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# *Effects of Continuous Positive-pressure Breathing on Functional Residual Capacity and Arterial Oxygenation during Intra-abdominal Operations:*

## *Studies in Man during Nitrous Oxide and d-Tubocurarine Anesthesia*

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**The effects of 0, 5, 10, and 15 cm $H_2O$ end-expiratory pressure (EEP) were studied in 24 patients receiving nitrous oxide–*d*-tubocurarine anesthesia for intra-abdominal operations. Functional residual capacity (FRC) of every patient was increased during continuous positive-pressure breathing (CPPB). $Pa_{O_2}$ increased an average of 1.6 torr/cm $H_2O$ EEP in the 12 patients with the lowest $Pa_{O_2}$'s (<100 torr at zero EEP). Non-obese patients more than 50 years old showed the greatest improvement in $Pa_{O_2}$. The other 12 patients ($Pa_{O_2}$ > 100 torr at zero EEP) had variable changes in $Pa_{O_2}$ averaging 0.2 torr/cm $H_2O$ EEP. No patient suffered an important decrease in $Pa_{O_2}$ with CPPB. Clinical signs suggesting interference with cardiac output did not occur in the absence of blood loss. (Key words: Continuous positive-pressure breathing; End-expiratory pressure; Functional residual capacity; $Pa_{O_2}$; Oxygenation.)**

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An increase in alveolar–arterial oxygen tension difference has been observed in anesthetized subjects.[1] When inhalation techniques employing high concentrations of nitrous oxide are used, arterial oxygen tensions may be much lower than expected.[2,3] Slater *et al.*[3] found lower $Pa_{O_2}$'s during intra-abdominal operations than during procedures performed on the extremities. Increasing inspired oxygen concentrations ($Ft_{O_2}$) to prevent hypoxia results in increased requirements for adjuvant drugs, each with its own disadvantages. Thus, other methods of improving oxygenation are worth considering.

Reductions in functional residual capacity (FRC) have been observed on changing from the sitting to the supine position,[4,5] during general anesthesia,[6,7] in obesity,[7-9] and following abdominal operations.[10] It has been suggested that such reductions of FRC are associated with airway closure and/or atelectasis. Since FRC is effectively increased with continuous positive-pressure breathing (CPPB), we suggest that this increase can evoke reductions in $A\text{-}aD_{O_2}$. The present study was designed to evaluate the effects of CPPB on FRC and $Pa_{O_2}$ during clinical anesthesia with nitrous oxide.

## Methods

Twelve male and twelve female patients undergoing elective intra-abdominal operations consented to participate in this study. All were free of acute cardiorespiratory disease,

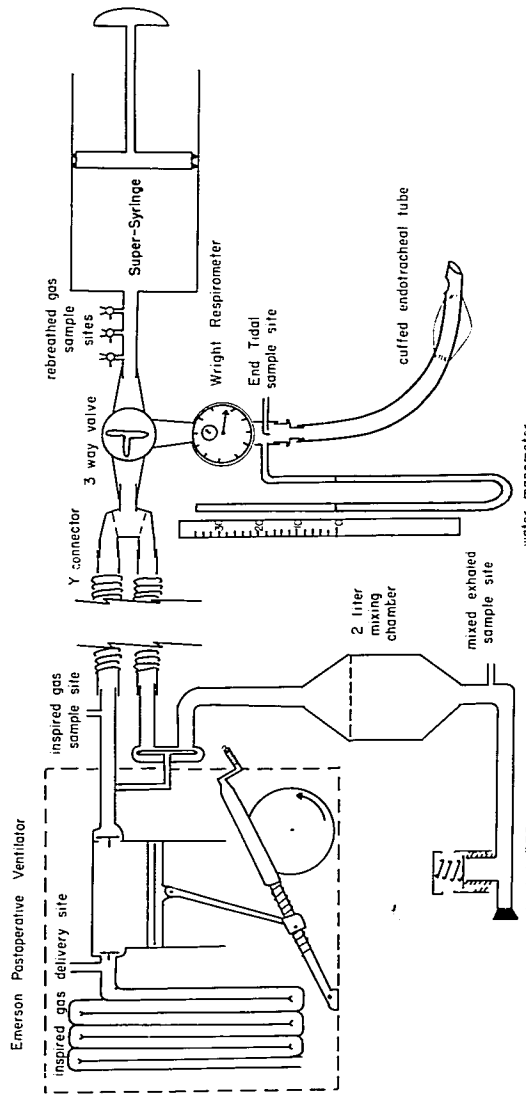


FIG. 1. Diagram of the respiratory circuit, including the super-syringe and three-way tap for the measurement of FRC.

