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Effect of Hyperventilation on Distal Colonic Motility and Rectal Sensitivity in Irritable Bowel Syndrome

D.G. Maxton, Alison Prior, P.J. Whorwell

Department of Medicine, University Hospital of South Manchester, UK

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Abstract. Hyperventilation is associated with some symptoms suggestive of irritable bowel syndrome and has been implicated in provoking excessive oesophageal contractility. Sixteen patients with irritable bowel syndrome were therefore studied in order to assess the effect of hyperventilation on distal colonic motility and rectal sensitivity. No significant change in either the amplitude or frequency of colonic contractile activity was noted following hyperventilation, nor was any alteration in rectal sensitivity observed. This study shows that acute hyperventilation does not affect colonic motor activity or visceral sensitivity and suggests that hyperventilation and irritable bowel syndrome are not causally related.

Hyperventilation may present with gastrointestinal, as well as neurological, respiratory and psychiatric symptomatology [1–3]. There is considerable overlap between the gastrointestinal manifestations of hyperventilation and irritable bowel syndrome (IBS) [3, 4], and this has led to the suggestion that symptomatic episodes of IBS may be provoked or worsened by hyperventilation [5]. The pathophysiological mechanisms underlying the symptomatology of IBS remain unclear, but it is believed that exaggerated motor activity [6–8] and enhanced visceral sensitivity [9–11] may both be of importance. It has recently been shown that hyperventilation enhances oesophageal contractility both in healthy controls [12] and in patients with noncardiac chest pain [13], but it is not known whether it modifies gut motility elsewhere. No information is available on the effect of hyperventilation on visceral sensitivity.

The aim of this study was to assess the influence of acute hyperventilation on spontaneous colonic contractions and rectal sensitivity in patients with IBS.

Material and Methods

Sixteen patients with IBS (all women: age range 18–44 years) were studied. IBS was diagnosed on the clinical history of abdominal pain, distension and a

disturbance of bowel habit in accordance with the guidelines of the International Working Party on IBS Rome 1988 [14]. All had either contrast radiology [10] or colonoscopy [6]. None gave a clear history of hyperventilation attacks. All patients completed a Hospital Anxiety and Depression Questionnaire with a score of greater than 8 for either anxiety or depression regarded as positive for that feature.

Distal Colonic Motility

Six patients took part in this study. Each subject was fasted overnight before the study and all medication discontinued for 48 h. Without bowel preparation a triple lumen polyethylene catheter was inserted using a colonoscope and the openings positioned 30, 25 and 20 cm above the anal verge. Each lumen of the catheter was perfused with water at 0.6 ml/min by an Arndorfer capillary infusion system.

Ten minutes after catheter insertion motility recording was commenced and a 30-min baseline period documented before hyperventilation. Each patient was hyperventilated for 3 min maintaining an end-tital pCO_2 below 3 kPa directly recorded on a capnograph (Capnograph Mark II, Gould Godart BV, Bilthoven, The Netherlands). End-tidal pCO_2 recording was continued until the prehyperventilation level was attained usually 4 min after cessation of hyperventilation. Changes in colonic intraluminal pressure were recorded on a Lectromed multichannel pen recorder (Ormed, Welwyn Garden City, UK). For each of the three recording levels the number of contractions and mean amplitude of contraction was calculated and from this a motility index (the sum of amplitude/2 $\times$ length of contraction) derived. All parameters were calculated for 5 min prehyperventilation, for 3 min during hyperventilation and for 5 min posthyperventilation. The indices during hyperventilation were then converted to 5-min epochs to allow direct comparison. By 5 min posthyperventilation end-tidal pCO_2 had recovered to baseline values in all patients.

Anorectal Manometry

Ten patients were studied. With the subject in the left lateral position a multilumen polyvinyl catheter was placed in the rectum with side holes at 1, 4 and 14 cm from the anal verge and connected to water-filled transducers. A 5-cm latex balloon was attached to the catheter between 6 and 11 cm with a side hole at 8.5 cm linked to an air-filled transducer. The same recording and perfusion equipment was used as for distal colonic motility.

