The consistent nature of the nociceptive modulation of jaw reflexes

GUILLAUME GARDON-MOLLARD, ANDREW MASON AND SAMUEL CADDEN. The Dental School, University of Dundee, Scotland.

Background and Aims

Background:

- Electrical stimulation of the lip can produce reflex inhibitions of activity in human jaw closing muscles at a latency of around 40 ms (e.g. refs[1, 2]).
- Painful stimulation elsewhere on the body can suppress this reflex (for review, see ref[3]).
- Other inhibitory jaw reflexes have been shown to vary between individuals but to be consistent in any one individual over a period of time.[4]

Aims:

To investigate, over a period of time:

- whether the inhibitory reflex evoked by stimulation of the lip is consistent in individual subjects, and more particularly:
- whether the modulation of this reflex by remote nociceptive stimuli is consistent in individual subjects.

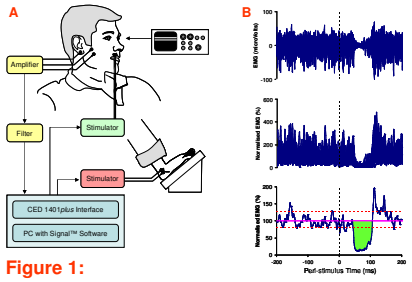

Figure 1:

(A) The experimental set up. (B) The analysis sequence: Top record: superimposed raw EMG sweeps; Middle record: following rectification, smoothing and normalisation to the mean pre-stimulus level of activity; lower record: the averaged record. The shaded area represents an inhibitory reflex.

Materials and Methods

- Experiments were performed on 8 volunteer subjects.
- Two identical experiments were carried out on each subject with an interval between experiments of at least 3 days.
- EMG recordings were made from a masseter muscle while the subjects maintained a level of approximately 12.5% of maximum with the aid of visual feedback (Fig. 1A).
- Electrical test stimuli (1ms duration) were applied to the hair-bearing skin of the upper lip. These stimuli were usually of an intensity equivalent to 2.2x the threshold for producing a sharp sensation - designated the "nociceptive threshold" (but see Results).
- Electrical conditioning stimuli (15mA, 1ms) were applied over the sural nerve, 100ms before the test stimulus. These were invariably described by the subjects as painful.
- Presentation of test stimuli alone or conditioning-test stimulus combinations were randomised to minimise the likelihood that the effects of condition were secondary to the stress of repeated painful stimuli.[5]
- Following rectification and averaging of the EMGs, inhibitory reflexes were quantified by integration (Fig 1B).
- Statistical comparisons were made using Paired students *t*-tests and relationships were investigated by determining Pearson's correlation co-efficient.
- Data are presented as means ± S.E.M.

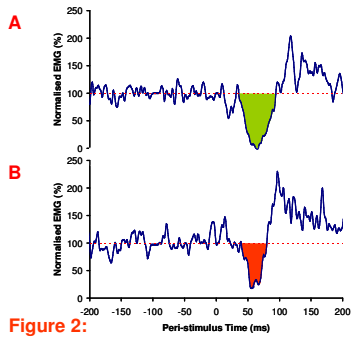

Figure 2:

Processed EMGs from a typical experiment for a single subject: (A) test stimuli alone and (B) conditioning-test combinations. The shaded areas represent the inhibitory reflex.

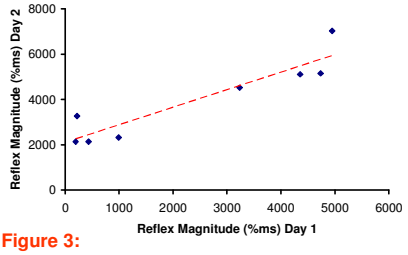

Figure 3:

A scatter plot (with regression line) of the control reflex sizes from day 1 and day 2.

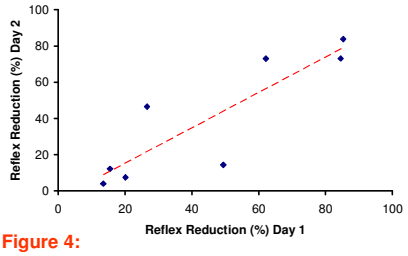

Figure 4:

A scatter plot (with regression line) of the condition-induced modulations from day 1 and day 2.

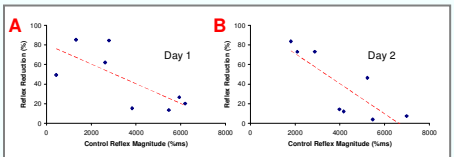

Figure 5:

Scatter plots (with regression lines) illustrating the relationships between control reflex magnitudes and modulatory effects on: (A) day 1 and (B) day 2.

Results

- Inhibitory reflexes (e.g. Fig. 2) were evoked in all 8 subjects.
- In 6 subjects, clear reflexes were evoked with the standard test stimulus intensity of 2.2× nociceptive threshold. However in the other 2 subjects a clear reflex was observed only when the test stimuli were higher than 2.2× the nociceptive threshold.
- In the first experiment on each subject, the reflex had a mean latency of 44 ± 3ms and a mean duration of 57 ± 9ms for the test-stimulus-only presentations in the randomised sequences. Although the corresponding figures in the second experiments were slightly different (latency, 39 ± 3ms; duration, 64 ± 7ms), these differences were not significant (P = 0.3 and P = 0.2 respectively).
- There was a strong correlation between the magnitudes of the reflexes for individual subjects recorded in the two experiments (Pearson's r = 0.832, P = 0.01). (Fig 3).
- In both experiments, the magnitude of the reflex was significantly smaller following conditioning-test stimulus combinations than following test stimuli alone (e.g. Fig 2).
- In the first experiment on each subject, this conditioning-induced reduction of the reflex was by 45 ± 11%. In the second experiments, the mean reduction was slightly less (by 39 ± 12%) but this difference was not significant (P = 0.4).
- There was a strong positive correlation (Pearson's r = 0.87, P = 0.005) between the extents of the conditioning-induced modulatory effects in the 2 experiments (Fig. 4).
- On each day, there was a negative correlation between the extents of the conditioning-induced modulatory effects and the unconditioned magnitude of the reflex (Experiment 1: Pearson's r = -0.72, P = 0.04, Fig. 5A; Experiment 2: Pearson's r = -0.78, P = 0.02, Fig. 5B).

Conclusions

From the data obtained in this short study, it appears that:

- The timing and size of inhibitory jaw reflexes evoked by stimulation of the human lip, are consistent in any one subject, at least over a short period of time.
- The nociceptive modulation of this inhibitory jaw reflex is also consistent over such a period of time.
- One possible explanation for this latter finding, is that stronger reflexes were more difficult to modulate and that subjects with strong reflexes on one day were likely to have these in the second experiment also. This hypothesis is supported by the current data - although it should be noted that the (negative) correlation between the modulatory effects and the reflex magnitude was rather weaker than the (positive) correlation between the extent of the inhibitory effects on the different days.

References

1: Yu S, Schmitt A & Sessle BJ. *Arch Oral Biol.* 1973; **18**: 861–870.

2: Cadden SW & Newton JP. *Arch Oral Biol.* 1988; **33**: 863–869.

3: Cadden SW. *Arch Oral Biol.* 2007; **52**: 370–373.

4: Lauweryns I, Van Cauwenberghe N, Carels C, van Steenberghe D, Vlietinck R. *Arch Oral Biol.* 1992; **37**: 979-982.

5: Mason AG, van der Glas HW, Scott BJJ & Cadden SW. *Exp Brain Res.* 2007; **182**: 81–91.