

PULMONARY RESPONSES DURING COLD INDUCED ACUTE PAIN

O. P. TANDON*, A. HIMANI AND S. SINGH

*Department of Physiology,
University College of Medical Sciences & G.T.B. Hospital,
Dilshad Garden, Delhi - 110 095*

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Abstract : Cold induced acute pain is associated with many autonomic responses of the cardiovascular system, skin conductance and pupil size. However, there are few reports suggesting changes in pulmonary function. Hence present study reports preliminary data on this. Acute pain was induced in 30 non-smoker males, 30-50 yrs of age by immersing hand in cold water and their respiratory rate (RR), tidal volume (TV), inspiratory and expiratory reserve volumes (IRV, ERV) and respective capacities, vital capacity (VC), force vital capacity (FVC), FEV₁%, peak flow and flow rates at 75%, 50% and 25% of expired volume (V_{75,50,25}) were measured. Acute pain parameters like pain threshold, tolerance and sensitivity were also recorded. Besides these, heart rate, blood pressure (systolic and diastolic), skin temperature of forehead and opposite palm were also measured. Comparisons were made between values recorded before, during and after cold induced pain. An increase in RR, RV, IC, EC, FVC, FVC%, FEV₁ was observed during cold induced pain reflecting an acute state of sympathetic dominance. Positive correlations between heart rate and respiratory rate, and other respiratory with pain parameters were seen during period of induction of acute pain. Hence the study indicates that alterations in pulmonary profile form a part of multidimensional responses observed during cold induced acute pain.

Key words : pulmonary responses
cold

acute pain
pain parameters

INTRODUCTION

Autonomic alterations form one of the obligatory components of the complex multidimensional pain response. Evidence for the possibility of differential temporal relationships between stages of pain processing and physiological correlates stems from the research on cortical evoked potentials to painful laser stimulation (1, 2). These findings suggest that the cortical response nociceptive stimuli consists of a direct (immediate) and on indirect (delayed) component. The former seems to reflect the immediate sensory and perceptual processing, while the latter correlates with affective and/or cognitive evaluation of the pain experience. Hence the autonomic responses to pain form part of the affective component of pain behaviour.

Acute pain can be associated with increased heart rate, blood pressure, skin conductance and pupil size (3, 4, 5). However, there are only few reports suggesting changes in pulmonary functions during pain induced autonomic responses. It is also not known as to which components of pulmonary response show specific changes secondary or concomitant to these autonomic alterations. Hence the present study was undertaken to answer some of these questions.

METHODS

Thirty non-smoker males of the age group 30-50 years were subjects for the study. They were free from any cardiorespiratory disease, allergic disorders, and other addictions. Acute

*Corresponding Author

pain was induced in these subjects by the standard cold pressor test. Experiments were conducted in the sound proof, airconditioned Cardio Respiratory Laboratory of the Department of Physiology, U.C.M.S. & G.T.B. Hospital.

All the subjects were tested upon between 9 a.m. to 12 noon in order to rule out any alterations imposed by diurnal variations in cardiorespiratory parameters. Relevant clinical history was taken and general physical examination was done. The subject was seated in a chair. SHARN skin temperature strips were applied to the forehead and inbetween the horizontal palmar creases of the dominant hand after cleaning the skin with plain water and drying it. The E.C.G. lead II electrodes were applied to record the heart rate. Sphygmomanometer cuff was applied on the right arm. The subject was made to dip his non-dominant hand upto the first proximal wrist crease in a beaker filled with tap water at room temperature. After a period of 10-15 minutes the baseline heart rate, blood pressure, forehead and opposite palm temperature was measured. Base line Forced Vital Capacity (FVC), FVC%, Forced Expiratory Volume in first second (FEV₁), FEV₁%, Flow Rates at 75%, 50% and 25% of expired volume (V₇₅, V₅₀, V₂₅) and Peak Flow (PF) were measured on the Autospiror. Spirometry was performed on Transfer Test Machine (PK Morgan, U.K.). Respiratory Rate (RR), Tidal Volume (TV), Inspiratory and Expiratory Reserve Volume and the respective capacities (IRV, ERV, IC and EC) and the vital capacity (VC) were recorded. After the baseline record the subject was informed about the cold pressor test i.e. he will be made to immerse his non-dominant hand in ice cold water at $4 \pm 1^\circ\text{C}$, contained in a beaker which was kept in a thermocol case surrounded by crushed ice.

