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Spirometry Measurements During an Episode of Exercise-Related Transient Abdominal Pain

Darren P. Morton and Robin Callister

Purpose: To determine whether changes in lung function are associated with exercise-related transient abdominal pain (ETAP). ***Methods:*** Twenty-eight subjects susceptible to ETAP performed a flow-volume loop before (pre) and after (post) treadmill exercise. Fourteen of the subjects developed symptoms of ETAP during the exercise and completed the flow-volume loop while the pain was present. The remaining 14 subjects reported no symptoms of ETAP. ***Results:*** Forced inspiratory vital capacity was essentially unchanged from pre to post in both groups (ETAP group –0.8% ± 5.1%, comparison group –0.9% ± 6.5%). Peak inspiratory-flow rate increased in both the ETAP group (12.4% ± 16.2%) and the comparison group (17.9% ± 16.6%), but the difference between groups (–4.6%, standardized effect size [EF] = –0.17) was trivial. Forced expiratory vital capacity decreased by approximately 4% in both groups (ETAP group –3.9% ± 3.3%, comparison group –4.0% ± 5.1%). Small differences in the mean change from pre to post between groups were recorded for peak expiratory-flow rate (–7.4%, EF = –0.28) and the forced expiratory volume in the first second of the test (–4.4%, EF = –0.44). ***Conclusions:*** ETAP does not appear to be associated with reduced inspiratory performance, suggesting that the diaphragm is not implicated directly in the etiology of ETAP. Expiratory power might be slightly reduced during an episode of ETAP, but the magnitude of this effect is unlikely to compromise exercise performance.

Key Words: injury management, lung function

The etiology of exercise-related transient abdominal pain (ETAP), commonly known as stitch or side ache, has attracted much speculation. Numerous theories have been proposed to explain ETAP, and recent attempts have been made to evaluate the integrity of the various theories.[1-4] One commonly purported origin of the pain is the diaphragm, and indeed diaphragmatic explanations for ETAP were among the first to appear in the literature.[5] Specifically, diaphragmatic ischemia or cramping has been argued as the cause of ETAP.[5]

Morton is with the Avondale Centre for Exercise Sciences, Avondale College, Cooranbong 2265 NSW, Australia. Callister is with the School of Biomedical Sciences, University of Newcastle, Callaghan 2308 NSW, Australia.

In 1932, a study[6] conducted at Guy's Hospital examined diaphragm function while ETAP was being experienced in 4 males. Using fluoroscopic observations, the study reported that diaphragm function was not impaired during an episode of the pain. Unfortunately, no spirometry or other data from this study were presented. Nearly a decade later, Capps[5] observed that the location of the pain was typically subcostal, even naming it "subcostal pain," and on this basis, Capps asserted that the diaphragm was implicated. Furthermore, Capps[5] argued that as the pain occurred during exercise it was likely to be an ischemic response. Since the work of Capps[5] until recent times, the diaphragm has been central to the proposed etiology of ETAP, and sports-medicine textbooks and popular publications commonly attribute the pain to diaphragmatic ischemia or cramping.

Recently, we provided evidence indicating that the diaphragm might not be involved in ETAP. The pain is not confined to the subcostal region and can occur as low in the abdomen as the iliac region.[2,7,8] In addition, the pain can manifest in activities of relatively low respiratory demand, such as horse riding[2,9] and walking.[8] It is also unclear how certain factors that have been demonstrated to influence the experience of the pain, such as the sugar content of fluid consumed, could relate to a diaphragmatic origin of the pain.[10] Finally, we observed no increase in surface electromyographic activity at the site of ETAP during an attack, despite the instrumentation being capable of measuring diaphragmatic electrophysiological output.[11]

The purpose of this study was to further examine the involvement of the diaphragm in the etiology of ETAP. We hypothesized that lung function, especially inspiratory performance, would be compromised during an episode of ETAP if the diaphragm or other muscles of respiration were involved in the etiology of the condition. Respiratory-muscle weakness and deficiency are known to result in restricted lung function, indicated particularly in measures of vital capacity.[12] Reductions in maximal flow rates are more characteristic of obstructive disorders such as bronchospasm and decreased lung compliance.[12] Hence, the aim of the present study was to determine whether spirometry changes indicative of compromised respiratory function occur with ETAP.

