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SYSTEMATIC REVIEW

Physical activity and gastro-oesophageal reflux

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Abstract

Objectives: Objective: The performance of many athletes is impaired by gastro-oesophageal reflux (GER), and if this reflux is repeated frequently, the regurgitation of the acid contents of the stomach into the oesophagus can cause the condition of gastro-oesophageal reflux disease (GERD), with inflammation, haemorrhage and even cancerous change in the cells lining the oesophagus. This article thus reviews the impact of acute exercise, regular habitual physical activity and competitive sport upon the risks of GER and GERD. *Methods:* A systematic Ovid/Medline search of the literature from 1996 to 2015 paired the terms physical activity/motor activity, exercise, and training/physical education with gastro-esophageal reflux and esophageal regurgitation; this information was supplemented by a review of reference lists, related articles listed on Pub Med and Google Scholar, and material obtained from the author's personal files. *Results:* Moderate exercise usually has no effect upon the likelihood of GER, although some studies have shown a small decrease in the volume and/or duration of reflux. However, GER is increased in a substantial proportion of individuals during athletic events and bouts of vigorous aerobic exercise at intensities >70% $\dot{V}O_{2max}$. Exercise that causes body oscillation (such as running), an increase of intra-abdominal pressures (as seen for instance in weight-lifting), and bending or adoption of the prone position (as in surfing) seem particularly liable to cause GER. The effects of regular, moderate physical activity are less certain, since almost all reports to date have been based upon questionnaires rather than objective monitoring of oesophageal pH; nevertheless, 8 of13 studies have concluded that regular moderate physical activity reduces the risk of GER. *Conclusions:* Very vigorous exercise increases the immediate risk of GER, and if such episodes recur frequently, there are serious dangers to health. The risk of reflux for the average person seems to be reduced by lifestyle changes, particularly control of obesity and regular moderate physical activity. Athletes who are severely affected by acid reflux may require treatment with proton pump inhibitors, although such medication is not always effective. **Health & Fitness Journal of Canada 2015;8(4):25-51.**

Keywords: Barrett's disease, Epigastric pain, Heart Burn, Lifestyle, Obesity, Oesophageal motility, Oesophageal pH, Oesophageal sphincter.

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Introduction

For some athletes, the regurgitation of gastric acid into the oesophagus and the associated "heartburn" are unpleasant consequences of vigorous exercise, with potentially dangerous complications. In this review, we will look briefly at available information on normal oesophageal function and how this is changed during exercise. We will explore how far the risk of gastro-oesophageal reflux is increased during physical activity and athletic events. Significant clinical problems associated with repeated gastro-oesophageal reflux will also be noted, including inflammation, ulceration and haemorrhage in the oesophagus, a permanent stricture, a squamous cell metaplasia of the oesophageal lining and the development of an oesophageal adenocarcinoma. We will examine the preventive value of moderate exercise and other changes in lifestyle, and will summarize current recommendations for the treatment of this condition.

Search Methods

A search of Ovid/Medline for the period 1996 to 2015 paired the terms *physical activity/motor activity, exercise*, and *training/physical education* with *gastro-esophageal reflux* and *esophageal regurgitation,* without restriction on the language of publication. Additional references were sought through Google Scholar and PubMed. This process yielded

a total of 220 entries. A review of the titles yielded 66 abstracts that were judged relevant to various aspects of the present review, and the full text of these articles was obtained. The primary foci of these papers and the corresponding number of citations were exercise and training (21), lifestyle (13), the athlete (4), clinical diagnosis and management (10), aetiology (7), and respiratory issues (11). Because of the limited total number of relevant articles, all were considered, although it should be emphasized that most of the studies relating to the effects of chronic physical activity upon gastro-oesophageal reflux were based on symptom reports rather than objective measures such as reductions of oesophageal pH. Supplementary material was gleaned from article reference lists and the author's extensive personal files.

Normal oesophageal function

Resting conditions

A muscular sphincter that is normally closed, to prevent the swallowing of air during breathing, guards the upper end of the oesophagus. At the lower end, a second sphincter restricts the reflux of acid and bile from the stomach. The second sphincter is also normally closed. During the swallowing of food or fluid, a wave of peristaltic contraction passes along the length of the oesophageal wall at a speed of about 2 m/s, propelling fluid or food forward, and the lower sphincter relaxes briefly as the ingested material reaches the entrance to the stomach (Ingelfinger, 1958). This process is coordinated by the medulla oblongata and vagal innervation. Disorders of oesophageal function can lead to difficulties in swallowing (dysphagia) and also to chest pain if a reflux of acid from the stomach reaches the oesophageal lining (Peters et al., 1988).

Effects of physical activity

Although many endurance athletes ingest fluids while they are competing, few studies discuss the effects of physical activity upon the mechanics of the healthy oesophagus (Józków et al., 2006; Moses, 1990; Moses, 1994; Peters et al., 1988; Shawdon, 1995; Shephard, 2013). Indeed, the classical review of (Ingelfinger, 1958) made no reference to any changes in oesophageal function that might be brought about by exercise.

More recently, six papers have examined oesophageal motility during physical activity. (Soffer et al., 1991; Soffer et al., 1993) evaluated the effects of graded exercise in a sample of 6 trained cyclists. Their protocol required subjects to undertake one hour of exercise at 60% of maximal aerobic power ($\dot{V}O_2max$), followed by 45 min at 75% of $\dot{V}O_2max$, and a final 10 min at 90% of maximum effort. The duration, frequency and amplitude of oesophageal contractions declined as the intensity of exercise was increased, with the change becoming statistically significant when subjects were cycling at 90% of their maximal oxygen intake. Episodes of gastro-oesophageal reflux, and the duration of acid exposure of the oesophagus were increased when subjects were exercising at 90% $\dot{V}O_2max$. Plasma concentrations of gastrin, motilin, glucagon, pancreatic polypeptide and vasoactive intestinal polypeptide were monitored during these studies, but the decreases in oesophageal motility seemed independent of changes in the levels of any of these hormones. Soffer et al. (1991, 1993) speculated that the cause of the observed changes might be a decrease of local blood flow to the

oesophagus, or simply an exercise-induced reduction in the volume of saliva that was swallowed. The same team of investigators made a parallel study of 9 untrained individuals who exercised on a cycle ergometer at 45%, 60%, 75% and 90% of their peak oxygen intake (Soffer et al., 1994). Again, the amplitude, duration and frequency of oesophageal contractions decreased during physical activity, with the changes becoming statistically significant when subjects were exercising at 90% of their peak heart rates.

vanNieuwenhoven and colleagues (1999) had a group of 10 subjects undertake cycle ergometer exercise at a loading that was decreased gradually from 70% to 60% of maximal work rate over a period of 90 min. The velocity of oesophageal peristalsis was increased by about a quarter while the subjects were cycling, However, the number of contractions was reduced from a resting average of 68 to only 24, and the duration of contractions and the peristaltic pressure were also somewhat decreased during the test activity (vanNieuwenhoven et al., 1999).

