POST-OPERATIVE EPIDURAL ANALGESIA: EFFECTS ON LUNG VOLUMES

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Functional residual capacity (FRC) is reduced following upper abdominal operations under general anaesthesia. The changes which have been reported[1–5] are summarized in Table I. A number of factors can be responsible for the changes observed.

The reduction of FRC which follows induction of general anaesthesia may persist. The cause for this reduction is still undetermined, but the fall is not progressive with time and is related to body size.[6,7] Loss of lung volume by atelectasis or œdema will reduce FRC, as will an increase in expiratory muscle activity. Abdominal distension by pneumoperitoneum and bowel distension and incisional pain can be responsible.

In all probability, all these factors play a role. The present study was undertaken to determine the role of post-operative pain in reducing FRC and vital capacity (VC) and the efficacy of epidural analgesia in reversing these changes.

Subject Material

Eight patients (five females and three males) scheduled for elective upper abdominal operations were studied. They were clinically free of cardiorespiratory disease, except for patient No. 5 who had mild chronic bronchitis. The ages ranged from 34 to 67 years (mean 42.5), height varied from 145 to 183 cm (mean 159 cm), and weight from 40.9 to 88 kg (mean 67.9 kg). Consent was obtained in each case following explanation of the purpose and methods of the study.

Experimental Protocol

All subjects were studied in the supine position in the Recovery Room. Preoperative (control) measurements were made on the day of operation with the patients fasted overnight and without premedication. Measurements of FRC and VC were made following the placement of lumbar epidural and intravenous catheters by standard techniques, without injection of any anaesthetic agent. During the operation, lidocaine epidural anaesthesia was supplemented with general anaesthesia using thiopentone, endotracheal N_2O: O_2, and meperidine. The

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TABLE I

CHANGES IN FRC FOLLOWING SURGERY

Source	Ref.	Reported Change (%)			Type of Surgery	Remarks
		Day 1 (%)	Day 2 (%)	Day 5 (%)		
Beecher	1	−22	—	—	Abdominal	Pooled Data: Variety of Procedures
Anscombe	2	−57	—	—	Abdominal	
Kitamura	3	−12	—	—	All Types	Older Age: 12/20 non-abdominal
Alexander	4	−25 to −35	−20	−5 to −11	Abdominal	Painfree: 4 Narcotic Analgesic Regimens
Spence	5	−30	−21	−7	Vagotomy	
		−29	−16	+3	Gallbladder	
		−14	−8	—	Ing. Hernia	
		−5	—	—	Varicose Veins	
Wahba	—	−22	—	—	Abdominal	In Pain: Present Study
Wahba	—	−17	—	—	Abdominal	Painfree: Unpublished Data
	—	−12.8	—	—	Peripheral	Painfree: Unpublished Data

dose of meperidine was in the range of 50 to 100 mgm. All subjects were ventilated mechanically during the operation.

Post-operatively, measurements were repeated during pain and following epidural analgesia. This was achieved by catheter injection of incremental doses of lidocaine, 1.5 per cent, to achieve analgesia to pin prick and ice up to the fourth thoracic segment (T_4). In a previous study, we reported insignificant changes in FRC and VC following epidural analgesia in a group of patients prior to surgery.[8] Patients Nos. 1 to 4 were studied on the day following their operation at least two hours after the last injection of analgesic, while patients Nos. 5 to 8 were studied not less than two hours post-operatively on the day of the operation.

Methods

FRC was measured by the helium dilution technique using a Palmer spirometer with a catherometer in the circuit (Cambridge Instruments Ltd.)[9] Nitrous oxide is known to affect the catherometer reading.[10] Although we did not measure expired nitrous oxide levels, we have assumed that all of the N_2O was eliminated by the time the measurements were made. The assumption is supported by the fact that there was no difference between the post-operative studies on the day of operation and those performed on the first post-operative day. Vital Capacity was measured by standard spirometry. The results were analyzed statistically by Student's test for paired groups using a programme prepared in the Cardio-Respiratory Research Laboratory, Royal Victoria Hospital, Montreal.

Results

Results are presented in Table II and in Figure 1.

(a) Functional Residual Capacity

The mean percentage change of FRC during pain was −21.7 per cent (±5.12 per cent) with partial restoration to −15.9 per cent (±4.1 per cent) following epidural analgesia: this represents a 26.7 per cent restoration. Statistically, the first change was significant with reference to control levels whereas the pain-free data were not significantly different from measurements during pain.

