Nasal dysfunction induced by chlorinate water in competitive swimmers*

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Summary

Aims: Swimmers commonly complain of nasal symptoms probably due to mucosal irritation caused by chlorinated water. The aim of the present prospective study was to investigate changes in nasal function and cytology in a cohort of 15 volunteer competitive swimmers, as compared with a control group of 15 competitive athletes practicing other sports.

Methods: Olfactory threshold for n-butanol was measured in a population of competitive swimmers. Changes in nasal function and cytology were compared between the two groups of volunteer competitive athletes.

Results: There were no significant differences between the two groups in terms of mean 20-Item Sino-Nasal Outcome Test scores, peak nasal inspiratory flow, pulmonary peak expiratory flow, or total nasal resistance on anterior active rhinomanometry. Nasal mucociliary transport time (MCTt) was significantly shorter for the non-swimmers than for the swimmers. The mean olfactory threshold for n-butanol in the swimmers was significantly lower than in the other group of athletes.

Conclusions: Data seem to confirm the utility of MCTt in studying nasal mucosa damage caused by chlorinated water. The present results also support the hypothesis of a role for the olfactory threshold in evaluating damage to the olfactory mucosa exposed to chlorinated water.

Key words: competitive swimmers, olfactory threshold, mucociliary transport time, nasal patency, nasal cytology

Introduction

Regular physical activity is recognized as an effective health promotion measure. In the general population, swimming is considered one of the preferable physical activities, after walking and cycling [(1)], but swimmers commonly complain of nasal obstruction, sneezing or itching, irritation of the conjunctiva, cough and wheezing, probably due to mucosal irritation by chlorinated water [(2)].

The aim of the present prospective study was to investigate nasal function changes, e.g. in respiratory flow, olfactory threshold, mucociliary transport time (MCTt), and morphological modifications identifiable by nasal cytology in a cohort of 15 volunteer competitive swimmers, and to compare them with findings in a control group of 15 competitive athletes practising other sports.

Materials and methods

Study design

The present investigation was a prospective study conducted in accordance with the 1996 Helsinki Declaration and was approved by the internal committee of the Section. All participants gave their written informed consent to involvement in the study. Thirty Caucasian competitive athletes were enrolled: the study group consisted of 15 competitive swimmers; the control group comprised 15 competitive athletes practising other sports (running, cycling, tennis, soccer, basketball, and volleyball). All the subjects involved had volunteered. They were non-smokers

and did not suffer from any type of allergy. None of them had a history of sinonasal surgery or loss of sense of smell, and none of them was taking any medication.

The athletes in the study group (12 males and 3 females; mean age 33.5 ± 9.5 years; mean height 176.7 ± 8.5 cm) had been swimming for 22.1 ± 9.8 years, and had been doing so competitively for 5.9 ± 5.4 years. They all trained at the same indoor swimming pool, without using a nose clip, for 4.5 ± 2.6 hours a week divided over two different days. The concentration of free chlorine in the pool ranged between 0.7 to 1.5 mg/L Cl_2, while the concentration of combined chlorine was 0.4 mg/L Cl_2, according to Italian legislation in terms of swimming pools. The chemical analysis on swimming pool water was serially repeated by both an internal and an independent committee. Swimmers were evaluated 2.8 ± 2.4 days after the last training session.

The volunteers in the control group (12 males and 3 females; mean age 32.7 ± 7.9 years; mean height 177.4 ± 7.1 cm) had been practising their respective sports for 13.3 ± 7.9 years and had been involved in competitions for 11.9 ± 7.6 years. They all trained for at least 4 hours a week (mean 9.1 ± 5.2 hours a week). Controls were evaluated after 2.2 ± 1.7 days after the last training session. None of those had swum in a swimming pool within at least 1 month before entering into the study.

All participants were asked to complete a SNOT 20 questionnaire (Sinonasal Outcome Test [3]) to record their nasal symptoms, and were then assessed in terms of peak nasal inspiratory flow (PNIF), pulmonary peak expiratory flow (PEF), basal anterior active rhinomanometry (AAR), *Nez du Vin*, odour threshold for *n*-butanol, nasal cytology, and MCTt.

Assessment of PNIF

A portable Youlten peak flow meter (Clement Clark International, Harlow, United Kingdom) was used to measure PNIF. Volunteers were encouraged to inhale as hard and fast as they could through the nose with their mouth tightly closed and the mask placed firmly over the face, starting from the end of a full expiration. All subjects were seated during the test. As in previous experiences [4,5], three satisfactory maximal inspirations were obtained for each subject and the highest value was considered as the basal PNIF value.