Choi et al. (2001) exercised their subjects on a treadmill at 40% and 70% of their maximal heart rates. At the higher of the two intensities of effort, they found a decrease in both the frequency and the pressure of oesophageal contractions. Further, there were then significant increases in the number of episodes of gastro-oesophageal reflux and the duration of exposure of the oesophagus to acids (Choi et al., 2001). Ravi and colleagues (2005) examined changes of oesophageal motility in response to moderate treadmill exercise (walking at a speed of 5.2 km/h) in 135 individuals, including normal subjects, a group with high amplitude peristaltic oesophageal waves (the so-called "nutcracker" group), a group with diffuse oesophageal spasm and a group with frank gastro-oesophageal reflux disease (GERD) (Ravi et al., 2005). All subjects ingested standardized boluses of water while they were exercising. The normal, nutcracker and GERD groups all showed a decrease of oesophageal peristaltic amplitude when walking, and the group with oesophageal spasm showed a similar (but statistically non-significant) trend to a decrease of peristalsis during exercise. Gastro-oesophageal reflux was also provoked in 13 of the 75 patients with GERD.

Budzyński (2010) examined 63 patients with recurrent angina-like pain; this symptom was unresponsive to a reduction of gastric acid secretion by proton-pump inhibitor drugs, and was thus somewhat atypical of GER. However, performance of a Bruce treadmill test again caused a decrease in the amplitude and effectiveness of oesophageal peristalsis in this group of individuals (Budzyński, 2010).

Thus, all six of these studies point to a decrease of oesophageal motility during acute physical activity, with the greatest effects being seen at the highest intensities of effort.

The clinical problems of gastro-oesophageal reflux and GERD

The occasional regurgitation of some of the gastric contents into the oesophagus (gastro-oesophageal reflux, GER) is an almost universal phenomenon, particularly immediately after a person has eaten. IF such reflux occurs during vigorous exercise, it has a negative effect upon athletic performance and the resulting symptoms can become a

disincentive to compliance with prescribed exercise. It is important to distinguish between GER and the more serious condition of GERD. The latter diagnosis is made if the reflux occurs frequently, and repeated exposure of the oesophagus to gastric acids causes sufficient symptoms to have an adverse effect upon an individual's quality of life. With GERD, there is a potential for mucosal injury and oesophageal complications that include ulceration (2 to 7% of patients), haemorrhage (<2% of patients), stricture (4 to 20% of patients), metaplasia of the stratified epithelium (Barrett's oesophagus, 10 to15% of patients), and the development of a very deadly adenocarcinoma (0.5% of patients)(Koppert et al., 2005; Modlin et al., 2004; Parmalee-Peters and Moeller, 2004; Rubenstein and Taylor, 2010; Shaheen and Richter, 2009). If such complications develop, a person's survival prospects can be affected (Ford et al., 2013).

Disagreement around diagnostic criteria and the reliance upon subjective reports have hampered clear estimates of the prevalence of GER and GERD. Under resting conditions, the reported incidence of GER has ranged from 9% (Italy) to 42% (USA)(Delaney, 2004), and the condition seems to be even more prevalent during some forms of vigorous exercise.

Underlying anatomy and physiology

GER plainly results from a malfunction of the lower oesophageal sphincter. The action of this sphincter is normally reinforced by the restraints imposed by the diaphragmatic crura and the phreno-oesophageal ligament (Mittal, 1990). Patients with GER and GERD show more frequent transient relaxations of the lower oesophageal sphincter than healthy individuals (Schneider et al., 2010), 2010). Oesophageal motility is poorly coordinated and ineffective, there is a loss of muscular tone at the lower oesophageal sphincter (Gómez Escudero et al., 2002), and chronic malfunction of the normal gastro-oesophageal barrier (vanHerwaarden. et al., 2000). Reflux of the gastric contents into the proximal part of the oesophagus is an important determinant of the severity of GER symptoms, and such reflux seems more likely when a person is active than when they are sedentary (Emerenziani et al., 2005).

Factors predisposing to GER

Genetic and demographic factors predispose to GER. Links have also been suggested to obesity and other lifestyle variables, as well as to specific pathologies.

Genetic and demographic influences

A large Swedish twin study found that heritability accounted for a substantial 31% of an individual's susceptibility to GERD (Cameron et al., 2002; Zheng et al., 2007). The common assertion of paediatricians that a child will "grow out" of GER thus appears unwarranted (Gold, 2004; Gold, 2006). Several authors have noted that the prevalence of GER and GERD is greater in women than in men (Dore et al., 2008; Hallan et al., 2015), increases with age (Fujiwara and Arakawa, 2009; Morozov et al., 2010) and is associated with a low educational level (Dore et al., 2008; Hallan et al., 2015). In one Swedish sample, the prevalence of GER in men peaked at an age of 50-70 years, but in women the likelihood of developing the condition increased into old age (Nilsson et al., 2004).

Lifestyle

Obesity and/or weight gain are important risk factors for onset of the GER that is seen in sedentary individuals under resting conditions (Anand and Katz, 2008; Çela et al., 2013; DeMarco and Passaglia, 2010; Dore et al., 2008; Eslick, 2012; Festi et al., 2009; Gómez Escudero et al., 2002; Hampel et al., 2005; Islami et al., 2014; Murao et al., 2011; Murray et al., 2003; Nandurkar et al., 2004; Nilsson and Lagergren, 2004; Nocon et al., 2006; Pandeya et al., 2012; Rey et al., 2006; Singh et al., 2013; Vart et al., 2011); a person's abdominal circumference is thus closely linked to the severity of GERD (Corley et al., 2007). In one report, severe obesity translated to a 3.3-fold increase in the risk of GER in men, and a 6.3-fold increase in women (Nilsson et al., 2003). A questionnaire-based study of 820 Swedes, 135 with symptoms that were interpreted as GER, failed to find the generally accepted association with obesity (Lagergren et al., 2000), but reasons for this anomaly other than the fallibility of questionnaire diagnoses remain unclear.

In most studies, obesity has persisted as a risk factor after adjusting for the influence of multiple covariates (Dore et al., 2008; ElSerag et al., 2005). Presumably because blood concentrations of the hormone ghrelin are inversely associated with obesity, there is also an inverse association between ghrelin levels, GER and GERD (Rubenstein et al., 2013). Obesity is thought to increase the prevalence of GER and GERD through mechanisms that include altered oesophageal and gastric motility, increased levels of sexual hormones, increased intra-gastric (De Vries et al.; Nilsson and Lagergren, 2004) and intra-abdominal (DeMarco and Passaglia, 2010) pressures, and a deficit of the parasympathetic activity that normally keeps the tonus of the oesophagus higher than that of the stomach (Devendran et al., 2014).