(b) Vital Capacity

Vital Capacity showed a mean decrease of −63 per cent (±3.4 per cent) from control values with pain. After pain relief, the mean was −44.5 per cent (±5 per cent) which represents a 29.4 per cent restoration. Both changes were statistically significant.

Discussion

The results will be discussed under three headings: the reasons for the incomplete restoration; comparison with reports using narcotic analgesic drugs; and clinical implications.

Incomplete Restoration of FRC

A number of reasons can explain our observation that FRC is only partially

TABLE II

RESULTS

	Functional Residual Capacity					Vital Capacity				
		Pain		Painfree			Pain		Painfree	
	Pre-Op.	(Litres)	(%)	(Litres)	(%)	Pre-Op.	(Litres)	(%)	(Litres)	(%)
1	1.74	1.14	−34.5	1.26	−26.6	3.885	0.96	−75.3	1.60	−58.8
2	2.38	2.22	−6.7	2.18	−8.4	3.890	1.55	−60.2	2.20	−43.4
3	1.27	0.65	−48.8	1.16	−8.7	2.250	0.90	−60.0	1.30	−42.2
4	2.01	1.64	−18.4	1.48	−26.4	4.320	1.21	−72.0	1.41	−67.4
5	1.87	1.69	−9.6	1.96	+4.8	3.260	1.61	−50.6	2.17	−33.4
6	2.56	1.80	−26.7	1.81	−29.3	3.740	1.80	−51.9	1.90	−49.2
7	1.30	1.04	−20.0	1.06	−18.5	2.600	0.67	−72.2	1.60	−38.5
8	1.98	1.80	−9.1	1.70	−14.1	2.890	1.26	−59.5	2.23	−22.9
Mean	1.88	1.50*	−21.7	1.58	−15.9	3.355	1.245*	−63.0	1.675*	−44.5
S.D.	0.16	0.18	5.1	0.14	4.1	0.250	0.190	3.4	0.130	5.0

*Significant Change, $P < 0.001$.

Restoration 1. FRC $= \frac{21.7 - 15.9}{21.7} \times 100 = 26.7\%$.

2. V.C. $= \frac{63 - 44.5}{63} \times 100 = 29.4\%$.

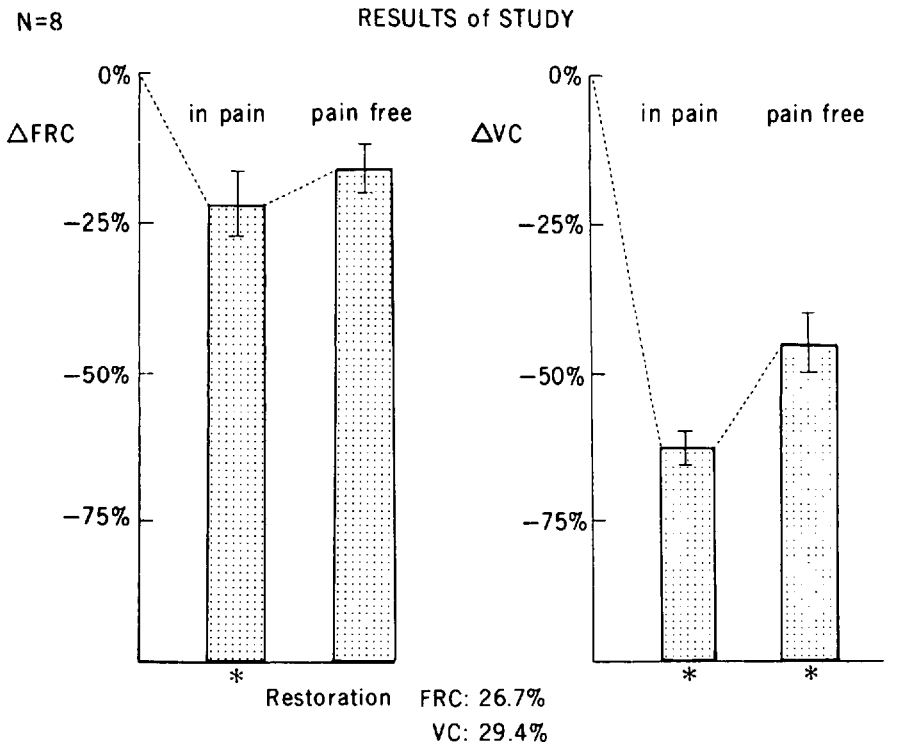

FIGURE 1. Results of Study: Changes ± S.E. For explanation see text.

