Swimming is widely promoted as a suitable exercise to maintain cardiovascular fitness, manage weight and improve joint health. It is the most common sport undertaken by the general public in England according to the latest figures from Sport England, with more than 2.5 million people swimming weekly in 2014/2015. Because of the weight-bearing properties of water it causes very few injuries, but there have recently been concerns around the link between chlorine exposure and upper respiratory tract problems.

There appears to be a discrepancy in the symptoms experienced between recreational and competitive swimmers. This paper will discuss the disorders experienced by competitive swimmers. These swimmers are training vigorously, for prolonged periods of time and as such these disorders are seen more frequently than in recreational swimmers.

THE SWIMMING ENVIRONMENT

Elite swimmers train in a unique environment compared to other elite athletes. An indoor swimming pool is a warm, humid environment containing a number of chemicals. Most indoor swimming pools use chlorine as the main disinfectant agent, although some newer pools use other techniques, such as saline chlorination units, ultra violet and ozone systems. Swimming pools are normally kept between 27 and 29°C for both water and air temperature and are naturally very humid environments, with levels of around 60% humidity commonly seen.

Chlorine reacts with nitrogen-containing products in the water, such as urine or sweat, or synthetic products like cosmetics or soap. This reaction releases chloramines and nitrogen trichloride as well as other gases into the atmosphere, which is what causes the traditional swimming pool smell, not
Chlorine as is widely believed. Chloramines are an irritant to the eyes, skin and the respiratory system and are responsible for most of the problems that swimmers complain of. Chloramines are heavy gases compared with air and sit just above the surface of the water. It is possible to measure the concentration of these gases and there are WHO guidelines on the management of water and air quality in pools to ensure a safe environment.

Confusingly, the release of chloramines is higher in a pool that is inadequately chlorinated for the actual usage of that pool, because if the chlorine level is not high enough to oxidise the nitrogenous waste products, the free chlorine combines with the waste products to create chloramines. The ventilation of the pool also needs to be sufficient to remove the chloramines produced from the air. All of these factors need to be taken into consideration if users of a particular pool start to report problems. One of the key, but often overlooked factors in maintaining a healthy pool environment is looking at the washing behaviour of the bathers prior to swimming, as well as the number of bathers in the pool to reduce the quantity of nitrogenous compounds being released into the pool water.

THE EFFECT OF SWIMMING ON THE RESPIRATORY SYSTEM

Nasal symptoms are likely to be the problem most commonly reported by elite swimmers, as they tend to affect everyday life and potentially contribute to the increased rate of upper respiratory tract infection in swimmers compared to other endurance athletes. Symptoms include rhinorrhoea, sneezing, itching and obstruction or congestion and have been reported in up to 72% of swimmers1.

On examination it is not uncommon to see nasal polyps, inflamed and enlarged turbinates as well as congested mucosa on nasal endoscopy or computed tomography examination of the nasal cavity.

Clinically, obstructed airways can lead to altered breathing patterns, with an increased tendency to mouth breathe, particularly during sleep. This leads to the swimmer inhaling dry, unfiltered air which may be one cause of reported increased infection rates2. Chronic obstruction may lead to decreased sinus drainage and an increased risk of sinus infection, which seem to be particularly troublesome in swimmers due to water pressure on the face when diving and turning, as well as the head down posture that is necessary to turn during freestyle and backstroke.

It has been shown that exercise itself causes increased rates of rhinitis in athletes3. Geraldi et al looked at nasal cytology in swimmers and found that 44% of swimmers with rhinitis had a pre-existing atopic tendency, but in those who were not atopic, 63% had predominately neutrophilic nasal inflammation, which decreased after using a nose clip for 30 days4. This may show a direct irritative role of chlorine on the nasal mucosa. In elite swimmers nose clips are unpopular due to the subsequent inability to exhale through the nose, which is the preferred breathing pattern.
Oral antihistamines, topical nasal steroids – either as spray or drops – and post-swimming nasal douches have all been reported to reduce symptoms and, anecdotally, decrease the rate of infections in swimmers. For troublesome symptoms, polypectomy and turbinectomy also decrease symptoms.

**ASTHMA IN SWIMMERS**

The link between swimming and asthma has been widely discussed in the literature, mainly among pool workers, children and recreational swimmers, but the results are not conclusive. However, there is now a body of evidence which indicates that the rates of exercise-induced asthma or exercise-induced bronchoconstriction (EIB) are much higher among elite swimmers, with reported rates ranging from 41% to 76%. It is also established that elite athletes have higher rates of EIB than the general public, with studies showing a prevalence of 21%. So it can be seen that the prevalence in swimmers is higher than in other endurance athletes.

Recent studies screening different sports have reinforced this, with members of the Great Britain boxing and swimming teams undergoing eucapnic voluntary hyperventilation tests (EVH) as a screen for bronchoconstriction. These groups included athletes with a previous diagnosis of asthma. The percentage of positive EVH tests was 77% (9 out of 39) in boxers and 69.7% (23 out of 33) in swimmers.

The existing body of literature seems to confirm a possible link between swimming and EIB or airway hypersensitivity; however this does not suggest that in the recreational swimmer we can conclude that swimming causes asthma.

