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Intraocular pressure fluctuations in professional brass and woodwind musicians during common playing conditions

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Abstract
Background We investigated the effects on intraocular pressure (IOP) and blood pressure (BP) of playing brass and woodwind instruments by monitoring IOP and BP in a representative group of professional musicians under a variety of common playing conditions.
Methods IOP and BP measurements were recorded from 37 brass and 15 woodwind instrument players, before and after playing tones of low, middle and high frequency. We also measured IOP and BP before and during playing common exercises of 10 minutes duration, as well as after playing a sustained high-pitched tone, to test for changes in IOP under conditions of maximum effort.
Results Playing tones on brass and woodwind instruments causes a temporary elevation in IOP and BP, depending on the tone frequency: brass instrument players showed a significant elevation after playing high and middle frequency tones ($p<0.0001$) whereas woodwind instrument players showed a significant increase only for high frequencies (e.g., oboe, 17±2.9 mm Hg to 21±4.4 mm Hg; $p=0.017$). Playing a typical exercise of 10 minutes temporarily increased IOP in both groups of musicians. Finally, playing a sustained tone of high pitch caused a significant elevation in IOP in brass instrument players only (16.6±3.5 mm Hg to 23.3±8.9 mm Hg; $p<0.0001$).
Conclusions The temporary and sometimes dramatic elevations and fluctuations in IOP observed in this study, coupled with daily exposure to instrument play, puts professional wind instrument players at increased risk of developing glaucoma. Consequently, these musicians should be monitored for signs of glaucoma, especially those with co-existing risk factors.

Keywords Intraocular pressure · Wind instruments · Glaucoma · Musicians · Blood pressure

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Introduction

Glaucoma, a progressive degeneration of retinal ganglion cell axons and cell bodies, is one of the most common ocular diseases leading to visual impairment and blindness [1, 2]. Although glaucoma is considered to be a complex genetic disease, increased intraocular pressure (IOP) is a critical risk factor (for excellent reviews, see Sultan et al. 2009 and Nassr et al. 2009 [2, 3]). Despite a lack of understanding of the precise pathological mechanisms leading to glaucoma, many recent studies have given an insight into the underlying etiopathology [4–10]. Further studies have indicated a correlation between systemic hypertension and open-angle glaucoma, and a positive correlation between blood pressure and IOP [11–14].

Pulmonary functions of wind instruments players have been studied extensively in the last 5 decades [15–18]. Playing wind instruments such as the French horn and trumpet causes cardiovascular changes resulting from blowing long notes or phrases using a single breath [19–23], which have been found to be similar to those caused by a classic Valsalva maneuver in tuba players [20, 24]. In contrast, studies by Harris and Larger and Ledoux found that the cardiovascular effects of the Valsalva did not match those of playing high-resistance wind instruments [22, 23].

Additionally, it has been suggested that playing wind instruments leads to elevated IOP through a rise in intrathoracic pressure and compression of the intrathoracic venous system. It is believed that the rise in venous pressure is transmitted to the choroid via the jugular, orbital and vortex veins, causing vascular engorgement, an increase in the choroidal volume, and a rise in IOP [25, 26].

Schumann et al. (2000) [26] recorded and compared IOP in high- and low-resistance wind instrument players while playing notes of different volumes and tone pitches. Furthermore, they used ultrasound biomicroscopy to measure uveal thickness in the posterior pars plana of the ciliary body before and during playing. Pneumatonometry showed an IOP elevation that was dependent on the force of blowing. The ultrasound biomicroscopy detected increased uveal thickness associated with IOP elevation, which strengthens the hypothesis of vascular engorgement and an increase in choroidal volume. The magnitude of elevation was found to be dependent on the amount of expiratory resistance provided by the particular instrument, where high-resistance wind instruments (oboe, trumpet) showed significantly higher elevations than low-resistance instruments (clarinet, saxophone). They also showed that high-resistance wind instrument players exhibit a small but significantly greater incidence of visual field loss than other musicians, which is related to life-hours of playing. Schumann et al. (2000) argued that the cumulative effect of long-term intermittent IOP elevation during high-resistance wind instrument playing might result in glaucomatous damage, which could be misdiagnosed as normal-tension glaucoma [26].

