THE CARDIO-RESPIRATORY EFFECTS OF THORACIC EPIDURAL ANAESTHESIA*

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THE USE OF epidural anaesthesia for pain relief in many situations has been reported in the literature.[1–6] The effects of this technique on some aspects of cardio-respiratory function have also been reported[8–15] and reviewed.[7] The aim of the present study was to add to the presently available knowledge of the cardio-respiratory effects of an epidural anaesthetic. The focus of our interest was on the Functional Residual Capacity (FRC) and the Alveolar-Arterial Oxygen Gradient ($A\text{-}aDo_2$) because studies of these two aspects of pulmonary function have not been reported. Because we were interested in studying these indices of cardio-pulmonary function in the context of pain relief following upper abdominal surgery, we used 1.5 per cent lidocaine in sufficient amounts to achieve sensory block to the level of the fourth thoracic segment, so as to avoid involving the cardiac sympathetics and to achieve mainly sensory blockade with minimal neuromuscular effects.

SUBJECT MATERIAL

Ten female and three male subjects who were scheduled for elective upper abdominal operations were studied. The procedures were described to them and their consent was obtained. All were clinically free of cardio-pulmonary disease. The pertinent patient data are given in Table I.

EXPERIMENTAL PROTOCOL AND METHODS

All the studies were performed in the Recovery Room in the pre-operative period. The subjects were fasting, supine and unpremedicated.

Following the preliminary steps of inserting the epidural, arterial and central venous catheters by standard techniques, the pre-epidural (control) study was carried out in the following sequence:

(1) *Determination of* FRC. This was measured by the closed circuit helium technique using a catharometer (Cambridge Instruments Co. Ltd) for the measure-

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TABLE I

PATIENT DATA

Group	Patient	Age yrs	Ht m	Wt Kg	Sex	Operation	Lidocaine ml	Lidocaine mgm	Epin. Ugr.	IV fluids L
A. Lidocaine 1.5 per cent	1 A.F.	44	1.65	55.5	F	cholecystectomy	19	285		0.90
	2 F.P.	26	1.73	70.0	M	pyloroplasty	26	390		1.00
	3 M.V.	40	1.62	100.5	F	cholecystectomy	20	300		0.95
	4 V.L.	35	1.54	88.0	F	cholecystectomy	33	495		1.00
	5 G.R.	45	1.68	82.0	F	cholecystectomy	22	330		1.00
	6 D.S.	34	1.83	84.0	M	cholecystectomy	17	255		1.00
	7 R.H.	67	1.51	58.0	F	cholecystectomy	8	120		0.60
	8 A.P.	36	1.52	55.5	F	cholecystectomy	15	225		0.60
B. Lidocaine 1.5 per cent + Epinephrine 1/200,000	9 P.G.	31	1.58	71.0	F	cholecystectomy	17	255	85	0.80
	10 M.C.	47	1.62	65.0	F	cholecystectomy	15	225	75	0.65
	11 H.L.	71	1.68	76.6	M	lt. hemicolectomy	14	210	70	0.75
	12 J.G.	20	1.65	65.0	F	cholecystectomy	23	245	115	0.70
	13 E.C.	59	1.57	65.0	F	cholecystectomy	15	225	75	0.80

ment of helium.[16] A Palmer spirometer was used to determine the other subdivisions of total lung capacity.

(2) *Determination of Cardiac Output (Qc).* This was measured in duplicate by the Hamilton Dye Dilution technique using Indocyanine Green.[17] Dye was injected into a central vein and sampled from a percutaneously placed arterial cannula. A Waters system (xC-302 cuvette, D-400 densimeter) was used, with a 10-inch recorder. (Honeywell Electronik 194). The curves obtained were analysed using a Stewart-Hamilton replot technique by a compensating polar planimeter (Keuffel-Esser 62005) to determine the area of the replotted primary curve.

