EFFECTS OF A SUSTAINED MUSCULAR CONTRACTION ON HUMAN INTRAOCULAR PRESSURE

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SUMMARY

1. Normal subjects performed fatiguing static hand-grip contraction at tensions of 20% and 55% of their maximum voluntary contraction (MVC). Intraocular pressure (IOP) was measured by applanation tonometry before, during and after the isometric exercise. Forearm blood samples were taken from the antecubital vein in both the exercised and non-exercised arm before and 2 min post-exercise for measurement of plasma lactate, osmolality, $Pv_{O_2}$, $Pv_{CO_2}$ and pH.

2. During hand grip the heart rate and blood pressure increased significantly, whereas the IOP remained unchanged from control in both the 20% and 55% MVC experiments.

3. In the recovery period heart rate and blood pressure returned to control values within 3 min and the IOP decreased significantly from control in both the 20% and 55% MVC experiments.

4. When an occlusion cuff was inflated on the exercising arm just before release of the 55% MVC grip, the decreased IOP could be delayed until the cuff was released.

5. Post-exercise blood samples showed elevated lactate concentrations and $Pv_{O_2}$ and decreased pH in the exercised arm; however, the values remained unchanged in the non-exercised arm. The decreased IOP after exercise may be related to an increased blood lactate concentration.

Key words: intraocular pressure, static exercise, lactate.

Dynamic exercise has been shown to produce a temporary decrease in intraocular pressure (Marcus, Krupin, Podos & Becker, 1970a, 1970b; Lempert & Cooper, 1967; Stewart, LeBlanc & Becker, 1970; Biro & Botar, 1962). This transient fall occurs in the recovery phase and has been attributed to a decrease in plasma pH, and an increase in plasma osmolality and lactate (Marcus et al., 1970a, 1970b).

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In experiments with dynamic exercise, it has not been possible to measure intraocular pressure during the exercise period because of the body movements. Since static (isometric) exercise using small muscle groups produces significant and predictable cardiovascular responses (Lind, McNicol & Donald, 1966) while causing minimal body movements, intraocular pressure may be measured throughout the exercise period.

The purpose of the present study was to determine the effects of static exercise on intraocular pressure. Changes in intraocular pressure were measured and correlated with blood pressures, blood gases, pH, plasma lactate concentration and osmolality during and after sustained static forearm contractions.

**METHODS AND MATERIALS**

Seven healthy male volunteers were studied. They were in good to average physical condition and had no previous training with static exercise. Complete ocular examinations, which included tonometry, tonography and fundus examinations, were performed to exclude ocular pathology.

Subjects performed sustained muscular contractions with a hydraulic hand-grip dynamometer (Van Patten & Rubenstein, 1970-71). Each subject gripped maximally several times (each grip less than 2 s with at least 3 min rest between grips) to establish their maximal voluntary contraction (MVC). Visual targets representing 20% and 55% MVC for test contractions were electronically displayed. Fatigue was defined as the point at which subjects could no longer maintain the required tension. Base-line blood pressure, heart rate and intraocular pressure (IOP) measurements were made before exercise and were repeated during the exercise and recovery periods.

The following experiments were conducted: (1) 55% MVC to fatigue; (2) 20% MVC to fatigue (maximal); (3) 20% MVC submaximal (one-half of the fatiguing 20% MVC time); (4) 55% MVC to fatigue with a blood pressure cuff placed on the exercising arm. This experiment was the same as the 55% MVC to fatigue, but 10 s before release of the grip a blood-pressure cuff was placed on the arm close to the axilla and was inflated to well over the maximum systolic pressure. The cuff was maintained above systolic for 3 min (the limit of endurance for most subjects). The recovery period consisted of two parts: (1) recovery with blood-pressure cuff inflated and (2) recovery with blood-pressure cuff deflated. Only one experiment was performed per day with each subject.

Subjects sat at a slit lamp throughout the exercise period and IOP measurements were made at appropriate intervals. Blood pressure was monitored from the non-exercising arm either by auscultation or with an automatic inflatable cuff and microphone connected to a Grass Polygraph (Grass Instrument Co., Quincy, Massachusetts). The electrocardiogram (ECG) was monitored by telemetry.