The subject was asked to inform when he started feeling the pain and take out his hand when the pain became intolerable. The subject

dipped his non-dominant hand in this beaker. As soon as he immersed his hand 2 stop watches were simultaneously started. As soon as the subject said that he had started feeling the pain one stop watch was stopped. The respiratory rate was recorded for 15-30 secs on the Morgan's Pulmotest machine. During this period heart rate, blood pressure and skin temperature were also recorded, and the subject was made to perform the vital capacity maneuver i.e. taking a deep breath and exhaling it forcefully with maximum effort. As soon as the pain became intolerable and subject took his hand out, the second stop watch was stopped. The subject's hand was wrapped in a towel and when he stopped feeling the pain he was again made to immerse his hand in the beaker containing tap water at room temperature. All the parameters were recorded again after a gap of 5-10 minutes. The subject was called again after a week and the cold pressor test was performed on him again and this time FVC, FVC%, FEV₁, FEV₁%, V₇₅, V₅₀, V₂₅ and PF were recorded, as soon as the patient started feeling the pain during the cold pressor test.

The time shown by the first stop watch gave the pain threshold i.e. when the subject just started feeling the pain, the time shown by the second stop watch gave pain tolerance i.e. the time for which the subject was able to tolerate the pain. Their difference gave the pain sensitivity. Pain threshold and pain tolerance were recorded both during spirometry and Autospiror measurements separately.

As done previously in the case of spirometry, Autospiror value were recorded again 5-10 minutes after the cold pressor test.

RESULTS

The mean age of subjects was 35.73 ± 4.61 years. The mean height and weight were 163.25 ± 7.16 cms. and 58.91 ± 10.93 kgs respectively. The pain thresholds in the subjects ranged from

2.6 to 163.1 secs, pain tolerance from 15.4 to 360.5 secs and pain sensitivity from 11.5 to 197.4 secs during the two cold pressor test sessions.

Analysis of variance (ANOVA) with Tukey Test at 5% level of significance was employed for comparing parameters before, during and after cold induced pain.

The basal heart rate was recorded on the ECG machine for 30 secs and was calculated with the help of RR interval. The heart rate recorded during cold induced acute pain was significantly higher (P value < 0.001) from both before and after cold pressor test. The systolic and diastolic blood pressures were significantly higher during the cold pressor test as compared to before and after the cold pressor test (P value <0.001) (Table I).

Thus, the cardiovascular parameters showed a net increase during the cold induced acute

pain as compared to those before and after the state of acute pain.

The mean respiratory rate recorded during acute pain was significantly higher (P value <0.001) than that recorded before and 5-10 minutes after cold pressor test. Tidal volume showed a significant increase from baseline during cold induced acute pain (P value <0.001). The mean tidal volume after cold pressor test was significantly higher than that recorded before acute pain (P value <0.001) and significantly lower than that recorded, during acute pain (P value < 0.001). The inspiratory and expiratory capacities and vital capacity showed a similar trend as that of respiratory rate and tidal volume during cold induced acute pain. The mean inspiratory reserve volume and expiratory reserve volume for the 3 situations were not significantly different from each other (Table II). The mean FVC and FVC% were

TABLE I: Cardiovascular parameters before, during and after cold induced acute pain.