Methods and Procedures

Study Design

Healthy subjects susceptible to ETAP performed a treadmill exercise session during which lung function was assessed using a flow-volume loop on 2 occasions: before commencing exercise and after treadmill running. Before the trial the subjects engaged in activities designed to increase their likelihood of developing ETAP. Subjects who experienced ETAP during the exercise constituted the ETAP group, and those who did not report symptoms of the pain formed the comparison group. The study design allowed comparisons to be made between the 2 groups in terms of the profile of change in the flow-volume-loop measures.

Sample

Thirty-five subjects who claimed to be susceptible to ETAP were initially recruited for the study and gave their written informed consent before participating. All

subjects were active and considered themselves to be in good physical condition. They reported no history of respiratory dysfunction, including asthma, and were free from symptoms of respiratory-tract infection and restriction at the time of the laboratory testing. The study was approved by the University of Newcastle Human Research Ethics Committee, and all research was conducted in accordance with this body.

Twenty-one of the 35 subjects (60%) experienced ETAP during the trial. In order for reliable testing to occur it was important that the subjects experience a "substantial" manifestation of the pain. When subjects reported symptoms of pain, they were asked if it was of a severity that would force them to slow down or stop if they were performing a recreational run. Of the 21 cases in which the pain manifested, 14 met this criterion. Thus, the symptomatic group comprised 14 subjects (ETAP group), with the 7 subjects who experienced "mild" symptoms of ETAP being discharged from the study. The 14 subjects that did not develop the pain still performed the testing procedures and served as the comparison group. Personal details and baseline measures of the subjects are presented in Table 1. The variability in the manifestation of the pain called for this unconventional approach to allocating the subjects to the ETAP or comparison group. As seen in Table 1, with the exception of the comparison group having a female skew, the groups were not dissimilar.

Strategies to Provoke ETAP

The subjects were instructed to perform any procedures before arriving at the laboratory that they believed would increase their likelihood of experiencing

Table 1 Descriptive Characteristics of Subjects and Baseline (Preexercise) Spirometry Measures*

	ETAP group	Comparison group	Combined
n	14	14	28
Age (y)	24.2 ± 5.8	22.2 ± 6.1	23.3 ± 5.9
Male/Female	11/3	9/5	20/8
Height (cm)	175.6 ± 9.9	172.7 ± 7.2	174.2 ± 8.6
Weight (kg)	66.8 ± 9.3	63.6 ± 9.7	65.2 ± 9.4
BMI (kg/m)	21.6 ± 1.8	21.2 ± 2.5	21.4 ± 2.2
FIVC (L)	4.69 ± 0.73	4.93 ± 0.77	4.81 ± 0.75
PIF (L/min)	7.50 ± 1.99	6.95 ± 2.21	7.23 ± 2.08
FEVC (L)	4.77 ± 0.80	5.02 ± 0.76	4.89 ± 0.78
PEF (L/min)	9.80 ± 3.16	9.80 ± 2.51	9.80 ± 2.80
$FEV_{1.0}$ (L/min)	4.09 ± 0.55	4.10 ± 0.54	4.09 ± 0.54

*ETAP indicates exercise-related transient abdominal pain; BMI, body-mass index; FIVC, forced inspiratory vital capacity; PIF, peak inspiratory-flow rate; FEVC, forced expiratory vital capacity; PEF, peak expiratory-flow rate; $FEV_{1.0}$, forced expiratory volume in the first second of maximal expiration. Data are presented as mean ± SD.

ETAP during the testing session. For all subjects this involved consuming food or drink shortly before the session. Before the first flow-volume loop, the subjects were familiarized with the protocol and given between 200 and 500 mL of carbonated soft drink. The volume depended on the amount of food and drink already consumed by the subject. Anecdotally, soft drinks were regarded by the subjects as particularly provocative of ETAP, which is consistent with our recent findings.[10]

Laboratory Testing Protocol

After the test briefing and fitting of a heart-rate monitor (Polar Electro Oy, Model 45920, Finland) the subjects were familiarized with the flow-volume-loop test by performing several trials. When comfortable with the procedure they completed the first test (pre). In order to derive a measure of test–retest reliability the subject then performed another flow-volume loop 2 minutes later in this preexercise state. All measures of lung function were highly reliable ($P < .01$), with a mean correlation coefficient of .92 ± .02.

