Quantitative Analysis of the Oculocardiac Reflex by Traction on Human Extraocular Muscle

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The oculocardiac reflex was quantitatively studied in 15 patients with strabismus. The reflex was observed in all patients when the medial rectus and inferior oblique muscles were stretched; the medial rectus muscle had a lower threshold than the inferior oblique. Bradycardia was evoked in 7 of the 15 patients when the lateral rectus was tractioned with tensions of 50 g and 600 g. The oculocardiac reflex was a graded phenomenon as a function of tension applied to the extraocular muscles. As tension was increased, bradycardia occurred rapidly and became deep. Systemic administration of atropine prevented completely the bradycardia from occurring. The results suggest that the response of the extraocular muscles to stretch are critically mediated through a polysynaptic path to the heart, resulting in suppression of the heart rate. Invest Ophthalmol Vis Sci 27:1160–1164, 1986

The oculocardiac reflex is a physiological response of the heart to physical stimulation of the eye or the ocular adnexa, characterized by bradycardia or arrhythmia, which sometimes leads to cardiac arrest.1 The major pathway mediating the oculocardiac reflex seems to consist of an afferent link through the ophthalmic portion of the trigeminal nerve to the vagus nuclei and an efferent link through the vagus nerve to the heart.2,3

It has been shown that the oculocardiac reflex occurred most frequently when the medial rectus was stimulated, but the incidence of the reflex with stimulation of the medial rectus has varied among previous reports.4,5 Such results suggest the reflex may differ in different people and for different muscles.

In order to understand the mechanism of the reflex and discrepancies on previous data, the relationship between input and output in the reflex should be quantitatively analyzed. Previous studies, however, have not explored the relationship between oculocardiac reflex and tension applied to extraocular muscles. We, therefore, have studied the oculocardiac reflex quantitatively during strabismic surgery. We measured heart rate as an instantaneous frequency and tension of traction on extraocular muscles, and have tried to clarify whether the depth of the oculocardiac reflex is related to the strength of tension applied to extraocular muscles.

Materials and Methods

The oculocardiac reflex was examined in 15 patients with strabismus under general anesthesia. Their ages ranged from 11 months–10 yr. Seven of the 15 patients were exotropes, and eight were esotropes with or without overaction of the inferior oblique muscle. None of the patients had cardiac or neurological disorders. The premedication for general anesthesia was diazepam (0.1 mg/Kg). Measurements were taken in all patients without preoperative administration of atropine, and atropine was used later in surgery in four patients. Informed consents were signed by all patients and their parents.

The heart rate was recorded from lead II of the electrocardiogram (ECG) which was presented as an instantaneous frequency by transforming from a QRS spike of the ECG to a pulse of 1 msec duration and calculating interspike intervals. The extraocular muscles were isolated from surrounding tissues and sutures were placed in the insertions of the muscles and pulled in a direction opposite the muscle field of action. Stepwise traction on the extraocular muscles was given manually. The tension of stretch was measured by a strain gauge (San Ei Sokki Co., Sapporo, Japan) fixed to suturing threads. Signals of instantaneous frequency of heart rate and tension were stored on magnetic tape. For analysis of the data, these signals were displayed on a polygraph (paper speed, 5 cm/sec). The latency of the oculocardiac reflex was measured from the onset of deflection of the tension curve to the pulse just prior to the beginning of instantaneous frequency curve, which declined continuously from the baseline level before the tension on the extraocular muscles, because the frequency was presented as the level of the following
interspike intervals. The measurements of latency in the present study, however, had errors larger than 0.5 sec as the maximum rate of heart beats was about 120 beats/min. The depth of the oculocardiac reflex was expressed as decrease ratio; i.e., the percentage of the difference between the lowest frequency in oculocardiac reflex and the initial frequency before the traction was applied to the initial frequency.