Parameter	Before acute pain (Mean±SD)	During acute pain (Mean±SD)	After acute pain (Mean±SD)	P value
Pulse(min)	75.77 ± 11.08	85.64 ± 13.3	75.60 ± 11.4	0.000
BP Syst. (mm of Hg)	115.03 ± 9.14	130.93 ± 17.52	115.2 ± 10.94	0.000
BP Diast. (mm of Hg)	79.60 ± 7.61	91.07 ± 10.82	80.53 ± 10.18	0.000

Significant P-value <0.05

TABLE II: Respiratory parameters before, during and after cold induced acute pain.

Parameter	Before acute pain (Mean±SD)	During acute pain (Mean±SD)	After acute pain (Mean±SD)	P value
Respiratory rate	18.35 ± 4.51	21.73 ± 4.5	18.27 ± 4.73	0.000
Tidal Volume (ml)	773.75 ± 203.88	967.5 ± 256.48	851.67 ± 230.15*	0.000
IRV (ml)	1273.08 ± 361.5	1216.17 ± 372.38	1247.5 ± 396.5	0.194
IC (ml)	2020.17 ± 451.22	2183.67 ± 448.48	2097.5 ± 487.97	0.000
ERV (ml)	834.42 ± 342.45	797.17 ± 296.07	796.67 ± 341.13	0.598
EC (ml)	1608.17 ± 391.23	1764.67 ± 333.83	1648.33 ± 376.12	0.000
VC (ml)	2881.25 ± 522.21	2980.83 ± 491.64	2895.83 ± 536.111	0.005

Significant P-value <0.05

*Tidal volume after acute pain is significantly different from before and during acute pain.

significantly higher during cold pressor test as compared to baseline. The mean FVC and FVC% recorded after acute pain were also significantly higher than the baseline but not different from

temperature of opposite palm increased to $32.18 \pm 2.55^\circ\text{C}$ but it was still lower than that recorded before cold pressor test.

TABLE III: Respiratory parameters before, during and after cold induced acute pain.

Parameter	Before acute pain (Mean \pm SD)	During acute pain (Mean \pm SD)	After acute pain (Mean \pm SD)	P value
Force Vital Capacity (ml)	2809 \pm 467.95*	2991.5 \pm 473.55	2925.67 \pm 531.22	0.000
FVC%	73.05 \pm 12.04*	77.65 \pm 12.14	75.9 \pm 13.58	0.000
FEV ₁ (ml)	2524.50 \pm 480.38*	2593 \pm 467.53	2602 \pm 515.76	0.014
FEV ₁ %	89.84 \pm 8.06	86.86 \pm 9.21	88.93 \pm 7.14	0.003
V ₇₅ (Lit/sec)	5.64 \pm 1.28	5.48 \pm 1.43	5.47 \pm 1.36	0.328
V ₅₀ (Lit/sec)	3.66 \pm 1.08	3.68 \pm 1.23	3.67 \pm 1.15	0.928
V ₂₅ (Lit/sec)	1.85 \pm 0.812	1.79 \pm 1.02	1.70 \pm 0.73	0.278
Peak Flow (Lit/sec)	6.36 \pm 1.11	6.31 \pm 1.16	6.32 \pm 1.32	0.868

Significant p-value <0.05

*Value before acute pain is significantly different from during and after acute pain.

that during acute pain. The mean basal forced expiratory volume in first second was significantly lower than the mean during and after cold pressor test. The FEV₁% was significantly lower during acute pain than before and after acute pain (Table III).

The various flow rates that is PF, and those at 75%, 50%, 25% of volume, i.e. V₇₅, V₅₀ and V₂₅ before, during and after cold pressor test showed no significant difference amongst each other.