Some investigators have found associations of GERD with other adverse lifestyle factors, including smoking (Çela et al., 2013; Friedenberg et al., 2013; Hallan et al., 2015; Islami et al., 2014; Nilsson et al., 2004; Nocon et al., 2007; Nocon et al., 2006; Vossoughinia et al., 2014), a heavy consumption of alcohol (Friedenberg et al., 2013; Nocon et al., 2007; Nocon et al., 2006; Veugelers et al., 2006), and an excessive intake of salt (Nilsson et al., 2004).

Association with other pathologies

GER and GERD have occasionally been linked with other pathologies, including the irritable bowel syndrome (Lovell and Ford, 2015),high serum levels of total cholesterol and triglycerides (Fujikawa et al., 2012), non-alcoholic fatty liver disease (Miele et al., 2012), psycho-social stress (Stanghellini, 1999) and a poor quality of sleep and irregular dietary habits (Yamamichi et al., 2012). The associations with blood lipids and fatty liver probably reflect the effects of obesity upon reflux, while the relationships with poor sleep and stress may indicate a low tolerance for pain and discomfort.

A large increase of the thoraco-abdominal pressure gradient is another factor increasing the likelihood of reflux. An increased gradient is particularly likely to develop when a person with asthma undertakes vigorous physical activity (Ayazi et al., 2011; Kiljander et al., 1999). The reflux of gastric acid into the oesophagus is likely to trigger coughing and/or an asthma attack, thus initiating a vicious cycle of increasing respiratory

effort and greater GER (Kahrilas et al., 2014). It has even been suggested that GER is one cause of exercise-induced asthma (Peterson et al., 2009). Some investigators have found no relationship between the severity of exercise-induced bronchial spasm and either GER or the administration of proton inhibitors (Ferrari et al., 2008; Weiner et al., 1998; Wright and Simons, 1996), but others have observed a reduction in the frequency of night-time asthma (Kiljander et al., 1999) and exercise-induced asthma (Peterson et al., 2009) following omeprazole treatment.

Finally, the action of gastric acid on the submucosal layers of the oesophagus can activate T-cells, liberating cytokines that in turn can cause abnormalities of immune function (Falk et al., 2011).

Diagnosis of GER and GERD

Epigastric complaints are common in the endurance athlete, but they do not necessarily reflect either GER or GERD. One study of 25,640 triathlon participants noted reports of nausea, epigastric pain or vomiting in 8.9% of those who were questioned (Lopez et al., 1994). Undoubtedly, such symptoms often reflect some degree of GER, but other possible causes include an excessive ingestion of "replacement" fluids, a slowing of gastric emptying, and gastric or myocardial ischemia. Several reports examining the prevalence of GER during competitive events have unfortunately based their diagnosis upon the response to questionnaires such as the gastro-intestinal symptom-rating scale (Norisue et al., 2009), probably with an over-estimate of the true prevalence of GER.

It is particularly important to be clear when the occasional gastro-oesophageal reflux that affects most athletes transitions into GERD. Pathological reflux may occur when a patient is erect, supine or both (Demeester et al., 1976). Often, it is unrelated to either meals or physical activity. It may occur at night, with two or more episodes of severe heartburn per week, and symptoms can adversely affect a person's well-being. Criteria differentiating GERD from GER include an oesophageal pH reading of <4.0 persisting >10 sec, and/or oesophageal spasm in more than 55% of spontaneous oesophageal contractions (Budzyński, 2010). Observation of oesophageal pH during a 1-hour period of dynamic posture changes may be helpful in diagnosing ambiguous cases (Schowengerdt, 2005).

About a third of GERD cases show an associated oesophagitis (Bretagne et al., 2005). However, endoscopy is not particularly helpful in detecting inflammation of the oesophageal lining; nor is diagnosis of GERD made easy by using oesophageal pressure manometers or barium ingestion to evaluate changes in local motility (Kahrilas et al., 1994; Lemire, 1997; Pandolfino and Kahrilas, 1995). Better diagnostic options include a decrease of symptoms in response to the administration of high doses of a proton-pump inhibiting drug such as omeprazole for one week (Botoman, 2002), and the detection of gastric acid reflux by the continuous 24-hour monitoring of esophageal pH (Kahrilas and Quigley, 1996; Kouklakis et al., 2005).

Different diagnosis of GERD

GERD must be differentiated not only from the less serious condition of GER, but also from other painful exercise-related conditions such as angina pectoris and visceral ischaemia, as well as a dysfunction of visceral pain perception.

One study concluded that as few as a a tenth of patients with a combination of chest pain and angiographically normal coronary arteries showed evidence of GERD (Cooke et al., 1994). Differentiation of GERD from angina is not always an easy task, particularly when the epigastric pain is associated with periods of vigorous exercise (Botoman, 2002; Budzyński, 2010; Lenfant, 2010; Liuzzo

and Ambrose, 2005; Ros et al., 1996; Shawdon, 1995; Sik et al., 2009; Singh and McGregor, 2005). The distal part of the oesophagus and the heart share a common afferent nerve pathway, and thus pain from either location is sensed over the same areas of the body surface. Moreover, regurgitation of acid into the oesophagus can precipitate a reflex spasm of the coronary arteries (Budzyński et al., 2000; Chauhan et al., 1996). This phenomenon can be simulated by deliberately instillating 0.1 M hydrochloric acid into the oesophagus, but the response is absent in patients with denervated hearts (for instance, after cardiac transplantation). A study of 52 patients with angina pectoris found 11 individuals had a high 24-h gastro-oesophageal reflux score; 10 of this group and 13 other patients all showed GER during performance of a Bruce treadmill test (Schofield et al., 1987).

Vigorous exercise induces a massive a redistribution of blood flow from the viscera to the muscles and the skin. The resulting gastric ischemia is a potential cause of severe epigastric pain (Michel et al., 1994; ter Steege et al., 2008), and this can be mistaken for GER. The differential diagnosis is complicated, since a reduction of local blood flow to the oesophagus may also be causing oesophageal dysfunction.

Nevertheless, many acute episodes of chest pain bear no apparent relationship to either a drop in oesophageal pH or to electrocardiographic abnormalities that indicate cardiac ischaemia. In many of these cases a dysfunction of visceral perception leading to a low pain threshold may be to blame (Paterson et al., 1993).

