The Effects of Mild, Moderate, and Severe Exercise on Intraocular Pressure in Glaucoma Patients

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Abstract Background: Previous studies have shown a reduction in intraocular pressure (IOP) from many means of exertion, ranging from walking to exhausting exercise in both normal and glaucoma subjects. The variability in their results may be due to several variables that can affect IOP significantly. Purpose: To investigate the effects of common means of exertion on IOP in normal and glaucoma subjects, after elimination of factors that can affect IOP. Subjects and methods: The effects of walking, jogging, and running fast until exhaustion on IOP were noted in seven normal and seven open-angle glaucoma subjects. Intraocular pressures were measured with the Goldmann applanation tonometer during and after exercise. Results: The mean differences between before and after walking, jogging, and running exercises were $-2.43 \pm 0.30$, $-3.85 \pm 0.55$, and $-4.0 \pm 0.37$ mmHg in normal subjects. In glaucoma patients these differences were $-7.72 \pm 1.25$, $-10.86 \pm 2.12$, and $-12.86 \pm 2.05$ mmHg, respectively. After 5 min of walking and jogging, in all subjects, IOP decreased significantly (by 56 to 61% of total decreases). Conclusion: Regardless of the means of exertion, in every subject tested there was a drop in intraocular pressure. Glaucoma patients had a greater drop and longer duration of post-exercise recovery as compared to normal subjects. It would seem reasonable, at present, not to discourage patients who have glaucoma from light exercises such as walking; perhaps, on the contrary, it should be encouraged.

Key words: exercise, glaucoma, ocular tension, tonometry.

Previous studies of normal volunteers [1–5] and open angle glaucoma patients [4, 6, 7] have shown that exercise temporarily lowers intraocular pressure. However, in glaucoma patients the percentage of decrease is highly different in different studies, ranging from 17.5 to 51% [4, 6, 7]. The variability in their results may be
due to several factors. In recent years, it has been noted that intraocular pressure is a dynamic function and is subject to many influences both acutely and over the long term. Many investigators have reported that IOP varies with age [8], sex [9], and diurnally [10]. It has been reported that drinking water, coffee, or alcohol has a significant effect on IOP [11]. Acute hyperglycaemia decreases [12], while chronic hyperglycaemia in diabetes increases IOP [13]. Moreover, seasonal variations [14] and systemic hypertension [15] both have significant effects on intraocular pressure.

The present study was planned to investigate the effects of common means of exertion on intraocular pressure of glaucoma patients and normal subjects after taking into account the above mentioned factors, neglected by previous studies.

