Chest Muscle Activity and Panic Anxiety: A Preliminary Investigation

PATRICK LYNCH, PHD, DONALD A. BAKAL, PHD,
WILLIAM WHITELAW, MD, AND TAK FUNG, PHD

This report represents a pilot investigation of the role of chest muscle electromyographic (EMG) activity in developing panic episodes. Chest EMG activity was obtained as part of a larger study examining ventilatory differences between panic sufferers and normal controls. Frontalis EMG, heart rate, and minute ventilation (breathing rate and tidal volume) were also obtained during the study. The ventilatory procedure involved exposing the subjects to three periods of carbon dioxide gas inhalations (1%, 3%, 5%; balance oxygen). Subjective measures of frightening cognitions and body sensations were obtained across the inhalation phases as well. The panic disorder subjects were divided, on the basis of subjective anxiety ratings obtained throughout the study, into high anxious (HA) and low anxious (LA) panic disorder groups. The HA panic disorder patients exhibited significantly higher chest EMG activity than the LA panic disorder patients and controls across all phases of the experiment. In addition, the chest EMG predicted, better than the other physiologic measures, the number of frightening cognitions and sensations reported by the subjects during the baseline and 5% CO_2 inhalation phases. Overall, the results were supportive of the further study of chest wall EMG activity in the pathogenesis of panic attacks.

INTRODUCTION

The multifaceted origins of panic disorder are recognized to include both cognitive and physiological determinants, with hypotheses surrounding central biological panic sites continuing to dominate the literature (1). There is growing appreciation, however, that factors outside the central nervous system per se contribute to both susceptibility and onset of panic attacks. Psychological or cognitive variables are recognized as part of the puzzle (2, 3) but purely psychological formulations have difficulty explaining the powerful physical symptoms of panic as well as accounting for attacks in the absence of provocation.

This study examined chest wall muscle activity as part of an investigation dealing with ventilatory correlates of panic susceptibility. The larger study examined two chronic hyperventilation hypotheses based on chemoreceptor sensitivity and respiratory after-discharge (4). The ventilatory hypotheses were tested using different levels of CO_2 inhalation and guided by the expectation that panic disorder patients would evidence greater increases than controls in minute ventilation in response to 1%, 3%, and 5% CO_2 concentrations. Chest electromyographic (EMG) activity was studied as a possible physiologic component of panic reactions to the experimental situation and manipulations.

From the Division of Psychology, Foothills Hospital (P.L.); and the Department of Psychology (D.A.B.), Respiratory Medicine (W.W.), and Academic Computing Services (T.F.), University of Calgary, Calgary, Alberta, Canada.

Address reprint requests to: Donald A. Bakal, 2500 University Drive N.W., Calgary, Alberta, T2N 1N4.

Received September 6, 1990; revision received October 2, 1990

0033-3174/91/5301-0080$03.00/0

The decision to monitor chest wall muscle activity during the provocation phases of the experiment was made on the basis of symptoms commonly reported by panic sufferers. Dyspnea is the most common respiratory symptom reported to precede and accompany panic episodes (2). However, the majority of panic sufferers also describe feelings of pressure, tightness, heaviness, and other sensations from the chest. Recent clinical observations of panic sufferers attest to the pervasive presence of chest pain as well as chest pressure and tightness prior to and during panic episodes (2, 4). The physiological origins of chest pain in panic disorder patients are unknown; however, cardiovascular disease does not appear to be implicated. Research has shown a high incidence of the diagnosis of panic disorder and normal coronary arteriograms in chest-pain patients presenting to cardiologists (5).

Chest wall muscle activity as reflected in electromyographic activity (EMG) from the region of the sternum was recorded across all phases of the experiment for 9 panic disorder patients and eight normal controls. Forehead EMG, heart rate, and minute ventilation were also monitored. Subjective measures of anxiety, frightening cognitions, and somatic symptoms were also obtained across all phases in order to examine the interplay between psychological and physiological components of panic.

SETTING AND SUBJECTS

The study was conducted at the Pulmonary Laboratory/Clinic of the Foothills Hospital, Calgary, Alberta. Female psychiatric outpatients making their initial presentation for treatment were interviewed by a clinical psychologist who, as part of a general diagnostic interview, applied DSM-III (6) criteria. Nine subjects with a primary diagnosis of panic disorder (with or without agoraphobia), who reported at least four of the 12 DSM-III symptoms to be moderately severe during episodes of anxiety, and a frequency of panic at least once per week, were selected for the study. Eight female normal controls were selected from hospital and university staff. Questionnaire assessment of panic symptoms was used as a validity check of the diagnosis. Each subject completed the Body Sensations Questionnaire (BSQ) (7), the Agoraphobic Cognitions Questionnaire (ACQ) (7), and the Fear Questionnaire (8). The ACQ consists of 14 items dealing with thoughts of physical catastrophe due to anxiety symptoms and with thoughts reflecting social or behavioral disaster from loss of control. The BSQ is a 17-item scale concerning the degree to which patients fear somatic symptoms commonly associated with panic. Total Phobia Score and Agoraphobia Score were selected from the Fear Questionnaire. The same clinical interview and questionnaires were given to female control subjects selected from a group of hospital and university staff volunteers. Only females were used as an attempt to reduce variability on the baseline pulmonary function measures.

All patients and controls received a brief medical examination to exclude subjects who might have organic respiratory or cardiac disease or hyperthyroidism. All subjects provided informed consent. No subjects withdrew during the course of the experiment.