Effects of moderate and vigorous exercise upon GER

Moderate physical activity and GER

There have been 9 reports examining the acute effects of moderate physical activity upon the likelihood of gastro-oesophageal reflux (Avidan et al., 2001; Choi et al., 2001; Karim et al., 2011; Mendes-Filho et al., 2014; Ravi et al., 2005; Schoeman et al., 1995; Sodhi et al., 2008; Soffer et al., 1994; Worobetz and Gerrard, 1986)(Table 1). With the exception of questionnaire observations on a post-dinner walk by 1825 Pakistanis (Karim et al., 2011), all investigators have followed small groups of individuals, assessing the extent of gastro-oesophageal reflux by pH monitoring and/or manometry. Most authors have found little evidence of GER except with efforts at toe-touching (Sodhi et al., 2008), and often moderate exercise has led to small improvements of gastro-oesophageal function.

In the study of Avidan and colleagues (2001), steady walking decreased the likelihood of GER by 17% during the first hour after a meal, although the same subjects obtained an even larger benefit simply from chewing gum. A multivariate analysis of the Pakistani data (Karim et al., 2011) showed that a post-dinner walk of unspecified length reduced the risk of GER by 34% relative to lying supine after a meal; benefit was also seen with a post-

Table 1: Relationships between moderate physical activity and gastro-oesophageal reflux.

Author	Population	Exercise	Diagnosis	Finding
Avidan et al., 2001	12 adults with GERD, 24 healthy	60 min of steady walking	pH monitoring	17% reduction in acid contact time in GERD group during 1st hr after meal
Choi et al., 2001	12 healthy males	Treadmill exercise at 40% and 70% of maximal heart rate after a meal	pH monitoring & manometry	Number & duration of reflux episodes, time when pH < 4 all increased with exercise at 70% max heart rate
Karim et al., 2011	1875 Pakistanis (689 with GERD)	Post-dinner walk	Questionnaire	34% reduction of risk with post-dinner walk
Mendes-Filho et al., 2014	29 patients with erosive GERD, 10 with non-erosive GERD	Cycle ergometry	pH monitoring & manometry	Light or brief exercise did not cause reflux; reflux in erosive group with exercise at >70% $\dot{V}O_{2max}$
Ravi et al., 2005	135 untrained Irish adults	Treadmill walk, 1 km/h, then 5 min at 4.2 km/h	pH monitoring & manometry	Oesophageal pressures reduced during exercise; reflux in 13/75 patients with GERD
Schoeman et al., 1995	10 healthy Australians	10 min treadmill walking and 10 min rapid walking	pH monitoring and manometry	Relaxation of lower oesophageal sphincter not increased by walking or moderately vigorous exercise
Sodhi et al., 2008	25 patients with GERD	Toe touching from supine, sitting & standing positions	pH monitoring	Bending exercise provokes GER
Soffer et al., 1994	9 untrained Americans	Cycle ergometry at 45%, 60%, & 75% of $\dot{V}O_{2peak}$	pH monitoring	GER not increased when exercising to 75% of their $\dot{V}O_{2peak}$
Worobetz and Gerrard, 1986	6 healthy male athletes	120 min treadmill exercise at 50% of peak aerobic power	Manometry	Immediately post-exercise, increase in lower oesophageal sphincter pressure, no change of oesophageal motility

dinner interval of at least 3 hr before lying down to sleep (Avidan et al., 2001).

Choi et al. (2001) found that treadmill walking at 40% of maximal heart rate had no impact upon GER following a meal. (Mendes-Filho et al., 2014) used a pH meter and manometry to examine 29 patients with erosive and 10 patients with non-erosive GERD; light or brief periods of exercise did not cause gastro-oesophageal reflux in this sample (Choi et al., 2001). Soffer and colleagues (1994) had nine untrained Americans perform cycle ergometry at 45%, 60% and 75% of $\dot{V}O_{2peak;}$ they found that GER was not increased by these intensities of exercise (Soffer et al., 1994). (Schoeman et al., 1995) noted that only 2 of the 123 episodes of GER in their series were associated with 10 min bouts of either "*steady*" or "*rapid-as-possible*" treadmill walking. Another study found that walking at a pace of 4.2 $km \cdot h^{-1}$ while engaging in wet swallows induced GER in only 13 of 75 participants with known GERD (Ravi et al., 2005). Finally, (Worobetz and Gerrard, 1986) had six healthy male athletes perform 120 min of treadmill exercise at 50% $\dot{V}O_2peak$; immediately post-exercise, oesophageal motility remained unchanged, and they observed an increase in pressure at the lower oesophageal sphincter.

Vigorous exercise and GER

In contrast to the limited effects of moderate physical activity, vigorous exercise generally provokes a decrease in the amplitude and effectiveness of oesophageal contractions with a decrease of oesophageal pressures (Budzyński, 2010). Moreover, vigorous exercise is often associated with an increase of GER (Choi et al., 2001; Clark et al., 1989; Kraus et al., 1990; Mendes-Filho et al., 2014; Motil et al., 1987; Soffer et al., 1994). Both the prevalence and the duration of GER increase with the intensity of effort (Choi et al., 2001; Collings et al., 2003; Motil et al., 1987; Norisue et al., 2009; Soffer et al., 1991; Soffer et al., 1994); the threshold for the development of GER is commonly around 70% of an individual's $\dot{V}O_2max$ (Clark et al., 1989; Collings et al., 2003; Peters et al., 1999b; Rehrer et al., 1992b; van Nieuwenhoven et al., 2004).

In one somewhat unlikely competitive scenario, exercise was performed at 70% of maximal heart rate only 30 min after a meal. In this situation, physical activity led to disorganized esophageal contractions, increases in the number and duration of gastro-oesophageal reflux episodes, and a prolongation of the total time when the oesophageal pH was < 4.0 (Choi et al., 2001). Most reports have described decreases in oesophageal sphincter pressures as the intensity of cycle ergometer exercise was increased beyond 70% $\dot{V}O_2peak$ (Józków et al., 2006; Kraus et al., 1990; Maddison et al., 2005; Peters et al., 2001; Peters et al., 1988; Soffer et al., 1991; Soffer et al., 1994), but investigators from one laboratory reported that 2 hours of treadmill running at 50% of maximal oxygen intake induced a modest increase of pressure at the lower esophageal sphincter (Worobetz and Gerrard, 1985, 1986). vanNieuwenhoven et al (1999) also saw an increase of oesophageal peristaltic velocity when their subjects were operating a cycle ergometer at 70-90% of $\dot{V}O_2max$ (vanNieuwenhoven et al., 1999)

In addition to the mechanical factors predisposing to GER during vigorous exercise, other potential causes of abdominal symptoms include a reduced gastro-intestinal blood flow, a slowing of gastric emptying, air-swallowing (Kraus et al., 1990), an increase in intra-abdominal pressures (Sik et al., 2009), and increases in the concentration of hormones such as catecholamines (Worobetz and Gerrard, 1986). However, Soffer et al. (1991) found no relationship between GER and the levels of hormones such as gastrin, motilin, glucagon, pancreatic polypeptide and vaso-active intestinal peptide during exercise (Soffer et al., 1991) .

