PULMONARY FUNCTION DURING SPINAL ANESTHESIA:
THE MECHANISM OF COUGH DEPRESSION

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THE effect of high spinal anesthesia upon pulmonary ventilation, as estimated by its effect upon blood gases, has been reported normal,[1-4] depressed,[5] or increased.[6] Confusion has arisen because different methods of measuring respiration have been used and because preoperative sedation affects respiration.[5]

As far as we know, no one has studied the effect of spinal anesthesia on the cough mechanism. Bucher[7] has stated that the force of cough depends on the abilities necessary to close the glottis, to breathe deeply and to increase intrapulmonary pressure. We have studied the effect of spinal anesthesia on respiratory function and on the ability to create changes in intrapulmonary and intra-abdominal pressure.

METHOD

Respiratory function was tested in 20 healthy men prior to elective operations on a leg or for inguinal hernia. On the afternoon before operation, each patient, while lying supine on an operating table, breathed into a 9-liter Collins spirometer. Rate of respiration, tidal volume, vital capacity, expiratory reserve volume, inspiratory capacity, and oxygen uptake were recorded. With the spirometer running at fast speed (166 mm. per minute) and the carbon dioxide absorber removed, one-second timed expiratory and inspiratory capacities and maximum breathing capacity (12 seconds) were determined. Intrapulmonary pressures were estimated with a face mask connected directly to an aneroid manometer.[8] Inspiratory negative pressures were obtained by sucking air from the mask as vigorously as possible, expiratory positive pressures, by blowing into the mask. The greatest deviation from atmospheric pressure

was considered maximum intrapulmonary pressure. Measuring expiratory positive pressure in this way is a crude estimate of intrapulmonary pressure since a subject blowing through his mouth may exert pressure by compressing his cheeks. This source of error was reduced by asking each subject to extend his neck and make his lips the shape of an O.

On the morning of operation, each patient received intramuscular injections of meperidine 100 mg., pentobarbital 100 mg., and atropine 0.4 mg. In the operating room, approximately 60 minutes later, a 30 French Bardex rectal catheter with balloon was inserted 8 to 10 inches into the rectum and inflated to exert a slight (around 5 mm. of mercury) pressure on the rectum. The patient was then asked to cough vigorously and the pressure created in the balloon was recorded on an aneroid manometer. Rectal pressures were also noted during subsequent measurements on intrapulmonary pressures (expiration obstructed by the manometer).

After recording the rectal pressures, spirometric testing was repeated. Spinal anesthesia was then administered with the patient in the lateral recumbent position; ephedrine 25 mg. was injected intramuscularly to prevent hypotension and 14 mg. tetracaine 1 per cent, 1.4 ml. of dextrose 10 per cent with 0.5 mg. epinephrine were injected into the subarachnoid space. The patient was returned to the supine position and the operating table tilted 10 degrees head down for about 20 minutes. Testing was repeated 30 minutes after induction of anesthesia. At this time the patients had analgesia to pin prick from the second to sixth thoracic dermatomes. One patient was able to move his feet; the remaining 19 patients could not.

Intra-abdominal and intrathoracic pressures were also measured after low spinal anesthesia (first sacral level) in 10 other subjects.

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TABLE 1

SPIROMETRIC CHANGES FOLLOWING PREANESTHETIC MEDICATION AND SPINAL ANESTHESIA
(AVERAGES ± STANDARD DEVIATION)

	Control	After Sedation	After Spinal
Rate (per minute)	14 + 3.6	13 ± 2.5*	13 ± 2.4*
Tidal volume (ml.)	698 ± 174	464 ± 115*	483 ± 99*
Vital capacity (liters)	4.11 ± .65	4.05 ± .81	3.73 ± .72†
Expiratory reserve volume (liters)	.72 ± .40	.74 ± .41	.50 ± .27†
Inspiratory capacity (liters)	3.38 ± .49	3.47 ± .56	3.35 ± .63
1-Second timed expiratory capacity (per cent of vital capacity)	81 ± 12	91 ± 8.5*	91 ± 7.5*
1-Second timed inspiratory capacity (per cent of vital capacity)	73 ± 21	70 ± 19	71 ± 18
Maximum breathing capacity (liters/minute)	101 ± 25	109 ± 27	110 ± 26
Oxygen uptake (ml./minute)	361 ± 92	276 ± 85*	267 ± 77*

* Significantly different from control.
† Significantly different from after sedation.