Treadmill velocity was then selected for the subjects according to their fitness level, with a mean velocity of 12.3 ± 1.7 km/h (range 11 to 15 km/h), which elicited an average heart rate of 172 ± 13 beats/min. The subjects commenced exercise on the treadmill at the set velocity and every 30 seconds were asked if they were experiencing any symptoms of ETAP. If symptoms had developed they were asked to rate the severity of the pain on a 10-point scale,[13] from *no pain* (0) to *pain as bad as it could be* (10). Other measures of the pain, including its localization, position, and sensation, were recorded. The sensation of the pain was recorded by the subjects choosing a descriptor from the following 5: aching, pulling, cramping, sharp, and stabbing. These descriptors have been shown to account for over 90% of ETAP pain sensations.[2,7]

In the subjects who developed symptoms of ETAP, the test was continued until the required severity was obtained. If the required pain severity was not obtained, the test was terminated after 15 minutes. If the required pain severity was achieved, the treadmill was stopped and the subjects performed the flow-volume loop (post). The flow-volume-loop test was completed within 10 to 20 seconds of the treadmill being stopped. On average the subjects rated the severity of ETAP as 5.1 ± 1.1 on the numerical rating scale before stopping for the flow-volume loop and 3.8 ± 1.4 immediately after the spirometry test. The severity rating of the pain after the tests had been completed still represented a pronounced manifestation of ETAP. If the subjects did not develop symptoms of the pain within the first 8 to 10 minutes of the testing session, they performed the flow-volume loop and were included as part of the comparison group.

Spirometry Measures

A standard flow-volume loop was employed for this study, because it is a comprehensive lung-function test that assesses both expiratory and inspiratory performance. Forced expiration relies heavily on the abdominal muscles[14-16] and internal intercostal muscles,[17] whereas forced inspiration recruits the diaphragm[18-20] and external intercostal muscles.[17]

The test was performed on a GOULD 9000 computerized pulmonary exercise system (GOULD, Inc, Model 229). Measures derived from the test included forced inspiratory vital capacity, forced expiratory vital capacity (FEVC), peak inspiratory-flow rate (PIF), peak expiratory-flow rate (PEF), and forced expiratory-flow rate in the first second of expiration ($FEV_{1.0}$).

Statistical Analyses

For analysis, the 5 measures of lung function were expressed relative to height to normalize the data.[12] Analysis was then completed on the log-transformed data, with changes expressed as percentages. A paired *t* test was used to determine the level of significance of within-group changes from pre to post. To assess between-groups effects, we used an Excel® spreadsheet prepared by Hopkins.[21] The spreadsheet calculated the percentage change from pre to post for each subject and then performed a *t* test to compare the mean change between the ETAP and comparison groups. A qualitative expression of the between-groups differences was assigned using a modification of Cohen's effect-size classification system.[22] The terms selected were *trivial* (0.0 to 0.2), *small* (0.2 to 0.6), *moderate* (0.6 to 1.2), *large* (1.2 to 2.0), and *very large* (2.0 to 4.0).

Analysis of variance (SPSS, version 11.5) was used to determine whether the severity of the pain was different when associated with the 5 pain descriptors used in the study. Reliability of the flow-volume-loop measures was assessed using the Pearson correlation coefficient. Descriptive analyses involved calculating changes and mean ± SD. A 90% confidence interval was adopted.

Results

Characteristics of ETAP

In the group that experienced ETAP during the testing session, the pain first appeared after 2.4 ± 1.5 minutes and was deemed of sufficient severity for testing after 7.2 ± 1.8 minutes. In 1 case the subject claimed that mild pain was present after only 30 seconds of treadmill running. In most cases the pain progressively developed until it achieved the desired severity, although in some instances it was completely relieved while the subject continued to exercise. Although it was difficult to determine exactly, the amount of time it took after the cessation of exercise for the pain to completely resolve was 45 seconds to 2 minutes.

Every subject who developed ETAP described the pain as well localized. In 8 of the 14 symptomatic cases, the pain was reported in more than 1 abdominal site, but the characteristics of the pain were identical in the different positions. Exactly half the symptomatic subjects described the pain as most severe on the right side of the abdomen, with the other half reporting the left side.

Only 3 of the 14 subjects who developed ETAP did not change the descriptor used to convey the sensation of the pain as the severity increased during the session. ANOVA confirmed a significant ($P < .01$) difference in the severity rating of the descriptors. *Stabbing pain* was associated with significantly greater pain severity (5.3 ± 1.5) than all other terms except *sharp pain* (4.6 ± 1.6). Sharp pain was rated as more severe than aching (1.7 ± 0.9) and pulling (1.8 ± 1.2)

but not cramping pain (3.2 ± 1.7). There was no difference among the 3 least severe terms.