restored to pre-operative values. One or more of the factors listed previously may be operative. It is possible that abdominal distension and pneumoperitoneum are principally responsible.[4,11] We base this belief on the fact that the return of FRC to pre-operative values closely follows the time course of the reduction in abdominal distension which accompanies the resumption of normal bowel activity. Arterial oxygenation also follows this pattern of a gradual return to normal over the course of two to five days.[3,12–16]

It is also probable that a variable degree of atelectasis is present. This would result from a protracted period of "guarded" breathing, central depression from narcotic analgesics, and the effects of abdominal distension acting from below the diaphragm. It would be interesting to determine the added effects of a number of deep breaths and vigorous pulmonary physiotherapy plus epidural analgesia on arterial blood gases. Such a combination could, quite possibly, result in a more dramatic improvement in oxygenation than that reported by Hollmen,[17] Muneyuki,[18] and Spence.[5]

Recent reports have stated that morphine, fentanyl, Innovar (R), and nitrous oxide cause an increase in expiratory muscle tone (EMT).[19–21] This could manifest itself as a post-operative reduction in FRC. Meperidine could conceivably do the same. However, the time interval between operation and our measurements would in all probability preclude this. The subjects studied on day 1 had received no meperidine for three hours prior to the study. Another factor contributing to

the low post-operative FRC may be the persistence of the fall in FRC following the induction of anaesthesia.[6,7] The reasons for this initial fall have not been fully clarified.

Comparison with Narcotic Analgesic Drugs

The post-operative reduction in FRC (−21.7 per cent) is similar to that reported by other authors. Epidural analgesia was followed by a partial restoration to −15.9 per cent below control. This is in sharp contrast with the results obtained by Alexander *et al.*[22] They report that FRC was reduced by −25 per cent to −35 per cent from control in a similar group of patients who received narcotic analgesic drugs.

Clinical Implications

The significance of FRC lies in its relationship to compliance and "Closing Volume" (CV). A number of recent reports dealt with the clinical aspects of this latter relationship.[23–28] Two recent studies report measurements of both FRC and CV in the post-operative period.

Alexander *et al.* report that CV is "more stable" than FRC since FRC showed a relatively bigger fall.[4,11] This might then place tidal breathing within closing volume. A restoration of FRC, as by epidural analgesia, would be beneficial for gas exchange by moving the end tidal position above closing volume. Such a situation is graphically illustrated in Figure 2.

We do not mean to imply that all subjects will show a dramatic improvement in oxygenation following epidural analgesia, but rather that this technique would be beneficial in the patient liable to develop severe post-operative pulmonary derangement.

We have reported that subjective pain relief resulted in a 26.7 per cent restoration in FRC and a 29.4 per cent restoration in VC. These figures indicate the "pain-component" in the post-operative reduction in these two lung capacities.

Deep breathing, opening up of atelectatic areas (with reversal of pulmonary shunting), and removal of expectorations can be accomplished more effectively because of the increase in Vital Capacity[29–30] in a pain-free situation. This would make chest physiotherapy and other measures more acceptable and beneficial to the patient. The purpose behind all this is to decrease the still relatively high incidence of post-operative pulmonary complications. In a recent report, Pflug *et al.*[31] report that X-ray evidence of atelectasis was lower in their epidural than in the morphine group and that convalescence was three days shorter in the epidural group.

Conclusions

Post-operative epidural analgesia is followed by partial restoration of FRC and VC towards preoperative values. This would be of value in subjects likely to develop post-operative pulmonary complications by avoiding closure of small airways during tidal breathing. Deep breathing and coughing can be performed more effectively with more satisfactory removal of secretion and reversal of atelectasis.