Asthma can be diagnosed clinically, with symptoms including wheezing, dyspnoea and chest tightness, with a drop in peak flow. On questioning those swimmers with a positive EVH test, there was no correlation between reporting of symptoms and the test results. There is also evidence that pre- and post-exercise peak flow readings do not drop in swimmers and have been shown to rise, so it is likely that we are looking at a different clinical condition in elite swimmers. From a public health perspective it is important to highlight this difference, as within our increasingly sedentary population, swimming as a form of exercise should not be discouraged in either children or adults. We also need to remember the safety benefits of being a competent swimmer in reducing drowning incidents. Thus the term ‘exercise-induced bronchoconstriction’ is preferable to ‘exercise-induced asthma’.

When the combination of high respiratory rates, breathing air close to the surface of the water – where the highest concentration of chloramines are found – high humidity levels and the prolonged exposure to these conditions (between 20 to 30 hours a week) are considered, we can see why swimmers are more prone to respiratory problems. As can be seen in other endurance athletes, increased ventilation over a prolonged period of time may affect the respiratory epithelial cells. This potential epithelial damage, in addition to osmotic stress, may lead to the release of inflammatory mediators and possibly initiate a repair process involving fibrogenic cytokines. It could be assumed that the high humidity and temperature of an indoor swimming pool may be protective, when compared with other outdoor endurance sports such as cross-country skiing, where high rates of EIB are seen with cold, dry air, however, repeated inhalation of the chloramines can contribute to allergic diseases and asthma in swimmers.

In recreational swimmers, children or pool staff, indoor chlorinated swimming swimmers can become over-reliant on the upper respiratory component of breathing, with over-utilisation of the upper trapezius and scalene muscles. This can lead to tension in the larynx and voice box, leading to spasm and inspiratory stridor.
pool attendance induces a rapid and transient disruption of the airway epithelium without development of asthma symptoms. Inhaled chloramines and other gases in chlorinated swimming pools could have the potential to cause structural changes in the airway epithelium, allowing allergens easier access to antigen-presenting cells, leading to allergen sensitisation. The mechanisms responsible for epithelial damage and airway hypersensitivity in swimmers remain incompletely understood. The increased oxidative stress observed in the upper airways of swimmers and the reduced antioxidant capacity may promote the release of inflammatory mediators and sensitisation of airway smooth muscle, contributing to the development of EIB. Certainly the rate of atopy, allergy, eczema and food allergies seems to be higher in elite swimmers. An audit of the British swimming team recently showed that four out of 65 swimmers at the 2015 Commonwealth Games carried an epipen for anaphylaxis.

No studies have been published on the possible improvement in performance of treating previously undiagnosed EIB in swimming. However, a study in professional rugby union players has shown an increase in aerobic performance in an on-field endurance test in previously undiagnosed EVH-positive players, when treated with inhaled corticosteroid, although the small group of players tested did not reach statistical significance. In a recent study Koch et al. showed that a one-off dose of salbutamol has no impact on cycling performance in previously undiagnosed EVH-positive athletes.

The margins of victory in swimming are so small that there may be a beneficial improvement in performance by treating rhinitis or bronchoconstriction. However, it is also important to consider that there is a potential health improvement for the swimmers if successfully diagnosed and treated, with fewer infections of the upper respiratory tract allowing for less training days missed and decreased unpleasant symptoms such as cough, rhinitis, increased mucus production and sinus congestion.

Currently, competitive swimmers are not routinely screened for upper airway dysfunction. Options could include questionnaires, spirometry, exercise tests, methacholine or EVH testing. There is no validated questionnaire that has been shown to be an effective screening tool and therefore use of a questionnaire could potentially lead to an inaccurate diagnosis. In the cohort of swimmers screened by Levai et al., six (26.1%) of the 23 EVH-positive participants reported no exercise respiratory symptoms during swimming. All 10 (30.3%) EVH-negative athletes reported at least one exercise respiratory symptom. There was no association between the experience of chest tightness, breathing difficulty and wheezing during or after training, with positive EVH-result. In swimmers, airway hyper reactivity can be found in the presence of a normal resting flow volume curve and normal peak flow, with no drop in peak flow during exercise, emphasising the need to use bronchial provocation or reversibility tests to assess airway function. Furthermore, two studies have shown that in some athletes, provocation tests with direct airway stimulation with methacholine can be negative, but at the same time have a positive EVH test, confirming EIB.

The concern of inaccurate diagnosis was shown in a recent study by Levai et
Three EVH-negative swimmers and seven EVH-positive swimmers reported having been previously diagnosed with asthma or EIB and were using one or a combination of short-acting β₂-agonists, long-acting inhaled β₂-agonists and inhaled corticosteroids. These previously-diagnosed, EVH-negative swimmers had not been formally tested or screened before. Their diagnosis had been made on a combination of signs, symptoms and – increasingly – fatigue and recurrent upper respiratory tract infections. The swimmers with a negative EVH test stopped their all of their inhalers and underwent a breathing re-training programme, with no reported decrease in respiratory function and no reduction in performance.