After a basal period of at least 15 min the rectal balloon was serially inflated with air at intervals of 30 s in 20 ml increments up to 100 ml and then in 50 ml increments to a maximum volume of 500 ml. After deflation of the rectal balloon, each patient was hyperventilated for 3 min as described above. Immediately after cessation of hyperventilation anorectal manometry was repeated. This ensured that the rectal sensitivity was assessed during a period of low pCO_2. The balloon volumes required to elicit sensations of gas, stool, urgency of defecation and rectal discomfort were thus recorded before and immediately after hyperventilation.

Statistical Analysis

Differences before, during and after hyperventilation were compared using a repeated measure analysis of variance with a square root transformation to normalise data or the non-parametric Freidman test as appropriate. Differences before and after hyperventilation were compared using the Wilcoxon matched pair test.

Results

All patients attained an end-tidal pCO_2 of less than 3 kPa within 30 s of commencing hyperventilation and usually maintained a level of around 2.5 kPa. Six of the 16 patients complained of dizziness and peripheral paraesthesiae although only 1 felt her gastrointestinal symptoms had been reproduced.

Distal Colonic Motility

Motility index and details of colonic contraction before, during and after hyperventilation are given in table 1. Assessment of motility during hyperventilation is hampered by a significant respiratory artefact. Despite this there was no significant change in any parameter of colonic motility during or after hyperventilation. In 3 individuals motility

Table 1. Effect of acute hyperventilation (HV) on distal colonic motility (per 5-min epoch)

	Pre-HV	HV	Post-HV
Number of contractions	4.5 (0.2/14.4)	2.2 (0.1/9.8)	3.9 (0.1/13.3)
Mean Ampl, cm H_2O	9.6 (6.0/16.8)	11.8 (11.3/19.6)	13.5 (10.0/23.8)
Motility index	204 (60/1,996)	152 (0/1,337)	355 (0/3,828)

Mean (95% CI); n = 6.

indices rose after hyperventilation and in 3 declined. There was no change in the percentage of stationary to propagated colonic contractions during any phase of the study with the majority being of the stationary type.

Anorectal Manometry

The rectal volumes required to elicit the sensations of gas, stool, urgency of defecation and discomfort in each patient are shown in figure 1. A similar rectal threshold for each sensation was recorded in the majority of patients, although there was a tendency for gas and stool to be perceived at slightly higher volumes after hyperventilation. However, as a group no significant change in rectal sensitivity was produced by hyperventilation.

Psychological Assessment

Six of the 16 patients were anxious and 2 depressed as assessed by the Hospital Anxiety and Depression scoring system. These patients did not behave differently from the remainder of the group.

Discussion

This study has failed to show any significant changes in colonic motility in patients with IBS following acute hyperventilation. These findings differ from those in the oesophagus, where hyperventilation has been reported to cause an increase in the amplitude of contractile activity [12, 13]. In these reports oesophageal motility was induced by swallowing, which is a predictable stimulus for motor activity. In the colon, stimuli of activity such as a meal or cholecystokinin are less reliable and a more prolonged period of recording is usually necessary. Unfortunately the maintenance of a low pCO_2 is such an unpleasant experience that of necessity recording periods have to be short. It would thus be extremely difficult to combine hyperventilation with any form of colonic stimulus. However by the time prehyperventilation pCO_2 levels had been regained at least 7 min had elapsed and in the oesophageal studies motility changes occurred both during and after hyperventilation and might therefore have been expected to have been observed here. The results of this study do not preclude a subtle effect of acute hyperventilation but a profound and obvious association between hyperventilation and colonic motility was not apparent. In addition changes during chronic rather than acute hyperventilation have not been excluded.