(3) *Determination of A-aDo$_2$.* A three minute expired gas collection, whose volume was determined by a dry gas meter, was used to measure the mixed expired P_{O_2} and P_{CO_2}. By applying the Ideal Alveolar Gas Equation, [18] the value for P_{AO_2} was found. Simultaneously, arterial blood was collected into iced, heparinized glass syringes and using conventional electrodes (Instrumentation Laboratories IL 113 system) the tensions of O_2 and CO_2 and pH were determined. Having in this manner determined alveolar and arterial P_{O_2} and P_{CO_2}, A-aD_{O_2}, V_D/V_T together with V_{O_2}, R, and V_A were calculated by applying the appropriate equations (see appendix).

(4) *Forced Expiratory Volume* FEV_1. This was measured using a Stead-Wells Spirometer. During this control study, 10 ml/kg body weight 5 per cent dextrose in normal saline was infused. Epidural anaesthesia was then instituted by injecting incremental doses of 1.5 per cent lidocaine to achieve a sensory block to the fourth thoracic segment for both ice and pinprick. The level is defined as the level of definite normal sensation above a zone of hypoaesthesia. The first 8 subjects were given plain 1.5 per cent lidocaine (Group A) while the last 5 were given 1.5 per cent lidocaine with epinephrine 1/200,000 (Group B). The post-epidural study was then performed in the same sequence as before and in this manner each patient acted as his own control.

Results

The results are presented in tabular and graphic form in Tables II to IV and Figures 1 to 6. Statistical analysis by Students' *t* test for paired groups, showed no statistical significance between the controls of the two groups. The changes both within each group and between the groups also had no statistical significance.

(A) *Functional Residual Capacity*

The changes in both FRC and Residual Volume (RV) were insignificant in either group of patients. The same holds true, therefore, with the Expiratory Reserve Volume (ERV) *since* FRC = ERV + RV. However, it must be noted that in individual patients, the changes differed in both the magnitude and direction of change (Figures 1–3).

(B) *Vital Capacity and Forced Expiratory Volume*

This did not change significantly in either group. Likewise the change in the mean FEV_1 was insignificant.

TABLE II

CHANGES IN TOTAL LUNG CAPACITY AND ITS SUBDIVISIONS

		FRC	RV	ERV	IC	VC	TLC	V_T	$FEV_{1.0}$
Group A	Mean pre-epidural	1.92	1.34	0.58	2.97	3.55	4.88	0.57	2.48
	Mean post-epidural	2.01	1.43	0.57	3.18	3.75	5.18	0.57	2.59
	Mean per cent change	+4.7	+6.9	+4.1	+6.5	+5.7	+6.2	+1.5	
	SE	0.03	0.05	0.11	0.03	0.02	0.02	0.04	
Group B	Mean pre-epidural	1.96	1.52	0.44	2.83	3.27	4.80	0.65	2.41
	Mean post-epidural	1.92	1.56	0.35	2.08	3.15	4.72	0.66	2.40
	Mean per cent change	+1.5	+4.7	−14.6	−0.9	+3.7	−1.1	+6.4	
	SE	0.03	0.05	0.11	0.07	0.07	0.04	0.09	

Group A: Lidocaine 1.5 per cent plain
Group B: Lidocaine 1.5 per cent + Epinephrine 1/200,000
SE: Standard Error (Standard Deviation of the Mean)

(All Volumes at BTPS)

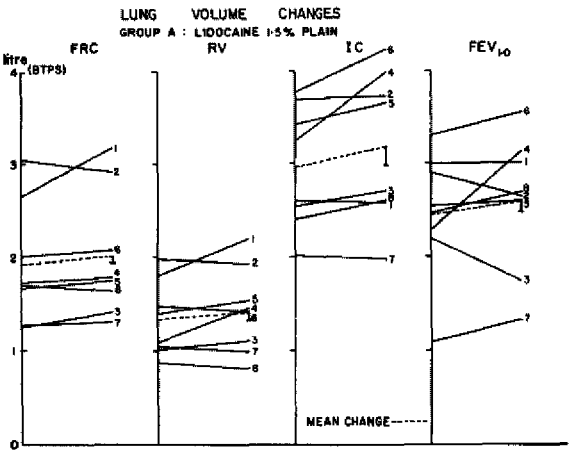

FIGURE 1. Changes in lung volumes in individual subjects of group A (plain lidocaine 1.5 per cent). The interrupted line indicates the mean change minus one standard error.

