unconditioned biceps reflex response to percussion alone. Broken trace=biceps response conditioned by triceps vibration for 70 ms before hammer firing. This record was made in a patient with head injury receiving intermittent pancuronium for ventilation. At the time of recording, no motor response to pain was discernible. During a temporary reduction in paralysis shortly before this record was taken, the patient localised a painful stimulus, but did not open his eyes (endotracheal tube in situ).

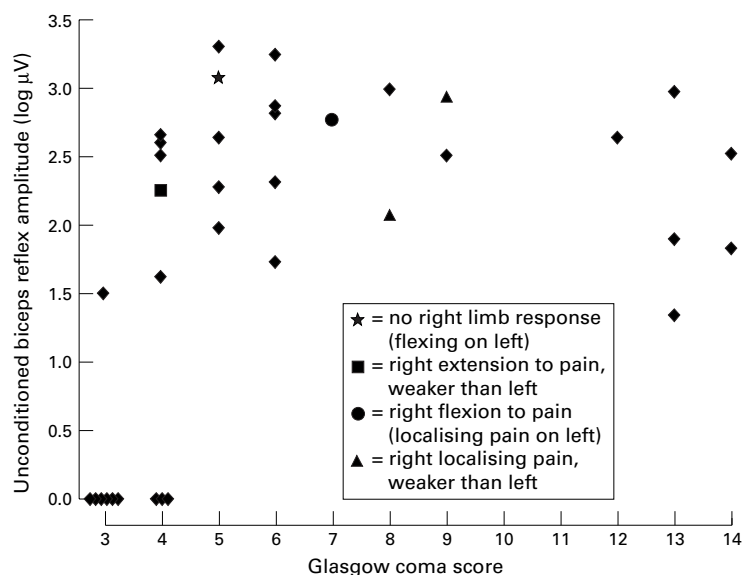


Figure 9 Scattergram of unconditioned biceps reflex amplitude in 36 patients with head injury. For each patient, the log transformed amplitude of the unconditioned biceps reflex is plotted against GCS as found immediately before reflex measurement. The diamonds denote most patients, in whom the clinical motor response in the right upper limb corresponded to the best motor response (used to derive GCS). Other symbols are described in the box.

few seconds between taps; the data presented in fig 4 suggest that this is unlikely to have influenced the amplitude of the reflex responses.

Standardising the conditioning test interval for triceps vibration

Vibration to triceps evoked inhibition of the biceps reflex in a normal subject is shown in fig 5. Figure 6 illustrates the mean time course of this effect in seven subjects. The conditioning-test interval associated with greatest mean inhibition was 80 ms. A 25 ms burst of triceps vibration beginning 80 ms before biceps tendon tap was therefore adopted as the standard conditioning stimulus for evoking antagonist inhibition. The conditioning effect at this interval is henceforth termed “ TI_{80} ” (triceps inhibition at 80 ms). TI_{80} is expressed as the percentage inhibition of the mean conditioned biceps reflex below the unconditioned value; for example, unconditioned and conditioned peak to peak amplitudes of 100 μV and 75 μV , respectively, are reported as $TI_{80}=25\%$. The frequency distribution of TI_{80} measurements in 16 normal subjects is shown in fig 7. Kurtosis for this sample is 0.84; skew 0.54.

Reproducibility of standardised reflex measurements

Average coefficients of variation for the immediate and daily reproducibility of unconditioned biceps reflex amplitude measurements were 6.2% and 8.0%, respectively. Corresponding values for TI_{80} were 16.1% and 15.2%, respectively.

STUDY IN PATIENTS WITH HEAD INJURY

Applicability of the reflex measuring technique to patients

The first objective of the patient study was attained by completing a measurement of unconditioned biceps reflex amplitude and antagonist inhibition in every recruited patient. In one violent patient, a successful measurement was only achieved at the second attempt. In all patients, except some exhibiting the lowest levels of consciousness (see later), biphasic EMG responses to biceps tendon tap could be recorded which were comparable in waveform and latency to those found in normal subjects. Similar biphasic responses were also recorded in the three patients who were studied while undergoing therapeutic paralysis by intermittent pancuronium infusion (fig 8).

Hypothesis test

From the 36 patient cross sectional study, scattergrams of reflex measurements plotted against GCS score are shown for log amplitude of the unconditioned biceps reflex (fig 9) and for TI_{80} (fig 10). No biceps EMG response to tendon tap could be discerned in nine patients, all of whom had GCS scores less than 5. For the purpose of analysis, both reflex variables were given zero values in each of these patients.