The mean skin temperature of forehead and opposite palm before the CPT was $35.35 \pm 0.85^\circ\text{C}$ and $32.54 \pm 2.23^\circ\text{C}$ respectively. The forehead skin temperature showed a significant increase to $35.58 \pm 0.93^\circ\text{C}$ (P value <0.05) and that of opposite palm showed a decrease to $32.05 \pm 2.42^\circ\text{C}$ during cold induced acute pain (P value <0.001). Though after cold pressor test the

DISCUSSION

Pain is a complex phenomenon which has a sensory discriminatory, motivational-affective and cognitive components (6). Cardiorespiratory neural control mechanisms form the efferent component of affective-motivational-behavioural dimension of the pain response. In fact the Spino (Trigemino) Ponto Amygdaloid system has been proposed to be involved in emotional-affective and autonomic (pupil dilatation, cardiorespiratory and adrenocortical responses) reactions to noxious events. Besides these Periaqueductal gray, nucleus Raphae Magnus, nucleus Tractus Solitarii, locus Coeruleus and A₅ cell groups which were primarily believed to be involved in the regulation of cardiovascular and autonomic functions, are now being documented to be having a role in modulation of spinal nociceptive transmission also. Some of

these are known to be monoaminergic in nature (7, 8).

Our results of increase in various cardiorespiratory parameters during cold induced acute pain support the notion of integrative function of these various neural components mentioned above. The cardiovascular responses observed in the present work i.e., an increase in the heart rate, systolic and diastolic blood pressures during cold induced acute pain are in line with those observed by Wolf and Hardy in 1941 (3) and form part of the classical cold pressor test response (9). During the cold induced acute pain, both rate and depth of respiration increased. The increase in rate and tidal volume indicates that cold induced pain has a stimulatory effect on ventilation.

There are two factors which could be responsible for this stimulatory effect on ventilation. One is the sensation of pain itself and the other could be due to the sensation of cold. The latter component was excluded, because in our experimental design we did not start recording the respiratory parameters, as soon as the subject immersed his hand in ice cold water and the recording was made only when the subject started getting pain. There are studies showing that blockade of pain during CPT did not produce the pressor response indicating that it is the sensation of pain which is responsible for the type of cardiorespiratory response observed.

Pain may cause either respiratory stimulation or inhibition, depending on its character, origin (visceral or somatic) and intensity. Nociceptive afferents can reflexly stimulate the respiratory centres. Behavioural responses of conscious animals confronted with stressful situations can be in the form of a "defence reaction" or a "playing dead" reaction.

In man usually the defence reaction is the main one. Tachycardia, increased force of heart beat, hypertension, venoconstriction and skeletal muscle vasodilatation are the preparatory responses for fight or flight. Since pain is also a form of stress, similar responses are very likely to be seen in individuals during a state of pain. This stimulation of respiration could be secondary to the excitatory inputs of the higher centres to cardiorespiratory centres in the lower brainstem.

The tidal volume showed an increase during acute pain but it was not more than 50% of the vital capacity recorded during acute pain. It is a known fact that in intact humans, breathing rate usually increases when tidal volume increases, to about half the vital capacity.

The forced vital capacity and the vital capacity showed significant increase during cold induced acute pain. The forced vital capacity expressed as a percentage of the predicted value also increased. This could be due to a better muscular and voluntary effort by the subject, coming into picture as a general arousal response to pain during cold stress. Another possibility could be that a change in the bronchomotor tone i.e., relaxation and dilatation of large airways. Interestingly FEV₁ is not effort dependent as FVC and VC and is affected by the calibre of large airways, this again points towards the possibility of bronchodilatation in response to cold induced acute pain. The FEV₁% showed a significant decrease which points towards the fact that FEV₁ did not increase to the extent to which FVC increased. The decrease in FEV₁% inspite of an increased FEV₁ indicates that the effect of increase in FVC which accompanied bronchodilatation nullified that due to an increase in airway calibre.