Results

Oculocardiac Reflex to Stretch on Extraocular Muscle

The heart rate was very regular under general anesthesia, ranging from 84 beats/min–123 beats/min. Stretch on the extraocular muscles produced changes in heart rate in the form of bradycardia and arrhythmia (Fig. 1). No tachycardia was observed. The bradycardia and arrhythmia were consistently observed during periods of traction on the medial rectus (Fig. 1A), the inferior oblique (Fig. 1B) and the lateral rectus muscles (Fig. 1C). Arrhythmia, however, occurred during steady traction but not during the trajectories of phasic changes in bradycardia. The analysis of instantaneous frequency of heart rate revealed that the arrhythmia appeared in order to compensate for a bradycardia, since low and high frequencies of heart rate during steady traction applied on the extraocular muscles tended to appear alternately.

The intensity of bradycardia changed with the strength of tension applied to the extraocular muscles. Heart rate decreased from 107 beats/min to 98 beats/min (8.6% in decrease ratio) and to 58 beats/min (45%) when the medial rectus muscles were stretched by tensions of 70 g and 350 g, respectively (Fig. 1A). The contours of bradycardia observed when the inferior oblique muscles were stretched were very similar to those when traction was applied to medial rectus muscles. Heart rate decreased 22.5% and 48% of the initial rate when the inferior oblique muscles were stretched by tensions of 170 g and 400 g, respectively (Fig. 1B). However, when lateral rectus muscles were tractioned with 150 g of tension, the heart rate did not alter during the period of traction (Fig. 1C). Bradycardia of 18% in decrease ratio appeared in association with traction on the lateral rectus muscle with tension increased to 400 g.

The occurrence and depth of bradycardia were related to the tension of stretch on the extraocular muscles. Figure 2 shows a quantitative analysis of the relationship between bradycardia and tension of stretch applied to the medial rectus. The tensions of 100 g, 150 g, 300 g, and 450 g induced 18%, 25%, 39%, and 44%, respectively, indicating that the depth of bradycardia increased with tension applied. The latencies of bradycardia ranged 0.67 sec–2.41 sec, which tended to be short with increase of tension. They, however, were not analyzed in detail any further because of errors larger than 0.5 sec on the measurements. Figure 2 also shows that, as the tension of traction increased from 100 g to 450 g, the responses of bradycardia to transient changes of traction became more rapid. The heart rate decreased gradually, although the tension of 100 g was applied stepwisely. A rapid decrease in heart rate, however, occurred to strong stimuli. The second QRS spike of bradycardia showed the lowest frequency when the tension of 300 g was applied and the first spike already reached the minimum rate of the heart rate at the tension of 450 g. On the other hand, the trajectories recovering from the bradycardia after the release of traction showed exponential curves which were identical, independent of the strength of tension.
bradycardia occurred in all 11 patients, as well as when the medial rectus muscles were stretched. The stretch on the inferior oblique muscles with tensions below 100 g evoked no changes in heart rate in all the patients. The lowest tension to evoke changes was 128 g, indicating that the threshold of the inferior oblique muscles is higher than that of the medial rectus muscles. On the other hand, no bradycardia occurred in 7 of the 15 patients when the lateral rectus was stretched with tensions to 600 g, suggesting that the incidence of the oculocardiac reflex with traction of the lateral rectus was low. The bradycardia induced with stretch of the lateral rectus was weak; the maximum decrease ratio was 19%.

Figure 3 shows the relationship between the depth of bradycardia and the tensions applied to the medial rectus (Fig. 3A), the inferior oblique (Fig. 3B), and the lateral rectus (Fig. 3C) in three patients who were ex-

**Relationship Between Depth of Bradycardia and Tension**

The relationship between the depth of bradycardia and the tension was studied in 15 medial rectus, 11 inferior oblique, and 15 lateral rectus muscles of the 15 patients. The muscles were tractioned with tension ranging from 50 g–600 g. Bradycardia was observed in all 15 patients when the medial rectus muscles were stretched, while, in 4 of 15 patients, no bradycardia occurred on traction with tensions less than 100 g. The lowest tension to evoke the bradycardia was 60 g. The depth of bradycardia, however, differed in different patients. When the inferior oblique was stretched, the
examined with various tensions. Case 1 (Fig. 3, filled circles) showed deep bradycardia to stretch on each muscle, whereas case 2 (Fig. 3, filled triangles) showed weak response of oculocardiac reflex to tensions, indicating that there was an interpersonal variation as well as a difference in depth of oculocardiac reflex among the extraocular muscles.