At least 15 reports have made laboratory evaluations of the effects of vigorous physical activity upon the incidence of GER (Choi et al., 2001; Clark et al., 1989; Collings et al., 2003; Kraus et al., 1990; Maddison et al., 2005; Mendes-Filho et al., 2014; Motil et al., 1987; Pandolfino et al., 2004; Peters et al., 1999b; Schoeman et al., 1995; Schofield et al., 1987; Soffer et al., 1991; van Nieuwenhoven et al., 2004; vanNieuwenhoven et al., 1999; Yazaki et al., 1996) (Table 2). All of these investigations diagnosed GER using an objective monitoring of oesophageal pH.

Table 2. Relationships between vigorous exercise and gastro-oesophageal reflux as seen in experimental studies.

Author	Population	Exercise	Diagnosis	Finding
Choi et al., 2001	12 healthy males	Treadmill exercise at 70% of maximal heart rate after a meal	pH monitoring & manometry	Number & duration of reflux episodes, time when pH < 4 all increased with exercise at 70% max heart rate
Clark et al., 1989	12 asymptomatic Americans (7M, 5F)	15 min cycling, 15 min rowing 15 min weight routine	pH monitoring	Association greatest for running, less for cycling, some effect from weights
Collings et al., 2003	American athletes (10 runners, 10 cyclists, 10 weightlifters)	Standardized exercise (60 min at 65% max, 20 min at 85% of max)	pH monitoring & symptom evaluation	Association greatest in weightlifters; mild reflux in cyclists, moderate in runners
Kraus et al., 1990	14 American runners	60 min running	pH monitoring	Reflux increased relative to baseline during running, reduced by 300mg ranitidine
Maddison et al., 2005	5M, 2F asymptomatic recreational cyclists	Four 5-min bouts of exercise at 90% of $\dot{V}O_2max$	Measurements of oesophageal, gastric and sphincter pressures	High intensity exercise reduces sphincter pressures, response unaffected by ingestion of sport drinks
Mendes-Filho et al., 2014	29 patients with erosive GERD, 10 with non-erosive GERD	Cycle ergometry	pH monitor & manometry	No reflux in erosive group with exercise at >70% $\dot{V}O_2max$
Motil et al., 1987	One athletic girl, aged 11 yr	Treadmill stress test	pH monitoring	No GER over 24 h, but induced by treadmill test
Pandolfino et al., 2004	20 Americans (10 with GERD)	30 min running & 30 min resistance exercise (intensity not specified)	Endoscopy & manometry	Exercise caused 3-fold increase of acid exposure in controls and in those with GERD
Peters et al., 1999b	7 Dutch triathletes	50 min of running and cycling at 75% $\dot{V}O_2max$ with intake of sports drinks	pH monitoring	Percent reflux time greater if running than cycling; GERD increased by drinking, sports drink>water, before exercise
Schoeman et al., 1995	10 healthy Australians	10 min vigorous cycle ergometry, 10 min treadmill walking and 10 min rapid walking	pH monitor and manometry	Relaxation of lower oesophageal sphincter not increased by walking or moderately vigorous exercise
Schofield et al., 1987	52 patients with angina pectoris	Bruce treadmill test	pH monitoring & manometry	10/11 patients with high 24-h reflux score, 13 other patients all showed reflux during exercise
Soffer et al., 1991	8 trained American cyclists	Cycle ergometry, 1 h at 60% + 45min at 75% & 10 min at 90% $\dot{V}O_2peak$	pH monitor	Oesophageal acid exposure increased when exercising at 90% $\dot{V}O_2max$
vanNieuwenhoven et al., 1999	10 healthy males	Cycling at 70% $\dot{V}O_2max$	pH monitor, pressure sensors	Peristaltic velocity increased while cycling, but no change in GERD
van Nieuwenhoven et al., 2004	10 symptomatic, 10 asymptomatic athletes	90 min cycling & running at 70% of aerobic power	pH monitor	GERD running>cycling; Symptomatic subjects had more frequent & longer lasting reflux
Yazaki et al., 1996	17 healthy adults	Rowing, fasted running, post-prandial running	pH monitor	Gastro-oesophageal reflux in 70% of rowers, 45% of fasted runners, 90% of fed runners

Plainly, a substantial proportion of athletes who are symptom-free at rest develop GER during vigorous exercise, and sometimes the symptoms are sufficiently severe as to cause a deterioration of their performance or even an abandonment of competition. Thus, (Peters et al., 1999) found that some participants dropped out of a 4-day walking event due to various gastro-intestinal symptoms, and the experimental infusion of 0.1 N hydrochloric acid into the oesophagus led to a deterioration in the treadmill test performance of well-trained runners relative to controls who received a sham infusion of acid (Rodriguez-Stanley et al., 2006). Nevertheless, one review concluded that the most common cause of abdominal discomfort in athletes was a local ischaemia of the gut wall rather than a reflux of gastric acid (deOliveira and Burini, 2009).

Studies of sport participation

Fifteen field studies have looked at the symptoms developed during vigorous exercise and participation in athletic events (Glace, Murphy, and McHugh, 2002; (Glace et al., 2002; Józków et al., 2007; Keeffe et al., 1984; Lopez et al., 1994; Norisue et al., 2009; Peters et al., 1999a; Peters et al., 1999; Rehrer et al., 1992a; Rehrer et al., 1989; Rehrer et al., 1992b; Riddoch and Trinick, 1988; Sullivan, 1987; Verbeek et al., 2014; Worobetz and Gerrard, 1985; Yazaki et al., 1996). Unfortunately, all except one of these investigations (Yazaki et al., 1996) relied upon symptom questionnaires that did not always differentiate clearly between GER and other upper and lower abdominal complaints (Table 3).

Upper abdominal symptoms have been found more commonly in women than in men, and more commonly in young than in older runners (Riddoch and Trinick, 1988). Some 8.9% of a large sample of 25,640 French triathletes reported symptoms such as nausea, epigastric pain or vomiting during competition (Lopez et al., 1994). Other observers have noted various types of gastro-intestinal complaints in at least a half of athletes during a competitive event (Brouns and Beckers, 1993; Moses, 1990; Waterman and Kapur, 2012), for example 83% of marathon runners (Riddoch and Trinick, 1988), 50% of ultramarathon participants (Glace et al., 2002), 83% of participants in a 3-peaks mountain biking event (58% of these contestants reporting upper abdominal symptoms)(Worobetz and Gerrard, 1985), and 24% of subjects who walked for 4 days (a distance of 203 km for the men, and 164 km for the women)(Peters et al., 1999). Nevertheless, in most of these studies, lower abdominal symptoms such as diarrhoea were more common than upper abdominal complaints likely to have been caused by GER.