Kendall’s rank coefficient, τ_b , for correlation of unconditioned biceps reflex log amplitude with GCS was 0.3939 ($p=0.002$). For correlation of TI_{80} with GCS, τ_b was 0.6531

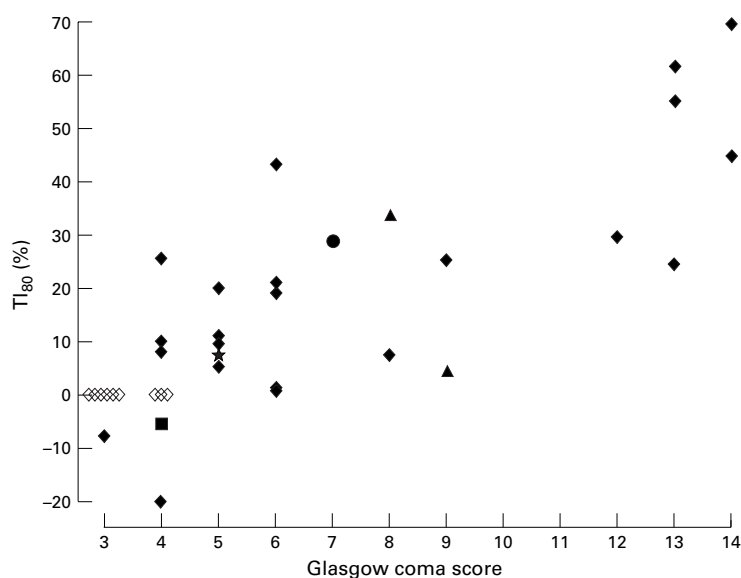


Figure 10 Scattergram of TI_{80} in 36 patients with head injury. Format and symbols as in fig 9. Also, the nine patients in whom no biceps reflex response could be recorded are distinguished by open diamonds: in these people TI_{80} has been set at zero.

($p < 0.001$); even if the TI_{80} values were disregarded for patients in whom no biphasic EMG response was recorded, τ_b only fell to 0.5323 ($p < 0.001$). As all of these coefficients were associated with null probabilities less than 0.01, the null hypothesis was dismissed. These data therefore support the hypothesis that cerebral dysfunction after head injury is reflected in myotatic reflex abnormalities.

In figs 9 and 10, the five patients are distinguished in whom the motor response of the right upper limb was found during clinical assessment to be poorer than the response of the left upper limb. Despite the fact that reflex measurement in these asymmetric cases was not made in the limb which determined the GCS score, there was no consistent tendency for reflex variables to regress as if the local right limb behaviour were the dominant influence upon reflex response.

Discussion

PRELIMINARY STUDY IN NORMAL SUBJECTS

The biphasic EMG response elicited in each subject by the experimental apparatus is assumed to represent the phasic stretch reflex in biceps brachii as it is comparable with published records of this for both waveform⁹ and latency.¹⁰ The positive skew of unconditioned reflex amplitude in the sample of 16 normal subjects is similar to that which has been reported for phasic reflexes in several upper and lower limb muscles.¹⁰ The prolonged antagonist inhibition of the biceps reflex which is evoked by triceps vibration has not previously been reported using tendon percussion, although a similar depression of human soleus H reflex has been shown to follow tibialis anterior vibration.¹¹ In the second case, evidence was obtained which suggested a component of presynaptic inhibition. It seems reasonable to postulate that this is evoked by activity in group Ia afferent fibres, as muscle vibration is known

to excite muscle spindle primary endings preferentially.⁵

Reproducibility of measurements

The standardised measure of antagonist inhibition from triceps, TI_{80} , is not as reproducible as the measure of unconditioned biceps reflex amplitude. This may reflect variability in the triceps group Ia afferent volley generated by the vibrator: there is some evidence that vibration excitation of muscle spindle primary endings is more critically dependent on stimulus variables (particularly the force with which the vibrator is applied to the skin) than had previously been appreciated.¹² This raises the prospect of improving TI_{80} reproducibility by employing more complex servo-controlled vibration delivery—in effect extending to the conditioning stimulus the same regulatory mechanisms that were incorporated to ensure consistency of tendon percussion in the face of patient restlessness.

STUDY IN PATIENTS WITH HEAD INJURY

Applicability of the reflex measuring technique to patients

It has been demonstrated that myotatic reflex measurements may be made on patients with head injury without requiring their cooperation. The ability to record reflex responses in patients rendered inaccessible to GCS assessment by moderate therapeutic paralysis is of particular interest. However, pharmacological effects on the myotatic reflex should be considered. Clearly, progressive deepening of neuromuscular blockade will diminish (and ultimately abolish) the EMG response accompanying a given reflex efferent volley from the biceps α -motor neuron pool. Although partial attenuation of the EMG response would diminish unconditioned reflex amplitude measurements, it would affect unconditioned and conditioned reflex responses equally and therefore leave TI_{80} measurements unaltered. Nonetheless, it is conceivable that paralytic agents might influence myotatic reflex mechanisms at points other than the output stage. In practice, central effects seem unlikely, as these agents tend not to cross the blood-brain barrier in significant quantities.¹³ However, non-depolarising blockade is known to extend to the intrafusal fibres of muscle spindles¹⁴; by unloading spindle receptors, this might theoretically reduce their sensitivity. For the reasons discussed above, a universal reduction in sensitivity to percussion would not affect TI_{80} , but reduced sensitivity to vibration would reduce TI_{80} . The influence of therapeutic paralysis on myotatic reflex measurements would require systematic investigation before this measurement technique could be applied clinically to the monitoring of electively ventilated patients. The influences of other medications employed in head injury management, such as sedatives, anticonvulsants, and analgesics, also require consideration (see below).

