LUNG MECHANICS AND BLOOD GASES DURING ANAESTHESIA WITH SPONTANEOUS RESPIRATION*

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DURING THE PAST YEARS several authors have documented the progressive impairment in the mechanical and gas exchange properties of the lungs observed during anaesthesia. However, recent work[1,2] has demonstrated that the main changes occur in the early period of the anaesthetic procedure and that lung parameters are not adversely affected in the later course.

In this study we have investigated the effects of anaesthesia on pulmonary efficiency, starting before premedication, and continuing into the recovery period. Each subject acts as his own control.

MATERIAL AND METHODS

The observations were made on 20 adult patients without clinical or radiological evidence of respiratory or cardiovascular disease (Table I), who were anaesthetized for cerebral angiographic procedures. The patients had remained in bed for the previous twelve hours. On arrival in the induction room a no. 15 central venous pressure catheter was placed in the superior vena cava through a basilic or cephalic vein and an intravenous infusion of 0.85 per cent saline was started. A radial artery was cannulated with a no. 20 Argyll plastic needle and kept patent with heparinized saline.

At the same time an oesophageal balloon (length 10 cm, perimeter 3.8 cm) sealed over a catheter (i.d. 0.14 cm, o.d. 0.19 cm, length 85 cm) was inserted through the nose to a position 45 cm from the nostril, and filled with 0.5 ml of air. This was connected to one part of a 268B Sanborn differential pressure transducer, the other part being attached to the patient's airway.

Respiratory flow was measured with a no. 2 Fleisch pneumotachograph and integrated electronically to show volume. Pressure, flow, and volume were displayed on a Honeywell UV Visicorder. Except for a short period of time following the intravenous injection of succinylcholine and the endotracheal intubation, all patients were allowed to breathe spontaneously from a non-rebreathing system using a Rudolph valve.

Inspired gas was sampled from the inspiratory limb of the anaesthetic machine; expired gas was collected simultaneously in a meteorologic bag. Blood and respiratory gases were analysed for Po_2 and Pco_2 in an IL 113 gas analyser within

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ten minutes of the sampling. Correction was made for temperature differences between the patient and the measuring electrode. The Po_2 electrode was assumed to under-read by 2 per cent when blood was to be analysed, and the values were adjusted. pH was measured using an Astrup semi-micro apparatus.

A Beckman cardiodensitometer coupled to a 906 Harvard pump recorded the curve produced by the intravenous injection of 5 mg indocyanine green, from which the cardiac output was calculated.

TABLE I

AGE, SEX, WEIGHT, AND HEIGHT OF THE PATIENTS

Patients	Age (years)	Sex	Weight (kg)	Height (cm)
1	53	M	90	185
2	41	M	88	190
3	41	F	57	160
4	27	M	89	176
5	50	M	70	179
6	29	M	82	182
7	42	M	82	190
8	54	M	72	170
9	34	M	83	178
10	23	F	47	157
11	48	F	71	170
12	53	F	43	165
13	29	M	75	168
14	57	F	50	160
15	36	F	97	165
16	47	M	62	168
17	52	F	44	152
18	33	M	89	183
19	49	M	103	175
20	48	F	49	160
Mean	42.3		72.2	172.0
S.D.	10.3		18.7	11.0

Assuming $PA_{CO_2} = Pa_{CO_2}$, PA_{O_2} was calculated by the following equation:

$$PA_{O_2} = PI_{O_2} - PA_{CO_2} \times \frac{PI_{O_2} - PE_{O_2}}{PE_{CO_2}}.$$

AaD_{O_2} and $a\bar{v}_{O_2}$ were derived by subtraction. Assuming $Pc_{O_2} = PA_{O_2}$, physiological shunt was computed thus:

$$\frac{\dot{Q}s}{\dot{Q}t} = \frac{Cc_{O_2} - Ca_{O_2}}{Cc_{O_2} - C\bar{v}_{O_2}}.$$

Physiological dead space was derived from Bohr's equation:

$$V_D = V_T \times \frac{Pa_{CO_2} - PE_{CO_2}}{Pa_{CO_2}} - V_{D_{apparatus}}.$$

Oxygen consumption was determined from Fick's equation:

$$\dot{V}_{O_2} = \dot{Q}t(Ca_{O_2} - C\bar{v}_{O_2}).$$

Dynamic lung compliance was taken as a mean of five successive measurements. The paired t-test was used to compare each set of measurements with the preceding one.

All measurements were carried out in the supine position; the patients were breathing air when awake and a 50:50 O_2/N_2O mixture when anaesthetized. Five conditions were considered:

1. Before atropine.

2. 37 ± 17 min after atropine (i.e. 0.015 mg/kg intravenous atropine).

