THE EFFECT OF THORACIC EXTRADURAL ANALGESIA ON PULMONARY GAS DISTRIBUTION, FUNCTIONAL RESIDUAL CAPACITY AND AIRWAY CLOSURE

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SUMMARY

Pulmonary gas distribution, functional residual capacity (FRC), closing capacity (CC), arterial oxygen tension \( (P_{A\text{O}_2}) \) and alveolar–arterial oxygen tension gradient \( (P_{A\text{O}_2} - P_{A\text{O}_2}) \) were measured in seven subjects before and after the induction of extradural analgesia for routine surgery. It was found that pulmonary gas distribution was within normal limits throughout the study, although there were two patients in whom airway closure occurred consistently within the tidal volume. In both cases this was associated with a low \( P_{A\text{O}_2} \), CC and FRC were substantially unchanged by the induction of extradural analgesia. Changes in \( (P_{A\text{O}_2} - P_{A\text{O}_2}) \) and \( P_{A\text{O}_2} \) were usually not large, and are apparently related to factors other than changes in lung geometry.

It is well known that the induction of anaesthesia is associated with a decrease in \( P_{A\text{O}_2} \) and an increase in the alveolar–arterial oxygen tension gradient \( (P_{A\text{O}_2} - P_{A\text{O}_2}) \) (Nunn, Bergman and Coleman, 1965; Sykes, Young and Robinson, 1965). A possible explanation for this relative hypoxaemia is the occurrence of airway closure within the tidal volume, brought about by a reduction in functional residual capacity (FRC) (Hedenstierna, McCarthy and Bergström, 1975). Since such hypoxaemia cannot be demonstrated after the induction of thoracic extradural analgesia (Sjögren and Wright, 1972), a study was undertaken to measure the effect of thoracic extradural analgesia on functional residual capacity, pulmonary gas distribution and airway closure.

PATIENTS AND METHODS

Seven patients undergoing extradural analgesia for routine surgery were studied. All were considered to be free from cardiovascular disease. Most were moderate or heavy smokers and three had a chronic unproductive morning cough. There was no other clinical or radiological evidence of lung disease. The nature and extent of the investigation had been explained to the subjects and their agreement was obtained. There were no complications. Further clinical details are shown in table I.

The measurement of FRC and intrapulmonary gas distribution

FRC was measured by means of a nitrogen washout technique, the expire being collected in a Douglas bag and its nitrogen content measured subsequently. Oxygen administration was discontinued when the nitrogen concentration was less than 2%. Corrections were made for gas impurities \(- 0.2\% N_2\) and for nitrogen dissolved in body fluids \(- 0.7\% N_2\). The final end-expiratory nitrogen concentration was assumed to correspond to that in the alveoli. This technique permitted also the quantitative determination of intrapulmonary gas distribution by measuring the end-expiratory nitrogen content of each breath, and plotting the values obtained on semilogarithmic paper. A fractional analysis could then be performed and a gas distribution index calculated (Fowler, Cornish and Kety, 1952).

Blood-gas tensions and the measurement of \( (P_{A\text{O}_2} - P_{A\text{O}_2}) \)

Arterial \( P_{O_2} \) and \( P_{CO_2} \) were measured from samples drawn from a Teflon catheter inserted previously into the femoral or radial artery. Two samples were taken for each estimation, at least 15 min after the insertion of the catheter. Blood-gas analysis was performed by means of an oxygen polarograph and a Severinghaus carbon dioxide electrode (E 5046, E 5036, Radiometer). The mean of the two estimations was used for a simplified calculation of \( (P_{A\text{O}_2} - P_{A\text{O}_2}) \) according to the following formula:

\[
(P_{A\text{O}_2} - P_{A\text{O}_2}) = P_{1\text{O}_2} - \frac{P_{A\text{CO}_2}}{R} - P_{A\text{O}_2},
\]

where \( P_{1\text{O}_2} \) is the oxygen tension in the inspirate and is taken to be 149 mm Hg, \( R \) is the respiratory exchange ratio and is taken to be 0.8.

Measurement of airway closure

Airway closure was measured according to the method of Anthonisen and colleagues (1970), using

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nitrogen as a tracer gas (measuring instrument: Nitralyzer 505, Med. Science), while gas volumes were measured by means of a pneumotachograph (flow head: Fleisch No. 1, Godardt; differential pressure meter EMT 32 and amplifier EMT 31, Siemens-Elema; with an integrator built in this department). Both signals were recorded on an X-Y writer (Bryans 26000). The inspirate could be changed from air to oxygen, and the expirate was collected in a Douglas bag, by turning a series of taps. The apparatus is described and illustrated more fully elsewhere (Hedenstierna and McCarthy, 1975). The pneumotachograph was calibrated with air, and the volumes of the oxygen-enriched expirate registered were divided by a factor of 1.05, to compensate for changes in viscosity resulting from oxygen enrichment of the expirate (Hedenstierna, McCarthy and Bergström, 1975).