METHOD

Physiologic Measures

The primary physiologic measures relevant to this report are chest electromyogram (EMG), frontalis EMG, minute ventilation, and electrocardiogram (EKG). Chest and forehead EMG measures were each obtained from sets of three Beckman silver/silver chloride electrodes. The skin was prepared with prepping paste and isopropyl alcohol and the electrodes were attached 1.0 cm apart by means of an electrode adhesive strip. The three chest electrodes were placed vertically on the sternum, with the lowest 5 cm above the xiphoid process. The EMG measures were obtained from Cyborg EMG J33 preamplifiers and a Cyborg dual EMG rms contour processing system (100–1000 Hz passband, time constant = 0.3 seconds). In order to minimize artifact

due to changes in the breathing cycle and the EKG, the lowest EMG recorded during the last 10 seconds of each minute of observation was used as the recorded value. This was an end-expiratory value. EKG was recorded using a five lead (left leg, right leg, left arm, right arm, chest) configuration, with the signals routed through a Hewlett-Packard amplifier. Expired air was continuously sampled from the mouthpiece through which the subject breathed. End tidal CO_2 was monitored using a Beckman LB-II Infra-Red Gas Analyzer, which was calibrated before data collection using room air and a calibration-grade medical gas mixture containing 5.02% carbon dioxide, 94.98% oxygen. Minute ventilation was determined on the basis of expired flow, which was measured by a Fleisch #2 pneumotachograph and a Validyne differential pressure transducer attached to the output side of an Otis-McKerrow valve, with the signal sent to a Hewlett-Packard Carrier amplifier. The gas mixtures were administered from a rubber reservoir bag connected to the input side of the Otis-McKerrow valve. The reservoir was kept partially full of gas through a continuous flow from the calibrated gas cylinder. With each change in gas concentration, the reservoir and connecting tubes were thoroughly flushed and filled with the next gas concentration.

Procedure

Prior to the laboratory session, subjects were informed that this was a study of respiratory parameters, and that they would be exposed to a series of CO_2/O_2 gases. They were informed that any changes in breathing during the inhalations would be a normal consequence of the gas inhalations. During the laboratory session, subjects were required to lie supine and breathe different mixtures of CO_2-O_2. In order to reduce anxiety, patients were informed in a session one week before the lab session and during the lab session that this was not a study of panic attacks, but of their general physiology, and were reassured that measures would be taken to reduce the possibility that they would panic in the laboratory. Patients and controls were informed in advance that inhalation of various mixtures of CO_2 might cause some physical symptoms similar to those associated with physical exercise (e.g., higher respiration rate, fatigue).

Each session began with the measurement of the subject's Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV1) while standing and breathing into a spirometer. Next, the subject was required to lie on a bed while the EMG and EKG electrodes were attached. The subject was then familiarized with the use of the mouthpiece, following which a noseclip was affixed so that all breathing would be entirely through the mouthpiece. Once everything was in place, the five-minute baseline period began during which time subjects were instructed merely to lie quietly and get used to the equipment while breathing room air. At the end of this period, the noseclip and mouthpiece were removed and the rating scales were completed. The baseline was followed by three successive five-minute CO_2 gas inhalation periods with 1%, 3%, and 5% CO_2 in oxygen, respectively. The subjective rating scales were completed at the end of each inhalation period. Calibration grade CO_2 gas mixtures were used throughout the study. The 5% CO_2 inhalation was followed by a five-minute recovery period.

Immediately after each of the five phases, the subject was prompted to rate her level of anxiety on a 10-point Likert scale ranging from '0' (very relaxed and sleepy) through '5' (moderately severe anxiety) to '10' (full-blown panic attack). Responses to this scale were used to group subjects into those who showed evidence of panic anxiety across the manipulations from those who did not. Subjects were also required to report on the intensity of a number of somatic and cognitive symptoms as measured by the BSQ and the ACQ. The instructional set given to subjects to complete these instruments was in reference to their experiences during the immediately preceding phase.

RESULTS

Pre-Baseline Comparisons

The clinical and control subjects did not differ on age, weight or height (Table 1). The groups were also similar in terms of forced vital capacity and forced expiratory volume. The groups were significantly different in terms of their scores on the Agoraphobic Cognitions Questionnaire (ACQ; $t = 4.03$, $df = 15$, $p < 0.002$) and the Body Sensations Questionnaire (BSQ; $t = 7.19$, $df = 15$, $p < 0.001$). The panic disorder subjects also reported significantly more phobic avoidance behavior than the controls as measured by the

TABLE 1. Comparison of Panic Disorder Patients and Controls on Pre-Experimental Physical and Psychological Characteristics*

	Panic Disorder		Controls	
	M	SD	M	SD
Age (years)	36.4	9.7	31.1	8.3
Weight (kg)	64.5	16.2	61.6	12.4
Height (cm)	163.6	6.3	161.8	7.3
FEV1 (liters)	3.6	0.5	3.5	0.6
FVC (liters)	4.2	0.6	4.1	0.6
Cognitions	35.9	11.4	17.8	3.2
Sensations	50.7	10.1	23.0	6.7
Total phobia	53.4	14.9	10.9	5.5
Agoraphobia	20.2	1.9	10.3	1.6
No. of panics	4.9	3.1	0.0	0.0

* FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; Cognitions, total score on Agoraphobic Cognitions Questionnaire (ACQ); Sensations, total score on Body Sensations Questionnaire (BSQ); Total Phobia, subscale scores from the Fear and Agoraphobia Questionnaire; No. of panics, number of panic attacks reported during last 7 days.

Total Phobia score ($t = 7.5$, $df = 15$, $p < 0.001$) and the Agoraphobia scale ($t = 4.9$, $df = 15$, $p < 0.001$) of the Fear Questionnaire.

Each subject was queried about medication use during the 24 hours prior to the laboratory session. One panic disorder subject reported minor tranquilizer use and two subjects reported tricyclic antidepressant use. None of the panic subjects reported use of monoamine oxidase inhibitors or beta-blockers. None of the controls reported use of any medications.