3. 16 ± 4 min after induction and intubation (i.e. 4.8 ± 1.4 mg/kg Penthotal and 1.1 ± 0.3 mg/kg succinylcholine, anaesthesia being maintained with 0.5–1.0 per cent halothane).

4. 96 ± 51 min after induction.

5. 38 ± 11 min after the end of the anaesthesia (i.e. when complete consciousness was regained).

RESULTS (TABLE II)

Dynamic lung compliance (C_{dyn}) was unaffected by atropine, but decreased significantly after induction, from 124 ml/cm H_2O (f = 16) to 84 ml/cm H_2O (f = 27). No further impairment was noticed with time. After recovery the compliance increased but did not return to preoperative values (98 ml/cm H_2O at f = 18). C_{dyn} measurements were repeated, before induction, at higher than normal frequencies (f = 26) in 13 patients. Although the preoperative values were lower at these high frequencies, a subsequent fall in C_{dyn} was observed after induction.

Atropine had no influence on the V_D/V_T (from 0.32 to 0.30), but induction caused a highly significant increase of this parameter (from 0.30 to 0.53). Following recovery the V_D/V_T returned to its preoperative level (0.32).

$\dot{Q}s/\dot{Q}t$ increased slightly after atropine (from 8.77 to 9.12 per cent) and markedly after induction (14.22 and 12.58 per cent). However, none of these changes reached the 5 per cent level of significance. After recovery no improvement was apparent.

The atropine effect on the $\dot{Q}t$ was of no significance. Induction caused a fall in $\dot{Q}t$ (from 4.19 L/min to 3.92 L/min), which was highly significant. Recovery increased the $\dot{Q}t$ to its preoperative level (4.64 L/min).

$\dot{V}o_2$ and $a\bar{v}o_2$ were not significantly affected by atropine, but during anaesthesia a decrease in $\dot{V}o_2$ (from 234 ml/min to 123 ml/min), and in $a\bar{v}o_2$ (from 4.55 to 3.02 vol per cent) was observed. These values tended to return to their initial levels during the recovery period.

When similar FI_{O_2} values were considered, i.e. in the awake state or during anaesthesia, no changes in $aaDo_2$ could be demonstrated. The increased $aaDo_2$ observed with induction is biased by the increase in FI_{O_2}. The importance of this effect is discussed elsewhere.[21]

DISCUSSION

C_{dyn} was found to be decreased early after induction, and this reduction was sustained during anaesthesia. The values found are in agreement with those of

TABLE II

OBSERVED AND DERIVED DATA: MEAN VALUES AND STANDARD DEVIATION

	Before premedication	After premedication	Early after induction	Late after induction	After recovery
V_T (ml)	662 ±201	520″ ±94	340″ ±94	356 ±71	492″ ±81
f (b.p.m.)	16.3 ±4.8	16.2 ±3.1	26.8″ ±6.0	24.1 ±3.6	17.7 ±3.7
$\dot{V}_I$ (L/min)	10.2 ±3.1	8.2 ±1.5	8.8 ±2.4	8.7 ±2.0	8.7 ±2.5
C_{dyn} (ml/cm H_2O)	126 ±48	124 ±44	84″ ±22	81 ±26	98″ ±29
C_{dyn} at increased f (ml/cm H_2O)	111 ±44	112 ±43	81″ ±22	76 ±25	94′ ±25
V_D (ml)	208 ±88	158′ ±48	179 ±56	194 ±56	162′ ±65
V_D/V_T	0.32 ±0.05	0.30 ±0.07	0.53″ ±0.11	0.53 ±0.11	0.32″ ±0.09
$\dot{Q}s/\dot{Q}t$ (%)	8.77 ±4.31	9.12 ±6.81	14.22 ±7.62	12.58 ±5.13	14.93 ±10.24
$\dot{Q}t$ (L/min)	4.76 ±1.12	5.08 ±1.24	4.19″ ±0.97	3.92′ ±1.12	4.64″ ±1.41
$a\bar{v}O_2$ (vol %)	5.12 ±1.32	4.55 ±1.24	3.02″ ±1.06	3.82″ ±0.73	4.54′ ±1.41
$\dot{V}O_2$ (ml)	242 ±90	234 ±78	123 ±56	140 ±40	210 ±94
PA_{O_2} (torr)	105.6 ±8.6	106.6 ±9.4	299.2 ±35.1	303.0 ±23.4	99.2 ±10.5
Pa_{O_2} (torr)	79.7 ±10.8	83.8 ±10.2	183.5 ±39.0	175.6 ±36.6	73.7 ±12.3
AaD_{O_2} (torr)	26.0 ±10.6	24.7 ±9.5	115.7 ±51.6	127.4 ±33.0	25.6 ±9.4
Pa_{CO_2} (torr)	36.8 ±3.7	36.1 ±4.3	45.0″ ±5.2	44.9 ±4.7	39.6″ ±5.9
pH	7.43 ±0.03	7.43 ±0.03	7.36″ ±0.04	7.35 ±0.03	7.39″ ±0.03
HR	74 ±9	87″ ±14	80′ ±11	78 ±8	83 ±8
BA_s (torr)	134 ±24	130 ±23	122 ±30	121 ±19	126 ±17