Blood samples were taken from the antecubital vein of the exercising arm before and 2 min after the completion of the 55% MVC exercise. On a subsequent day the experiment was repeated and blood samples were taken from the non-exercising arm. Plasma osmolality was determined on a Fiske Osmometer by the freezing-point depression method, a probe being used for 0.2 ml samples enabling measurements of ±2 mosmol/l. $P_{v,\text{O}_2}$, $P_{v,\text{CO}_2}$ and pH were measured with a blood-gas analyser (Radiometer, Copenhagen). Lactate was measured by the lactic dehydrogenase enzymatic method (Sigma Chemical Co., St Louis, Missouri).
Intraocular pressure was measured by Goldman applanation tonometry after topical corneal anaesthesia with benoxinate HCl (0.4%) and sodium fluorescein (0.25%) as the disclosing agent (Fluress, Barnes and Hind Co., Sunny Vale, California). Statistics were analysed by using Student's t-test for group changes and paired t-test for individual changes. The criterion for significance for all tests was the 0.05 level.

RESULTS

55% MVC to fatigue

The average time to fatigue was 1.5 min (range 1.2–2 min) for a contraction of 55% MVC. During the exercise period there was no change in intraocular pressure, but systolic and diastolic blood pressures, breathing frequency and heart rate increased significantly during exercise (Fig. 1).

Fig. 1. Average changes in intraocular pressure (IOP), blood pressure (BP), pulse and respiratory rate (mean ± SEM, P values from base-line) induced by 55% MVC maximal static exercise in six human subjects. IOP values (twelve eyes) are presented as kPa and mmHg.
During the recovery phase, intraocular pressure fell rapidly and remained significantly lower than base-line values at 3 and 5 min. After 5 min the intraocular pressure slowly increased towards the pre-exercise value. Systolic blood pressure returned to pre-exercise levels by 3 min. Diastolic pressure, heart rate and breathing frequency returned to resting values within 1 min. Measurements of these parameters were discontinued when they returned to pre-exercise values.

Blood taken from the exercised arm had a significantly lower pH, was higher in lactate concentration and had a higher $P_{V, O_2}$ after exercise. Plasma osmolality and $P_{V, CO_2}$ were not significantly changed. Blood samples taken from the non-exercised arm showed no significant changes in pH, osmolality, lactate concentration, $P_{V, O_2}$ and $P_{V, CO_2}$ after exercise (Table 1).

**Table 1. Changes in blood pH, plasma osmolality, plasma lactate concentration, and $P_{V, O_2}$ and $P_{V, CO_2}$ from exercised and non-exercised arms with 55% MVC static exercise to fatigue (maximal) of six human subjects.**

Results shown are mean values ± SEM.

<table>
<thead>
<tr>
<th></th>
<th>Exercised arm</th>
<th>Opposite arm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Base-line</td>
<td>Post-exercise (3 min)</td>
</tr>
<tr>
<td><strong>Plasma pH</strong></td>
<td>7.35 ± 0.007</td>
<td>7.24$^{(1)}$ ± 0.018</td>
</tr>
<tr>
<td><strong>Osmolality (mosmol/l)</strong></td>
<td>302 ± 5</td>
<td>306 ± 5</td>
</tr>
<tr>
<td><strong>Lactate (mmol/l)</strong></td>
<td>0.65 ± 0.23</td>
<td>3.73$^{(1)}$ ± 0.75</td>
</tr>
<tr>
<td>$P_{V, O_2}$ kPa</td>
<td>4.13 ± 0.13 (31 ± 1)</td>
<td>6.40$^{(1)}$ ± 0.53 (48 ± 4)</td>
</tr>
<tr>
<td>$P_{V, CO_2}$ kPa</td>
<td>8.26 ± 0.39 (62 ± 3)</td>
<td>9.20 ± 0.39 (69 ± 3)</td>
</tr>
</tbody>
</table>

$^{(1)}$ Significant difference from base-line value ($P < 0.05$).

20% MVC to fatigue

The average time of exercise to fatigue was 9 min (range 7–10 min) for 20% MVC. Intraocular pressure did not change during the exercise period although blood pressure was significantly elevated at 4, 6 and 8 min. Heart rate was significantly increased only at 8 min.