The aim of the current study was to determine the effect of playing specific tone pitches (frequencies) on high- and low-resistance brass and woodwind instruments on blood pressure (BP) and IOP under everyday playing conditions. We further recorded the dynamics of BP and IOP during common playing exercises with a length of 10 minutes.

General methods

Subjects

Thirty-seven (30 males, seven females) professional brass instruments players (nine trumpet, 11 horn, ten trombone and seven tuba players) participated in the first part of this study. The mean age was 28.4±8.0 years, with a range of 19–56 years. All subjects had normal general health, and none was using any medications. Twelve subjects were smokers (≥3 cigarettes per day).

The second group of musicians consisted of 15 professional woodwind instrument players (seven oboes, eight clarinets) with a mean age of 26±8.5 years (range 20–54 years). None of these subjects were smokers, and all had normal general health with no medications. All participants gave informed consent to take part in the study, which was performed with approval of the Ethics committee of the Friedrich Schiller University in Jena, Germany. This study adheres to the tenets of the Declaration of Helsinki.

Procedures

Subjects played their own instruments while seated in a room of normal temperature.

IOP was measured with an iCare® rebound tonometer from each subject's accessible eye (right eye for trombone, tuba, clarinet, and oboe; left eye for trumpet and horn). Systolic and diastolic BP was recorded using either a Siemens Sirecust 610 ® on the accessable upper arm (right arm for trumpet and horn; left arm for trombone and tuba) or a Medisana® MTP (right arm for oboe and clarinet).

For the first part of experiment 1, subjects were asked to play a tone of middle frequency (see Table 1) with increasing volume (crescendo) and to sustain it for as long as possible. IOP and BP were taken before playing began, immediately after playing ceased, and for a further 2 minutes at 20-second intervals. This was repeated for tones of low and high pitch (Table 1). Appropriate breaks for

Table 1 Frequencies for different types of wind instruments used in this study

Instrument	Low pitch	Middle pitch	High pitch
Trumpet	220 Hz	440 Hz	880 Hz
Horn	220 Hz	440 Hz	880 Hz
Trombone	110 Hz	220 Hz	440 Hz
Tuba	55 Hz	110 Hz	220 Hz
Oboe	220 Hz	440 Hz	880 Hz
Clarinet	220 Hz	440 Hz	880 Hz

recovery between each condition were provided and tested by a control measurement of IOP and BP.

To investigate changes in IOP and BP during common playing conditions, musicians were asked to play exercise compositions lasting 10 minutes (Table 2). Due to the wide range of brass instruments, each having its own individual gamut and characteristics, different warm-up exercises and compositions were chosen under the supervision of experienced musicians. Nonetheless, we ensured to guarantee similar pitch ranges by determining the tone range (e.g., A–e'''). Baseline values of IOP and BP were taken before play began, and further measurements were recorded at 2-minute intervals during play. To further evaluate the behaviour of BP and IOP after maximum expiratory effort, subjects were asked to choose another high pitch and to sustain it with maximal exertion. IOP measurements were taken during the playing of this tone, directly after playing was ceased, and for a further 4 minutes at 20-second intervals. BP was recorded directly after the high pitch, and for a further 4 minutes at 2-minute intervals.

Statistics

Results here and throughout are expressed as mean ± standard error of the mean. Data were analyzed globally by a non-parametric Friedman test. Wilcoxon tests were used to compare data points within a single condition (e.g., IOP base value vs IOP directly after tone of increasing volume). For statistical analyzes between instruments, Mann–Whitney U tests were performed (e.g., IOP basic value clarinet vs IOP basic value oboe). To test for correlations between IOP and BP, Spearman's rank correlation coefficients were determined. The error bars throughout this paper represent the standard errors.