TABLE III

CHANGES IN ALVEOLAR GAS EXCHANGE

		$A - aD_{O_2}$	Q_C	Q_S/Q_T%	V_D/V_T
Group A	Mean pre-epidural	27.3	5.7	11.5	0.29
	Mean post-epidural	26.2	6.0	9.9	0.29
	Difference of means	−1.1	+0.3	−1.6	+0.00
	Mean per cent change	+0.5	+5.0	−5.1	+17.6
	SE	0.12	0.07	0.15	0.23
Group B	Mean pre-epidural	33.2	5.9	11.5	0.30
	Mean post-epidural	32.6	6.0	10.1	0.31
	Difference of means	−0.6	+0.1	−1.4	+0.01
	Mean per cent change	+4.3	+0.9	−3.8	+3.6
	SE	0.11	0.16	0.28	0.16

Group A: Lidocaine 1.5 per cent plain
Group B: Lidocaine 1.5 per cent + Epinephrine 1/200,000
SE: Standard Error (Standard Deviation of the Mean)

(C) *Alveolar Ventilation, Oxygen Consumption and Respiratory Quotient*

In order to validate comparison between pre-epidural and post-epidural values for gas exchange parameters, the values for R must be similar and must also be less than 1.0. These values are shown in Table 4. The fact that $\dot{V}_A$, $\dot{V}_{O_2}$, and R showed very small and insignificant changes in both groups makes comparison between the gas exchange figures valid.

(D) *Alveolar Gas Exchange*

This can be analyzed in terms of its three components: dead space ventilation (V_D/V_T), shunt (Q_S/Q_t) and ventilation/perfusion scatter, the latter two being responsible for the $A\text{-}aD_{O_2}$. Because any significant changes in cardiac output

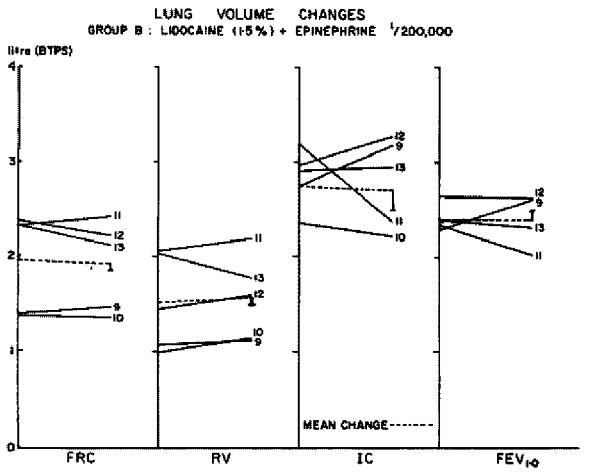

FIGURE 2. Changes in lung volumes in individual subjects of group B (lidocaine with epinephrine).

TABLE IV

CHANGES IN RQ AND OXYGEN CONSUMPTION

		$\dot{V}_{O2}$	$\dot{V}_A$	RQ
Group A	Mean pre-epidural	224	5.2	0.90
	Mean post-epidural	226	5.4	0.90
	Difference of means	+2.0	+0.2	+0.00
	Mean per cent change	+0.4	+10.6	+3.0
	SE	0.7	0.16	0.07
Group B	Mean pre-epidural	201	4.9	0.97
	Mean post-epidural	193	5.3	0.97
	Difference of means	−8.0	+0.4	+0.00
	Mean per cent change	−4.0	+6.9	+0.2
	SE	0.05	0.13	0.08

will affect the A-aDo_2, the changes in this parameter are presented on the same figures (4–6). Cardiac output determinations were only possible in five patients in Group A and three in Group B. The changes in the means of these four parameters were insignificant in either of the two groups, with individual variations being observed in both direction and magnitude.