During the recovery period, intraocular pressure was significantly lower at 3 min. Heart rate, systolic and diastolic blood pressure all fell quickly to pre-exercise levels (Fig. 2).

20% MVC, sub-maximal

The average time of exercise was 4.5 min (range 3.5–5 min). During exercise and recovery,
Muscular contraction and intraocular pressure

Intraocular pressure was unchanged from the pre-exercise levels. Systolic blood pressure was significantly elevated only at 4 min of exercise, and diastolic blood pressure was elevated at 2 and 4 min of exercise. Recovery blood pressures were similar to pre-exercise values. Heart rate was unchanged during both the exercise and recovery periods (Fig. 3).

55% MVC to fatigue with 3 min occlusion of circulation

When the blood-pressure cuff was inflated on the exercised arm 10 s before release of the grip and maintained above systolic blood pressure, there was no immediate decrease in intraocular pressure and it remained the same as base-line values (recovery cuff inflated). However, when the blood-pressure cuff was released (recovery cuff deflated) the IOP showed a decrease within 2 min and was significantly lower than base-line value 5 min after the release of the cuff (Table 2).

Fig. 2. Average changes in intraocular pressure (IOP), blood pressure (BP), pulse and respiratory rate (mean ± SEM, P values from base-line) induced by 20% MVC maximal fatiguing static exercise in seven human subjects. IOP values (fourteen eyes) are presented as kPa and mmHg.
Fig. 3. Average changes in intraocular pressure (IOP), blood pressure (BP), pulse and respiratory rate (mean ± SEM) induced by 20% MVC sub-maximal static exercise performed for one half the fatiguing time of a 20% MVC in six human subjects. IOP values (twelve eyes) are presented as kPa and mmHg.

DISCUSSION

These results show that after static exercise there is a significant fall in intraocular pressure. This fall is transient and returns within 15 min to pre-exercise levels. A significant observation was that, during the time blood pressure was increasing, the intraocular pressure remained constant throughout the period of static exercise and fell only during the recovery period, with the greatest decrease occurring from 3 to 5 min after the completion of exercise.

The various levels of exercise in this study were selected for different reasons; 55% MVC was used to show a marked cardiovascular response in a short period of time. The exercise period, however, permitted only one measurement of intraocular pressure since no accurate measurement was obtainable during the last half minute of exercise. The 20% MVC maximal grip was selected to achieve a long exercise period during which multiple measurements of intraocular pressure could easily be made. Despite the fact that it took much longer to reach


Muscular contraction and intraocular pressure

TABLE 2. Changes in intraocular pressure of six subjects (twelve eyes) after 55% MVC static exercise before and after occlusion of circulation in the exercised arm

Results shown are mean values ± SEM. BP, Blood pressure.

<table>
<thead>
<tr>
<th>Recovery time (min)</th>
<th>Base-line&lt;br&gt;Pre-exercise</th>
<th>Exercise</th>
<th>BP cuff inflated</th>
<th>BP cuff deflated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>kPa</td>
<td>1.76</td>
<td>1.71</td>
<td>1.69</td>
</tr>
<tr>
<td>(mmHg)</td>
<td>(13.25)</td>
<td>±0.05</td>
<td>±0.06</td>
<td>±0.06</td>
</tr>
<tr>
<td></td>
<td>(±0.39)</td>
<td>±0.46</td>
<td>±0.49</td>
<td>±0.81</td>
</tr>
</tbody>
</table>

(1) Significant difference from base-line value (*P <0.002*).

fatigue, the magnitude of the response (blood pressure, pulse) was the same as the 55% MVC near the fatigue point. The 20% MVC sub-maximal exercise was used to determine whether minimal blood pressure changes could produce changes in intraocular pressure during or after exercise. This level of exercise was below the duration necessary to change intraocular pressure.