Thus, it can be hypothesized that bronchodilatation is occurring during cold

induced acute pain. The reason for this bronchodilatation could be due to the following: It could be in response to one of the components of Herring Breuer inflation reflex which brings about a relaxation of tracheobronchial tree smooth muscle (10). Possibly this may modulate airway calibre. The reason for bronchodilatation could be due to sympathoadrenal discharge leading on to an increase in the circulating catecholamine levels during the cold pressor test, which is a well documented fact (11, 12, 13). The increase in circulating Epinephrine which acts on the β -adrenoceptors in the airway may be responsible for bronchodilatation and the type of responses which we got during cold induced acute pain in our study. Another possibility could be that during cold induced acute pain increased sympathetic discharge to the aortic and carotid chemoreceptors may improve their sensitivity and augment their response. Increased circulating catecholamines may further increase their firing by directly acting on the chemoreceptors.

The expiratory and inspiratory capacities also showed a significant increase during cold

induced acute pain. Since inspiratory and expiratory reserve volumes did not show any change increase in these capacities is secondary to the increase in the tidal volume.

These findings indicate to a large extent that stimulation of respiration should form an integral part of cold induced pain. The respiratory rate showed a positive correlation with heart rate during acute pain and pain sensitivity with tidal volume (Fig.1, 2). This shows that cardiovascular and respiratory responses go hand in hand in response to stimulation of higher centres to prepare the individual optimally for a stressful situation like pain.

We also observed an increase in the skin temperature of forehead during cold induced acute pain. This has also been documented by Hampf in 1989 (14). The decrease in the skin temperature of the opposite palm observed in our study has also been documented before from our own laboratory (15). These findings show that alteration in blood flow to different skin areas do occur during cold induced pain and

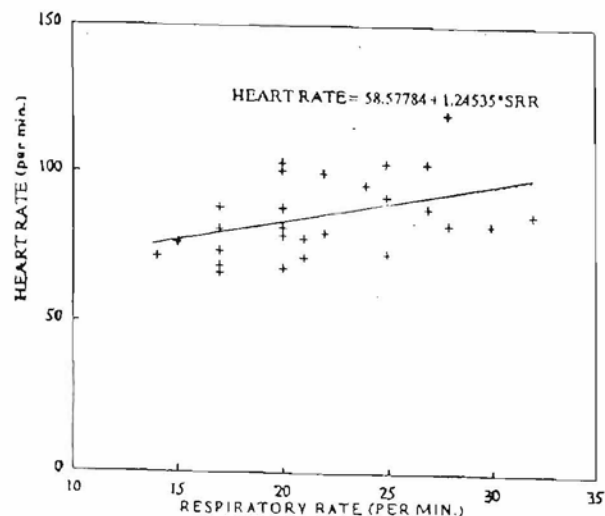


Fig. 1: Correlation between respiratory rate and heart rate during cold pain. Coeff. of correlation, $r = 0.4215$

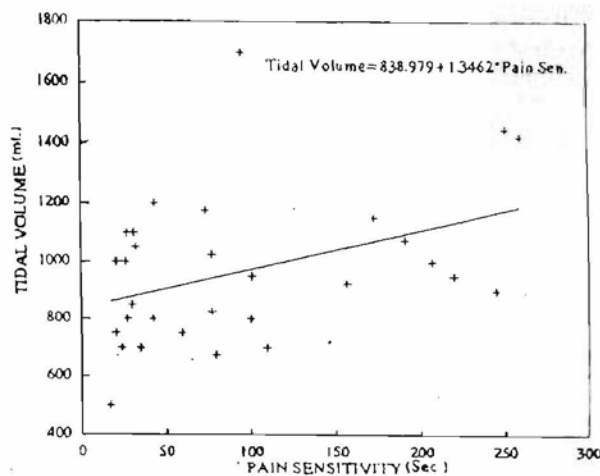


Fig. 2: Correlation between pain sensitivity and tidal volume during cold pain. Coeff. of correlation $r = 0.4215$

these alterations in the regional blood flow are not uniform throughout the body.

Hence, our findings point towards autonomic adjustments suggesting more

of sympathetic overactivity during cold induced acute pain. Further, studies are required to work out the exact level of integration of these pulmonary responses during acute pain.

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