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Effect of Intercostal Nerve Blockade on Respiratory Mechanics and CO_2 Chemosensitivity at Rest and Exercise

Bernice R. Hecker, M.D., F.A.C.C., Robert Bjurstrom, M.D.,† Robert B. Schoene, M.D.‡*

Lower intercostal and abdominal muscles interact with other respiratory muscles to produce inspiration as well as expiration. Intercostal nerve blockade from T_6–T_{12} was produced in seven healthy males to study its effect on: 1) supine pulmonary function, 2) inspiratory effort, 3) hypercapnic ventilatory response, including mouth occlusion pressures with and without an expiratory load, and 4) ventilation during progressive exercise on a cycle ergometer. Studies during control and blocked states were performed on different days. Lower chest and abdominal wall paralysis was documented with electromyography. Findings include a minimal decrease in peak expiratory flows with intercostal blockade ($P = 0.02$), but no other changes in supine resting pulmonary function tests, inspiratory effort, or hypercapnic ventilatory response slopes. Minute ventilation, respiratory rate, and V_T/T_I during exercise were also minimally increased, indicating an increase in the drive to breathe, which was unrelated to a change in metabolic rate. During exercise, total time to exhaustion was decreased following intercostal nerve blockade. Bilateral intercostal nerve blockade produced minimal decreases in peak expiratory flow at rest in supine subjects. During seated exercise, there was a slight increase in respiratory drive, probably due to minor alterations in the mechanics of breathing induced by intercostal blockade. The authors conclude that, in healthy young subjects, intercostal nerve blockade does not exert a clinically significant adverse affect on pulmonary mechanics and that ventilatory function is well-maintained even at extremes of ventilatory demand. (Key words: Anesthetic techniques, intercostal block: chemosensitivity; exercise; ventilatory drive.)

INTERCOSTAL NERVE BLOCKADE (ICB) producing deafferentation and lower chest and abdominal wall muscle paralysis is used in clinical practice for acute and chronic pain control and for anesthesia and motor blockade during surgery.[1–3] Despite the frequent use of these blocks, the isolated effects of ICB on lung mechanics, ventilatory drive, and the ability to increase ventilation has not been rigorously studied. In this study, we attempted to define further the effects of ICB, on subjects at rest in the supine position, on pulmonary function tests and hypercapnic ventilatory drive with and without expiratory resistive loading, and with the subjects in the seated position, on respiratory mechanics at the extremes of ventilation produced by exercise.

* Staff Anesthesiologist, Virginia Mason Clinic.

† Senior Research Fellow, University of Washington.

‡ Associate Professor of Medicine, University of Washington.

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Address reprint requests to Dr. Hecker: Department of Anesthesiology, Virginia Mason Medical Center, P.O. Box 900, 1100 Ninth Avenue, Seattle, Washington 98111.

Materials and Methods

Seven healthy adult males aged 25–38 yr were studied after giving informed consent as approved by the University of Washington Human Subjects Review Committee. Subjects were blinded as to anticipated results. All subjects were non-smokers with no history of cardiopulmonary disease or recent medication intake. Each subject was studied on two separate experimental days with and without bilateral ICB at levels T_{6-12}. Half of the subjects were studied in the control state prior to the blocked state and vice versa. Subjects fasted for 8 h prior to each session. Intercostal blocks were performed as previously described by Moore and Bridenbaugh[2] using 1% lidocaine and 1:250,000 epinephrine. Serum lidocaine levels were assayed by gas chromatography using a Hewlett-Packard® 5750 with a flame ionization detector immediately post-block and every 15 min thereafter until the end of the experimental session. Lower intercostal and abdominal muscle neural blockade was evaluated throughout the experiment by checking the response to pin-prick and ability to perform both sit-up and head-lift.[4] Absence of significant muscle activity was further documented by integrated electromyography, (TD 10, TECA Corp., Pleasantville, NY). Skin electrodes were placed across the abdomen vertically and horizontally at T_6, T_8, T_{10}, and T_{12} dermatomes.[5,6]

Pulmonary function tests were performed with subjects in the supine position. These included measurement of lung volumes with functional residual capacity (FRC) determined by helium dilution, spirometry by volume displacement, flow-volume loops from total lung capacity (TLC), and maximal voluntary ventilation (MVV). All tests were performed on a Cybermedic® 310 system (Boulder, CO). Maximal negative inspiratory pressures (−IE, cm H_2O) were measured using a hand-held Wright's manometer. Excessive use of buccinator muscles was inhibited by manual compression with flattening of the cheeks. Following completion of pulmonary function tests with mouthpiece and nose clips in place, stable resting ventilation ($\dot{V}_E$, $1 \cdot min^{-1}$) was documented using constant end-tidal P_{CO_2} (PET_{CO_2}) as a determinant of resting $\dot{V}_E$.