At any given intensity of effort, GER as monitored objectively by oesophageal pH is less frequent during cycle ergometry (where body movement is relatively limited) than when the body is oscillating (as in treadmill running)(Clark et al., 1989), or if a Valsalva manoeuvre is undertaken [as in weight-lifters, (Collings et al., 2003)]. Nevertheless, GER can occur even in cyclists (van Nieuwenhoven et al., 2004), particularly if a stooped racing posture is adopted (Verbeek et al., 2014), GER is also is seen in 70% of rowers (Yazaki et al., 1996); a raised intra-abdominal pressure is suggested as a

Table 3: Relationship between vigorous exercise and abdominal symptoms and/or gastro-oesophageal reflux as seen in field studies of athletes.

Author	Population	Exercise	Diagnosis	Finding
Glace et al. (2002)	19 volunteers	160 km ultra-marathon	Questionnaire	Gastro-intestinal symptoms in 50%
Józków et al. (2007)	100 patients with GERD	3 levels of habitual physical activity	IPAQ questionnaire	Neither self-reported symptoms nor GER differed between 3 activity groups.
Keeffe et al. (1984)	707 marathoners	A "hard run"	Questionnaire	Heartburn 9.5%, nausea 11.6%, vomiting 1.8%
Lopez et al. (1994)	25,640 triathlon participants	French triathon competition	Questionnaire	8.9% had upper gastric symptoms (nausea, epigastric pain or vomiting)
Peters et al. (1999a)	185 long-distance runners, 173 cyclists & 149 triathletes	Recent sport participation	Questionnaire	Runners- mainly lower GI symptoms cyclists upper & lower GI symptoms, frequently caused drop-out
Peters et al. (1999b)	Long-distance walkers, 79M, 76F	Walk 203 km (M), 164 km (F) over 4 days	Questionnaire	24% reported symptoms, commonly nausea, headache & flatulence
Rehrer et al. (1989)	44 untrained individuals	Training for marathon	Questionnaire	52% developed GI distress during marathon, related to dehydration rather than fluid ingestion
Rehrer et al. (1992a)	172 ultra-endurance runners	67 km run with 1900 m change of altitude to highest point	Questionnaire	43% complained of gastro-intestinal distress
Rehrer et al. (1992b)	55 male triathletes	Half Iron-man triathlon	Questionnaire	52% reported eructation, 48% flatulence, symptoms greatest when running
Riddoch and Trinick, (1988)	471 marathoners	Marathon run	Questionnaire	Heartburn in 13%, nausea in 20%, vomiting in 4%
Sullivan (1987)	110 triathletes	Triathlon	Questionnaire	Heartburn, nausea & vomiting in 24%
Verbeek et al. (2014)	196 rowers, 439 other athletes	Rowing events	Questionnaire	GER symptoms in 51% of rowers
Worobetz and Gerrard (1985)	70 endurance runners	Dunedin "Enduro" event	Questionnaire	58% developed upper gastrointestinal symptoms
Yamake et al. (1996)	17 healthy adults	Rowing, fasted & post-prandial running	pH monitoring	GER in 70% of rowers, 45% of fasted runners, 90% of fed runners

causal factor in this group of athletes (Bi and Triadafilopoulos, 2003; Peters et al., 1999a; Waterman and Kapur, 2012; Worobetz and Gerrard, 1985). Exercise in the prone position predisposes to GER, and complaints are very common in surf-boarders, particularly if they use short boards (Norisue et al., 2009). The odds ratio of GERD (defined as episodes of GER > 2 times per week) was 4.6 times higher in surfers than in non-surfer athletes, and the likelihood of GERD increased with the frequency and duration of surfing. Ingestion of a meal (Clark et al., 1989;

Table 4: Habitual physical activity, occupational activity and aerobic fitness in relation to gastro-oesophageal reflux.

Author	Population	Exercise	Diagnosis	Finding
Habitual physical activity				
Çela et al. (2013)	345 men, 500 women	3-level activity classification	Symptom-reporting	6.3 fold increase in odds of GERD in low vs. high activity individuals (adjusted for BMI, smoking & alcohol)
Djärv et al. (2012)	4910 Swedish adults, 472 with GERD	3-level classification of frequency of physical activity	Questionnaire	Physical activity has no effect on GERD in normal weight individuals, but moderate frequency of exercise reduces risk in multivariate analysis for obese
Józków et al. (2007)	100 patients with GERD	3 levels of physical activity defined by IPAQ	pH monitoring and questionnaire	Number of self-reported symptoms did not differ between 3 activity groups
Murao et al. (2011)	2853 Japanese	Exercising less than once/month	Questionnaire	26% decrease in risk of GERD in exercisers with multivariate analysis
Nandukar et al. (2004)	211 Americans	Energy expenditure 7.3 MJ/d by questionnaire; 3-level PA classification	GERD questionnaire	Association of GER with exercise disappears if BMI and diet included as co-variates
Nilsson et al. (2004)	3153 cases, 40210 controls	Habitual exercise bouts > 30 min 1-3 times/wk	Severe or recurrent heartburn or regurgitation	Odds ratio of GERD 0.5-0.7 in exercisers vs. control group
Nocon et al. (2006; 2007)	7124 Germans	3 level questioning of sports participation	Questionnaire	Sports >2h/wk gives 25% protection against GERD
Norisue et al., 2009	185 Haiwaian surfers, 178 athletic non-surfers		Questionnaire	Odds ratio of GERD >2/week 4.6 in surfers; prevalence also related to frequency & duration of surfing
Pandeya et al. (2012)	1580 Australian adults	3-level physical activity index	Questionnaire	Occasional reflux 11% higher with low physical activity; frequent reflux 32% lower with low than with high physical activity
Stake-Nilsson et al., 2013	18-year follow-up of 85F, 52 M	Exercise questionnaire	Questionnaire	Odds ratio 3.05 for decrease of GERD with increase of exercise
Waško-Cnopnik et al. (2013)	100 patients with GERD symptoms	3 levels of habitual physical activity by IPAQ	pH monitor & manometry	No relationship of activity to reported habitual activity
Yamamichi et al. (2012)	19,864 healthy Japanese	Exercising <30 min/day (questionnaire)	Questionnaire	GERD significantly related to low physical activity in multivariate analysis
Zheng et al. (2007)	4083 twins with GERD, 21,383 controls	Physical activity at work, recreational physical activity	Questionnaire	"Much recreational physical activity" reduced risk of GER by 40%
Occupation				
Chen et al. (2005)	1468 M, 1870 F in South China		Questionnaire	Odds ratio of 3.43 for those with strenuous work
Zheng et al. (2007)	4083 twins with GERD, 21,383 controls	Physical activity at work, recreational physical activity	Questionnaire	Strenuous physical activity at work increased risk of GERD by 20%,
Fitness Level				
Hawryłkiewicz et al. (2008)	6 F, 12 M with obstructive sleep apnoea	6-min walk test	12 of 18 patients had GERD, 14 had oesophagitis	Distance walked reduced 22% relative to normal controls