Procedure

The subject breathed air and a normal spirogram was obtained, volume being recorded on the X-axis. The subject then breathed out to residual volume (RV) and inhaled fully, oxygen being administered on the inspiratory limb at maximum expiration. He was then asked to exhale slowly and completely, while the nitrogen content of the expirate was recorded on the Y-axis. This was made possible by a simple switch on the X-Y writer. The commencement of airway closure was taken to be that point on a “best fit” line through the alveolar plateau where a secondary consistent increase in nitrogen concentration commenced (Phase IV). The volume at which this secondary increase takes place, as measured from RV, is closing volume (CV) and the sum of CV and RV closing capacity (CC) (nomenclature according to Anthonisen). CV (or CC) could thus be compared directly with the resting expiratory level (corresponding to FRC) on the spirogram.

Extradural analgesia

The T5–T6 interspace was identified, and an 18-s.w.g. Tuohy needle was inserted laterally into the extradural space, which was identified by the loss of resistance to the injection of air. A “Portex” catheter was inserted cranially to a distance of 2–3 cm from the tip of the needle. The needle was then withdrawn and the catheter strapped in position. Extradural blockade was induced by the injection of 9–10 ml of 2% mepivacaine (Carbocaine, Bofors) without adrenaline, through the extradural catheter. By this means, analgesia was induced to a level of T2 or T3, extending to T12 or L1, as assessed by the loss of temperature discrimination on spraying the skin with ethyl chloride. In one patient (case 1), extradural analgesia was induced, using a catheter at T12–L1 inserted cranially to a distance of approximately 10 cm, by injecting mepivacaine 2% 19 ml; analgesia extended from T2 to L4–5.

The investigations were performed in the morning, a dose of diazepam 0.2 mg/kg having been administered 1 hr previously. After the commencement of an i.v. infusion of 10% invertose (Inverdes, Pharmacia), a polythene catheter was inserted into the thoracic extradural space, as described. A Teflon catheter was inserted into the radial or femoral artery under local anaesthesia, and the patient was allowed to rest for a period of 10–15 min. Samples of arterial blood were then withdrawn, and the nitrogen washout performed twice. After a further 10–15 min while the subject breathed air, airway closure was measured on four or five occasions. Analgesia was then induced through the extradural catheter, and, after a further period of 25–30 min, when extradural analgesia was complete,
the investigations were repeated. A physiological electrolyte solution (Ringerdex, Pharmacia) was administered, to maintain the arterial pressure within an acceptable range. After all investigations had been performed, a further suitable dose of mepivacaine was administered and surgery was performed. In some cases nitrous oxide and oxygen were administered during surgery, supplemented in two cases (2 and 7) with neuromuscular blocking drugs. The extradural catheter was retained for analgesia after surgery.

RESULTS

Arterial oxygenation

The initial values of \((PaO_2 - PaO_2)\) and \(PaO_2\) were within normal limits in all patients (Raine and Bishop, 1963). In two cases, an appreciable increase in \((PaO_2 - PaO_2)\) occurred following the induction of extradural analgesia. In one case, an appreciable decrease in \((PaO_2 - PaO_2)\) occurred. Otherwise, changes in \((PaO_2 - PaO_2)\) and \(PaO_2\) were small.

Gas distribution

Intrapulmonary gas distribution was within normal limits throughout the investigations (Fowler, Cornish and Kety, 1952; Bouhuys, 1963). The results of fractional analysis performed on nitrogen washout curves showed, in the majority of cases, a completely uniform gas distribution both before and after the induction of extradural analgesia; no division into different lung compartments could be made.

FRC and vital capacity (VC)

FRC and VC measured before the induction of extradural analgesia were both within normal limits for recumbent subjects (Kaltreider, Fray and Hyde, 1938). They were substantially unchanged by the induction of extradural analgesia.

Airway closure

CC was approximately the same before and after the induction of extradural analgesia. Airway closure occurred above FRC in two patients (cases 3 and 7) both before and during extradural analgesia. Both patients were aged more than 60 yr, cigarette smokers, with comparatively high values of \((PaO_2 - PaO_2)\) and low values of \(PaO_2\).