TABLE 1. Pulmonary Function Tests

	Control*	Block*	P
TLC (l)	7.13 ± 0.49	7.07 ± 0.81	N.S.
FRC (l)	2.42 ± 0.24	2.51 ± 0.51	N.S.
RV (l)	1.39 ± 0.16	1.72 ± 0.52	N.S.
ERV (l)	1.05 ± 0.20	0.78 ± 0.21	N.S.
FVC (l)	5.53 ± 0.58	5.18 ± 0.68	N.S.
FEV_1 (l/sec)	4.40 ± 0.32	4.29 ± 0.45	N.S.
MVV (l/min)	197 ± 17.4	199 ± 26.3	N.S.
PEF (l/sec)	11.23 ± 1.8	9.25 ± 2.5	<0.02
FEF_{25-75} (l/sec)	4.37 ± 1.4	4.80 ± 1.4	N.S.
IE (cm H_2O)	−99.4 ± 11.8	−100.8 ± 15.9	N.S.

* Mean ± S.D.
N.S. = no significant difference between control and blocked states.

The modified rebreathing technique of Read with a 7% CO_2, 40% O_2, and balance nitrogen mixture was used to measure hypercapnic ventilatory response.[7] During ventilatory drive testing mouth occlusion pressures ($P_{0.1}$, cm H_2O) were measured approximately every 20 s with inaudible closure of the inspiratory portion of the airway by a pneumatically driven solenoid shutter. Mouth occlusion pressure was measured as the pressure generated at 100 msec, which was electronically timed, from initial inspiration. Output was recorded on an oscilloscope calibrated prior to each study with a water manometer. Each subject underwent two hypercapnic ventilatory response tests with $\dot{V}_E$ reaching approximately 30–35 $l \cdot min^{-1}$. The slope ($\dot{V}_E/PET_{CO_2}$) was determined after estimated alveolar equilibration with mixed venous blood. The first test was performed in standard fashion followed by a rest period during which stable resting minute ventilation was re-established.[7] The second test was performed with the addition of an expiratory resistive load, 6-0 endotracheal tube in the expiratory limb of the system, ≥15 cm $H_2O \cdot l^{-1} \cdot s^{-1}$.[8]

Following a 10-min period of rest, the subject sat upright on a cycle ergometer with an electrocardiographic monitor, nose clips, and mouthpiece in place. After exercising for 3–5 min to allow CO_2 to equilibrate, the subject blinded to exercise state and time, performed a progressive exercise protocol to exhaustion. Exercise increments were 400 $kpm \cdot min^{-1}$ every 2 min through 1600 kpm/min, then 200 $kpm \cdot min^{-1}$ each 2 min to exhaustion. Respiratory rate, end-tidal gas concentrations, $\dot{V}_E$, tidal-volume (V_T), inspiratory flow (V_T/T_I), duty cycle (T_I/T_{Tot}), CO_2 production, and O_2 consumption were collected on a breath-by-breath basis throughout each exercise interval.

Exercise and ventilatory drive data were obtained on a Medical Graphics® System 2000 incorporating computer programs to measure metabolic variables as well as ventilatory drives. Within this system, $\dot{V}_E$ and V_Ts were measured by a Rudolph pneumotachometer that was calibrated with a 3-l syringe. End-tidal oxygen and carbon dioxide concentrations were monitored at the mouth by a gas sampling line which lead to an infrared CO_2 analyzer and an electrochemical cell O_2 analyzer. Both analyzers were calibrated before each study with a gas of known O_2 and CO_2 concentrations. A waveform analyzer received analog information from the flow module, gas analyzers, and the heart rate monitor, and transferred the data to a Tektronix® 4052 computer system for digital and graphical display, tape storage, and computation.