Table 2 Common compositions and warm-up exercises with an average duration of 10 minutes were taken to investigate the dynamics of IOP and BP. Due to the variety of brass instruments with their different individual pitch ranges, different pieces of compositions with similar compasses of tones had to be chosen

Instruments		Composition
Brass	Trumpet	James Stamp; Warm up exercises; Nos. 4b, 5, 6
	Horn	B. Müller; B Études, Op. 64; Volume 1, No. 1
	Trombone	Skolar; Warm up exercises; No. 5a – 5e
	Tuba	Walter Hilgers; Daily Exercises
Woodwind	Oboe	Mozart's Oboe Concerto in C major; K. 314; 1st movement (Allegro aperto)
	Clarinet	Mozart's Clarinet Concerto in A major; K. 622; 1st movement (Allegro)

Experiment 1 — brass instruments

Results

Temporary playing of tones of different frequency

While playing a middle frequency tone in crescendo (mean playing duration 13.2 s±5.17), the mean IOP for brass instrument players (n=7) increased significantly from 16± 3.9 mm Hg to 18.8±3.7 mm Hg (p<0.0001). This is consistent with an elevation rate (ER) of 0.160 mm Hg/ second. Playing the deep pitch led to a significant increase in IOP from 14.7±3.3 mm Hg to 16.4±4.0 mm Hg (p= 0.004) (duration: 10.68 s±4.21; ER: 0.159 mm Hg/second). Playing the high pitch resulted in a significant elevation of mean IOP from 15.2±3.5 mm Hg to 18.3±4.8 mm Hg directly after playing (p<0.0001) (duration: 10.84 s±4.337; ER: 0.284 mm Hg/second) (Fig. 1).

Playing middle frequency tones caused an IOP elevation ≥5 mm Hg (compared to the base value) in ten of 37 (27%) brass instrument players. One musician showed an increase of ≥10 mm Hg. Eleven (30%) brass wind instrument musicians showed alarming peak IOP values, which fell within the ocular hypertension range (IOP>21 mm Hg). In one individual case (tuba player), IOP increased from 19 mm Hg to 48 mm Hg within 40 s. Deep frequency pitches caused elevations of ≥5 mm Hg in nine cases (24%), and two musicians (5%) showed increases of ≥10 mm Hg. Here, IOP values in the ocular

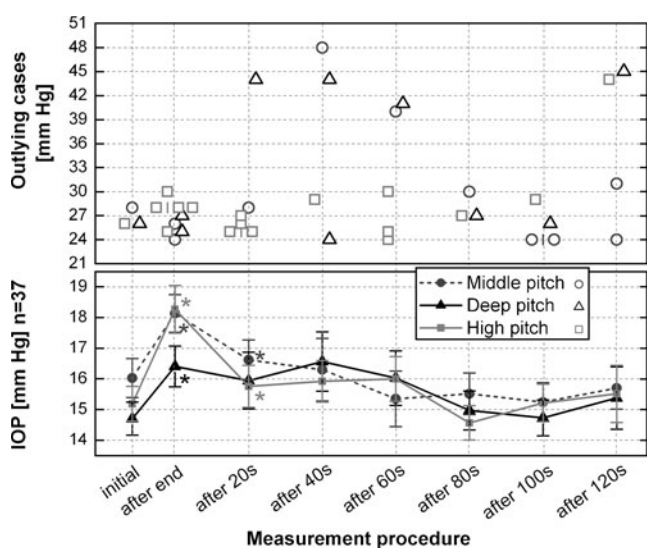

Fig. 1 Course of changes in mean IOP for brass instruments at three different frequencies. IOP increases dramatically after playing the tone with increasing volume and typically decreases to the base value after 40 to 80 seconds. Significant outliers are spread horizontally about the vertical time points to enable visibility. Here and throughout, *error bars* represent standard errors. Significant changes in IOP are indicated by *

hypertension range could be measured in six brass musicians (16%), with peak values of up to 44 mm Hg. The high frequency tones led to increases ≥5 mm Hg in 11 of the brass musicians (30%), and six (16%) even showed an elevation ≥10 mm Hg. Nine of the brass wind instrumentalists (24%) showed alarming hypertension pressures, with peak values of 29 mm Hg when playing high frequency pitches. Significant outliers are shown separately and spread horizontally to enable visibility (top part Fig. 1). The general characteristic in the change of IOP was similar for all three tone frequencies. IOP typically increased during playing, and decreased slowly within 20 seconds after playing ceased, reaching the approximate level of the base value after 2 minutes.