Sub-Grouping of Panic Disorder Subjects

Although efforts were made to minimize the occurrence of anticipatory anxiety and panic, the subjects proved to be highly variable in their subjective reactions to the laboratory situation, both at baseline and across the various CO_2 inhalation phases. Prior to the analyses, the subjects were grouped on the basis of how "anxious" or "panicky" they became during the various phases of the experiment. The grouping was made on the basis of each subject's average response across the five administrations of the 10-point subjective anxiety scale. Previous research (9) demonstrated that the average panic attack occurs at a moderate intensity (5.6 on a 0–10 scale). Subjects with an average scale score of 5 or greater ($n = 6$) were placed in the High Anxious (HA) group while subjects with an average score less than 5 were placed in the Low Anxious (LA) group ($n = 3$). None of the control subjects ($n = 8$) obtained an average anxiety score above 5. All three panic disorder subjects reporting medication use were in the HA group. An additional analysis using the Marks and Mathews (8) Fear Questionnaire indicated that the three groups also differed in phobic severity outside the experimental situation (Table 2). HA subjects had significantly greater Total Phobia scores, $F(1,14) = 6.96$, $p < 0.02$, and significantly greater Agoraphobia scores, $F(1,14) = 7.63$, $p < 0.02$, than the LA group. The LA group also had significantly higher scores on the Total Phobia subscale, $F(1,14) = 21.35$, $p < 0.001$, and the Agoraphobia subscale,

TABLE 2. Group Means for the total Phobia and Agoraphobia Subscales of the Fear Questionnaire

	Total Phobia		Agoraphobia	
	M	SD	M	SD
HA panic disorder	59.5	13.2	24.3	9.0
LA panic disorder	41.3	11.0	12.0	8.2
Controls	10.9	5.5	1.9	1.6

$F(1,14) = 5.61$, $p < 0.05$, than the Control group.

Physiologic Responses to Carbon Dioxide

The physiologic data were grouped and analyzed across the baseline, CO_2 inhalation, and recovery phases. The data were reduced to one-minute averages for the time periods defining each phase. The group means for chest EMG, forehead EMG, heart rate and minute ventilation are presented in Figure 1. The primary variable of interest in this study was chest EMG activity. However, given the potential interdependence of the physiological variables, a three-way repeated measures MANOVA (Groups, Phases, Time Within Phase) was carried out using a total of six physiological measures (breathing rate and tidal volume were included as variables separate from minute ventilation). The multivariate effects for Phase, $F(24,179) = 4.91$, $p < 0.01$, and Time Within Phase, $F(24,179) = 2.83$, $p < 0.01$, were significant. The univariate F's for the three ventilatory measures were significant for both the Phase and Time effects ($p < 0.01$) indicating that the CO_2 manipulations were effective in altering breathing. Heart rate also showed a significant univariate F for Phases, $F = 2,31$, $p < 0.001$, but not for Time. Chest and forehead EMG however were not associated with significant univariate F's for Phases or Time.

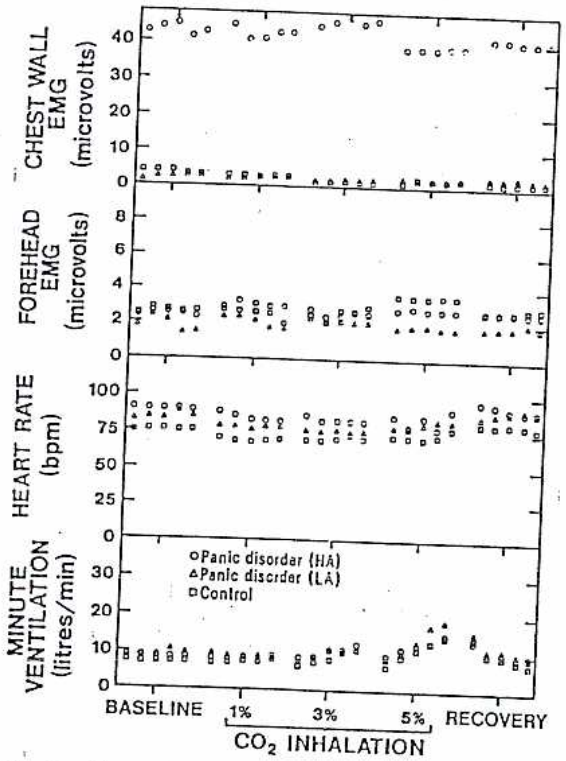

Fig. 1. Physiologic average scores of high anxious (HA) panic disorder subjects, low anxious (LA) panic disorder subjects, and controls.

The multivariate F for Groups was nonsignificant. The a priori hypothesis regarding chest EMG was supported with a significant univariate result, $F(2,14) = 6.58$, $p < 0.01$. The Group univariate F's for the remaining physiological variables were all nonsignificant. Post-hoc analyses revealed that the HA panic group had significantly higher chest EMG than both the LA panic, $F(1,14) = 6.83$, $p < 0.02$, and control groups, $F(1,14) = 11.58$, $p < 0.01$. The LA group did not differ significantly from the control group on this measure. In summary, heightened chest muscle activity was observed only in the panic disorder subjects who were also showing evidence of anxiety and/or panic. The group differences were independent of the CO_2 manipulations.

As a further examination of possible ventilatory differences, a separate one-way ANOVA was conducted on the final minute of each of the three CO_2 inhalation phases. This minute represents the period of maximal provocation within

each gas inhalation phase and the best approximation of steady-state responding achieved in the study. None of the comparisons was significant.

Psychologic Responses to Carbon Dioxide

The modified BSQ and ACQ questionnaires were administered at the end of the baseline and immediately after each of the three carbon dioxide inhalation phases. A two way (Groups, Phases) MANOVA performed on the total scores of these questionnaires revealed significant main effects for Groups ($F(4,26) = 2.75$, $p < 0.05$) and for Phases ($F(6,82) = 2.42$, $p < 0.05$). Insufficient degrees of freedom ruled out calculation of the interaction effects. The Group univariate F for BSQ scores was significant, $F(2,14) = 5.45$, $p < 0.02$, while the Group univariate F for ACQ scores was nonsignificant. Post-hoc analyses revealed significant BSQ score differences between the HA Panic Group and Controls, $F(1,13) = 3.39$, $p < 0.01$. The remaining comparisons were nonsignificant.

The Phase univariate F for BSQ scores was significant, $F(2,32) = 3.35$, $p < 0.05$) while the Phase univariate F for ACQ scores was nonsignificant. As shown in Figure 2, the three groups showed a decrease or no change in their BSQ scores from baseline through to 3% CO_2 inhalation. Following 5% CO_2 inhalation, the groups showed an increase in their BSQ scores.