Reproducibility of measurements

The reproducibility of myotatic reflex measurements in patients with head injury requires further investigation. As well as the issue of vibration delivery discussed above, the reproducibility of reflex measurements may presumably also be degraded by restlessness in certain patients. To some extent servo-regulation of stimulus delivery (particularly if extended to vibration) should minimise the variability introduced by actual limb movement. However, variable reinforcement of the myotatic reflex may be anticipated in restless patients, either through contraction of the biceps and triceps muscles themselves¹⁵ or of remote muscles.¹⁶ These central effects may impose a limit on the reproducibility of reflex measurements in restless patients. The reproducibility of reflex measurements in patients could not be assessed with the present bulky apparatus, as it would have interfered with nursing care if left at the bedside. Future miniaturised apparatus might be left attached to the limb; if this were coupled with data processing equipment capable of recording individual responses, then variability might be quantified and continuous moving-window averaging employed over a period selected for the desired degree of reproducibility.

Hypothesis test

The cross-sectional study of 36 patients supports the hypothesis that cerebral dysfunction after head injury is accompanied by myotatic reflex abnormalities. For the unconditioned biceps reflex, the abnormality is confined to patients with GCS scores less than 5, among whom there is a distinct subgroup in which the biceps reflex is unrecordable. For TI_{80} , there is a continuum of abnormality across the patient group, greater in those with lower GCS.

It might be argued that differences in medication could account for the differences in reflex values between patients of differing conscious level. In particular, selective use of sedation for some more severely injured patients might confound a study of this nature. As discussed above, the effects of sedatives and other medications on reflex variables certainly require further investigation. However, it is noteworthy that none of the patients reported here with GCS less than 5 were receiving sedation at the time of reflex measurement.

Utility of myotatic reflex measurements in head injury monitoring

The data obtained thus far do not permit direct inference of a patient's absolute level of consciousness (GCS) from a single reflex measurement. Direct inference of GCS from TI_{80} measurements might be possible in future if reproducibility could be enhanced by servo-regulated vibration delivery (see above). Although measurement of unconditioned biceps reflex amplitude would seem to be of little value in isolation, the association of absent biceps response with low GCS might be exploited in TI_{80} monitoring by setting $TI_{80}=0$

where biceps response is unrecordable (as shown by the open data points of fig 10).

Ultimately, the clinical utility of a cerebral function measurement in head injury monitoring depends more on its ability to reflect changes in clinical state than on its absolute correlation with GCS. A longitudinal study is required using the miniaturised apparatus proposed above to provide repeated TI_{80} measurements for correlation with any changes in GCS.

Mechanisms of reflex derangement in head injury

No attempt has been made here to investigate the pathophysiology of the abnormal myotatic reflex responses seen in the patients with more severe head injury. The abnormalities are consistent with disruption of tonic descending facilitation of the phasic stretch reflex and antagonist presynaptic inhibitory mechanisms. Animal studies have long since demonstrated that the reticulospinal, vestibulospinal, rubrospinal, and corticospinal tracts make direct and indirect excitatory connections with α -motor neurons whereby they might facilitate the phasic stretch reflex.⁴ In humans, there is evidence of corticospinal facilitation of α -motor neurons of some muscle groups.¹⁷ However, it is not known which, if any, facilitatory descending tracts are tonically active in healthy, resting humans. Moreover, indirect inhibitory influences on the phasic stretch reflexes of some muscles (particularly soleus) have been shown in humans to arise from the cerebral cortex and the vestibular system.^{18, 19} Thus a putative loss of descending facilitation in some patients with head injury would presumably represent a shift in net balance between excitatory and inhibitory influences, rather than pure loss of facilitation.

Similar arguments apply to presynaptic Ia inhibitory mechanisms. In the cat, interneurons in the pathway of Ia presynaptic inhibition are known to receive both excitatory and inhibitory descending connections.²⁰ In humans, galvanic stimulation of the vestibular system facilitates Ia presynaptic inhibition of the soleus H reflex¹⁹; transcranial magnetic stimulation of the corticospinal tract decreases the same.²¹ The vestibular system is an attractive candidate for the putative source of tonic facilitation of presynaptic inhibition which might be reduced in severe head injury, as the centripetal hypothesis of concussion syndrome²² would predict greater disruption of mesencephalic vestibular circuitry in patients with greater disturbance of cerebral function after acute head injury. Perhaps in the five patients exhibiting clinical asymmetry this was due to focal diencephalic dysfunction which was unrelated to mesencephalic injury and therefore had little influence on the reflex responses found.

Conclusion

It is concluded that abnormalities of the myotatic reflex are seen in head injury as a consequence of disordered descending supraspinal control of spinal reflex circuits. Because a technique has been demonstrated

for quantifying such abnormalities at the bedside, this may be a means of supplementing conventional clinical monitoring of cerebral function in patients with head injury if reproducibility can be sufficiently enhanced.

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