Playing tones with increasing volumes resulted in an increase in systolic BP in all conditions (significant for middle frequencies (p=0.014) and deep frequencies (p= 0.003), whereas the diastolic BP usually remained constant. Both BP and IOP typically decreased to the base value after 2 minutes. Data for BP components are not shown graphically in this paper.

Long-term playing

During the long-term condition, where subjects were asked to play a common exercise lasting 10 minutes, the mean IOP increased significantly from 15.2±4.3 mm Hg to 16.6±3.5 mm Hg (p=0.004). During the subsequent tone with maximum intensity, IOP (see Fig. 2) shows a significant increase to 23.3±8.9 mm Hg (p<0.0001). Compared to the base value, 23 of 37 brass instrumentalists (62%) showed IOP elevations ≥5 mm Hg, and 15 (41%) showed increases of ≥10 mm Hg. The most dramatic elevations were typically reached while playing the high pitch with maximal effort. Here, 21 (57%) of the musicians reached peak IOP values in the hypertension range, with peak values of up to 51 mm Hg. The outlying cases are separately illustrated as single data points within Fig. 2 (top part). Although each instrument class played slightly different compositions, the course of changes of IOP consistently followed the same pattern. IOP typically increased within the first 2 minutes and reached a plateau, increased markedly while playing the tone of maximum intensity, and then decreased rapidly to the initial base level after playing ceases.

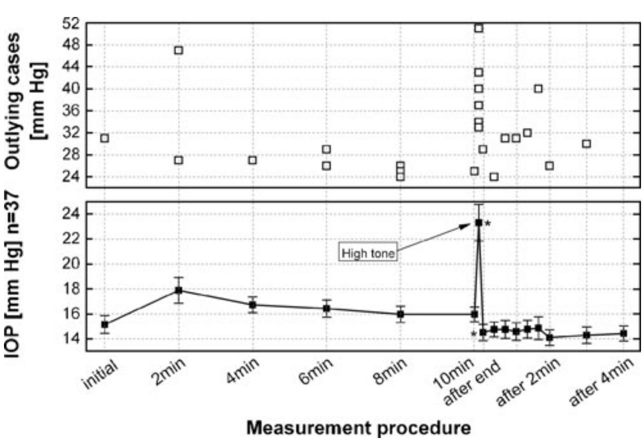

Fig. 2 Course of changes in mean IOP for brass instruments during a 10-minute common exercise with a subsequent tone of maximal intensity and volume after 10 minutes. IOP typically increases within 2 minutes, followed by a plateau and then a smooth decrease. Notable outliers are illustrated separately by single data points (*top part*). The high frequency tone causes a significant elevation of IOP and decreases to base value after 4 minutes

Systolic and diastolic blood pressure increased significantly from 122.8±15.5 mm Hg (systole; p<0.0001) and 75.5±8.9 mm Hg (Diastole; p<0.0001) to 148.3±17.1 mm Hg and 97.2±16.1 mm Hg respectively. There was no significant increase in BP under conditions of maximum effort. After playing ceased, both systolic and diastolic BP significantly decreased again (128.7±23.7 mm Hg and 72.4±11.5 mm Hg (p<0.0001), finally reverting to baseline values after 4 minutes.

Correlation between IOP and BP

There was a small but mostly non-significant correlation between systolic BP and IOP. However, an increase in systolic BP was generally accompanied by an increase in IOP.

IOP dynamics separated for brass instrument class

There were no significant differences between high-resistance (trumpet and horn; n=20) and low-resistance brass instruments (trombone and tuba; n=17) for short-term playing. However, compared with low-resistance instruments (17.1±4.2 mm Hg and 17.2±3.9 mm Hg), high-resistance instruments showed a higher increase in IOP for middle frequency (18.9±3.1 mm Hg) and high frequency tones (19.2±5.3 mm Hg).