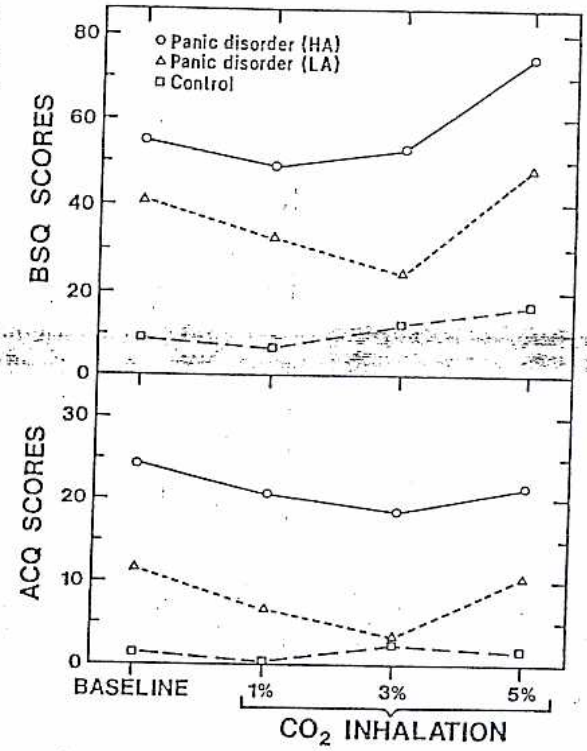

Fig. 2. Body Sensation Questionnaire (BSQ) and Agoraphobic Cognition Questionnaire (ACQ) scores obtained at the end of baseline and following each of the carbon dioxide inhalation phases.

Physiologic Predictors of Anxiety, Cognitions, and Bodily Sensations

The relationships between the physiologic and subjective measures during baseline and 5% CO_2 were examined with regression analyses. For each phase, a separate analysis was performed using BSQ scores and ACQ scores as the dependent variables, respectively. The independent variables were chest EMG, forehead EMG, minute ventilation, and heart rate. The physiologic data used in these analyses were obtained from the last minute of baseline and of 5% CO_2 inhalation. The results of these analyses are presented in Tables 3 and 4. The combination of chest EMG, forehead EMG, minute ventilation, and heart rate yielded significant R2 for

TABLE 3. Physiologic Predictors of BSQ and ACQ Scores During Baseline

Independent Variables	Dependent Variables	
	BSQ	ACQ
	Regression Coefficient	
Chest EMG	0.55*	0.76†
Forehead EMG	1.59	0.28
Minute vent	4.96*	2.31*
Heart rate	0.92*	0.26

* $p < 0.05$.
† $p < 0.001$.

TABLE 4. Physiologic Predictors of BSQ and ACQ Scores Following 5% CO2 Inhalation

Independent Variables	Dependent Variables	
	BSQ	ACQ
	Regression Coefficient	
Chest EMG	1.26‡	0.88†
Forehead EMG	0.54	−0.36
Minute vent	0.24	1.29
Heart rate	1.28	0.33

‡ $p < 0.01$.
† $p < 0.001$.

BSQ scores for both phases: baseline, $F(4,9) = 8.20$, $p < 0.005$; 5% CO_2 inhalation, $F(4,9) = 5.53$, $p < 0.02$. A similar pattern emerged for the ACQ scores with significant R2 emerging for both baseline, $F(4,9) = 18.18$, $p < 0.001$, and 5% CO_2 inhalation, $F(4,9) = 38.18$, $p < 0.001$. Chest EMG accounted for significant variance for BSQ and ACQ scores ($p < 0.05$) during both phases. Minute ventilation accounted for significant variance associated with the BSQ and ACQ scores during baseline ($p < 0.05$) but not during 5% CO_2 inhalation. Heart rate contributed significantly ($p < 0.05$) to baseline BSQ scores only. The contributions of forehead EMG were nonsignificant.

DISCUSSION

Preliminary data are presented indicating that heightened chest EMG activity recorded from the region of the sternum constitutes a physiologic component of panic episodes and a strong predictor of panic-based somatic and cognitive symptoms. By dividing panic disorder subjects on the basis of anxiety level, it was observed that the heightened chest EMG activity was specific to the panic subjects who experienced moderate to severe anxiety during the course of the study. The EMG activity observed in the chest region was not matched by similar EMG activity in the forehead. Heart rate was variable and not significantly different across the groups. Minute ventilation group differences were nonsignificant. These findings need to be interpreted with considerable caution as the group sample sizes were extremely small.

Group differences in chest EMG occurred across all phases of the experiment, including the initial baseline period. The chest EMG measure was nonresponsive to the experimental procedures as there was no increase from baseline during the CO_2 inhalation phases. We cannot determine, from the available data, whether the heightened chest EMG activity was chronic in nature or specific to the experimental situation. Other researchers (10) have noted high anxiety and physiological arousal in panic disorder patients during baseline periods of provocation paradigms. We suspect that the pulmonary laboratory setting proved to be highly anxiety provoking for the

more severe patients, despite the use of antipanic instructions and reassurances.

The HA and LA panic disorder subjects, although grouped on the basis of their anxiety levels during the experimental situation, also differed significantly on a pre-experimental measure of total phobic behavior and agoraphobic avoidance activity. A hallmark clinical feature of agoraphobia is fear of situations that prevent easy escape. Entering the pulmonary lab and having the physiologic transducers attached certainly made a rapid or unnoticed exit difficult. Thus, the severity of the condition may have prompted the panic-related chest EMG activity observed during the session.

In a review of the sodium lactate infusion literature, Margraf et al. (10) commented that baseline differences in arousal may explain why some subjects panic to lactate infusion and other subjects do not. The reviewers also suggested that the baseline differences between panickers and nonpanickers may reflect acute anticipatory anxiety rather than chronic differences in level, with the lactate pushing more highly aroused subjects across a tolerance threshold. We propose, in a similar fashion, that the HA panic disorder subjects in the present study were initially exhibiting high levels of anticipatory anxiety and panic-related somatic and cognitive symptoms. The accompanying chest EMG activity, within this context, was likely a physiological component of the developing panic attack. Panic subjects who showed no anxiety exhibited low levels of chest EMG activity and were very similar to the controls on this measure.