Assessment of PEF

PEF was measured with a portable peak flow meter (TruZone, Trudell Medical, Ontario, Canada). Volunteers were encouraged to exhale through the mouth as hard and fast as they could into the mouthpiece of the instrument, starting from the end of a full inspiration. Three satisfactory maximal expirations were obtained and the highest value was considered as the basal PEF value [5].

Assessment of nasal patency

Nasal patency was also evaluated using AAR (Rhinolab, Rendsburg, Germany) according to the International Committee on Standardisation of Rhinomanometry [6]. At least five breaths were recorded at a fixed transnasal pressure of 150 Pa during quiet breathing with the mouth closed and the subject in a seated position. Airflow values were expressed in ml/sec. Total nasal resistance was calculated by combining the two separate of nasal resistance values for each nasal passage using the following equation:

$R_{total} = R_{left} \times R_{right} / (R_{left} + R_{right})$

Values were expressed in Pascal (Pa).

Assessment of sense of smell

The participants' normal sense of smell was first evaluated using the *Nez du Vin* method [7], which is a screening test of olfactory capacity that allows to quickly exclude hyposmic subjects. Since none of the participants gave more than one wrong answer, they underwent the Sniffin' Sticks test [8] only to establish their olfactory threshold for *n*-butanol (Burghart Medical Technology, Wedel, Germany), as described in a previous study [9]. To measure olfactory threshold, the odorants were presented in felt-tip pens to the subjects, who were blindfolded to prevent them from visually distinguishing the pens.

Cytology

Nasal cytology was performed by anterior rhinoscopy, using a nasal speculum. The collection technique consisted of scrapings from the middle portion of the inferior turbinate, using a Rhino-Probe (Arlington Scientific Inc., Springville, UT, USA) nasal curette. The specimens were fixed in 100% alcohol and underwent May-Grünwald-Giemsa staining. All specimens were examined under the light microscope by the same operator (GO), who was unaware of the sport practised by each subject. The cytologic variables considered were: the total number of ciliated cells, with and without hyperchromatic supranuclear stria (HSS), as described in a previous study [10] and the total number of inflammatory cells (neutrophil granulocytes and eosinophil granulocytes) counted for each specimen in 5 separate high-power fields (HPF, original magnification x100). The MCTt was established by positioning charcoal powder at the head of the inferior turbinate [11] and ascertaining its transit from the nasal fossa to the rear wall by direct pharyngoscopy.

Statistical analysis

A Welch test was used to compare all the variables between the two groups, assuming a different variance between the two cohorts. A p-value <0.05 was considered statistically significant.

Spearman's rank correlation test was used to correlate the olfactory threshold with both PNIF and AAR in each group. The R: a language and environment for statistical computing

(R Foundation for Statistical Computing, Vienna, Austria) was used for all analyses.

Results

No significant differences emerged in the mean SNOT 20 scores, PNIF, PEF, or total nasal resistances on AAR between then two groups of athletes (Table 1). In particular, none of the swimmers or other athletes scored more than 1 in the SNOT 20.

All volunteers were tested for their olfactory threshold for *n*-butanol based on the Sniffin' Sticks test battery. The mean olfactory threshold for *n*-butanol in the group of swimmers was 8.2 ± 3.7, while for the other athletes it was 10.9 ± 0.5; this difference proved statistically significant (p = 0.017). The nasal function test results are summarized in Table 1. Nasal MCTt was significantly shorter for the non-swimmers than in the group of swimmers (p = 0.00000159). In particular, the mean MCTt among the swimmers was 20.2 ± 2.5 minutes, while for the other athletes it was 17.0 ± 0.9 minutes (Table 2).

The cytological study on nasal mucus showed that the total number of ciliated cells (with or without HSS) (Figure 1A) did not differ statistically between the swimmers and the other athletes (p = 0.24). The neutrophil count (Figure 1B) was nil for 8 cases in the group of swimmers and 8 athletes in the control group. The eosinophil count was nil for 12 swimmers and 14 control athletes. The mean neutrophil count was also not significantly different between the two groups (p = 0.27). The results of nasal cytology are summarized in Table 2.

Finally, Spearman's rank correlation test failed to identify any significant correlation in either group between the athletes' olfactory threshold and their PNIF (p = 0.36 and 0.57 for swimmers and non-swimmers, respectively) or total nasal resistance by AAR (p = 0.79 and 0.41 for swimmers and non-swimmers, respectively).