Rehrer et al., 1992; Yazaki et al., 1996) or a sports drink rather than water shortly before exercising (Peters et al., 1999) predisposes to GER. Moreover, the problem is exacerbated by gastric distension and air swallowing (Dent et al., 1988). However, dehydration can also lead to gastro-intestinal complaints (although not necessarily GER) during distance running (Rehrer et al., 1989).

Effects of habitual physical activity upon GER

The effects of habitual physical activity upon oesophageal function and GER can be examined by relating the prevalence of the disorder to questionnaire assessments of leisure activity, making comparisons between occupational categories with differing levels of energy expenditure, or correlating the prevalence of GER with assessments of aerobic fitness (using the latter as a surrogate of habitual physical activity).

Any apparent benefit from regular physical activity (Festi et al., 2009; Nilsson et al., 2004) could reflect in part the impact of increased physical activity upon other components of personal lifestyle such as obesity, smoking and stress (Festi et al., 2009; Vart et al., 2011). Physical activity is significantly associated with a lean body build, and with abstinence from cigarettes. Moderate physical activity also seems likely to encourage a reduction of stress, but on the other hand intensive athletic competition could augment stress levels. Many (but not all) studies of habitual physical activity have largely eliminated the influence of other aspects of personal lifestyle through extensive multivariate analyses. Their main weakness is reliance upon questionnaires to diagnose GER.

Habitual recreational physical activity and gastro-oesophageal reflux

At least 13 reports have examined associations between habitual recreational physical activity and GER (Çela et al., 2013; Djärv et al., 2012; Józków et al., 2007; Murao et al., 2011; Murray et al., 2003; Nandurkar et al., 2004; Nilsson et al., 2004; Nocon et al., 2007; Pandeya et al., 2012; Stake-Nilsson et al., 2013; Waško-Cnopnik et al., 2013; Yamamichi et al., 2012; Zheng et al., 2007). In general, a several-level classification of habitual leisure activity has been based upon questionnaire responses, and in all except 2 studies from the same laboratory (Józków et al., 2007; Waško-Cnopnik et al., 2013) ,the diagnosis of GER and/or GERD was also determined by questionnaire (Table 4).
Eight of the 13 studies (Çela et al., 2013; Murao et al., 2011; Nilsson et al., 2004; Nocon et al., 2007; Pandeya et al., 2012; Stake-Nilsson et al., 2013; Yamamichi et al., 2012; Zheng et al., 2007) found benefit from participation in regular moderate physical activity, although the extent of benefit varied widely between investigations. One multivariate analysis of 345 men and 500 women that included BMI, smoking and the use of alcohol as covariates (Çela et al., 2013) found a 6.3 fold decrease in the odds of the Montreal definition of GERD when it compared individuals reporting high and low levels of physical activity. Likewise, a multivariate analysis of data for 3153 cases and 40,210 controls from Norway (Nilsson et al., 2004) demonstrated that the risk of GER (here defined by reports of severe or recurrent heartburn or regurgitation) was significantly diminished by participating in vigorous exercise (jogging, cross-country skiing or swimming) for 30 min or more 1, 2, or 3

times per week (with respective odds ratios of 0.5, 0.6, and 0.7. after statistical adjustment for the effects of age, sex, body mass index, use of tobacco and coffee, table salt intake and dietary fibre in bread). Study of a Japanese population (Murao et al., 2011) suggested that even exercising as infrequently as once per month was associated with a 26% decrease in the risk of GERD as documented by responses to the Carlsson-Dent questionnaire for this condition. One report also underlined that a moderate frequency of physical activity reduced the risk of GERD in those who were obese, but not in those of normal body mass (Djärv et al., 2012).

One investigation examined the effects of the intensity of habitual physical activity. Relative to those engaging in high intensity effort, frequent GER was 32% less common in those taking light physical activity, and 54% less in those engaging in moderate physical activity (Pandeya et al., 2012).

An 18-yr follow-up study suggested that an increase of habitual physical activity over the period of observation was associated with a substantially decreased risk of GER (odds ratio, 3.05), although apparently the data in this study were not adjusted for covariates such as obesity(Stake-Nilsson et al., 2013).

Two negative reports (Józków et al., 2007; Waško-Cnopnik et al., 2013) were from the same laboratory; they found no relationship between habitual physical activity and either lower oesophageal pressures or GER (as assessed objectively by pH monitoring). A third investigation with negative findings (Nandurkar et al., 2004) was based upon a relatively small sample of 211 individuals. It found that an apparent association of GER with habitual physical activity disappeared if diet and body mass index were introduced into the analysis as co-variates. Another negative report was based on a large sample (2035 men and 2350 women, including 472 people with GERD)(Peters et al., 1999a); a 3-level classification of habitual physical activity showed no relationship to GERD in those with a normal body mass, but a moderate frequency of physical activity did reduce the risk of GERD in those who were obese. The final report was based upon the prone exercise of surf-boarding; here, frequent surfing led to a large increase in the incidence of GER (Norisue et al., 2009).

Occupational physical activity and gastro-oesophageal reflux

Two reports have studied the impact of heavy occupational activity upon the incidence of GERD (Chen et al., 2005; Zheng et al., 2007). The first of these surveys included 3338 Chinese villagers. It found that relative to those with a "mild" burden of physical work, the odds ratio of manifesting GERD as assessed by a validated Chinese version of the reflux disease questionnaire was 1.29 for those with a "moderate" burden, and 3.43 for those with a "severe" burden of occupational activity (Chen et al., 2005). The second investigation was based on twins living in Sweden, and it examined GER rather than GERD; in a multivariate analysis. it found benefit from "active" employment. However, the overall intensity of occupational work was probably lower in Sweden than in China, and activity patterns were classified in a different fashion; the odds ratios for manifesting GER relative to a corresponding twin involved in sedentary employment was 0.85 for jobs that required standing and walking, 0.68 for jobs that required standing, lifting and

carrying, and 0.74 for work that was classed as "physically strenuous" (Zheng et al., 2007).