It may have been that the symptoms which they had experienced at the time of diagnosis could have been due to impaired breathing patterning, inspiratory stridor, vocal cord dysfunction or anxiety, some of which can be worsened by inhaler use and can lead to an unhelpful diagnosis of asthma from primary care and sports physicians, which then prevents further investigation.

Exhaled nitric oxide (ENO) has been suggested as a simple non-invasive screening method to assess swimmers for EIB, but currently the evidence in the literature is mixed. It has been previously established that ENO levels are higher in asthmatics, however there appears to be no change in ENO levels pre- and post-exposure (swim training) and in children there is no correlation between swimming exposure and ENO levels. In competitive swimmers no link has been shown between positive EVH test and ENO levels.

INSPIRATORY STRIDOR AND BREATHING DYSFUNCTION

Swimmers often complain of post-exertion cough, sore throat with a hoarse voice, noisy breathing or ‘wheezing’ and feeling short of breath during training. If they have been investigated for rhinitis and exercise-induced bronchospasm and no cause found then it is necessary to look at their biomechanics and breathing patterning. Inspiratory stridor is commonly seen in swimmers, as it is in other endurance athletes and should also be considered.

Swimming is essentially an overhead sport and the arms provide the majority of the propulsive effort, but swimmers need excellent core stability to provide the link between the upper and lower body and to keep them flat in the water to reduce drag and this can cause splinting of the diaphragm. Of the four strokes, butterfly can lead to increased stiffness in the thoracic spine, leading to decreased rib excursion during breathing. A combination of these two factors mean that swimmers can become over-reliant on the upper respiratory component of breathing, with over-utilisation of the upper trapezius and scalene muscles. This can then lead to tension in the larynx and voice box, leading to spasm and inspiratory stridor, as opposed to the expiratory wheeze seen in asthma. This can be shown using video recordings during hard interval training and after racing. The added anxiety associated with racing can exacerbate muscle tension and spasm, making the problem worse. If these swimmers have been incorrectly diagnosed as being asthmatic during childhood, they will often use β₂-agonist inhalers repeatedly during training, with no improvement in symptoms. As well as the video observation discussed earlier, direct laryngoscopy may show incomplete closure of the larynx on inspiration, helping to confirm the diagnosis.

There is currently very little published around biomechanics and breathing in swimming, but in the author’s experience, altering breathing mechanics to increase diaphragm activity, increase thoracic mobility and reduce tension/spasm in and around the shoulder and neck muscles has been beneficial in helping to improve stridor.

Another factor that has been shown to cause inspiratory stridor is gastro-oesophageal reflux disease. Both the head-down position associated with dives and turns and lying flat in the water when swimming, which may increase reflux. When this is combined with the possible use of mildly acidic sports drinks it is possible to see how this could be a problem for swimmers. In those where a negative EVH test has been performed and breathing re-training has failed to help, an antacid before intensive training may be beneficial. This reflux may cause some of the cough and sore throat symptoms in swimmers who have no demonstrable bronchospasm on exertion.

 increased exposure to chlorine may lead to airway remodelling and inflammation
Breathing patterns in swimming are unusual compared to other endurance sports, as exhalation usually occurs under water, using ‘trickle breathing’, primarily via the nose, particularly during aerobic, steady state training. This means that swimmers are breathing out against the resistance of the water, in addition to the resistance of the nasal airways. This may lead to a degree of air trapping and hyperexpansion of the lungs, which will alter buoyancy in the water, but as yet it is not apparent if this is beneficial or detrimental to performance.

SUMMARY
• Elite swimmers can be considered a different population to recreational swimmers, as a result of their differing exposure patterns. It may be that the increased exposure to chlorine leads to an airway remodelling and inflammatory process, giving a positive EVH test. One potential problem is the dry air used for EVH tests at only 2% humidity, versus the 60% humidity seen in a typical swimming pool, which may affect airway responsiveness during the testing process. As yet this has not been accounted for in the research.
• Further work needs to be performed to look at the difference in individual swimmers when they change training environments, to see if this alters the EVH response and possibly ENO levels. This may give further clues as to whether the environment is the cause of the higher EVH-positive rates.
• It is currently not established whether treating athletes with previously undiagnosed EIB leads to an increase in performance.
• Treatment of nasal symptoms probably decreases the rates of unpleasant symptoms and increases the quality of life for swimmers. It may also decrease the incidence of upper respiratory tract infection, but this has not been confirmed.
• In those swimmers with respiratory symptoms, but a negative EVH test, breathing re-training exercises may be beneficial.
• To help guide treatment options in swimmers presenting with breathing dysfunction, an EVH test is invaluable to determine the presence of EIB and should be considered by sports and primary care physicians.
• The increased rate of positive EIB tests in elite athletes should not be used as evidence that swimming causes asthma in children or recreational swimmers, due to the different exposure patterns between these groups.

References
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Jon Greenwell M.B.B.S., M.Sc., F.F.S.E.M.
Specialist in Sport and Exercise Medicine
Medical Officer, British Swimming
Leeds, UK
Contact: Jon.Greenwell@swimming.org