Discussion

Nasal respiration is needed to condition inhaled air and provide a defensive barrier. The role of nasal ventilation becomes even more important in athletes, and an impaired nasal function can affect their physical performance [12]. Various authors have reported that chlorine and other components of swimming pool water (such as chloramines, chloroform, formaldehyde, acetaldehyde and halogenated hydrocarbons) may damage the respiratory mucosa in swimmers, causing nasal obstruction, sneezing and nasal serosa secretion [2,12,13]. To avoid any bias due to the variable chemical characteristics of the water in different swimming pools, we investigated a group of swimmers who all trained in the same pool.

In quantitative terms, nasal patency did not seem to decrease and, likewise, nasal resistances did not increase, in our group exposed to chlorinated water. Bougault et al. [14] recently measured PNIF in 39 swimmers and a control group of non-swimmers, and were also unable to find any significant difference. Similar results were also obtained by Alves and Martins [13], who measured PNIF in a group of competitive swimmers before and after swimming. Ondolo et al. [15] measured nasal resistances by AAR in a group of 30 competitive swimmers before and after a training session in the pool, and again found no significant differences. The present study seems to confirm these earlier results, as we were unable to find any significant differences between the PNIF or nasal resistances measured by AAR in competitive swimmers and those of other competitive athletes. On the other hand, Clearie

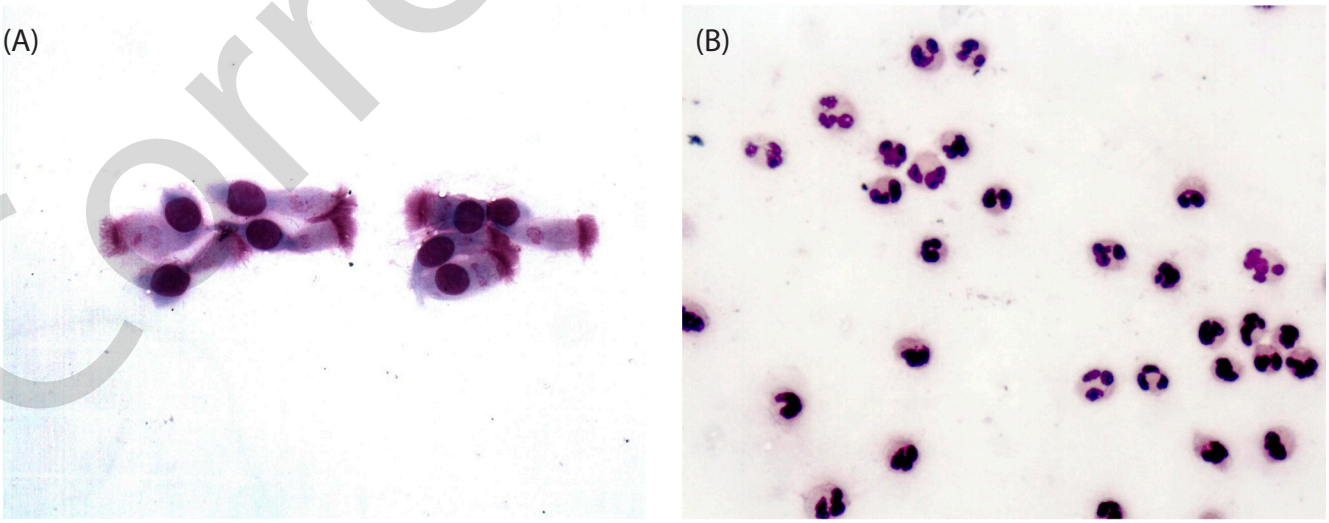

Figure 1. (A) Ciliated cells (May-Grünwald-Giemsa stain, original magnification ×40). (B) Neutrophils (May-Grünwald-Giemsa stain, original magnification ×60).

Table 1. Nasal function parameters in groups of swimmers and non-swimmers.

Variables	Swimmers	Non-swimmers	p
SNOT 20	0.2 ± 0.2	0.2 ± 0.1	0.40
PNIF (L/min)	233.3 ± 58.0	258.0 ± 66.2	0.29
PEF (L/min)	601.3 ± 99.4	601.3 ± 96.5	1.00
ARR mean total nasal resistance (Pa*sec/ml)	0.1 ± 0.1	0.1 ± 0.1	0.85
Olfactory threshold	8.2 ± 3.7	10.9 ± 0.5	0.017

Table 2. Nasal cytology and MCTt results in groups of swimmers and non-swimmers.