DISCUSSION

The first manoeuvre in the technique used for measuring airway closure is expiration to RV. This might be expected to be hampered by the induction of extradural analgesia with accompanying motor block. However, this was so in only one patient (No. 7), when RV was increased by 140 ml. Even in this case, the inability to expire fully cannot have altered the registration of airway closure, since it has been shown (Hedenstierna, McCarthy and Bergström, 1975) that expiration to a point midway between FRC and RV does not render the subsequent measurement of airway closure unreliable. Secondly, the method used requires inspiration of oxygen to VC. Our patients were all premedicated, and in the supine position. Whether the reduction in VC brought about by this position (Comroe et al., 1962) alters the ensuing measurement of airway closure is not clear. There was, however, no further reduction in VC following the induction of extradural analgesia (fig. 1), a finding in agreement with the observations of Sjögren and Wright (1972). So it may reasonably be assumed that the measurements of airway closure are reliable, in spite of the motor blockade associated with extradural analgesia.

<table>
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<tr>
<th>Patient</th>
<th>(PaO_2) (mm Hg)</th>
<th>((PaO_2 - PaO_2)) (mm Hg)</th>
<th>FRC (litre)</th>
<th>CC (litre)</th>
<th>VC (litre)</th>
<th>NWOD (min)</th>
<th>Arterial pressure (mm Hg)</th>
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</table>

NWOD = nitrogen washout delay.
NO THORACIC ANALGESIA EXTRADURAL

FIG. 1. Mean functional residual capacity (FRC), closing capacity (CC), vital capacity (VC), nitrogen washout delay (NWOD), alveolar-arterial oxygen gradient \((P_{\text{A}O_2} - P_{\text{a}O_2})\) before and after the induction of thoracic extradural analgesia. J = SEM.

\((P_{\text{A}O_2} - P_{\text{a}O_2}), P_{\text{a}O_2} \text{ and extradural analgesia}\)

In two subjects (3 and 7), both cigarette smokers with a chronic cough, a comparatively low arterial oxygen tension was recorded after premedication. It is the author’s practice to use thoracic extradural analgesia in such patients undergoing upper abdominal surgery, which explains the high proportion of such patients in the material presented.

An appreciable decrease in \(P_{\text{a}O_2}\) was recorded in two patients under extradural analgesia (cases 1 and 5).

**Case 1** was a healthy lady, a non-smoker aged 27 yr. After premedication \(P_{\text{a}O_2}\) was 97 mm Hg, and \((P_{\text{A}O_2} - P_{\text{a}O_2})\) 4 mm Hg. After the induction of thoracic extradural analgesia \(P_{\text{a}O_2}\) decreased to 85 mm Hg, and \((P_{\text{A}O_2} - P_{\text{a}O_2})\) increased to 15 mm Hg. The arterial pressure decreased from 115/80 mm Hg to 75/50 mm Hg.

**Case 5**, a male aged 34 yr, had chronic pancreatitis, and was a heavy smoker with a chronic morning cough. After premedication \(P_{\text{a}O_2}\) was 83 mm Hg, but decreased to 67 mm Hg after the induction of extradural analgesia. \((P_{\text{A}O_2} - P_{\text{a}O_2})\) increased from 23 mm Hg to 39 mm Hg. This was accompanied by a decrease in arterial pressure from 120/80 mm Hg to 85/60 mm Hg.

It may be that a hypokinetic circulation reflected by the decrease in arterial pressure provides an adequate explanation for the decrease in arterial oxygenation in both cases (Eckenhoff et al., 1963). On the other hand, an appreciable increase in arterial oxygenation occurred in one patient (case 7).

**Case 7**, a 64-year-old male, a moderate smoker with a chronic morning cough, was sleepy after premedication. \(P_{\text{a}O_2}\) was 74 mm Hg. After the induction of extradural analgesia, \(P_{\text{a}O_2}\) had increased to 88 mm Hg and \((P_{\text{A}O_2} - P_{\text{a}O_2})\) decreased from 32 mm Hg to 21 mm Hg. The patient had become more alert as tests for loss of sensation were performed, and had presumably cleared his airways of secretions.

It is thus apparent that the comparatively small changes in arterial oxygen tension recorded can be explained by factors other than changes in respiratory parameters. The finding that changes in \(P_{\text{a}O_2}\) following the induction of extradural analgesia are slight is in agreement with the findings of other workers (Sjögren and Wright, 1972).

**FRC and airway closure**

FRC was within normal limits for healthy recumbent subjects (Kaltreider, Fray and Hyde, 1938). The changes induced by extradural analgesia were slight, and are within the tolerance of accuracy of the method used. There appears to be no published data on the effect of thoracic extradural analgesia on FRC, although Bromage has stated that changes are minimal (Bromage, 1967). The results of the measurement of airway closure after premedication are in agreement with previously published data (Hedenstierna, McCarthy and Bergström, 1975). Airway closure occurred above FRC (within a normal tidal volume) in two patients out of seven. Both (cases 3 and 7) were smokers aged over 60 yr and thus belonged to a category of subjects in whom a large CC was to be expected (McCarthy et al., 1972). The occurrence of airway closure within a normal tidal volume was associated with low arterial oxygen tension and high \((P_{\text{A}O_2} - P_{\text{a}O_2})\) in both cases.