DATA ANALYSIS

Pulmonary function data were compared in the control and blocked state using absolute values. Hypercapnic ventilatory response was analyzed from the equation $\dot{V}_E = S(PA_{CO_2} - B)$ with S being the slope of the regression line of $\dot{V}_E$ plotted against PET_{CO_2} and B = x-axis intercept.[7] Pulmonary function and ventilatory response data were analyzed using paired *t* tests. Exercise data were averaged over the last half of each exercise interval for comparison and analyzed using two-way analysis of variance for repeated measures with Tukey's *post hoc* test (SPSS Inc., Marija J. Norusis, Chicago, IL). Significance was defined as $P \leq 0.05$. All data are shown as means ± standard deviations.

Results

NERVE BLOCKADE

Bilateral ICB was successfully achieved in all subjects as evidenced by iso-electric electromyographic traces and clinical examination of subjects. The absence of hypesthesia was present in only two subjects at two dermatome levels each, T_{12} bilaterally and T_{10} (R) plus T_{11} (L), respectively. These subjects were, nonetheless, unable to raise their shoulders off the stretcher or sit up, and no significant abdominal muscle tension could be ascertained on direct physical examination. At the completion of the study protocol, all subjects noted mild difficulty maintaining posture while erect, and no subject could sit up from the supine position. However, perception of pinprick had returned in the upper and lower-most dermatomes in four of the subjects.

SUPINE PULMONARY FUNCTION

No significant differences between the control and blocked state were found in forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), the ratio of forced expiratory volume to forced vital capacity (FEV_1/FVC), slow vital capacity (SVC), FRC, residual volume (RV), TLC, IE, MVV, or flow-volume loops (table 1). A statistically significant ($P < 0.01$) decrease in peak

TABLE 2. Effect of ICB on Ventilatory Response to CO_2 (Mean ± SEM)

	Control	Block	
V_E/P_{CO_2}	2.22 ± .28	1.92 ± .20	N.S.
$P_{.1}/P_{CO_2}$	.60 ± .09	.47 ± .06	N.S.
	Resistive	**Resistive**	
V_E/P_{CO_2}	1.97 ± .20	2.21 ± .29	N.S.
$P_{.1}/P_{CO_2}$	.72 ± .14	.55 ± .11	N.S.

ICB = intercostal blockade; N.S. = no significant difference between control and blocked state.

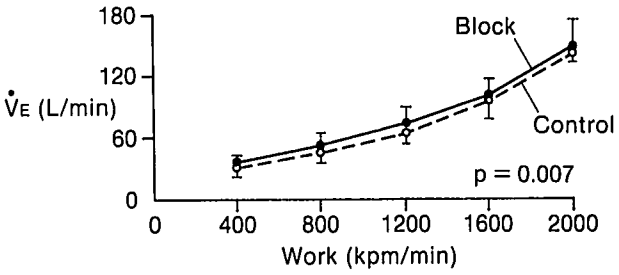

FIG. 1. Ventilation ($1 \cdot min^{-1}$) is increased at each work stage in the blocked state. Circles represent means ± SD; open circles, control group; closed circles, blocked group.

expiratory flow (PEF) rates in the blocked state was found. The flow-volume loops were otherwise unaffected by ICB.

CARBON DIOXIDE CHEMOSENSITIVITY

No significant difference in either slope ($\dot{V}_E/PET_{CO_2}$ and $P_{0.1}/PET_{CO_2}$) or x-intercept was found between groups in ventilatory response lines with or without a respiratory resistive load (table 2).

EXERCISE

All subjects exercised to exhaustion. The lowest maximal workload that any subject completed was 1600 $kpm \cdot min^{-1}$, 480 s. Total exercise time was decreased in every subject after intercostal blockade (control = 813.0 ± 152.4, blocked = 729.6 ± 138.5 s, mean ± SD; $P < 0.0001$). Exercise ventilation was significantly increased at comparable levels of work in the blocked state (fig. 1) characterized by an increased frequency response (fig. 2). Inspiratory times, expiratory times, and total respiratory cycle were all shortened in the blocked state. Tidal volume did not differ between groups. Throughout exercise, there was an increase in inspiratory flow (V_T/T_I) in the blocked state (fig. 3). The duty cycle (T_I/T_{Tot}) did not differ between groups.