The average values of duplicate determinations were used for statistical analysis except for the timed capacities when the higher figure was used. The effects of preanesthetic medication and of spinal anesthesia were subjected to statistical evaluation using Student's t test.[9]

RESULTS

The average age of the subjects was 28.9 years (range 18 to 43); average height 70.6 inches (67 to 74); average weight 173 pounds (140 to 215). All patients were slightly drowsy after sedation. Blood pressures and heart rates remained within normal limits during the studies.

Results of spirometry are shown in table 1. Following sedation, mean respiratory rate was significantly $(P < .05)$ less than during control measurements; tidal volume was less $(P < .01)$; timed expiratory capacity improved $(P < .01)$; oxygen uptake was less $(P < .05)$. After spinal anesthesia, respiratory rate and tidal volume remained approximately the same as after sedation alone; vital capacity was less $(P < .05)$ because of a decreased $(P < .01)$ expiratory reserve volume. Spinal block had no significant effect on other spirometer measurements.

High spinal anesthesia profoundly depressed expiratory positive intrapulmonary and rectal pressures $(P < .01$, table 2). The depression

TABLE 2

INTRAPULMONARY AND INTRA-ABDOMINAL PRESSURE CHANGES FOLLOWING
PREANESTHETIC MEDICATION AND HIGH SPINAL ANESTHESIA
(AVERAGES ± STANDARD DEVIATIONS)

	Control	After Sedation	After Spinal
Maximum inspiratory negative intrapulmonary pressure (mm. Hg)	−90 ± 27	−102 ± 27*	−94 ± 24
Maximum expiratory positive intrapulmonary pressure (mm. Hg)	78 ± 18	87 ± 21	41 ± 22*†
Maximum rectal pressure during vigorous expiration (mm. Hg)		53 + 20	7 + 11†
Maximum rectal pressure during cough (mm. Hg)	—	95 ± 43	4 ± 4†

* Significantly different from control.
† Significantly different from after sedation.

TABLE 3

THE EFFECT OF PARALYSIS OF THE PERINEAL
MUSCLES ON INTRAPULMONARY AND INTRA-
ABDOMINAL PRESSURES
(AVERAGES ± STANDARD DEVIATIONS)

	After Sedation	After Spinal (Level S1)
Maximum inspiratory negative intrapulmonary pressure (mm. Hg)	−91 ±22	−100 ±31
Maximum expiratory positive intrapulmonary pressure (mm. Hg)	79 ±24	83 ±35
Maximum rectal pressure during vigorous expiration (mm. Hg)	57 ±24	46 ±24
Maximum rectal pressure during cough (mm. Hg)	105 ±37	69 ±23*

* Significantly different from after sedation.

in the maximum rectal pressure during forced expiration and during coughing is much greater in patients with high spinal than in patients with low spinal anesthesia (table 3). Spinal anesthesia had no significant effect on the patients' ability to create negative pulmonary pressures. An increase in both negative and positive pulmonary pressures after preanesthetic medication may have been influenced by learning from the previous day's experience [8] or by improved relaxation following sedation.[10]