Treatment of individuals with gastro-oesophageal reflux

To date, no treatment of gastro-oesophageal reflux has been entirely successful. The main focus has been upon prevention, sometimes with recourse also to drug treatment, and some authors have suggested benefit from programmes designed to strengthen the muscles of the diaphragm. It is particularly important to prevent progression of GER by clearing reurgitated stomach acid from the oesophagus; this can be done by encouraging salivation and resultant oesophageal peristalsis (Kahrilas and Lee, 2005).

General preventive measures

Recommendations to prevent GER may include sleeping with the head elevated (not always easy to arrange), and lifestyle changes such as altering the diet and the timing of meals, together with attempts to reduce body mass in those who are obese (Csendes and Burdiles, 2007; Kendrick and Houghtonb, 2006; Nilsson and Lagergren, 2004; Peters et al., 1999c; Reavis, 2011).

A reduced frequency of GER often follows a reduction in body mass (Anand and Katz, 2010; Festi et al., 2009; Friedenberg et al., 2008; Kaltenbach et al., 2006; Ness-Jensen et al., 2013; Singh et al., 2013). Other simple tactics to reduce reflux include chewing gum and standing following a meal (Bujanda et al., 2007).

One meta-analysis found no reduction of GER from a cessation of smoking, a reduction of alcohol consumption or other modifications of diet (Kaltenbach et al., 2006). A second report also noted little improvement of oesophageal function from the cessation of smoking (Ness-Jensen et al., 2014).

If a recreational exerciser is complaining of acute GER during bouts of physical activity, the problem can sometimes be corrected by changing the form of exercise (for instance, substituting cycling for running), reducing the intensity and/or the duration of effort (Collings et al., 2003; Parmalee-Peters and Moeller, 2004), or altering the timing and composition of meals relative to periods of exercise. Substantial meals should be taken at least 3 hours before exercising, while taking care to maintain fluid balance subsequently (Parmalee-Peters and Moeller, 2004). One review concluded that GER was more likely if exercisers ingested hypertonic carbohydrate or salt-containing beverages rather than water (Festi et al., 2009).

Pharmacological measures

If exercise-induced gastric pain is frequent, severe, and does not respond to modifications of diet and training, pharmacological measures may be proposed, although none are uniformly effective. Drugs that are currently prescribed include antacids, histamine H_2-receptor blockers such as cimetidine or ranitidine (these are intended to inhibit stomach acid production and thus protect the oesophagus from further damage)(Kraus et al., 1990), proton pump inhibitors such as omeprazole or rabeprazole (these also limit the secretion of acid by the stomach)(Holtmann et al., 2004; Jian et al., 2007; Kinoshita, 2004; Kraus et al., 1990; Peters et al., 1999a; Peters et al., 1999c; Shawdon, 1995; Simons and Kennedy, 2004; Tytgat, 2002) and possibly drugs designed to inhibit

lower oesophageal relaxation (Coron et al., 2007).

Proton pump inhibitors are at present the most popular choice of medication. In France, about 88% of cases of GERD are treated with proton pump inhibitors, and 73% of these patients are also given lifestyle advice (Bretagne et al., 2005). Unfortunately, proton pump inhibitors drugs are costly (Rubenstein and Chen, 2014), and although they often reduce symptoms, it has yet to be demonstrated that they avert the long-term complications of GERD (Delaney, 2004). Moreover, although the gastric acid secretion is reduced by such drugs, they do not necessarily correct any impairment of athletic performance (Rodriguez-Stanley et al., 2006).

Diaphragmatic training

One further option is a course of training to strengthen the muscles of the diaphragm. If the patient is willing to invest the effort, such exercises are said to help increase oesophageal sphincter pressures and thus reduce reflux (Chaves et al., 2012; daSilva et al., 2013; Ding et al., 2013; Eherer, 2014; Nobre e Souza et al., 2013).

Areas for further research

Much of the research to date has been based upon questionnaires that have looked at gastro-intestinal symptoms. Although there have been attempts to standardize such questionnaires, this is not a very reliable way to determine either the prevalence of gastro-oesophageal reflux or factors modifying its incidence. There is thus a need for more objective laboratory research, based upon measurements of pH and pressures within the oesophagus.There also remains great scope for further research to find an effective remedy for those who suffer from gastro-oesophageal reflux, and have not benefitted from either simple lifestyle changes or proton pump inhibitors. Given that the oesophagus penetrates the diaphragm, there seems logic in the suggestion that diaphragmatic training may be helpful, and this form of treatment also merits further investigation.

Practical implications and conclusions

Although moderate intensities of physical activity usually improve oesophageal function, vigorous exercise commonly reduces oesophageal motility and leads to an inappropriate relaxation of the sphincter at the lower end of the oesophagus, with reflux of the acid contents of the stomach. Occasional reflux of this type is a common enough phenomenon, and is commonly reported by both athletes and sedentary individuals. In itself, it may be no more than a painful annoyance, but if the reflux is frequent and is left untreated it can progress to gastro-oesophageal refluc disease, with more serious clinical manifestations that include ulceration, haemorrhage and stricture of the oesophagus, a cancerous change in the squamous-cells of the oesophageal lining (Barrett's oesophaus), and a very deadly oesophageal adenocarcinoma.

The tendency to gastro-oesophageal reflux is exacerbated by obesity (in sedentary individuals) and in athletes by bouts of exercise at intensities >70% of the individual's $\dot{V}O_{2max}$; the prevalence of GER is particularly marked during activities that involve rhythmic body movement (such as running), a prone position (for instance, surfing) and/or an increase of intra-abdominal pressure (as in weight-lifting).

The main focus of prevention is upon simple changes of lifestyle- a decrease of body mass if a person is obese, sleeping with the head raised, and altering the timing of meals relative to bouts of exercise. Regular moderate physical activity seems to reduce GER, and such activity should thus be a component of the treatment plan for anyone who currently has a sedentary lifestyle. If the symptoms of reflux are frequent during bouts of vigorous effort, a recreational exerciser may consider changing the type and/or intensity of physical activity that he or she practices. However, a change of sport is usually less feasible for the serious athlete. Encouraging salivation may reduce exposure of the oesophagus to acid during episodes of GER. The administration of proton pump inhibitors can often reduce symptoms, but it does not necessarily correct impaired physical performance. Exercises to strengthen the diaphragm and thus increase oesophageal sphincter pressures may also be helpful.

Author's Qualifications

The author's qualifications are as follows: Roy J. Shephard, C.M., M.B.B.S., M.D. [Lond.], Ph.D., D.P.E., LL.D., D.Sc., FACSM, FFISM.

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