Spirometry

Within-group and between-groups changes in the spirometry measures are presented in Table 2. Trivial differences were observed between the groups in the inspiratory measures. An increase in PIF was observed in both groups. Although the increase in PIF was 4.6% less on average in the ETAP group, the magnitude of the increase was highly variable, and the effect of this difference between groups is unclear (Table 2).

FEVC decreased by approximately 4% in both groups, with only a trivial difference between them. Although there was considerable variability in the pre–post changes in PEF and $FEV_{1.0}$ in both groups, on average these values tended to decrease in the ETAP group and increase in the comparison group. Consequently, there were small differences between the groups for PEF and $FEV_{1.0}$.

Discussion

Characteristics of ETAP

Sinclair[9] claimed that, with ingestion of food or drink before exercise, ETAP could be evoked in 30% to 35% of cases. In the present study ETAP was provoked in 60% of cases, with 40% attaining the severity required for assessment. Allowing the subjects to select procedures to provoke the pain was clearly a successful strategy. The consumption of carbonated soft drink shortly before exercise appeared to be particularly provocative of ETAP, which is consistent with a recent report that fluids with high sugar content should be avoided by susceptible individuals.[10] Sinclair[9] observed that after ingestion, the pain progressed from the left side of the abdomen to the right side. In the present study there did not appear to be any pattern of progression with respect to the position of the pain.

The characteristics of ETAP observed in the subjects who developed the pain were consistent with our previous definition of the pain,[2] namely, that it is well localized and most prevalent in, although not confined to, the lateral aspects of the midabdomen. The localized nature of the pain reported by every subject in the present study is noteworthy because it suggests a somatic rather than visceral origin.[2]

Although more than half the symptomatic subjects experienced ETAP in more than 1 position, the pain description was consistent at the different sites. This suggests that a common tissue is responsible for the pain, and the tissue must be located throughout the entire abdomen. This observation alone provides evidence against the pain originating from the diaphragm.

Several studies have suggested that ETAP is twice as common on the right side of the abdomen as on the left.[2,7,9] The equal distribution of right- and left-side pain observed in the present study is likely a factor of the relatively small number of subjects. The progression from pulling or aching to stabbing pain with increasing severity is precisely the same as that observed in our previous work.[2,7]

Table 2 Within- and Between-Groups Changes in the Spirometry Measures From Pre to Post*

	% Change Pre to Post					
	ETAP group	Comparison group	% Difference in means between groups (90% CL)	*P*	ES (90% CL)	Descriptor
FIVC (L)	–0.8 ± 5.1	–0.9 ± 6.5	0.1 (–3.5 – 3.9)	0.62	0.01 (–0.28 to 0.30)	trivial
PIF (L/min)	12.4 ± 16.2	17.9 ± 16.6	–4.6 (–13.5 to 5.2)	0.42	–0.17 (–0.53 to 0.19)	trivial
FEVC (L)	–3.9 ± 3.3	–4.0 ± 5.1	0.1 (–2.6 to 2.9)	0.95	0.01 (–0.21 to 0.22)	trivial
PEF (L/min)	–4.3 ± 14.4	3.4 ± 10.7	–7.4 (–14.3 to 0.0)	0.10	–0.28 (–0.55 to 0.00)	small
$FEV_{1.0}$ (L/min)	–2.9 ± 7.5	1.6 ± 7.1	–4.4 (0.1 to –8.6)	0.10	–0.44 (–0.88 to 0.01)	small

*ETAP indicates exercise-related transient abdominal pain; CL, confidence limits; ES, effect size; FIVC, forced inspiratory vital capacity; PIF, peak inspiratory-flow

Breathing maneuvers including deep exhalation, forced exhalation, "belly breathing," and shallow respirations have been proposed to facilitate the relief of ETAP.[23-25] Although a decrease in pain severity was noted over the duration of the spirometry assessment, the pain subsides quickly when activity is ceased, regardless. Hence is not possible to determine from these data whether the breathing maneuvers of the flow-volume loop facilitated the pain relief.