IOP dynamics as a function of smoking

Cigarette smoking is positively linked to many systemic diseases and some ocular conditions such as cataract [27] and age-related macular degeneration [28]. There is as yet no clear evidence as to whether smoking cigarettes (i.e., nicotine) leads to increased or decreased IOP. In the Blue Mountain Eye Study, only a modest IOP elevation was found among smokers [29]. A multitude of other case-control, cross-sectional and epidemiological studies have failed to establish a direct relationship between cigarette smoking and elevated IOP [11, 30–37].

Here, we analyzed differences between non-smokers (n=25) and smokers (n=12). Forty seconds after the playing of a middle frequency tone was ceased, the IOP of smokers (17.7±3.5 mm Hg) was significantly higher (p=0.01) than that of non-smokers (15.6±7.1 mm Hg). At low and middle frequencies and during long-term playing, the IOP of smokers was typically higher. For example, during high, loud tones with maximal effort, IOP was increased up to 26.0±11.5 mm Hg in smokers, and up to 22.0±7.2 mm Hg in non-smokers.

Experiment 2 — woodwind instruments

Results

Temporary playing of tones of different frequency

While playing a middle frequency tone in crescendo, the mean IOP for clarinet players (n=7) increased significantly from 19±3.3 mm Hg to 21±3.3 mm Hg (p=0.034), and increased slightly for oboe players (20±4.0 mm Hg to 21±3.2 mm Hg). Two of eight clarinet players (25%) and two of seven oboists (29%) showed increases of ≥5 mm Hg compared to the base IOP. Two clarinet musicians and one oboist showed elevations of ≥5 mm Hg while playing low frequency pitches. Three (20 %) of all woodwind instrument musicians showed alarming peak values in the ocular hypertension range (IOP>21 mm Hg). Playing the high pitch led to a significant increase in IOP for both instruments, where the mean IOP for the high-resistance wind instrument players (oboe) rose from 17±2.9 mm Hg to 21±4.4 mm Hg (p=0.017) (ER: 0.13 mm Hg/second). The clarinet players showed an elevation of mean IOP from 16±2.7 mm Hg to 18±2.0 mm Hg directly after playing (p=0.05) and 19±2.7 mm Hg after 20 seconds (ER: 0.09 mm Hg/second). Here, one clarinet player and five oboe players (63%) showed dramatic increases of ≥5 mm Hg. Twenty percent of woodwind musicians reached IOP in the hypertension range. Thirteen percent of woodwind instrumentalists showed alarming elevations in IOP, with peak values of up to 29 mm Hg when playing high frequency pitches. The characteristic of the general change of IOP was similar for all three tone frequencies, where IOP typically increased during playing and decreased slowly within 20 seconds after playing ceased, reaching the approximate level of the base value after 2 minutes. Figure 3 shows the fluctuations of the mean IOP with outlying peak values illustrated separately.

The mean systolic BP generally increased when playing began, and returned to the base value after 2 minutes. While playing high pitches on an oboe, a significant elevation of systolic BP from 121±13.0 mm Hg to 131±18.7 mm Hg (p=

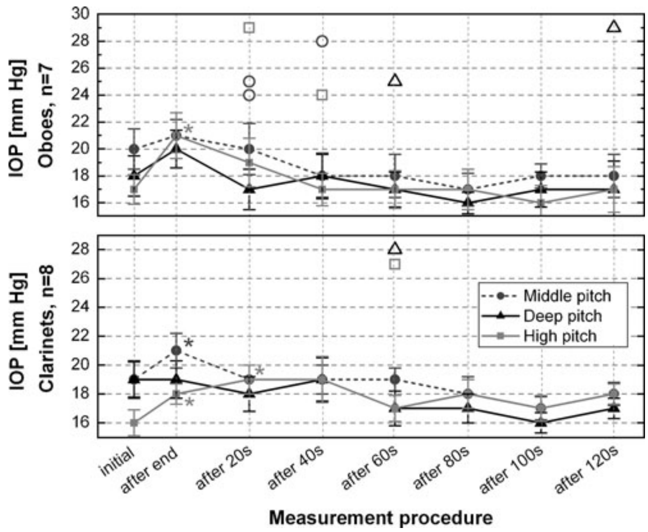

Fig. 3 Course of changes in mean IOP for woodwind instruments at three different frequencies. IOP increases dramatically after playing the tone with increasing volume, and typically decreases to the base value. High pitches lead to a significant elevation in IOP. The increase is typically followed by a slow decrease and reaches the base value after 2 minutes. Significant outliers are illustrated separately as single data points

0.028) was recorded. The diastolic BP remained constant during observation.