There was little change in CC after the induction of extradural analgesia in any of the subjects. In a previous publication (Hedenstierna, McCarthy and
Bergström, 1975) it was suggested that the increased \((P\text{O}_2 - P\text{A}O_2)\) known to occur following the induction of anaesthesia and artificial ventilation may be explained by the occurrence of airway closure within a normal tidal volume, as a result of a reduction in FRC.

It is a common clinical impression that thoracic extradural analgesia is a suitable method for abdominal surgery in the presence of chronic lung disease. The evidence presented here supports this contention. It may be that with extradural analgesia the maintenance of a normal FRC accounts for the fact that arterial oxygen changes are usually small.

REFERENCES


EFFET DE L’ANALGESIE EXTRADURALE THORACIQUE SUR LA REPARTITION DU GAZ PULMONAIRE, LA CAPACITÉ RESIDUELLE FONCTIONNELLE ET SUR LA FERMETURE DES PASSAGES D’AIR

RESUME

La répartition du gaz pulmonaire, la capacité résiduelle fonctionnelle, la capacité de fermeture, la tension d’oxygène artériel \((P\text{A}O_2)\) ainsi que la courbe de la tension de l’oxygène artériel-alvéolaire \((P\text{A}O_2 - P\text{A}O_2)\) ont été mesurées sur sept sujets avant et après l’induction de l’analgésie extradurale pour les interventions chirurgicales courantes. On a trouvé que la répartition de gaz pulmonaire était dans les limites normales pendant toute la durée de l’étude, bien qu’il y avait deux patients chez lesquels la fermeture des passages d’air se trouvait constamment dans les limites du volume courant. Dans ces deux cas, cela a été associé à une faible tension d’oxygène artériel \((P\text{A}O_2)\). La capacité de fermeture et la capacité résiduelle fonctionnelle sont restées virtuellement inchangées par l’induction de l’analgésie extradurale. Les variations dans le \((P\text{A}O_2 - P\text{A}O_2)\) n’ont pas été très importantes et elles sont apparemment liées à des facteurs autres que les variations dans la géométrie des poumons.

ZUSAMMENFASSUNG

Pulmonale Gasverbreitung, funktionelle Residualluft-Kapazität (FRC), Verschlusskapazität (CC), arterieller Sauerstoffdruck \((P\text{A}O_2)\) und alveolar-arterieller Sauerstoffdruck \((P\text{A}O_2 - P\text{A}O_2)\) wurden bei sieben Patienten vor und nach Einleitung extraduraler Analgesie zwecks Routinechirurgie ermessenen. Während der Untersuchung konnte festgestellt werden, dass sich die Lungengas-Verteilung innerhalb normale Grenzen befand, obwohl bei zwei Patienten durchweg Luftwechselverschleiß innerhalb des Tidevolumens auftrat. Bei beiden Fällen bezog sich dies auf den niedrigeren \(P\text{A}O_2\). CC und FRC blieben verhältnismässig unverändert bei Anwendung von extraduraler Analgesie. Die Veränderungen im \((P\text{A}O_2 - P\text{A}O_2)\) und \(P\text{A}O_2\) waren allgemein nicht gross und sind anscheinend mit anderen Faktoren als denen der Lungengeometrie verbunden.

EFECTO DE LA ANALGESIA EXTRADURAL TORACICA EN LA DISTRIBUCION DE GAS PULMONAR, CAPACIDAD RESIDUAL FUNCIONAL Y CIERRE DEL CONDUCTO DE AIRE

SUMARIO

Se midió en siete pacientes antes y después de la inducción de analgesia extradural para cirugía de rutina, la distribución de gas pulmonar, la capacidad residual funcional (FRC), la capacidad de cierre (CC), la tensión de oxígeno arterial \((P\text{A}O_2)\) y el declive de tensión de oxígeno alveolar-arterial \((P\text{A}O_2 - P\text{A}O_2)\). Se halló que la distribución de gas pulmonar estaba dentro de los límites normales a lo largo del estudio, aunque en dos pacientes...
el cierre del conducto de aire sucedía constantemente dentro del alcance del volumen de la corriente. En ambos casos esto iba acompañado de un \( P_{aO_2} \) bajo. CC y FRC no cambiaron esencialmente por la inducción de analgesia extradural. Los cambios en \( P_{aO_2} \) y \( P_{aO_2} \) no fueron en general grandes, y están ligados aparentemente a factores distintos de los cambios en la geometría del pulmón.