′Significant difference: $0.01 < p < 0.05$.
″Highly significant difference: $p < 0.01$.

previous studies in anaesthetized, spontaneously breathing subjects.[1,3-6] Gold and Helrich[4,5] showed the decrease in C_{dyn} to appear six and ten minutes after induction. Wu, Miller, and Luhn[6] observed a similar fall two hours after induction, as did Colgan and Whang,[1] who, however, did not specify the time of their measurements.

In keeping with studies by Gold et al.,[4,5] we noticed C_{dyn} to decrease with lower V_T values. Shallow tidal ventilation may in itself cause a fall in C_{dyn} due to the closure of small airways at low inflation volumes.[7] When the trapped

gases are oxygen and nitrous oxide, as in the present study, rapid absorption and atelectasis are likely to occur. In the presence of airway closure and atelectasis, without changes in regional perfusion, one expects an increase in $\dot{Q}s/\dot{Q}t$. Increased physiological shunts of the same order of magnitude as the compliance changes were not detected in this study.

One is left with the possibility that the reduction of C_{dyn} was related to distribution changes inside the airways. The anaesthetic drugs might affect the broncho-motor tone and the time constants of the millions of respiratory units, and/or might have an action on the surfactant with an increase in the surface tension inside the alveoli. In both situations one would expect physiological dead space to be increased, though $\dot{Q}s/\dot{Q}t$ need not be affected, if there were concomitant falls in $\dot{Q}t$ and $\dot{V}o_2$ as observed in the present work. Previous studies in man have not demonstrated any effect of anaesthetic agents on surfactant in similar conditions.[8,9]

No further deterioration in C_{dyn} was demonstrated up to 96 minutes after induction, a finding in keeping with the report of Colgan et al.,[1] who were unable to show any significant change up to 60 minutes after induction.

Endotracheal intubation is known to cut down the $V_{D_{anat}}$ by about 70 ml.[10] As V_D did not change after induction, an increase in $V_{D_{alv}}$ must be accepted. The latter can be due to the above mentioned distribution changes and/or to irregu-larities in the $\dot{V}A/\dot{Q}c$ relationship. Indeed, the observed fall in $\dot{Q}t$ could result in a decreased perfusion of the horizontally upper regions of the lungs, causing some alveoli to be well ventilated and poorly perfused.[11]

No progressive change in Pa_{O_2} could be demonstrated with anaesthesia, a finding in agreement with the reports of Gold et al.[5] and Colgan et al.[1] The mean Pa_{O_2} measured in the postoperative period was lower than the preoperative value, as was PA_{O_2}; as a result $AaDo_2$ did not change. $\dot{Q}t$ and $\dot{V}o_2$ were on the average back to the preoperative values; Pa_{CO_2} was slightly higher. The most likely explanations for this fall in Pa_{O_2} are hypoventilation and/or the nitrous oxide elimination phenomenon, diluting the oxygen in the alveoli. Our postopera-tive results agree with those of Stephen and Talton,[12] who reported arterial hypoxaemia in all their patients allowed to breathe room air in the immediate postoperative period, as well as with the findings of Bay, Nunn, and Prys-Roberts[13] and Nunn and Payne.[14]

The lack of progressive changes in $\dot{Q}s/\dot{Q}t$ and $AaDo_2$ observed up to 96 minutes in the anaesthetized state is in agreement with the reports of Marshall[15] and Panday and Nunn,[2] who were unable to detect any significant deterioration of these parameters for periods up to 70 and 60 minutes respectively. However, it should be emphasized that, although no significant difference could be demon-strated between any set of $\dot{Q}s/\dot{Q}t$ measurements and its preceding one, 13 of 16 patients showed an increase in this parameter when the set obtained late after induction was compared with those obtained before induction.

$\dot{Q}s/\dot{Q}t$ values in awake subjects breathing air are the result of both $\dot{V}A/\dot{Q}c$ inequalities and physiological shunt, as diffusion limitation does not play an important role with PA_{O_2} at or above 100 torr.[16] When the patient breathes 50 per cent oxygen, the distribution component is thought to be eliminated.[17]

Although $\dot{Q}s/\dot{Q}t$ did not change significantly during anaesthesia, the possibility remains that some atelectatic areas developed, but that their effect on the venous admixture was offset by the decrease in the distribution component after O_2 breathing. This hypothesis is not supported by the absence of changes in $aaDo_2$ results, which we reported elsewhere.[21] As a distribution component cannot be invoked, one must therefore consider that the same degree of atelectasis, if any, is present before and after induction.