DISCUSSION

(A) *Vital Capacity*

The results of our study agree with those reported by Freund *et al.*,[11] if one considers only those of their volunteers whose block was at T_4 and not higher. That the changes in VC are insignificant can be anticipated by reviewing the role of the different muscle groups involved in a vital capacity breath. Using electro-

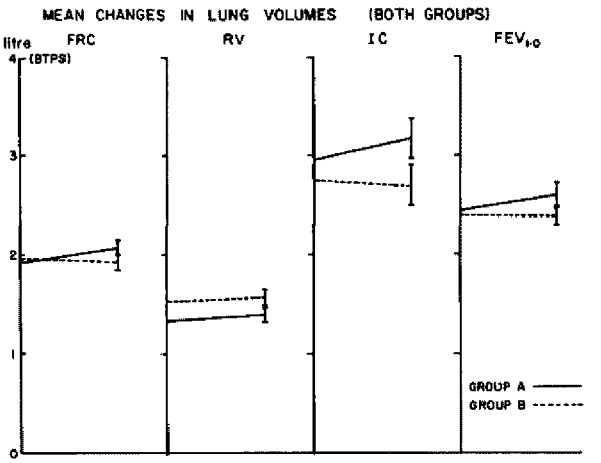

FIGURE 3. Mean changes in lung volumes in both group A (plain lidocaine – continuous line) and group B (lidocaine with epinephrine – interrupted line).

myography to detect muscle activity in awake subjects, Campbell[19,20] has shown that inspiration is due to the action of the diaphragm and the fifth to ninth external intercostal muscles. In an expiratory effort the main muscles involved are the diaphragm and the anterior abdominal wall muscles. Freund[11] also using electromyography in subjects who had been given an epidural anaesthetic, reported that the level of motor blockade was 4.6 neurotomes below the sensory level. Thus a sensory block up to T_4 should not involve the muscles of deep inspiration because the level of motor block in such a situation would only be at T_8–T_9. Also, the use of 1.5 per cent Lidocaine should have minimal or no effects on voluntary muscle tone.[21] These considerations explain why we found no change in vital capacity.

(B) *FEV*

The manoeuvres involved in this test are quite similar to those required for a forceful cough. The fact that, in spite of it being a test requiring patient cooperation, it did not change significantly is important, since it denotes that both muscle power and airway calibre are unaffected. Thus an epidural anaesthetic in itself, will not adversely affect coughing and deep breathing.

(C) *FRC*

Our findings of no significant change in this parameter are important. Because of the significance of FRC in relation to airway closure[22] maintenance of a normal FRC is important for both adequate gas exchange and lung compliance. A low FRC has been shown to promote airway closure[23] and this will lead to arterial hypoxaemia. If an epidural anaesthetic is given for pain relief, it will, by relieving the pain, allow more comfortable breathing and a possible correction of the low-

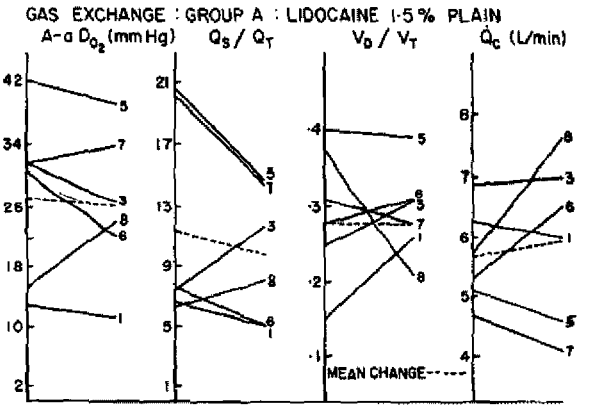

FIGURE 4. Changes in gas exchange data in individual subjects of group A (plain lidocaine 1.5 per cent). The interrupted line indicates the mean change.

ered FRC that is seen after abdominal surgery.[24,25] This will obviously be beneficial to the patient for both gas exchange and coughing and deep breathing. The fact that we have observed no significant change can be interpreted to mean that the epidural anaesthetic had no effect on either the thoracic wall expansion or the pulmonary retractive forces which interact to determine the FRC. Since FRC did not change, one need not anticipate a derangement of gas exchange due to a significant change in FRC in either direction. This we have observed, in the essentially unchanged mean A-aD_{O_2}.