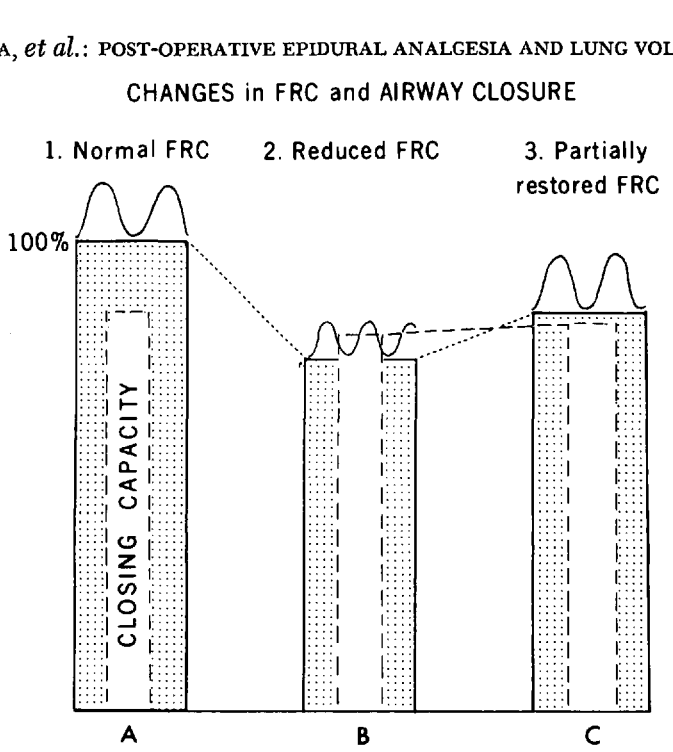

FIGURE 2. Changes in FRC and their role in Hypoxaemia. For explanation see text.

SUMMARY

A study was undertaken to assess the role of post-operative pain in reducing Functional Residual Capacity (FRC) and Vital Capacity (VC). The efficacy of post-operative epidural analgesia in reversing these changes was measured in eight subjects after upper abdominal operations.

With pain, FRC and VC were 78 per cent and 37 per cent of control respectively. Following epidural analgesia, the values were 84 per cent and 55 per cent. These figures indicate the pain component in the post-operative reduction of these two lung capacities. This partial restoration may be of value in the prevention of post-operative pulmonary complications by avoiding closure of small airways during tidal breathing and by increasing the effectiveness of deep breathing and coughing in the removal of secretions and the reversal of atelectasis.

RÉSUMÉ

Les auteurs ont entrepris une étude pour déterminer le rôle de la douleur dans la diminution de la Capacité Residuelle Fonctionnelle (CRF) et la Capacité Vitale (CV) dans la periode postopératoire. Cela ainsi que l'efficacité de l'analgesie peridurale postopératoire pour rétablir les valeurs de ces deux capacités vers la valeur de contrôle fut determiné chez huit malades ayant subi des interventions intra-abdominales majeures. Les deux capacités étaient de 78 pour cent et 37 pour cent CRF et CV respectivement pendant la douleur; suivant l'analgesie peridu-

rale ces deux capacités étaient à 84 pour cent et 55 pour cent. La différence indique la composante "douleur" dans la réduction universellement observée dans ces deux fonctions pulmonaires. Cette restoration incomplète pourrait quand même être utile dans la prévention des complications pulmonaires durant la phase postopératoire. Ceci ce ferait en empêchant la fermeture prématurée des petites voies aèriennes pendant la ventilation normale et aussi en augmentant l'efficacité des manœuvres habituelles pour degager les secretions et re-ouvrir les alvéoles.

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REFERENCES

1. BEECHER, H.K. Effect of laparotomy on lung volume. Demonstration of a new type of pulmonary collapse. J. Clin. Invest. *12*: 651 (1933).
2. ANSCOMBE, A.R. Pulmonary complications of abdominal surgery. Chicago, Year Book Medical Publishers (1957).
3. KITAMURA, H., SAWA, T. & IKEZONO, E. Post-operative hypoxemia. Anesthesiol. *36*: 244 (1972).
4. ALEXANDER, J.I., SPENCE, A.A., PARIKH, R.K. & STUART, B. The role of airway closure in post-operative hypoxaemia. Brit. J. Anaesth. *45*: 34 (1973).
5. SPENCE, A.A. & SMITH, G. Post-operative analgesia and lung function: a comparison of morphine with extradural block. Brit. J. Anaesth. *43*: 144 (1971).
6. LAWS, A.K. Effects of induction of anaesthesia and muscle paralysis on function residual capacity of the lungs. Can. Anaes. Soc. J. *15*: 325 (1968).
7. DON, H.F., WAHBA, W.M., CUADRADO, L., & KELKAR, K. The effects of anesthesia and 100 per cent oxygen on functional residual capacity of the lungs. Anesthesiol. *32*: 521 (1970).
8. WAHBA, W.M., CRAIG, D.B., DON, H.F., & BECKLAKE, M.R. The cardiorespiratory effects of thoracic epidural anaesthesia. Can. Anaes. Soc. J. *19*: 8 (1972).
9. MENEELY, G.R. & KALTREIDER, N.L. The volume of the lung determined by relium dilution. Description of the method and comparison with other procedures. J. Clin. Invest. *28*: 129–139 (1949).
10. COLGAN, F.J. & WHANG, T.B. A method for measuring the functional residual capacity and dynamic lung compliance during oxygen and halothane inhalation. Anesthesiol. *28*: 559 (1967).
11. ALEXANDER, J.I., HORTON, P.W., MILLAR, W.T., PARIKH, R.K., & SPENCE, A.A. The effect of upper abdominal surgery on the relationship of airway closing point to end tidal position. Clin. Sci. *43*: 137 (1972).
12. DIAMENT, M.L. & PALMER, K.N.V. Venous/arterial pulmonary shunting as the principal cause of post-operative hypoxaemia. Lancet *1*: 15 (1967).
13. GEORG, J., HORNUM, J., & MELLEMGAARD, I.C. The mechanism of hypoxaemia after laparotomy. Thorax *22*: 382 (1967).
14. GORDH, T., LINDERHOLM, H., & NORLANDER, O. Pulmonary function in relation to anesthesia and surgery evaluated by analysis of oxygent tension of arterial blood. Acta. Anaesthesiol. Scand. *2*: 15 (1958).
15. MUNEYUKI, M., UEDA, Y., URABE, N., KATO, H., SHIRAI, K., & INAMOTO, A. Oxygen breathing and shunting during post-operative pain relief in man. Can Anaes. Soc. J. *19*: 230 (1972).
16. SILER, J.N., ROSENBERG, H., MULL, T.D., KAPLAN, J., BARDIN, H., & MARSHALL, B.E. Hypoxemia after abdominal surgery. Ann. Surg. *179*: 149 (1974).