Long-term playing

Neither high-resistance (oboe) nor low-resistance wind instrument (clarinet) players showed a significant elevation in IOP and BP while playing a common exercise of 10 minutes. The mean IOP typically increased within the first 2 minutes, reaching a plateau for several minutes, until reverting to the base value. While playing the high pitch with maximal effort, IOP generally increased and returned to the base value at the end of the measurement procedure. However, two clarinet players (25%) and two oboe players (29%) showed elevations of ≥5 mm Hg. Additionally, an increase of ≥10 mm Hg was measured for two oboists. Twenty-six percent of woodwind instrument players reached peak IOP values in the hypertension range, with a maximum value of 32 mm Hg. The highest elevations were typically reached after the maximum effort task.

A similar pattern of increased pressure within the first minutes of playing, a subsequent plateau and a rapid decrease after the end of playing was found for the systolic BP, whereas the diastolic BP remained relatively constant.

IOP dynamics separated for wood instrument class

There was no significant difference between high-resistance and low-resistance woodwind instruments.

General discussion

Ocular hypertension is recognised as a critical risk factor in the development of open-angle glaucoma [2, 3]. To investigate changes in IOP and BP while playing brass and woodwind instruments, we took measurements of both under a variety of common playing conditions. Professional brass and woodwind instrument players were asked to play tones with increasing volume and intensity in three distinct frequencies (low, middle and high, depending on the gamut; see Table 1). In a second experiment, they played a common exercise lasting 10 minutes with a subsequent high pitch tone with maximum effort, to assess the change in IOP and BP during workaday playing conditions.

Playing wind instruments has been shown to cause cardiovascular changes [19–23] similar to those caused by a Valsalva maneuver [24]. It is suggested that a Valsalva maneuver leads to an elevated IOP through a rise in intrathoracic pressure and compression of the intrathoracic venous system. The rise in venous pressure is transmitted to the choroid through the jugular, orbital and vortex veins, causing vascular engorgement, an increase in the choroidal volume and a rise in IOP [26]. Does playing wind instruments cause significant changes in IOP?

Playing sustained tones with increasing intensity is an everyday routine for professional musicians, and is typically one of a series of warm-up exercises.

Our data suggest that playing long sustained tones on brass and woodwind instruments causes a significant temporary elevation in IOP and BP. Brass instrument players showed a significant increase in IOP for low, middle and high frequency tones. The extent of increase is dependent on the frequency of the played tone, as high pitches (ER: 0.284 mm Hg/second) and middle pitches (ER: 0.160 mm Hg/second) lead to a significantly higher elevation than deep tones (ER: 0.159 mm Hg/second). This interdependence was also evident for woodwind instrument players. IOP typically increased in all conditions. However, playing the high pitches caused a significant IOP elevation in oboe players (ER: 0.13 mm Hg/second) and clarinet players (ER: 0.09 mm Hg/second). These findings confirm observations made by Grewal et al. (1995) [38], who measured IOP in musicians after 1 minute of blowing sustained notes in certain frequencies and found that IOP increased dramatically for high notes, but did not change significantly for low or middle tones.

Due to the significantly higher intraoral pressures that are needed for high-resistance wind instruments (e.g., oboe) [39], one would intuitively expect higher elevations for this class of instruments. However, significant differences between high- and low-resistance wind instruments, proposed by Schuman et al. (2000) [26] were not evident in our study. Dramatic ERs for both high-resistance and low-resistance wind instruments, as reported by Schuman and colleagues, cannot be verified by our data, which are typically smaller than those proposed by Schuman et al. (2000) [26]. Our focus in this current study was the examination of the change in IOP. Nevertheless, we recorded BP and found an increase in systolic BP, similar to that recently published by Elghozi et al. (2008) [24].