SUBJECTS AND METHODS

Seven primary open-angle glaucoma patients aged between 40 and 50 years (46.14±3.23, mean±SD), were chosen from outpatients seen at the Department of Ophthalmology, Jinnah Postgraduate Medical Centre, Karachi, Pakistan. To compare the effect of exercise on IOP between glaucoma patients and normal subjects, seven healthy sedentary male volunteers of the same age group (44.42±3.15) working in the same hospital, were also studied. Fifty years of age was chosen as the maximum, since older patients would be more likely to have medical problems precluding exercise. Patients were instructed not to take their glaucoma medication on the morning of the exercise test. The criteria met by the subjects were an absence of any history of eye surgery or diabetes, and normal body temperature and blood pressure. To avoid the effect of acute hyperglycaemia, the subjects were asked not to have breakfast or any form of drink before the test. A transport service was provided to each subject to avoid any exertion, and they were asked not to do any hard work after awakening. Tests were performed after a complete rest of 15 min in the supine position. Before the start of the test, blood pressure was measured in the supine position. Each test was started at a fixed time (0800 A.M.) to minimize the effect of diurnal variations. After installation of 0.25% fluorescein sodium and 0.4% benoxinate hydrochloride (fluress) eye drops, IOP was measured with the Goldmann applanation tonometer, first in the right eye and then in the left. Three consecutive readings of each eye were taken. An average of the three readings was computed separately for each eye. Each subject was tested three times by different exercise tests at an interval of 4 d. These tests were: test 1, walking for 1 h; test 2, jogging for 1 h; and test 3, running as fast as possible until volitional exhaustion. In the first two tests, intraocular pressures were measured at the beginning and in the last 30 s of the 5th, 20th, 40th, and 60th minutes of the test, but in test 3, only at the beginning and end of the test. A compulsory 5-min “cool down” period was given to all subjects followed by IOP measurements every 10 min until the IOP returned to pre-exercise levels (±1 mmHg). The effects of all tests were similar on both eyes, so the data were pooled.
Table 1. Effect of exercise on intraocular pressure in normal and glaucoma subjects.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Glaucoma patients</th>
<th>Normal subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IOP</td>
<td>% decrease</td>
</tr>
<tr>
<td>Effect of walking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>33.43±2.19</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>29.14±1.77</td>
<td>12.83</td>
</tr>
<tr>
<td>20</td>
<td>27.43±1.53</td>
<td>17.95</td>
</tr>
<tr>
<td>40</td>
<td>26.14±1.32</td>
<td>21.81</td>
</tr>
<tr>
<td>60</td>
<td>25.71±1.23</td>
<td>23.09</td>
</tr>
<tr>
<td>Effect of jogging</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>33.29±2.24</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>26.71±1.04</td>
<td>19.77</td>
</tr>
<tr>
<td>20</td>
<td>25.71±0.87</td>
<td>22.77</td>
</tr>
<tr>
<td>40</td>
<td>23.57±1.11</td>
<td>29.20</td>
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<tr>
<td>60</td>
<td>22.43±1.04</td>
<td>32.62</td>
</tr>
<tr>
<td>Effect of running</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>32.86±2.13</td>
<td></td>
</tr>
<tr>
<td>After</td>
<td>20.00±0.82</td>
<td>39.14</td>
</tr>
<tr>
<td>Post-exercise mean recovery time (min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking</td>
<td>54.28±8.41</td>
<td></td>
</tr>
<tr>
<td>Jogging</td>
<td>71.43±11.43</td>
<td></td>
</tr>
<tr>
<td>Running</td>
<td>84.29±13.43</td>
<td></td>
</tr>
</tbody>
</table>

*p value as compared to previous reading.

for statistical analysis. A two-tailed paired Student’s t-test was used for statistical analysis.

RESULTS

The results are summarized in Table 1, which shows that the mean differences between before and after walking, jogging, and running exercises were $-2.43\pm0.30$, $-3.85\pm0.55$, and $-4.0\pm0.37\text{mmHg}$ in normal subjects. In glaucoma patients, these differences were $-7.72\pm1.25$, $-10.86\pm2.12$, and $-12.86\pm2.05\text{mmHg}$, respectively. The total decreases of 58.84 and 55.56% during walking and of 59.22 and 60.6% during jogging occurred after 5 min in normal and glaucoma patients, respectively. The mean times of fast running were $9.43\pm0.65$ and $8.29\pm0.97\text{min}$ in normal subjects and glaucoma patients, respectively. After running until exhaustion, IOP dropped 26.42 and 39.14% in normal subjects and in glaucoma patients, respectively.

DISCUSSION

The bicycle ergometer is frequently used for testing the effect of exercise on
intraocular pressure, but in the present study the common means of exertion were used which, of course, are of more practical importance. Moreover, this is the first study in which the effects of mild, moderate, and severe exercise on intraocular pressure were noted in the same individuals. Thus, in this study, all factors that may affect intraocular pressure were eliminated. Regardless of the means of exertion, in every subject tested there was a drop in intraocular pressure, although there was considerable variation in the drop in pressure and the duration of post-exercise recovery.

In this study, mean differences between before and after walking, jogging, and running exercises were $-2.43 \pm 0.30$, $-3.85 \pm 0.55$, and $-4.0 \pm 0.37$ mmHg in normal subjects. In glaucoma patients, these differences were $-7.72 \pm 1.25$, $-10.86 \pm 2.12$, and $-12.86 \pm 2.05$ mmHg, respectively. This suggests that more severe exercise might be associated with a greater fall in intraocular pressure. The drop in intraocular pressures due to walking and jogging were 3.18 and 2.82 times higher in glaucoma patients than in normal subjects, respectively. Similarly, the effect of running as fast as possible until volitional exhaustion was 3.22 times higher in the glaucoma patients than in normal subjects. This result suggests that a high initial intraocular pressure might be associated with a greater fall in intraocular pressure. In glaucoma patients, the drop in intraocular pressure after walking and jogging was 7.2 and 7.19% greater than the drop in normal subjects, respectively, which are nearly the same amounts. This similarity, may be related to the duration of exercise because these two exercises were of the same duration i.e., 1 h. The drop in glaucoma patients after running, which is more severe exercise than walking or jogging, was 12.72% greater than the drop in normal subjects. This result suggests that mild exercise like walking and moderate exercise like jogging, if continued for the same duration of time, not less than 1 h, may produce the same percentage decrease in intraocular pressure of glaucoma patients.