Variables	Swimmers Mean ± SD	Non–swimmers Mean ± SD	p
Mean ciliated cell count	12.6 ± 12.0	15.5 ± 7.4	0.24
Mean neutrophil count	15.2 ± 36.2	4.3 ± 9.9	0.27
MCTt (min)	20.2 ± 2.5	17.0 ± 0.8	$1.59e^{-5}$

et al. [16] measured PNIF in a group of 36 adolescent competitive swimmers before and after training, and surprisingly found a significant increase in nasal patency after swimming, attributing this to a "nasal douche effect". This last study supports the hypothesis that chlorinated water does not negatively affect nasal flows. Moreover, also in the current study, as previously reported [17], the toxic irritation of the nasal mucosa induced by the chlorinate water exposure produces an inter-individual variability of reaction (as confirmed by our large SD in ARR) that could also explain the unchanged nasal patency of the swimmers included in the study.

Nasal cytology is a field of increasing interest to rhinologists. Gelardi et al. [18] recently showed a significant increase in the neutrophil count in swimmers complaining of nasal symptoms (e.g. rhinorrhea, itching, sneezing, obstruction) as compared with a control group. These results do not necessarily contrast with our findings, since none of the swimmers involved in our study complained of rhinitic symptoms (SNOT 20 score ≤1), which would reasonably explain their relatively low neutrophil count.

As in previous reports [12,19], we found a significantly shorter nasal MCTt in non-swimmers than in swimmers, indicating that assessing MCTt would be a sensitive method for detecting damage to the nasal mucosa induced by prolonged exposure to chlorinated water. Oddly enough, our control group also revealed a longer MCTt than the normal values available in the literature [11]. Since our control group consisted mainly of runners, this result could well be an expression of the so-called "athlete's nose" [12]. Exercise, and running especially, has the effect of reducing nasal mucosa congestion in much the same way as a nasal decongestant, and is believed to be a consequence of changes in arterial pCO_2, mediated by the autonomic innervation of the nasal vasculature [9]. If these alterations persist, they could cause dryness of the nasal mucosa, justifying the altered MCTt.

To the best of our knowledge, this is the first time the olfactory threshold for *n*-butanol has been measured in a population of competitive swimmers. Our results indicate that regular swimmers have a significantly lower olfactory acuity than non-swimmers as confirmed by swimmers' lower odour threshold according to the Sniffin' Sticks test; like the MCTt for the nasal respiratory mucosa, this seems to be the consequence of the olfactory mucosa being damaged by the chlorinated water. In our opinion, odour threshold and MCTt are together the first most sensitive signs of toxic epithelial damage respectively to the olfactory mucosa and to the respiratory nasal mucosa. There is a difference between swimmers *Nez du Vin* and *n*-butanol olfactory threshold results, as the former did not show significant differences between the groups as the latter did. In our opinion, this may be explained by the fact that the *Nez du Vin* test is just a screening test based on the identification of six suprathreshold odorants, while the odour threshold test is based on the identification of the lowest concentration of an odorant presented. Obviously, odour threshold is a more sensitive test as demonstrated by the fact that olfactory thresholds decrease more significantly with age than odour identification [8]. Moreover, our results are based on a limited sample of subjects so the present results should be considered as preliminary.

Finally, no correlations emerged between PNIF or AAR findings and the olfactory thresholds in either of our groups. A recent work by Philpott and coworkers [20] produced similar results when the authors studied the correlation between nasal flows in terms of PNIF and the subjects' olfactory threshold to eucalyptol, finding none. The authors concluded that their results could be explained by the highly variable local airflows at olfactory cleft level, which are difficult to quantify using PNIF measurements. The same study nonetheless identified a direct correlation between nasal flows and olfactory threshold to phenethyl alcohol (PEA). These different findings can probably be explained by the molecules' different vapour tension and different olfactory receptor sensitivity.

Conclusions

Our data seem to confirm the usefulness of MCTt for studying nasal mucosa damage caused by chlorinated water. Our preliminary findings also support the hypothesis of a role of the olfactory threshold in identifying damage to the olfactory mucosa exposed to chlorinated water.

Further investigations on larger series of swimmers are needed to confirm our results. In particular, the MCT data should be confirmed using other objective methods able to assess mucociliary clearance such as the study of the ciliary beat frequency using inverted phase-contrast light microscope [21] or rhinoscintigraphy[22]. Furthermore, the olfactory threshold result should be investigated also by electrophysiological methods.

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Author contribution

Each of the authors has contributed to this manuscript.

Conflict of interest

None.

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