Group differences in heart rate were nonsignificant. Other investigators (9) have observed a lack of relationship between heart rate and subjective reports of panic. The respiratory measure of minute ventilation also failed, in the present study, to statistically differentiate the panic groups and controls. Minute ventilation was predictive of somatic symptoms and cognitions associated with panic during baseline. However, there was no evidence for differences between the groups in terms of responsiveness to the different levels of CO_2 inhalation. The use of the 5-minute inhalation period may not have been sufficient for all subjects to have reached asymptotic levels of minute ventilation. In addition, the small number of subjects prevents reaching a conclusion that there are no differences in CO_2 responses between panic patients and controls.

Given the diagnostic procedures used, we cannot be certain that the present findings are specific or unique to panic disorder. The subject selection/diagnostic procedure did not rule out the possibility of other Axis I conditions. Such a possibility would not, however, change the basic conclusion that chest wall EMG activity is a component of panic anxiety. The group differences in chest wall EMG activity were discernible in spite of the fact that three of the six high anxious subjects were receiving medications for panic. It is possible that these medications may have attenuated group differences in the other physiological parameters.

Freeman and Nixon (11) proposed that overuse and fatigue of the intercostal muscles is responsible for the chest symptoms experienced by panic sufferers. Surface electrodes placed over the sternum can pick up activity from pectoral and parasternal intercostal muscles. In normal subjects, pectoral muscles are used in respiration only for vigorous inspiratory ef-

forts, such as when there is mechanical impedance to breathing. The parasternal intercostal muscles are used for inspiration in normal quiet breathing, but are silent in expiration (12, 13). The EMG reported in this study was the trough of the time-varying signal and corresponded to end-expiration. The sustained EMG activity seen in expiration implies an inspiratory activity maintained throughout the whole breathing cycle. That is, these muscles are holding the chest, in a tonic fashion, at a volume higher than its relaxed volume, plus making repeated inspiratory efforts to increase volume further for each breath. Such activity would be quite abnormal and serve no physiological purpose and could potentially be quite tiring.

Biologic models of panic are viewed by an increasing number of investigators as too narrow and in need of expansion to include both psychological and physiological factors. It has been suggested that biologic challenge tests, such as CO_2 inhalation, rather than directly activating biologic panic sites, operate by eliciting somatic sensations to which patients respond with fear (2); i.e., carbon dioxide challenge leading to bodily sensations that are perceived by panic sufferers as frightening. Furthermore, this viewpoint implies that such challenge tests produce similar physiological responses in everyone and it is the hypervigilance for accompanying somatic sensations which is the significant determinant of panic. The present findings indicate that there may be a musculoskeletal substrate for some of the sensations. Replication and elucidation of the chest wall EMG findings, under conditions of threat and non-threat, would add to our understanding of this condition.

SUMMARY

As a pilot investigation, the level of EMG activity was measured from the sternum of panic disorder patients and controls who were participating in a larger ventilatory study. All subjects were exposed to three concentrations of carbon dioxide inhalation during a single session. Chest muscle activity was recorded from surface electrodes throughout the session. Additional physiological measures recorded during the session included forehead EMG, heart rate, and minute ventilation. Psychological data were obtained prior to the laboratory session and across the experimental phases within the session.

Prior to conducting the main statistical analyses, the panic disorder patients were sub-grouped into High Anxious (HA) and Low Anxious (LA) panic disorder groups on the basis of their average anxiety scores reported during the session. HA panic patients ($n = 6$) had significantly higher chest EMG activity than LA panic patients ($n = 3$) and controls ($n = 8$) across all phases of the study, including baseline. The groups showed no differences in terms of frontalis EMG activity. Group differences on the heart rate and minute ventilation measures were also nonsignificant. A series of regression analyses revealed that chest EMG activity proved to be the strongest physiologic predictor of somatic and cognitive symptoms measured during baseline and in response to 5% CO_2 provocation.

The findings, although very preliminary, were interpreted as support for chest EMG activity as a physiologic component of the panic episode. The activity is believed to be partially independent of the respiratory cycle and may have parasternal and pectoralis muscle origins.

REFERENCES

1. Gorman JM, Liebowitz MR, Fyer AJ, Stein J: A neuroanatomical hypothesis for panic disorder. Am J Psychiatry 146:148-161, 1989
2. Barlow DH: Anxiety and its disorders: The nature and treatment of anxiety and panic. New York, Guilford, 1988
3. Ottaviani R, Beck AT: Cognitive aspects of panic disorders. J Anxiety Dis 1:15–28, 1987
4. Bakal DA, Lynch PB, Whitelaw WA: Carbon dioxide recovery in panic disorder patients and controls. Paper presented at the Annual meeting of the American Psychosomatic Society, Boston, 1990
5. Katon W, Hall ML, Russo J, Cormier L, Hollifield M, Vitaliano PP, Beitman BD: Chest pain: Relationship of psychiatric illness to coronary arteriographic results. Am J Med 84:1–9, 1988
6. American Psychiatric Association: Diagnostic and Statistical Manual, Third Edition. Washington, DC, American Psychiatric Association, 1980
7. Chambless DL, Caputo GC, Bright P, Gallagher R: The assessment of fear of fear in agoraphobics: The body sensations questionnaire and the agoraphobic cognitions questionnaire. J Consult Clin Psychol 52:1090–1097, 1984
8. Marks IM, Mathews AM: Brief standard self-rating scale for phobic patients. Behav Res Ther 17:263–267, 1979
9. Margraf J, Taylor B, Ehlers A, Roth WT, Agras WS: Panic attacks in the natural environment. J Nerv Ment Dis 175:558–565, 1987
10. Margraf J, Ehlers A, Roth WT: Sodium lactate infusion and panic attacks: A review and critique. Psychosom Med 48:23–51, 1986
11. Freeman LJ, Nixon PGF: Chest pain and the hyperventilation syndrome—Some aetiological considerations. Postgrad Med J 61:957–961, 1985
12. DeTroyer A, Sampson MC: Activation of parasternal intercostals during breathing efforts in human subjects. J Appl Physiol 52:524–529, 1982
13. Taylor A: The contribution of the intercostal muscles to the effort of respiration in man. J Physiol (Lond) 151:390–402, 1960