SERUM LIDOCAINE CONCENTRATIONS

Serum lidocaine concentrations ranged from 1.09 to 2.79 $\mu g \cdot ml^{-1}$ (table 3). Highest serum levels were obtained 15 and 30 min after blockade, 2.03 and 1.97 $\mu g \cdot ml^{-1}$, respectively; lowest serum levels were obtained immediately after ICBs were completed and 90 min later, 1.37 and 1.28 $\mu g \cdot ml^{-1}$, respectively.

Discussion

The results of this study in healthy young men show that, in the supine position, which was chosen to mimic clinical conditions, muscle paralysis induced by intercostal nerve blockade causes a minimal reduction in peak expiratory flow rates, but otherwise does not affect static or dynamic indices of pulmonary function at rest. Similarly, hypercapnic ventilatory response with or without expiratory resistive loading was also unaffected. However, during exercise, producing higher levels of ventilation, $\dot{V}_E$, respiratory rate, and V_T/T_I were increased at each work load; and exhaustion occurred at lower workloads following bilateral ICB.

PULMONARY FUNCTION TESTS

During restful breathing, ventilation is normally primarily dependent upon the diaphragm.[9] However, other muscle groups also function as primary muscles of inspiration. Tonic/phasic activity of scalene, intercostal, and abdominal muscles occurs in the erect, sitting, and supine positions and expands the rib cage and lung.[10–14] As ventilation increases, there is increased activation of these muscle groups. In addition, the activity and interaction of the intercostal and abdominal muscles reduce the load on the diaphragm, preserving in part the mechanically advantageous muscle fiber length and radius of curvature of the diaphragm so that optimal tension, and, hence, contraction, can occur.[9,13,15] Thus, the overall mechanical displacement of the respiratory system and chest wall configuration is significantly influenced by muscles of the

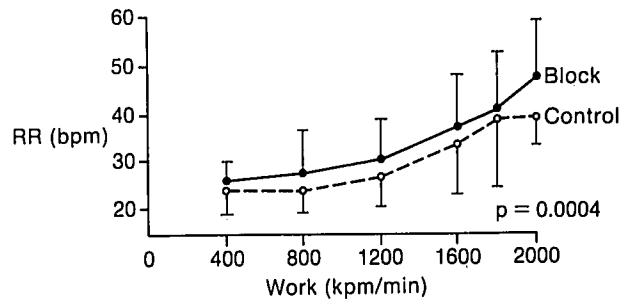

FIG. 2. Respiratory rate (breaths $\cdot min^{-1}$) during exercise is increased at each work stage in the blocked state. Circles represent means ± SD; open circles, control group; closed circles, blocked group.

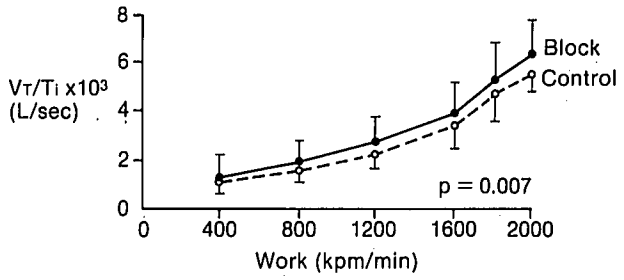

FIG. 3. Inspiratory flow, as represented by $V_T/T_I(1 \cdot s^{-1})$, is increased at each work stage in the blocked state during exercise. Circles represent means ± SD; open circles, control group; closed circles, blocked group.

chest wall other than the diaphragm. Indeed, abdominal muscle paralysis, induced by ICB, has been reported by Jakobson *et al*[16,17] to produce decreases in inspiratory capacity. In semi-recumbent volunteers, these authors observed small decreases in VC and FRC, but no change in lung recoil. However, in our subjects in the supine position, our findings of unaltered FVC, IE, MVV, and flow-volume loops indicate that inspiratory strength was unaffected by lower intercostal and abdominal muscle paralysis induced by ICB. In supine subjects partially paralyzed with neuromuscular relaxants (as opposed to sitting subjects[18]), Gal and Goldberg established that FVC and inspiratory capacity are similarly maintained.[19]

Functional residual capacity, ERV, and RV were also preserved after ICB. The constancy of FRC with consequent stability of the length-tension relationship of the diaphragm also validates both the accuracy and interpretability of $P_{0.1}$ measurements obtained in the blocked state.[20] Stability of VC and FRC also explains the absence of change in lung recoil directly established in Jakobson's work,[16] and is consistent with the observations of Freund and Bonica[21] Wahba and Craig,[22] and McCarthy.[23] These researchers found no significant changes in VC or FRC with motor blockade to the sixth dermatomal level induced by epidural anesthesia.