Spirometry

The present study was unconventional in its approach to measuring exercise-related changes in spirometry in that the posttesting occurred immediately after the subject ceased exercise so that we could take the measurements while the pain was present. Many studies have examined the magnitude of exercise-induced changes in lung function in both normal and asthmatic populations, but the convention, recommended by the American Thoracic Society, is to perform initial posttesting 10 minutes after exercise has been terminated.[26-28] Studies have required subjects to perform posttests as soon as 4 minutes after exercise,[26] but immediately postexercise data are not available for trend comparison with the present study.

When the 10-minute postexercise protocol is adopted, decreases in spirometry measures of more than 10% are typically considered functionally significant and indicative of obstructive disorders such as bronchospasm,[26,28] and it has been suggested that this criterion should be reduced to as low as 6% to 7% for the elite athletic population.[26,28] In neither group did the mean within-group changes in the present study meet either of these criteria, although 2 individuals in each group did. This compromise in lung function in these individuals does not appear to be related to the presence of ETAP because it was also observed in the comparison group and might simply reflect mild undiagnosed postexercise bronchospasm in these individuals.

In the postexercise test, both groups demonstrated a substantial increase in PIF and a slight decrease in FEVC. The increase in PIF could be explained by an increased lung compliance associated with exercise-induced surfactant production.[29] The small decrease in FEVC might have been the result of the subjects' finding the flow-volume loop challenging when performed immediately after ceasing the treadmill exercise. The subjects' ventilations were high at this time, and for this reason they found it taxing to hold the forced expiration for the 5 seconds required by the testing protocol.

The analyses most relevant to the primary focus of the present study are the comparison data between the groups because they enable the effect of ETAP to be elucidated. Clearly, the episode of ETAP did not result in substantial effects on inspiratory or expiratory capacity. Although there was an average of 4.6% difference in the increase in PIF pre–post between the groups, both groups increased considerably more than 4.6% from pre to post. This suggests that ETAP is unlikely to result in a restrictive impairment of lung function.

In terms of an obstructive respiratory disorder, the between-groups differences in the expiratory measures are of interest. Similar reductions in FEVC postexercise were observed in both groups, but on average, the ETAP group experienced a reduction in PEF and $FEV_{1.0}$ after exercise, whereas the comparison group improved. If this is a real effect attributable to ETAP and not merely some

individuals experiencing mild postexercise bronchospasm, 2 explanations seem plausible for a possible reduction in expiratory power associated with ETAP. First, the pain being experienced in the ETAP group might have an effect on forced expiration. Immediate, sudden pain can cause acute apnea, but little information is available on the effect of more persistent pain on spirometry measures. Pain is typically associated with some pathology, and separating the 2 is difficult if not impossible. The second possible explanation is that the mechanism responsible for ETAP imposes obstructive limitations on lung function. $FEV_{1.0}$ and PEF can be reduced by an increase in airway resistance or a decrease in the lung's elastic recoil.[12] A mechanism for such acute obstructive changes is unclear, and if this is the case, it is also unclear why expiratory capacity (FEVC) is unaffected.

The measures of inspiratory performance were the most relevant to the primary aim of the present study because inspiration is dominated by diaphragm activity.[18-20] The results suggest that inspiratory performance is unlikely to be compromised during an episode of ETAP, indicating that diaphragm function is not substantially impaired during an episode of ETAP. This observation is consistent with the Guy's Hospital report.[6] Compromised diaphragm function would be anticipated if the diaphragm were anoxic or in spasm, which is commonly asserted as the mechanism responsible for ETAP.

Practical Applications

The following practical applications arise from the present study:

- Carbonated drinks with high sugar content appear to be highly provocative of ETAP. Hence, these fluids should be avoided by individuals prone to ETAP to reduce their likelihood of experiencing the pain. Conversely, the use of these fluids is of value to researchers wanting to evoke the pain for investigative purposes.
- Diminished exercise performance associated with ETAP is not likely the result of compromised lung function.
- Because diaphragm function is not substantially affected during an attack of ETAP, pain-management strategies specifically designed to target the diaphragm are likely to be ineffective. Such procedures have included "belly breathing," in which the abdomen is distended during inspiration; deep breathing; and forced expiration through pursed lips. If these techniques assist with pain relief it would seem that their effect is for reasons other than their influence on the diaphragm.

Conclusions

The findings of the present study indicate that diaphragm function is unlikely to be compromised during an episode of ETAP, which is enlightening with respect to the etiology of the pain. Whether the abdominal muscles are implicated in the pain requires further investigation.

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