British Journal of Obstetrics and Gynaecology
January 1991, Vol. 98, pp. 84–91

A randomized controlled trial of early amniotomy

WILLIAM D. FRASER, REG SAUVE, I. JOHN PARBOOSINGH, TAK FUNG, ROBERT SOKOL, DAVID PERSAUD

Abstract

Objective—To determine if a policy of early amniotomy resulted in a reduction in mean labour duration when compared to a policy of conservation of the membranes.
Design—A single-centre randomized controlled trial.
Setting—A tertiary care teaching hospital in Alberta, Canada.
Subjects—Ninety-seven term nulliparae in spontaneous labour, baby in cephalic presentation.
Intervention——Early amniotomy versus intent to keep membranes intact.
Main outcome measures—Interval from randomization to delivery, rate of abnormalities of fetal heart rate tracings, cord artery blood pH, Apgar scores.
Results—The mean interval from randomization to delivery was 390·9 (SE 29·1) min in the amniotomy group and 442·9 (SE 34·1) min in the control group ($P = 0{\cdot}251$). There were no differences between groups in the occurrence of fetal heart rate tracing abnormalities, nor was there a difference in the proportion of babies with abnormal Apgar scores, or abnormal cord pH ($< 7{\cdot}20$).
Conclusion—The results of the study fail to support the long held belief that early amniotomy is an effective method for reducing labour duration.

Amniotomy was introduced to obstetric practice by Kreis (1928) who, on the basis of data from case series, argued that it was an effective method to prevent prolonged labour. Amniotomy has recently been promoted as a component of the active management of labour, a protocol designed to reduce both the duration of labour and the occurrence of dystocia (O'Driscoll *et al.* 1984). The effects of amniotomy on the duration of labour have not been adequately assessed. The primary objective of this randomized trial of labour management was to determine the effectiveness of early amniotomy in reducing the duration of labour. The effects of the policy of membrane management on indicators of fetal and neonatal status were also assessed.

University of Calgary, Department of Obstetrics and Gynaecology and Community Health Sciences
W. D. FRASER
I. J. PARBOOSINGH
D. PERSAUD
Community Health Sciences and Pediatrics
R. SAUVE

Academic Computing Services
T. FUNG

Wayne State University
Department of Obstetrics and Gynecology
R. SOKOL

Correspondence: Dr William Fraser, Périnatologie, Hôpital St-François d'Assise, 10 rue de l'Espinay, Québec, QC Canada G1L 3L5.

Subjects and methods

Subjects were eligible if they met the following criteria: nulliparous, spontaneous labour, single fetus in cephalic presentation, term pregnancy (≥ 38 weeks), intact membranes, and a normal fetal heart monitor tracing at admission. Exclusion criteria were a history of genital herpes, the presence of proteinuria or hypertension, or a cervical dilatation of ≥ 5 cm at admission. Demographic information on eligible non-participants was obtained from the hospital's computerized data base.

The study was approved by the University

Medical Ethics Committee and consent was obtained at the time of admission. A non-stratified randomization technique was employed using the block method of Zelen (1974). A series of numbered, sealed, opaque envelopes was prepared containing allocation. Allocation was later verified against the master list. Women with cervical dilatation of < 3 cm were randomized when the fetal head was fixed in the pelvis and the cervix had undergone a change in dilatation after admission. Women with a cervical dilatation of ≥ 3 cm were randomized when the fetal head was fixed in the pelvis.

Women in the amniotomy group underwent the procedure as soon as possible while those in the control group had membranes conserved unless intervention was dictated by the judgment of the treating physician. Amniotomy during labour was permissible in the control group if there was a fetal heart rate abnormality, if there had been an arrest of cervical dilatation for ≥ 2 h, or when full dilatation was achieved. Physicians were requested not to augment labour with oxytocin unless there had been an arrest of cervical dilatation for ≥ 2 h. The frequency and duration of post-randomization electronic fetal monitoring was left to the treating physician. The time from randomization to delivery was the outcome of interest.

Fetal heart rate tracings were later analyzed by two trained observers who worked independently and who were blinded to allocation. The following definitions were used as guidelines in the interpretation of the tracings. Bradycardia: < 110 bpm; tachycardia: > 160 bpm; decreased variability: ≤ 5 bpm about baseline. Decelerations were classified as (1) early≥ 10 bpm below the baseline rate, onset within 20 s of the onset of contraction, recovery to baseline rate within 20 s of the end of the contraction; (2) late: repeated decreases in fetal heart rate, onset > 20 s after onset of contractions, recovery to baseline rate > 20 s after end of contraction; (3) variable: sharp decrease in rate from baseline and a variable relation to contractions. These were subclassified as (i) severe variable—one of the following criteria had to be met: ≥ 60 s duration; ≥ 60 bmp below baseline; a rate of < 60 bmp at its lowest point; (ii) mild variable: ≥ 15 bpm below the baseline and ≥ 15 s duration but failing to meet the criteria for severe variable decelerations. Variable-type decelerations which were < 15 s duration were disregarded. Repeated variable decelerations were counted as separate events only if the fetal heart rate returned to the baseline between decelerations. A dichotomous outcome (normal versus abnormal) was assigned to each hour during which at least 10 min of interpretable tracing was obtained. The segment of tracing was considered 'abnormal' if any of the following features were noted: decreased variability, severe variable or late decelerations, baseline tachycardia or bradycardia. The proportion of 'abnormal' hours was determined for both the pre-randomization and post-randomization periods. For each hour of tracing, the two observers were considered to agree on interpretation if the difference between observers in the total number of decelerations counted for that hour was no more than one, and if there was complete agreement on the normality or abnormality of the baseline fetal heart rate and of its variability. There was agreement between the observers for 87·5% of the hours assessed. When there were disagreements, the tracings were reviewed by the two observers together and a consensus on the interpretation of the tracings was achieved for all hours.

Umbilical artery blood was collected in a heparinized syringe by the delivery physician and was analyzed on a Corning Blood Gas Analyzer within 30 min of delivery. The Apgar score was assigned by the delivery room nurse or by a member of the paediatric staff, if one was present at delivery.