17. HOLLMEN, A., SAUKKONEN, J. Effects of post-operative epidural analgesia vs centrally acting opiate on physiological shunt after upper abdominal operation. Acta Anaesth. Scand. *16*: 147 (1972).
18. MUNEYUKI, M., UEDA, Y., URABE, N., TAKESHITA, H., & INAMOTO, A. Post-operative pain relief and respiratory function in man. Anesthesiol. *29*: 304 (1968).
19. FREUND, F., ROOS, A., & DODD, R. Expiratory activity of the abdominal muscles in man during general anesthesia. J. App. Physiol. *19*: 693 (1964).
20. FREUND, F., MARTIN, W.E., WONG, K.C., & HORNBEIN, T.F. Abdominal muscle rigidity induced by morphine and nitrous oxide. Anesthesiol. *38*: 358 (1973).
21. HEWLETT, A.M., KAUL, S.U., HULANDS, G.H., ANDERSON, M., & NUNN, J.F. Expiratory muscle activity and changes in functional residual capacity during anesthesia. Brit. J. Anaes. *45*: 114 (1973).
22. ALEXANDER, J.I., PARIKH, R.K., & SPENCE, A.A. Post-operative analgesia and lung function: a comparison of narcotic analgesic regimens. Brit. J. Anaesth. *45*: 346 (1973).
23. CRAIG, D.B., WAHBA, W.M., DON, H.F., COUTURE, J.G., & BECKLAKE, M.R. "Closing volume" and its relationship to gas exchange in seated and supine positions. J. Appl. Physiol. *31*: 717 (1971).
24. CRAIG, D.B., WAHBA, W.M., & DON, H.F. Airway closure and lung volumes in surgical positions. Can. Anaes. Soc. J. *18*: 92 (1971).
25. CRAIG, D.B., WAHBA, W.M., DON, H.F., & BECKLAKE, M.R. The effect of posture and airway closure on gas exchange. Clin. Res. *18*: 742 (1970).
26. DON, H.F., WAHBA, W.M., & CRAIG, D.B. Airway closure, gas trapping and the functional residual capacity in anesthesia. Anesthesiol. *36*: 533 (1972).
27. DON, H.F., CRAIG, D.B., WAHBA, W.M., & COUTURE, J.G. The measurement of gas trapped in the lungs at functional residual capacity and the effects of posture. Anesthesiol. *35*: 582 (1971).
28. FAIRLEY, H.B. Editorial: Airway closure. Anesthesiol. *36*: 529 (1972).
29. BOUTROS, A.R. Management of severe respiratory insufficiency associated with painful conditions in the thorax and abdomen. Can. Anaes. Soc. J. *9*: 246 (1962).
30. BROMAGE, P.R. Extradural analgesia for pain relief. Brit. J. Anaesth. *39*: 721 (1967).
31. PFLUG, A.E., MURPHY, T.M., BUTLER, S.H., & TUCKER, G.T. The effects of post-operative peridural analgesia on pulmonary therapy and pulmonary complications. Anesthesiol. *41*: 8 (1974).