There has recently been increased interest in the role of visceral sensitivity in the pathophysiology and symptomatology of IBS [10, 11]. In patients with visceral hyper-

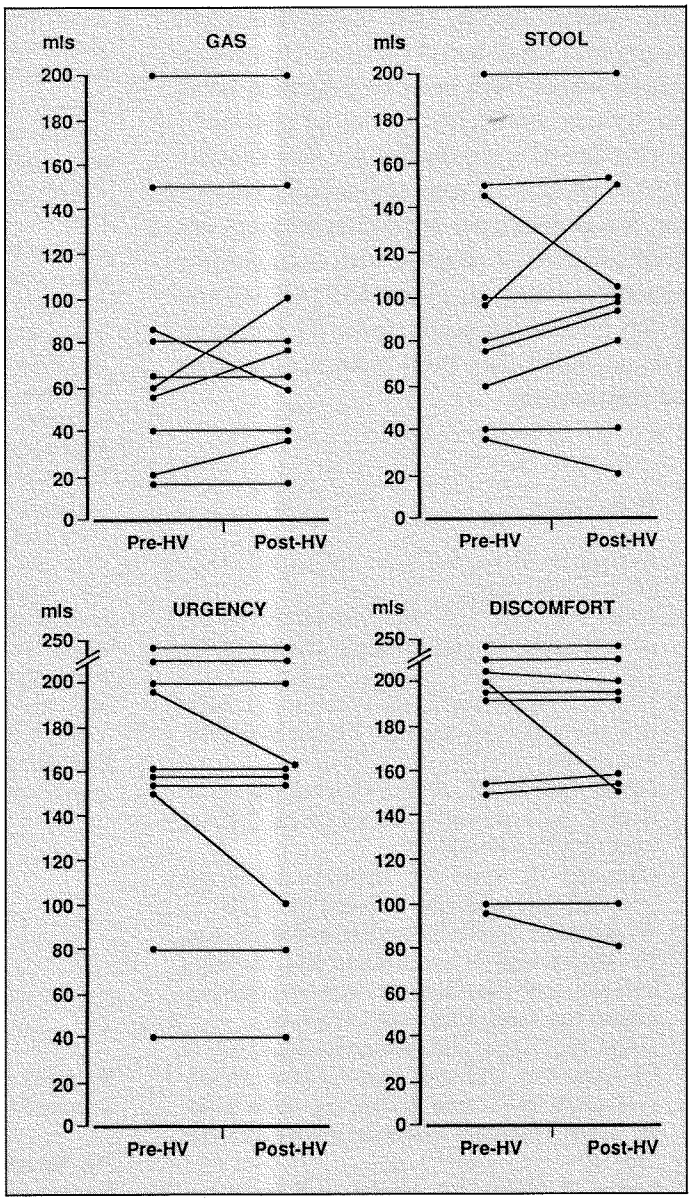

Fig. 1. Volumes required to elicit the rectal sensations of gas, stool present, urgent desire to defecate and discomfort before and after hyperventilation (HV).

sensitivity, thresholds for the appreciation of painful stimuli such as contractions or intraluminal distension may be lowered. As hyperventilation did not stimulate colonic contractile activity it seemed reasonable to assess its effect on visceral sensitivity but again the results were negative.

If hyperventilation does not influence colonic motility or visceral sensitivity why is it associated with symptoms suggestive of IBS?

One possibility is that hyperventilation might alter small bowel motility, abnormalities of which have been suggested to be of importance in IBS [8]. The other possibility is that the two conditions are not causally linked but share a common aetiological factor. A high incidence of psychopathology has been reported in patients with both chronic hyperventilation [1, 3] and IBS [15, 16] and it may be that this accounts for the symptom overlap rather than there being a direct causal relationship.

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P.J. Whorwell, MD
Department of Medicine
University Hospital of South Manchester
Nell Lane, Didsbury
Manchester M20 8LR (UK)