The comparisons of group means were performed using Student's *t*-test. The χ^2 test was used for comparison of proportions, except where the expected value for a cell was less than 5. In this case Fisher's exact test was used. The multiple linear regression analyses used Minitab (Ryan 1985). BMDP-2L (Hoplans 1985) was used to apply Cox's proportional hazards model to the survival data. The power analysis was performed using the statistical package Power (1985).

Results

The study was conducted at Foothills Hospital, a tertiary care teaching centre associated with the University of Calgary, between Sept 11, 1987 and Sept 11, 1988. Of the 1503 nulliparous women delivered during the study period, 278 (18%) were eligible to participate. The reasons for ineligibility and their frequencies are shown in Table 1. Of the 278 women eligible to partici-

Table 1. Reasons for exclusions in 1503 nulliparous women delivered during the study period

	N	(%)
Total no. of women delivered	1503	(100)
Total no. excluded		
Reasons for exclusion:	1225	(81·5)
Spontaneous rupture of membranes	373	(24·8)
Induction of labour	335	(22·3)
Cervical dilation > 5 cm at admission	113	(7·5)
Malpresentation	67	(4·5)
< 266 days gestation	157	(10·4)
Suspected fetal distress	52	(3·5)
Other (including med. complication)	128	(8·5)
Total no. eligible	278	(18·5)

pate 97 (35%) entered the study. The reasons for non-participation of eligible women were patient refusal (44%), doctor refusal (5%) and failure to offer participation by the nursing staff (16%). The mean gestational age at delivery was 39·9% (SD 0·1) weeks for non-participants and 40·0 (SD 0·1) weeks for participants. The mean maternal age was 27·5 (SD 0·3) years for non-participants and 26·7 (SD 0·6) years for participants. Oxytocin use for labour augmentation was similar in the two groups (non-participants 27·6%, participants 30·9%), as was the frequency distribution of the methods of delivery (data for non-participants not shown). A total of 100 women were randomized, 49 to the amniotomy group and 51 to the control group. Eligible patients did not change treatment groups after randomization, regardless of the evolution of their membrane status. Three women who did not meet the eligibility criteria were randomized in error (1 breech, 2 inductions of labour). Of the 97 eligible women who were randomized, 47 were allocated to the amniotomy group and 50 to the control group (conservation of the membranes). As seen in Table 2, the mean prepregnancy weight, the mean admission weight and the mean birthweight were somewhat greater in the amniotomy group than in the control group. The groups were similar with respect to maternal height education, and gestational age. The mean cervical dilatation at admission was somewhat less in the amniotomy group than in the control group (Table 2). A similar proportion of women in the two groups smoked during pregnancy, attended prenatal classes, was accompanied by a significant other (usually the husband), and claimed English as their mother tongue (data not shown).

Of the 47 women in the amniotomy group two had a spontaneous rupture of the membrane before amniotomy. Of the 50 women in the control group 19 had an amniotomy before full dilatation. The indications for amniotomy in the control group were labour augmentation (11), and suspected fetal distress (8). The mean interval from labour onset to rupture of the membranes was 431 (SE 36) min in the amniotomy group and 592 (SE 37) min in the control group ($t_{95df} = 3·13$, $P = 0·002$). Figure 1 is an histogram of the distribution of cervical dilatations at the time of membrane rupture for the two groups. Of the women in the control group 60% achieved ≥ 8 cm cervical dilatation before membrane rupture compared with only 2% in the amniotomy group.

Fifteen women in each group had labour augmented with oxytocin. There were eight caesarean sections in the amniotomy group and four in the control group. Similar proportions of women in each group were delivered by low forceps or vacuum extractor (amniotomy 23%, control 28%), and by mid-forceps (8% in each group).

The mean interval from randomization to

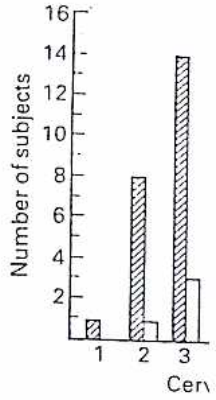

Fig. 1. Frequency dist
the time of rupture
(shaded) and control

delivery was 390·9
tomy group and 44
trol group (t_{95df} =
analysis of labour d
group of women wh
mean duration of la
labour for these wo
ment group in Tabl
domization to full
significantly betwee

The greater mean
at admission and
mean cervical dila
amniotomy group
longer labours in
between these varia
randomization to
delivered vaginall
multiple linear regre
sion coefficients that
able were obtained
the last one in the
treatment policy of
not a statistically s
duration of the inter

To better describ
treatment groups w

Table 3. Duration of la

Onset-delivery (min)
Onset-admission (min)
Admission-randomizat
Randomization to full
Second stage (min)

Table 2. Maternal demographic data for study patients: amniotomy and control groups

	Amniotomy (n = 47)		Control (n = 50)	
	Mean	(SE)	Mean	(SE)
Years of education	13·3	(0·3)	14·3	(0·4)
Prepregnancy weight (kg)	61·1	(1·75)	57·3	(7·1)
Admission weight (kg)	76·3	(1·7)	71·8	(1·0)
Height (cm)	164·8	(0·9)	165·3	(0·9)
Birthweight (g)	3493·1	(73·0)	3322·1	(64·0)
Gestational age (days)	281·1	(0·8)	279·5	(1·0)
Cervical dilatation first exam (cm)	2·4	(0·02)	2·8	(0·02)
Onset of labour to randomization (min)	399·0	(31·0)	331·0	(28·0)

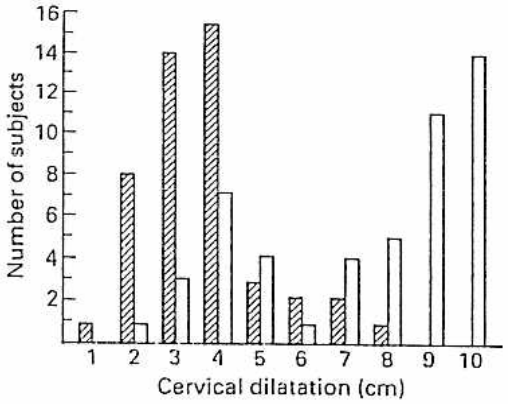

Fig. 1. Frequency distribution of cervical dilatation at the time of rupture of membranes in amniotomy (shaded) and control (unshaded) groups.

delivery was 390·9 (SE 29·1) min in the amniotomy group and 442·9 (SE 34·1) min in the control group ($t_{95df} = 1·15$, $P = 0·251$). A further analysis of labour duration was based on a subgroup of women who had vaginal deliveries. The mean duration of labour and the sub-intervals of labour for these women are displayed by treatment group in Table 3. The interval from randomization to full dilatation did not differ significantly between the two groups.