(D) *Oxygen Consumption and Respiratory Quotient*

From our results, it seems that there is no gross change in metabolic status. The shivering that is sometimes observed following an epidural anaesthetic was noted in one case in whom there was a concomitant increase in both Qc and V_A but a very slight drop in V_{O_2}.

(E) *Alveolar Gas Exchange*

A fall in Qc would result in an enlarged A-aD_{O_2}. If peripheral pooling following an epidural were extensive enough then one would observe an enlarged A-aD_{O_2} and increased V_D/V_T ratio due to ventilation of non-perfused areas. We did not observe any of these changes which could be anticipated with marked peripheral pooling. Moreover we found insignificant changes in the mean Qc, in contrast to the findings of Bonica *et al.*[26] They found, using 2 per cent plain lidocaine that Qc fell by 5 per cent. However, the upper level in their series is described only as rising to at least T_5 and does not exclude a block of the cardiac sympathetics.

A knowledge of the exact level is important in view of the findings reported by Wugmeister and Hehre.[27] These authors have observed that there was no significant difference between the levels of sensory and sympathetic blockade during epidural anaesthesia, in contrast to the two segment difference during

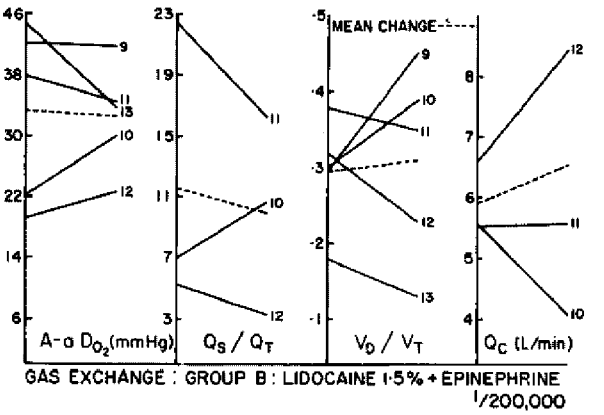

FIGURE 5. Changes in gas exchange data in individual subjects of group B (lidocaine 1.5 per cent with epinephrine). The interrupted line indicates the mean change.

spinal anaesthesia reported by Greene.[28] Therefore exact knowledge of the level of sensory blockade is important in order to determine whether the cardiac sympathetics have been blocked or not. The cell stations for these fibres arise from the upper four thoracic segments.[29] In the present study, by achieving a sensory block to T_4 only, we can assume, on the basis of Wugmeister's study, no cardiac sympathetic blockade and therefore no significant change in Qc. This assumption is borne out by our results. The incorporation of adrenaline in the anaesthetic solution has a role of its own. After vascular uptake adrenaline will have a dose-related effect on cardiovascular dynamics. Ward *et al.*[12] report a 30 per cent rise in Qc when they used 2 per cent lidocaine with epinephrine 1/200,000. An effect due to the epinephrine can be anticipated when one considers that their dosage range was between 125–200/μg. In the 3 subjects we studied using epinephrine and in whom we measured cardiac output we observed a mean percent change of only +0.9 per cent. Our dosage was 70–115 micrograms. This difference in dosage could explain the different results.

Another factor that is not mentioned in Bonica's study is the state of hydration or intravenous fluid management during the test. Our subjects received 10 ml/kg Dextrose in normal saline during the control study. This could also play a role in the results and perhaps stresses the importance of adequate hydration.

In a recent article Bonica and his colleagues[31] report no significant change in cardiac output when the level spread incrementally up to T_{4-5}. In these subjects intravenous fluids were given and lidocaine was injected by increments. These results are very similar to our findings, specially when subject (8) who was shivering at the end of the measurements, is excluded. It is interesting to note a significant increase in cardiac output when the block extended beyond T_{4-5} in that study. This was mainly due to an increase in heart rate and was accompanied by a fall in arterial P_{CO_2}, suggesting, therefore, an indirect effect through hyperventilation due to apprehension with cephalad spread of the block above T_4.