However, the somewhat unlikely possibility cannot be excluded of a progressive increase in venous admixture occurring along with a decrease in the distribution component, as low $\dot{V}a/\dot{Q}c$ areas keep washing out part of their nitrogen.

A decrease in $\dot{Q}t$ will magnify the effects of any given $\dot{Q}s/\dot{Q}t$ on $aaDo_2$, provided the $\dot{V}o_2$ is constant.[18] If both $\dot{Q}t$ and $\dot{V}o_2$ fall at the same rate, the difference $Ca_{O_2} - C\bar{v}_{O_2}$ will remain unchanged. Actually a decrease in both parameters was found, and this was consistent with the failure to demonstrate any significant change in $\dot{Q}s/\dot{Q}t$. The $\dot{V}o_2$, however, fell proportionally more than the $\dot{Q}t$, as demonstrated by the decrease in $Ca_{O_2} - C\bar{v}_{O_2}$ with anaesthesia; but the expected decrease in $aaDo_2$ was not observed. This may be the consequence of assuming the oxygen content of the svc to be equal to the $C\bar{v}_{O_2}$. The direction of changes, however, in $C\bar{v}_{O_2}$ may validly be sensed by samples obtained from the svc.[19]

The lack of correlation between the changes in C_{dyn} and $\dot{Q}s/\dot{Q}t$ confirms the findings of Collier and Mead in dogs.[20]

In conclusion, three major observations were made:

1. Induction of anaesthesia was followed by a fall in C_{dyn}, probably due to a change in the time constants of the different parts of the lungs. Atropine and progression of anaesthesia up to 96 minutes did not alter C_{dyn} to any significant degree.

2. $\dot{Q}s/\dot{Q}t$ measurements were not different at any stage of the study, despite the fact that $\dot{Q}t$ fell during anaesthesia. The concomitant and probably proportional decrease in $\dot{V}o_2$ seems a valid explanation for this finding. It should be stressed, however, that 13 of 16 patients showed an increased $\dot{Q}s/\dot{Q}t$ late after induction, when values were compared with the preinduction ones. A closure of some units is thus suggested.

3. No significant changes in $aaDo_2$ were observed with atropine during the course of anaesthesia or after recovery, at identical F_{IO_2}.

SUMMARY

Lung mechanics and blood gases were studied throughout the course of anaesthesia in 20 patients undergoing cerebral angiographic procedures. Measurements started before premedication and continued into the recovery period, each subject acting as his own control.

Premedication (0.015 mg/kg atropine) did not affect C_{dyn}, $\dot{Q}s/\dot{Q}t$, and $aaDo_2$. Induction of anaesthesia resulted in a significant fall in C_{dyn}, but no alteration in $\dot{Q}s/\dot{Q}t$. There were no changes in C_{dyn}, $\dot{Q}s/\dot{Q}t$, and $aaDo_2$ up to 96 minutes after induction. C_{dyn} returned to preoperative values during the recovery period, and $\dot{Q}s/\dot{Q}t$ and $aaDo_2$ remained unaffected.

The fall in C_{dyn} was probably due to a change in the time constants of the different parts of the lungs. The concomitant and probably proportional decrease in $\dot{Q}t$ and $\dot{V}O_2$ seems a valid explanation for the lack of changes in $\dot{Q}s/\dot{Q}t$. Thirteen out of sixteen patients, however, showed an increased $\dot{Q}s/\dot{Q}t$ late after induction, when values were compared with the preinduction ones. A closure of some units is thus suggested.

RÉSUMÉ

Les valeurs de compliance pulmonaire et des gaz sanguins furent relevées chez 20 patients anesthésiés pour des examens angiographiques cérébraux. Les observations commencèrent avant toute prémédication et se poursuivirent jusqu'à la période du réveil dans chaque cas.

L'injection intraveineuse d'atropine n'eut aucun effet sur la compliance dynamique, le shunt ou le gradient alvéolo-capillaire. L'induction anesthésique provoqua une diminution de la compliance, mais n'affecta pas le shunt. Les valeurs de la compliance, du shunt et du gradient alvéolo-capillaire, relevées après 96 minutes d'anesthésie, ne présentèrent aucune altération subséquente. Au réveil, la compliance redevint normale, tandis que le shunt et le gradient restaient inchangés.

La diminution de la compliance est imputable aux variations de constante de temps à l'intérieur des poumons. Les changements simultanés et parallèles du débit cardiaque et de la consommation d'oxygène fournissent une explication satisfaisante à l'absence de variation observée dans le shunt.

Treize patients sur seize cependant présentèrent une augmentation du shunt lorsque les valeurs peranesthésiques furent comparées aux valeurs de contrôle. Ceci semble indiquer la fermeture de certains segments pulmonaires.

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