DISCUSSION

Spinal anesthesia has little effect upon the ability of healthy young men to breathe normally; in our patients, spinal anesthesia had less effect on respiratory function than did preoperative sedation which agrees with Johnson's data.[5] Why then is spinal anesthesia not a useful technique for avoiding atelectasis after operation?[16] An ineffective cough seems a likely explanation. Previous reports [11, 12, 16] have stressed the importance of the site of the incision, the general condition of the patient and the presence of infection elsewhere. Upper abdominal and chest wounds cause pain when the patient takes a deep breath or coughs;[11] he tries to brace his abdominal wall in order to reduce its motion. Whether coughing or deep breathing is more important in preventing atelectasis is not known. Mead and Collier [13] discuss the importance of a deep sigh to open occluded alveoli; they present data to show that narcotics depress this physiologic sigh. Semiconscious patients recovering from general anesthesia may not take deep

breaths. In our patients with spinal anesthesia, effective coughing was impossible; deep breathing was easy. This difference may explain the observation of Dripps and Deming [16] that more respiratory complications occurred after general anesthesia than after spinal anesthesia until active recovery room care was begun; after this, the complication rate was about the same after either type of anesthesia.

Spinal anesthesia does not completely suppress coughing, probably because of the elastic recoil of the lungs against the closed glottis; intrapulmonary pressure is raised in this way.[7] Other possible factors seem less likely. For example: (1) peristalsis of the bronchi does not occur;[14] (2) the accessory muscles of respiration (sternocleidomastoids and scalenes) serve only to stabilize the ribs against the pull of the abdominal muscles and probably have no direct effect in raising intrapulmonary pressure;[15] (3) the upper intercostal muscles, even if not paralyzed, play a minor part in forceful expiratory activity.[15] Campbell [15] has reported that the abdominal muscles are the most important muscles for coughing. We measured intrapulmonary pressures in several patients after operation. A return to normal was more closely related to the return of motor function than to the return of sensory function. Our data, therefore, support Campbell [15] as well as Bucher;[7] the abdominal muscles are important but are not the only mechanism for raising intrapulmonary pressure.

That adequate ventilation continues during high spinal anesthesia in the unoperated healthy young man probably does not guarantee adequate ventilation in other types of patients or in patients during operation. For example, a patient with emphysema uses his intercostal muscles to compensate for the downward displacement of the diaphragm;[15] he may not breathe adequately when these muscles are paralyzed. Any situation where decreased diaphragmatic activity might occur would cause a depression of ventilation during spinal anesthesia. Normally, the costal fibers of the diaphragm pull the ribs up and out using the viscera as a fulcrum; if the viscera are displaced as at laparotomy, the costal portion of the diaphragm may pull the ribs in.[15] Sancetta et al.[3] described a patient whose arterial oxygen concentration decreased dur-

ing exploration of the abdomen; he became anxious, his blood pressure fell, but cardiac output increased 200 per cent. Although the hypotension and anxiety may be attributed to reflexes from traction on the viscera, the low arterial oxygen content could be caused only by hypoventilation. That tissue hypoxia can occur in spite of normal ventilation has been well demonstrated by Kety and associates [6] who, by measuring arterial and jugular venous blood gas concentrations in patients anesthetized to the second thoracic level, found arterial carbon dioxide concentrations to be low, which suggests hyperventilation; nevertheless, the jugular vein oxygen concentration fell significantly. Hypoxia may occur during spinal anesthesia because of a sluggish peripheral blood flow as well as because of surgical manipulations.

The decrease in expiratory reserve volume after spinal anesthesia is probably caused by the same mechanism as the depression of cough. In order to perform maximal expiration, the abdominal wall muscles must contract.[15]

SUMMARY

Respiratory function was tested before and after spinal anesthesia to the second to sixth thoracic levels in 20 healthy men. Spinal block had less effect upon respiratory function tests that did preanesthetic medication.

The subjects' ability to raise intra-abdominal and intrapulmonary pressures was considerably depressed by high spinal anesthesia. Paralysis of the abdominal wall muscles is probably the mechanism of inadequate coughing after spinal anesthesia. The inability to cough forcefully during spinal anesthesia may explain the incidence of postoperative respiratory complications.

The opinions contained herein are those of the authors and do not necessarily reflect those of the Department of the Navy.

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