The greater mean values for maternal weight at admission and birthweight and the lower mean cervical dilatation at admission in the amniotomy group could have biased toward longer labours in that group. The relations between these variables and the interval from randomization to delivery in the women delivered vaginally were investigated by multiple linear regression (Table 4). The regression coefficients that are expressed for each variable were obtained by entering that variable as the last one in the regression equation. The treatment policy of membrane management was not a statistically significant predictor of the duration of the interval.

To better describe the behaviour of the two treatment groups with respect to the interval from randomization to delivery, survival curves were created. For the purposes of this analysis, the critical event was defined as vaginal delivery. Women delivered by caesarean section were considered as censored following the procedure. Cox's proportional hazards model was applied to the data to control for differences in co-variates that could influence labour duration. The variables included in the model along with their regression coefficients are shown in Table 5. A positive coefficient indicates an increase in the hazard function and an inverse relation with survival (continuation in labour). The graphic representations of the adjusted estimates of the survival functions are shown in Fig 2. Trial status (amniotomy versus conservation of the membranes) was not a statistically significant predictor of survival function, adjusting for birthweight, cervical dilatation at admission and maternal admission weight.

With respect to maternal post-partum morbidity, no women required blood transfusion. One woman in the amniotomy group developed a wound cellulitis after caesarean section and was treated with oral antibiotics. No woman met the study criteria for febrile morbidity (two episodes of fever > 38° C on two occasions, excluding the first 24 h after delivery).

Fifty five women in the amniotomy group and 47 women in the control group had post-randomization fetal heart tracings. The mean duration of electronic fetal monitoring was similar in the two treatment groups (amniotomy: 7·73 SE 0·47 h; control: 8·57, SE 0·48 h). Concerning the dichotomous fetal heart rate tracing outcome, the mean proportion of hours during which the tracings were 'abnormal' was similar in the two groups (post-randomization: amniotomy: mean 0·41, SE 0·05; control mean 0.44, SE 0.04; $t = 0·50$, $P = 0·620$).

The two treatment groups were similar with respect to the frequency of early, mild variable, severe variable and late decelaration during the pre-randomization period. The mean hourly

Table 3. Duration of labour (minutes) in women delivered vaginally in the amniotomy and control groups

	Amniotomy ($n = 39$)		Control ($n = 46$)				
	Mean	(SE)	Mean	(SE)	t	df	P
Onset-delivery (min)	776	(52)	763	(48)	0·21	83	0·842
Onset-admission (min)	201	(26)	192	(31)	0·21	83	0·842
Admission randomization (min)	192	(29)	149	(16)	1·37	83	0·175
Randomization to full dilatation (min)	311	(20)	346	(30)	0·82	83	0·422
Second stage (min)	72	(9)	75	(6)	0·24	83	0·807

Table 4. Multiple linear regression examining several variables for effect on interval from randomization to delivery in the women delivered vaginally

Variable	Regression coefficient	*t*-ratio	*P*
Constant	264·7		
X1			
Birthweight	0·14	2·88	0·005*
X2			
Cervical dilatation at admission	−39·10	−1·71	0·091
X3			
Maternal admission weight	−2·64	−0·98	0·332
X4			
Trial status (0 = control, 1 = amniotomy)	−80·10	−1·67	0·099

Interval (randomization to delivery) = 264·7 + 0·14(X1) − 39·1 (X2) − 2·64 (X3) − 80·10 (X4)

*Significant at the alpha = 0·01 level

rates of the four types of deceleration during the post-randomization period are shown in Table 6. There were no statistically significant differences between the two groups in the rates of deceleration after randomization.

The results of the variables which are indicators for condition at birth are summarized in Table 7. The proportions of babies with a 1-min Apgar score of < 6 and a 5-min Apgar score of < 8 did not differ significantly between the two groups. Umbilical artery blood pH was measured in 42 of the 47 babies born in the amniotomy group and in 47 of the 50 babies born in the control group. No statistically significant difference was found between the two groups in the proportion of infants with pH < 7·20. The mean cord artery pH was 7·254 (SE 0·07) in the amniotomy group and 7·256 (SE 0·08) in the control group. Six infants in each group were provided with assisted ventilation at birth. Neonatal cephalohaematoma was considered present if the diagnosis was recorded in the baby's chart by the treating physician. This occurred in one infant in the control group who was born by mid-forceps after a prolonged second stage. There was no abnormality noted on skull x-ray. This infant was cared for in the normal nursery and was discharged from hospital without further complications. All babies left hospital alive and in good condition.

Discussion

Several controlled studies which assess the effect of amniotomy on labour duration have been reported. Schwarcz & Caldeyro-Barcia (1982) reported a multi-centre study of 1124 nulliparous and multiparous women. The mean duration of the active phase of labour (5 cm to full dilatation) was 165 min in women who had an amniotomy compared with 213 min in a control group ($P < 0·001$, effect size = 0·77). This study suffers from methodological problems as recently reviewed by Keirse (1989). A smaller randomized trial by Stewart *et al.* (1982) found mean labour duration (admission to full dilatation) to be 4·9 h in the experimental group and 7·0 h in the control group ($P < 0·02$, effect

Table 5. Analysis using Cox's proportional hazards model to assess the effects of variables on survival function

Variable	Coefficient	Coefficient/SE	*P*
X1			
Birthweight	−0·001	−2·476	0·013*
X2			
Cervical dilatation at admission	0·118	1·158	0·2468
X3			
Maternal admission weight	0·012	1·005	0·3188
X4			
Trial status (0 = control,	0·231	0·960	0·337

* Significant at the alpha = 0·05 level.