A statistically significant difference was found in PEF. However, clinically, a decrease of the degree observed would have a negligible effect on the ventilatory capacity of healthy subjects. This reduction is most probably accounted for by the inability of subjects to raise voluntarily intra-abdominal pressure to control levels.

TABLE 3. Serum Lidocaine Levels

Time (Min)	n	μg/ml (Mean)	μg/ml (Range)
0	5	1.37	1.12–1.49
15	6	2.03	1.47–2.79
30	6	1.97	1.60–2.84
45	7	1.76	1.45–1.91
60	7	1.60	1.29–1.81
75	5	1.49	1.09–1.70
90	4	1.28	1.14–1.62

Preservation of these lung volumes suggests that, in healthy subjects, gas exchange would be negligibly affected by the muscle paralysis affected by ICB. Also, atelectasis would not be expected to occur secondary to ICB alone. Retained secretions, alterations in respiratory pattern, and early airway closure secondary to a decrease in ERV relative to closing volume are, in ascending order of importance, contributing factors to atelectasis.[24] A decrease in ERV far exceeds the importance of other factors and may be, moreover, the only factor required for the development of atelectasis. With ICB in our subjects, respiratory pattern and mechanics, inspiratory capacity air flow, and maximal voluntary ventilation were essentially unchanged and lung volumes, ERV, and FRC were well maintained. However, since airway closure occurs earlier in expiration during general anesthesia and in patients with obesity or lung disease, atelectasis may be present after ICB performed with one or more of these conditions present, although not caused by ICB *per se*.

Preservation of inspiratory function, as well as static volumes at rest, may be partially explained by the supine positioning of our subjects where diaphragmatic length is optimized by the effect of gravity on the abdominal contents minimizing any impact of chest wall musculature on diaphragmatic function. (Measurement of transdiaphragmatic pressures or abdominal and chest wall configurations could have confirmed this hypothesis.) In addition, the activity of parasternal (uppermost intercostals) or scalene muscles is entirely unaffected because ICB from T_{6-12} does not produce paralysis of these inspiratory muscles and, in the supine position, abdominal muscle activity does not normally occur until ventilation exceeds 45 l/min.[25] Thus, decreases in inspiratory capacity secondary to paralysis of the lower intercostal and abdominal muscle groups would not be expected and were not seen.

Expiratory function is also preserved. That lung volumes, especially RV, were not increased in the blocked state suggests that supine subjects with intercostal nerve blockade may recruit other muscles during expiration, as well as during inspiration. De Troyer *et al.*[26] have recently demonstrated the importance of the triangularis sterni in expiration, activation of which appears to be neurally coupled to abdominal muscle activation. When development of abdominal muscle tension is inhibited, the triangularis sterni alone produce "a marked decrease in rib cage anterior/posterior dimension and an increase in pleural pressure."

Since complete paralysis of the muscles of the lower thoracic and abdominal walls can be attained with intercostal, spinal, or epidural blockade, we believe that the findings of our study may be reasonably extended to apply to any young healthy patient undergoing conduction

anesthesia with motor blockade to and including the sixth thoracic dermatome. Any differences in the intensity of block once the patient is clinically paralyzed would be irrelevant.

As opposed to regional anesthesia, it is difficult to extrapolate our data, obtained in healthy subjects, to patients with respiratory mechanical dysfunction. Nonetheless, one may speculate that, in patients with COPD whose ventilation is adversely affected both by out-flow obstruction and diaphragmatic mechanical disadvantage, ICB may further impair effective gas exchange and cough. The downward displacement of the diaphragm seen in patients with COPD reduces the effectiveness of diaphragmatic contraction and increases the dependence on the chest wall musculature to produce inspiration. Sharp *et al.*[27] have shown that patients with COPD may depend more on the rib-cage musculature than the diaphragm to produce tidal ventilation compared to healthy persons. Intercostal block with attendant abdominal muscle paralysis inhibits this compensatory activity, as well as the lever-fulcrum relationship of the diaphragm and abdominal viscera, and optimization of whatever diaphragmatic tension development and contractile force could otherwise be attained. The decrease in PEF observed in this study may also adversely affect the cough mechanism and impair clearance of secretions creating further out-flow obstruction. These effects may be additive or even potentiate existing dysfunction resulting in respiratory failure. Indeed, postoperative respiratory failure after ICB in a patient with COPD has been reported by Cory and Mulroy.[28] Further studies are necessary to elucidate this possibility.