The results of this study showed (Table 1) that in both glaucoma patients and normal subjects, the post-exercise recovery time depends upon the severity of the exercise. In glaucoma patients, the post-exercise recovery times were higher than in normal subjects. These results suggest that the time required for recovery is related to the severity of the exercise and the initial intraocular pressure. In summary, the results of this study suggest that the effect of exercise on intraocular pressure may depend upon three factors which, in order of importance, are: 1) a high initial intraocular pressure might be associated, as has been shown above, with a greater fall in intraocular pressure; 2) severity of exercise might be associated with the degree of the fall in intraocular pressure; and 3) mild exercise like walking and moderate exercise like jogging, if continued for the same duration of time, not less than 1 h, may produce the same percentage decrease in intraocular pressure of glaucoma patients.

As in this study, previous investigators have also found a decrease in intraocular pressure after walking, both in normal subjects [1] and glaucoma patients [6]. Although the previous studies used different methods of exertion, they also
reported a consistent fall after moderate and severe exercise, both in normal subjects [2–5] and glaucoma patients [4, 7]. Three studies reported that intraocular pressure decreases in glaucoma patients after exercise [4, 6, 7]. In these studies, the maximum decrease varied between 17.8 and 51%. This large difference is due to several variables, such as means and duration of exercise, timing of intraocular pressure measurement, diurnal variation, age, sex, and seasonal variation. Differences in reported results were also due to variations among protocols. Previous studies also did not control for the drinking of water or alcohol, acute hyperglycaemia, diabetes, and systemic hypertension of the subjects. One of these reported a difference in the measurements taken before and after the test [6]. This method does not account for diurnal variation, which can occur over as short a time as 20 min and may be as great as the changes noted during exercise [16]. In this study, running until exhaustion decreased intraocular pressure 39.14% in glaucoma patients, while Lempert et al. [4] reported a decrease of 51%. In their study, glaucoma patients used epinephrine 2% before exercise as medication. The greater drop in intraocular pressure may be due to the combined effect of exercise and epinephrine, which decreases intraocular pressure [17].

The physiological mechanisms responsible for the decrease of intraocular pressure during exercise are not clearly known [18], although a number of possible mechanisms can be postulated. Podos et al. [19] reported that a direct relationship exists between intraocular pressure and episcleral venous pressure. Exercise produces significant changes in systemic vascular dynamics and could possibly alter episcleral venous pressure. However, Stewart et al. [20] did not note any significant change in episcleral venous pressure after exercise. The decrease in IOP after exercise has been attributed to an increase in blood lactate and a decrease in blood pH [21]. However, Kielar et al. [18] found no significant differences in intraocular pressure reduction when comparing standardized aerobic and anaerobic exercise, despite significant differences in blood pH and lactate measurements. Intraocular pressure is known to be altered by sudden changes in plasma osmolarity [22]. Following exercise, a consistent increase in serum osmolarity occurs [23]. However, Stewart et al. [20] noted that exercise induces greater changes in intraocular pressure than does oral doses of glycerin for the same change in serum osmolarity. It also has been suggested that exercise increases the facility of outflow. The outflow channels of the eye, especially around Schlemm's canal, show fibrinolytic activity [24]. Such fibrinolysis can be postulated to assist in preventing obstruction of the aqueous outflow pathways, and thus participate in the regulation of intraocular pressure. Since exercise increases systemic fibrinolytic activity [25], one can speculate that exercise decreases intraocular pressure by increasing the facility of outflow, although one study revealed no change in the facility of outflow when measured immediately following exercise [20]. Passo et al. [3] have attempted to associate decreased intraocular pressures with pre- and post-exercise hemodynamic factors such as heart rate or maximum systolic or diastolic blood pressure, but no such relationship has been confirmed.
The existing literature shows the influence of hormones upon intraocular pressure. There is evidence that corticotropin, vasopressin, thyroxin, insulin, glucocorticoids, and mineralocorticoids may play a role in the physiologic regulation of intraocular pressure. Growth hormone, melanocyte stimulating hormone, progesterone, estrogen, chorionic gonadotropin, and relaxin may influence intraocular pressure when administered in pharmacologic doses. Some of these hormones increase, while others decrease intraocular pressure [26]. In addition to these hormones, the effects of serotonin and angiotensin have been reported. Decreased serum levels of serotonin have been noted in glaucoma patients [27]. Also intracarotid, subconjunctival and retrobulbar injections of serotonin have been shown to decrease intraocular pressure in dogs and rabbits. This effect was unrelated to any change in systemic blood pressure [28]. It has also been reported that intravitreal injection of angiotensin in rabbits led to a decreased intraocular pressure without a change in the facility of outflow. Intravitreal injection of angiotensin in cats produced an initial increase in intraocular pressure as blood pressure rose, followed by a lowered intraocular pressure associated with blanching of the iris vessels [29].