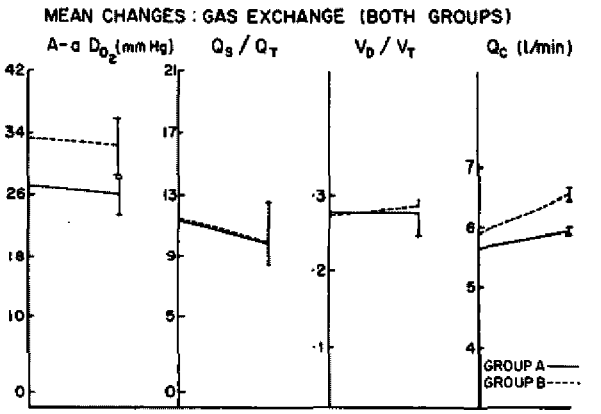

FIGURE 6. Mean changes in gas exchange parameters in both group A (plain lidocaine – straight line) and group B (lidocaine with epinephrine 1/200,000 – interrupted line).

From our results, we deduce that one of the important determinants of A-aDo_2, namely Qc, did not change to any significant degree. Although the mean value for venous admixture did decrease this fall was insignificant. We were impressed with the fact that by attempting to achieve a sensory block only to T_4, with adequate hydration, no derangement of oxygenation was observed.

The index of wasted ventilation, v_D/v_T, was insignificantly affected. In spite of the possibility of peripheral pooling and bronchodilatation due to lidocaine,[30] there was no net increase in dead-space ventilation, and therefore of CO_2 exchange in the lungs.

CONCLUSION

From our data, we conclude that an epidural anaesthetic given in the stated circumstances (sensory level to T_4, using 1.5 per cent lidocaine and with adequate hydration) will not result in a derangement of ventilatory capacity, will have no effect on FRC, and will not interfere with alveolar gas exchange as characterized by the A-aDo_2.

SUMMARY

The cardio-respiratory effects of an epidural anaesthetic given to 13 subjects pre-operatively are reported. They were fasting, unpremedicated and supine. A sensory block up to the 4th thoracic segment was not followed by any significant change in FRC, FEV_1, VC, A-aDo_2, Qs/Qt or cardiac output.

RÉSUMÉ

Les auteurs rapportent leurs observations sur les effets cardio-respiratoires lors d'une anesthésie péridurale thoracique haute (au niveau du T_4). Leur rapport est surtout concentré sur la capacité résiduelle fonctionnelle (CRF) et le gradient alvéolo-artériel de Po_2 (A-aDo_2), ces paramètres n'étant pas rapportés dans la

littérature à date. Les cas étudiés étaient 13 candidats à la chirurgie abdominale haute. Ils rapportent des changements sans importance clinique ou statistique dans la capacité vitale (CV), CRF, FEV i.o de même que dans le A-aDo_2, le shunt (Qs/QT) et débit cardiaque (Qc) ainsi que dans le rapport VD/VT. Ces observations suivirent une hydratation adéquate (10 ml/kg de dextrose à 5 pour cent dans sérum physiologique), l'emploie de lidocaïne 1.5 pour cent et un dosage qs pour atteindre une analgésie à la piqûre et à la glace de T_4 seulement.

En conclusion ils soulignent l'importance des précautions à prendre vis-à-vis le dosage et l'hydration pour en arriver à un résultat clinique satisfaisant de cette technique.

APPENDIX

(1) *Ideal Alveolar Gas Equation*:

$$P_AO_2 = P_IO_2 - PaCO_2(P_IO_2 - P_EO_2)/P_ECO_2$$

(2) *"Shunt" Equation:*

$$Q_s/Q_t = (Cc_{O_2} - Ca_{O_2})/(Cc_{O_2} - C\bar{v}_{O_2})$$

(3) *Physiological Dead Space:*

$$V_D = V_T(PaCO_2 - P_{\bar{E}}CO_2)/PaCO_2$$

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