Carbon Dioxide Chemosensitivity

Intercostal blockade did not alter hypercapnic ventilatory response to a $P_{ET_{CO_2}}$ of approximately 55 mmHg with $\dot{V}_E \geq 30$–$35\ l \cdot min^{-1}$. In the supine position, this level of ventilation may have been too low to have invoked lower intercostal and abdominal muscle recruitment. However, expiratory resistive loading that does produce recruitment at less than $20\ l \cdot min^{-1}$,[29] also failed to affect hypercapnic ventilatory response. Finding no divergence in the slopes for $P_{0.1}$ and $\dot{V}_E$ compared to $P_{ET_{CO_2}}$, we conclude that ventilatory and neuromuscular response was maintained in blocked subjects before and after expiratory resistive loading. These findings corroborate those of Lopata *et al.*[30] who demonstrated that expiratory loading either decreased or did not affect ventilatory and occlusion pressure response to CO_2 re-breathing. These results imply that an increased expiratory resistive load was not centrally perceived, and no significant impairment of lung mechanics occurred with ICB at rest.

Lack of effect upon hypercapnic ventilatory response supports our contention that these findings are unaffected by the local anesthetic agent. Lidocaine, in the serum concentrations measured in this study, has not been documented to affect hypercapnic ventilatory response significantly.[31] Although Gross *et al.* postulated that serum lidocaine levels obtained after ICB would be equal to those reached after intravenous bolus injection (approximately $8\ \mu g \cdot ml^{-1}$) and would affect CO_2 responsiveness when ICBs are done with epinephrine added to the local anesthetic, serum lidocaine concentrations are much lower, ranging in our study from 1.12 to $2.84\ \mu g \cdot ml^{-1}$. These levels are consistent with those previously reported for similar drugs[32] and well below the threshold that alters CO_2 chemosensitivity.

Exercise

We attempted to increase the demand on the respiratory system by stressing subjects with progressive exercise to exhaustion on the cycle ergometer. The maximum work level attained was the same in both control and blocked states, but blocked subjects were not able to exercise as long in the last stage, complaining of difficulty in breathing. The cause(s) of this lower endurance are not entirely clear. However, early fatigue of the respiratory muscles, either intercostals, diaphragm, or both, due to an increase in the work of breathing may explain the decrease in exercise time.

Ventilation during exercise appears to be altered minimally in the blocked state. An increase in minute ventilation ($\dot{V}_E$, $l \cdot min^{-1}$) was seen at each workload in the blocked state that was achieved by increases in respiratory rate without a change in V_T until higher levels of exercise when V_T actually decreased. Centrally perceived mechanical inefficiency may have produced or contributed to the alterations in respiratory pattern observed. An increase in respiratory drive during exercise in the blocked state, with $\dot{V}_E \geq 150\ l \cdot min^{-1}$, can be inferred from the increase in V_T/T_I; T_I/T_{Tot}, the other parameter of the duty cycle, was not different. Since metabolic rate (oxygen consumption) was significantly lower at two mid-level workloads and slightly lower at other work levels (possibly secondary to paralysis and lack of activity of the abdominal and trunk muscles), augmented chemical stimulation of drive would not have occurred.

We conclude that ICB appears to produce a modest increase in CO_2 responsiveness during progressive work with higher levels of ventilation. Whether this apparent increase in respiratory drive is related to diaphragmatic dysfunction secondary to a relative destabilization of the diaphragm is not clear. Transdiaphragmatic pressure measurements would be helpful to elucidate this issue. The results of the exercise testing seem to indicate that, although the lower chest wall does play a role in ventilation during periods of sustained high respiratory demand,

other muscles of respiration can compensate for functional impairment caused by ICB.

In summary, our data suggest that ventilatory function is well-preserved in normal subjects despite lower thoracic and abdominal wall paralysis until the extremes of ventilatory demand are reached when respiration is minimally impaired. Intercostal blockade for anesthesia or analgesia appears to be a safe technique in healthy individuals. Further studies need to be performed with direct measurement of diaphragmatic pump function.

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