There are significant differences between static and dynamic exercise. Static exercise produces an increase in mean systolic (132 to 152 mmHg) and mean diastolic blood pressure (82 to 106 mmHg), whereas dynamic exercise (Marcus et al., 1970a) increases mean systolic (111 to 135 mmHg) and decreases mean diastolic blood pressure (77 to 65 mmHg) 1 min after exercise. The increase in post-exercise lactate concentrations was lower in static exercise (3.7 ±0.8 mmol/l) than in dynamic exercise (7.6 ±0.6 mmol/l) (Marcus et al., 1970a), and the recovery times for intraocular pressure were shorter in static exercise (15 min) when compared with dynamic exercise (30 min) (Marcus et al., 1970a). The exercising muscle mass in static exercise is much smaller, which probably accounts for the differences in lactic acid concentration in the two exercise forms.

Changes in blood pressure and heart rate in this study are in accord with the results of other workers (Lind et al., 1966; Van Patten & Rubenstein, 1970–71; Whipp & Phillips, 1970; Lind & McNicol, 1967; Wiley & Lind, 1971). Similar changes in blood pH, lactate, $P_{v, o_2}$, $P_{v, CO_2}$ were reported by Astrand, Guharoy & Wahren (1968). The significantly elevated blood $P_{v, O_2}$ in the exercising arms of our subjects in recovery can be attributed to hyperaemia, which normally occurs after static forearm exercise.

The increased breathing frequency in static exercise reported here also has been noted by others (Wiley & Lind, 1971) and its mechanism is not understood. Breathing frequency was monitored during our studies and no alterations in rhythm were found. These results agree with those of Wiley & Lind (1971), who measured oesophageal pressure during sustained contractions and found no increase such as would have occurred if a Valsalva manoeuvre had been performed. A Valsalva manoeuvre, however, would cause an increase in intraocular
pressure rather than a decrease (Rosen & Johnston, 1959). Although there was an increase in breathing frequency during the exercise period, the subjects were not hyperventilating enough to change their \( P_{v,CO_2} \) in either the exercised or non-exercised arm (Table 1). Therefore the fall in IOP that occurs after exercise is unlikely to be the result of hyperventilation, which can cause a fall in IOP (Kaufmann, Schotti & Holtmann, 1971).

Various explanations have been proposed for the sudden and marked vascular changes that are seen with static exercise, but none has been entirely satisfactory. The blood pressure changes are thought to be reflex in origin but the receptors have not as yet been defined.

The lactate concentrations from the exercised arm are comparable with those found by other investigators (Astrand et al., 1968) in static exercise and are about the same magnitude as those found for a man hammering with a standard-weight hammer above his head (Astrand et al., 1968). Cardiovascular changes in our study were also comparable with changes found in subjects holding 20 kg weights in each hand (Lind & McNicol, 1968).

The mechanism of the decrease in intraocular pressure in static exercise is difficult to explain with existing evidence. The relationship to the lactic acid production, as previously suggested for the response after dynamic exercise (Marcus et al., 1970b), must be considered. The ocular hypotension does not appear to be related to blood pressure, since the 20% MVC sub-maximal exercise significantly increased blood pressure and did not alter intraocular pressure. The increase in lactate and decrease in pH from the exercised arm could possibly implicate lactic acid production as the cause of the decrease in intraocular pressure. However, blood samples taken from the opposite arm did not show increased lactate concentrations. In addition, osmolality from either arm was unchanged after exercise. It has been demonstrated that small amounts of hypertonic urea in quantities too small to affect systemic osmolality can lower intraocular pressure (Podos, Krupin & Becker, 1971). Therefore lactate or other products of exercise, released to the blood from the exercised arm, may be preferentially affecting 'ocular receptor sites' resulting in a fall in intraocular pressure. Sodium lactate infusion in human volunteers (325 ml of 0.5 mol/l solution infused over 6 min) has been shown to cause a decrease in intraocular pressure (13.2 to 8.6 mmHg) (Marcus et al., 1970a). This has also been confirmed in rabbits (Marcus et al., 1970b).

The dependence of the fall in intraocular pressure on the release of metabolites from exercising muscle is further suggested by the experiments at 55% MVC where the fall in intraocular pressure was delayed by inflation of a cuff on the exercising arm. Upon release of the blood-pressure cuff a significant fall in intraocular pressure occurs, which is comparable with the fall in pressure seen at the same exercise level when no cuff was used, but delayed by the time-interval the cuff was on the arm.

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Muscular contraction and intraocular pressure