Previous studies [24, 26] have concentrated on changes in IOP during particular tasks which, in our opinion, do not reflect the normal playing conditions of professional wind musicians. Therefore, we asked subjects to play well-known compositions that are frequently played during warm-up exercises. Our data typically show an elevation of IOP during the first 2 minutes, followed by a plateau and a then small decrease in IOP. When asked to play a high pitch with maximum effort, the IOP increased dramatically for brass as well as for woodwind instruments, with peak values clearly in the ocular hypertension range.

Although mean IOPs might indicate only moderate risks to wind instrument players, individual peaks in IOP can be dramatic. For clinical purposes, individual IOP profiles are crucial and have to be considered in association to further glaucoma risk factors such as family history or cardiovascular disease. Our findings clearly suggest that professional wind instrument players are a risk group for developing glaucoma due to frequent elevations and, more importantly, large fluctuations in IOP. Because of their daily exposure to conditions similar to those used in this study, wind instrument players merit continuous ophthalmic monitoring for signs of glaucoma, especially those musicians with additional risk factors. It is an important task for clinicians to identify those high-risk patients.

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References

1. Pizzarello L, Abiose A, Ffytche T, Duerksen R, Thulasiraj R, Taylor H, Faal H, Rao G, Kocur I, Resnikoff S (2004) VISION 2020: The Right to Sight: a global initiative to eliminate avoidable blindness. Arch Ophthalmol 122:615–620
2. Sultan MB, Mansberger SL, Lee PP (2009) Understanding the importance of IOP variables in glaucoma: a systematic review. Surv Ophthalmol 54:643–662
3. Nassr MA, Morris CL, Netland PA, Karcioglu ZA (2009) Intraocular pressure change in orbital disease. Surv Ophthalmol 54:519–544
4. Morrison JC, Nylander KB, Lauer AK, Cepurna WO, Johnson E (1998) Glaucoma drops control intraocular pressure and protect optic nerves in a rat model of glaucoma. Invest Ophthalmol Vis Sci 39:526–531
5. Heijl A, Leske MC, Bengtsson B, Hyman L, Hussein M (2002) Reduction of intraocular pressure and glaucoma progression:

results from the Early Manifest Glaucoma Trial. Arch Ophthalmol 120:1268–1279
6. Kass MA, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP, Parrish RK 2nd, Wilson MR, Gordon MO (2002) The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. Arch Ophthalmol 120:701–713, discussion 829-730
7. Leske MC, Heijl A, Hussein M, Bengtsson B, Hyman L, Komaroff E (2003) Factors for glaucoma progression and the effect of treatment: the early manifest glaucoma trial. Arch Ophthalmol 121:48–56
8. Johnson EC, Cepurna WO, Jia L, Morrison JC (2006) The use of cyclodialysis to limit exposure to elevated intraocular pressure in rat glaucoma models. Exp Eye Res 83:51–60
9. Nickells RW (2007) From ocular hypertension to ganglion cell death: a theoretical sequence of events leading to glaucoma. Can J Ophthalmol 42:278–287
10. Nickells RW, Schlamp CL, Li Y, Kaufman PL, Heatley G, Peterson JC, Faha B, Ver Hoeve JN (2007) Surgical lowering of elevated intraocular pressure in monkeys prevents progression of glaucomatous disease. Exp Eye Res 84:729–736
11. Leske MC, Warheit-Roberts L, Wu SY (1996) Open-angle glaucoma and ocular hypertension: the Long Island Glaucoma Case-control Study. Ophthalmic Epidemiol 3:85–96
12. Bulpitt CJ, Hodes C, Everitt MG (1975) Intraocular pressure and systemic blood pressure in the elderly. Br J Ophthalmol 59:717–720
13. Klein BE, Klein R (1981) Intraocular pressure and cardiovascular risk variables. Arch Ophthalmol 99:837–839
14. Klein BE, Klein R, Knudtson MD (2005) Intraocular pressure and systemic blood pressure: longitudinal perspective: the Beaver Dam Eye Study. Br J Ophthalmol 89:284–287
15. Bouhuys A (1964) Lung volumes and breathing patterns in wind-instrument players. J Appl Physiol 19:967–975
16. Fiz JA, Aguilar J, Carreras A, Teixido A, Haro M, Rodenstein DO, Morera J (1993) Maximum respiratory pressures in trumpet players. Chest 104:1203–1204
17. Schorr-Lesnick B, Teirstein AS, Brown LK, Miller A (1985) Pulmonary function in singers and wind-instrument players. Chest 88:201–205
18. Herer B (2001) Music and respiratory pathology. Rev Mal Respir 18:115–122
19. Borgia JF, Horvath SM, Dunn FR, von Phul PV, Nizet PM (1975) Some physiological observations on French horn musicians. J Occup Med 17:696–701
20. Dimsdale JE, Nelesen RA (1995) French-horn hypertension. N Engl J Med 333:326–327
21. Faulkner M, Sharpey-Schafer EP (1959) Circulatory effects of trumpet playing. Br Med J 1:685–686
22. Harris LR (1996) Horn playing and blood pressure. Lancet 348:1042
23. Larger E, Ledoux S (1996) Cardiovascular effects of French horn playing. Lancet 348:1528
24. Elghozi JL, Girard A, Fritsch P, Laude D, Petitprez JL (2008) Tuba players reproduce a Valsalva maneuver while playing high notes. Clin Auton Res 18:96–104
25. Rosen DA, Johnston VC (1959) Ocular pressure patterns in the Valsalva maneuver. Arch Ophthalmol 62:810–816
26. Schuman JS, Massicotte EC, Connolly S, Hertzmark E, Mukherji B, Kunen MZ (2000) Increased intraocular pressure and visual field defects in high resistance wind instrument players. Ophthalmology 107:127–133
27. Abraham AG, Condon NG, West Gower E (2006) The new epidemiology of cataract. Ophthalmol Clin North Am 19:415–425
28. Thornton J, Edwards R, Mitchell P, Harrison RA, Buchan I, Kelly SP (2005) Smoking and age-related macular degeneration: a review of association. Eye (Lond) 19:935–944
29. Lee AJ, Rochtchina E, Wang JJ, Healey PR, Mitchell P (2003) Does smoking affect intraocular pressure? Findings from the Blue Mountains Eye Study. J Glaucoma 12:209–212
30. Morgan RW, Drance SM (1975) Chronic open-angle glaucoma and ocular hypertension. An epidemiological study. Br J Ophthalmol 59:211–215
31. Reynolds DC (1977) Relative risk factors in chronic open-angle glaucoma: an epidemiological study. Am J Optom Physiol Opt 54:116–120
32. Wilson MR, Hertzmark E, Walker AM, Childs-Shaw K, Epstein DL (1987) A case-control study of risk factors in open angle glaucoma. Arch Ophthalmol 105:1066–1071
33. Katz J, Sommer A (1988) Risk factors for primary open angle glaucoma. Am J Prev Med 4:110–114
34. Klein BE, Klein R, Ritter LL (1993) Relationship of drinking alcohol and smoking to prevalence of open-angle glaucoma. The Beaver Dam Eye Study. Ophthalmology 100:1609–1613
35. Quigley HA, Enger C, Katz J, Sommer A, Scott R, Gilbert D (1994) Risk factors for the development of glaucomatous visual field loss in ocular hypertension. Arch Ophthalmol 112:644–649
36. Stewart WC, Crinkley CM, Murrell HP (1994) Cigarette-smoking in normal subjects, ocular hypertensive, and chronic open-angle glaucoma patients. Am J Ophthalmol 117:267–268
37. Kang JH, Pasquale LR, Rosner BA, Willett WC, Egan KM, Faberowski N, Hankinson SE (2003) Prospective study of cigarette smoking and the risk of primary open-angle glaucoma. Arch Ophthalmol 121:1762–1768
38. Grewal RK, Karalekas DP, Kraff C, Hawkinson DW, Krupin T (1995) Intraocular pressure change with increased pulmonary outflow. Investig Ophthalmol Vis Sci 36:295–387
39. Schwab B, Schultze-Florey A (2004) Investigations into intraoral pressure in woodwind and brass musicians. Musikerphysiologie und Musikermedizin 11:183–194