Stimulation of the sympathetic nervous system in anticipation of and during the stress of exercise is well documented. This causes the release of large quantities of epinephrine and norepinephrine from adrenal medulla [30, 31]. Epinephrine, an adrenergic agonist, is widely used as an ocular hypotensive drug for the treatment of glaucoma. The fact that epinephrine lowers intraocular pressure in humans is undisputed, but the mechanism of this action is not yet clear [17]. Many published studies support the idea that epinephrine reduces intraocular pressure by lowering outflow resistances and by lowering the rate of aqueous formation [31], but many inconsistencies appear in the results of these published studies [32]. In two studies of the effect of epinephrine on aqueous humor formation in the human eye, where fluorescein tracer techniques were used, it was concluded that epinephrine reduced the rate of aqueous humor formation [33, 34]. In another study, the opposite effect was observed [35]. One major difference in the three studies was the time between the drug instillation and the flow measurement. In the studies where suppression of aqueous humor formation was found, there was a longer lapse of time after the drug was given before the most critical data points were measured. In the study where stimulation of aqueous humor formation was found, a relatively short time lapse was used. However, Nagataki and Brubaker [17] failed to demonstrate any time dependency of epinephrine’s effect on aqueous humor formation. Stewart et al. [20] reported that when patients were pretreated with topical epinephrine in only one eye, there was no significant difference in the percentage reduction of intraocular pressure between fellow eyes after short-term exertion.

It has been demonstrated that patients with primary open-angle glaucoma are more responsive to epinephrine than normotensive persons. The lymphocytes from patients with primary open-angle glaucoma are also more responsive in vitro to epinephrine than those from nonglaucomatous persons [36]. Epinephrine produces
many of its effects by stimulating the synthesis of cyclic adenosine monophosphate (cAMP). cAMP regulates the activity of protein kinases. These, in turn, phosphorylate and thereby activate or inhibit key enzymes that control intracellular metabolic pathways [37]. It has been shown that activation of cAMP decreases intraocular pressure by decreasing aqueous humor production [38]. As mentioned above, in this study glaucoma patients had a greater drop in IOP and a longer duration of post-exercise recovery as compared to normal subjects. It is quite possible that in glaucoma patients the enzymes such as adenylate cyclase become more sensitive to hormones and this may be the cause of the greater drop and longer duration of post-exercise recovery of intraocular pressure as compared to normal subjects.

It is amazing that almost any type of stress, whether physical or neurogenic, will cause an immediate and marked increase in ACTH secretion. Even a small amount of ACTH is enough to permit the adrenal glands to secrete whatever amount of aldosterone is required [30]. The effects of ACTH, aldosterone and important catecholamines, including norepinephrine, have not been investigated, and since exercise changes their blood concentrations, they are more likely to affect intraocular pressure.

I consider it quite possible that a decrease in intraocular pressure during exercise is effected primarily through a hormonal mechanism; an effect on electrolytes or electrolyte transport enzymes may be involved as well. Two enzyme systems, Na/K-ATPase and carbonic anhydrase, are involved in aqueous humor secretion [39]. Therefore, antagonists of these enzyme systems can reduce aqueous formation and hence, lower the IOP [40]. Hormonal changes and metabolites produced during exercise can act as antagonists of these enzyme systems. At this time, investigation to elucidate such factors may prove a fruitful area for ophthalmic research. It would seem reasonable at present not to discourage patients who have glaucoma from walking; perhaps, on the contrary, it should be encouraged.

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