Effect of Atropine on Occurrence of Bradycardia

The oculocardiac reflex was also examined before and after administration of atropine (0.01 mg/Kg, intravenous) when the medial rectus was stretched with a tension of 300 g (Fig. 4). When the bradycardia was deep, a supraventricular premature contraction occurred, which had a normal duration. Atropine evoked an increase in heart rate from 108 beats/min to 150 beats/min. No bradycardia was observed in association with stretch on the medial rectus with a tension of 300 g 2 min after administration of atropine. The effect of atropine on preventing the bradycardia from occurring for more than 30 min.

Discussion

Stretch on extraocular muscles produced changes in heart rate such as bradycardia and arrhythmia. Bradycardia with arrhythmia was observed in all patients examined.

A striking finding was that the bradycardia was a graded phenomenon as a function of the tension applied to the extraocular muscles. In addition to the depth of bradycardia, the period of time to reach the minimum heart rate also became shortened as the tension increased. These results suggest that the depth and occurrence of bradycardia in the oculocardiac reflex were closely related to the strength of tension, and that the responses of extraocular muscles to stretch were quantitatively transmitted to the heart and then suppressed the heart rate.

The relationship between depth of bradycardia and the tension showed that the occurrence of the oculocardiac reflex differed in the medial rectus, the inferior oblique, and the lateral rectus muscles. Stretch on the medial rectus and the inferior oblique evoked the reflex in all the patients tested, whereas, in the lateral rectus, the reflex could be induced in only about 50% of patients. The incidence of oculocardiac reflex, however, has been reported to range from 16% to 95%.\textsuperscript{1,4,5,6,7} It seems likely that the difference in incidence between previous data and ours resulted mainly from differences in the technique analysis of measurement in heart rate and strength of tension applied. In the present study, the reflex was clearly observed even when the decrease ratio was 2%, whereas other reports defined a bradycardia of 10% or greater as a positive response of the oculocardiac reflex.

The present study also showed that there were differences of depth and threshold of the reflex as well as incidence among the medial rectus, the inferior oblique, and the lateral rectus muscles. No bradycardia occurred on stretching the medial rectus and the inferior oblique by tensions less than 60 g and 128 g, respectively, indicating that the medial rectus had a lower threshold than the inferior oblique. The traction of the lateral rectus even by tension less than 100 g, however, evoked bradycardia in two of the eight patients who showed oculocardiac reflex with tensions of 50 g to 600 g, but their bradycardia was significantly weaker than that of the other two muscles. What components of the oculocardiac reflex arc do the differences among the medial rectus, the inferior oblique, and the lateral rectus result from? The afferent system is responsible for their differences, rather than the efferent system including the vagus nuclei, since the response patterns were identical among the three muscles; the bradycardia occurred rapidly and recovered exponentially after release. It has been believed that the receptors for the reflex are trigeminal nerve terminals, muscle spindles,\textsuperscript{8,9} or Golgi tendon organs.\textsuperscript{10} However, it seems likely that the tri-
geminal nerve terminals may be considered as the receptor, because the threshold of the reflex with 60 g is too high for that of muscle spindles, although physiological characteristics of trigeminal nerve terminals and Golgi tendon organs in extraocular muscles are still obscure, and because the same bradycardia as the reflex could be induced by stimulations of eye lids, face, and oral cavity.

Arrhythmia was always observed in association with bradycardia during the period of steady traction on extraocular muscles. The heart rate in arrhythmia showed that the heart contracted soon after the preceding low frequency contraction, and a supraventricular premature contraction occurred when the heart rate reached around 60 beats/min. The arrhythmia, therefore, may occur in order to compensate for bradycardia. Systemic administration of atropine prevented completely the bradycardia and arrhythmia from occurring. This was in good agreement with previous reports and suggested that the bradycardia in the reflex is mediated through the vagus nerve.

Key words: oculocardiac reflex, extraocular muscle, bradycardia, arrhythmia, vagus nerve

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References