TABLE 1. General Information about the 24 Patients in the Study

| | Age (Years) | Sex | Body Weight (kg) | Height (cm) | Operation | Preoperative $Pa_{O_2}$ (torr) |
|---|---|---|---|---|---|---|
| Patient 1 | 23 | F | 169 | 178 | Cholecystectomy | 97.8 |
| Patient 2 | 49 | M | 116 | 175 | Partial colectomy | 66.9 |
| Patient 3 | 51 | F | 79 | 170 | Cholecystectomy | 72.9 |
| Patient 4 | 52 | M | 100 | 185 | Cystectomy | 80.1 |
| Patient 5 | 53 | F | 120 | 155 | Ventral herniorrhaphy | 62.8 |
| Patient 6 | 59 | M | 47 | 170 | Gastrostomy | — |
| Patient 7 | 59 | M | 72 | 168 | Gastrectomy | 62.5 |
| Patient 8 | 62 | M | 55 | 160 | Cholecystectomy | 82.7 |
| Patient 9 | 63 | M | 55 | 178 | Exploration CBD | 89.3 |
| Patient 10 | 64 | M | 72 | 168 | Whipple | 64.0 |
| Patient 11 | 66 | M | 70 | 175 | Colectomy | 78.5 |
| Patient 12 | 75 | M | 59 | 170 | Removal of jejunal bezoar | 61.5 |
| Patient 13 | 20 | M | 72 | 170 | Gastrectomy | 91.4 |
| Patient 14 | 25 | F | 50 | 152 | Oophorectomy | 107.5 |
| Patient 15 | 28 | F | 52 | 168 | Tuboplasty | — |
| Patient 16 | 32 | F | 46 | 160 | D & C; myomectomy | 79.2 |
| Patient 17 | 33 | F | 53 | 152 | Hysterectomy | 89.1 |
| Patient 18 | 34 | F | 46 | 160 | Hysterectomy | — |
| Patient 19 | 35 | F | 68 | 168 | Hysterectomy | 81.1 |
| Patient 20 | 36 | F | 62 | 173 | Cystectomy | 88.2 |
| Patient 21 | 40 | F | 64 | 163 | Cholecystectomy | 90.0 |
| Patient 22 | 51 | F | 60 | 168 | Transverse colostomy | 89.6 |
| Patient 23 | 56 | M | 55 | 173 | Ureterolithotomy | 90.3 |
| Patient 24 | 63 | M | 61 | 170 | Hemicolectomy | 77.3 |

although most were smokers. Their ages ranged from 20 to 75 years, heights from 152 to 185 cm, and body weights from 46 to 169 kg.

A radial or brachial artery was cannulated prior to induction of anesthesia, except in Patients 6, 15, and 18, in each of whom an arterial cannula was introduced after induction. Preoperative $Pa_{O_2}$ values obtained with the patients supine, breathing air, and premedicated are listed in table 1. Arterial blood pressure, electrocardiogram and nasopharyngeal or esophageal temperature were monitored continuously.

Morphine, 0.1 mg/kg (Patient 11 received Innovar, 1.5 ml), and atropine, 0.5 mg (average dose), were administered an hour before induction of anesthesia. After preoxygenation, anesthesia was induced with sodium thiopental, 4 mg/kg (Patient 11 received Innovar, 7.5 ml). Following the administration of succinylcholine, 1 mg/kg, the trachea was intubated with a cuffed endotracheal tube and a tight seal obtained. Anesthesia was maintained with 70 per cent $N_2O$ and 30 per cent $O_2$ (measured with a Beckman oxygen analyzer). Adjuvant drugs consisted of morphine or fentanyl for analgesia when clinically indicated and *d*-tubocurarine to maintain respiratory paralysis.

Respirations were controlled with an Emerson respirator at a constant tidal volume of 7 ml/kg (measured during expiration at the endotracheal tube with a Wright respirometer), with rates adjusted to maintain a constant arterial carbon dioxide tension ($Pa_{CO_2}$) between 30 and 40 torr. The respiratory circuit is diagrammed in figure 1.

The study period began after the surgeons had packed the abdominal contents and positioned the retractors. A spring-loaded expiratory valve was adjusted to produce 0, 5, 10, or 15 cm $H_2O$ EEP (measured at the trachea with a water manometer) for 15-minute periods in a predetermined, random order for

each patient. In the fifteenth minute of each period, a sample of arterial blood was obtained and sent for immediate analysis.

FRC was measured immediately after obtaining the blood sample. At end-expiration, a three-way stopcock (fig. 1) was turned without changing EEP, and the patient was ventilated with a super-syringe at a rate of 40 breaths/min. One and a half liters of a mixture containing 69.6 per cent $N_2O$, 30 per cent $O_2$, and 0.4 per cent neon were introduced and ventilation maintained with 1-liter tidal volumes. Gas samples of 20 ml were drawn through a manifold during the eighth and tenth breaths. The stopcock was turned again, re-establishing mechanical ventilation, and the next level of EEP was set.

## Analytic Methods

Arterial blood was analyzed for $P_{O_2}$, $P_{CO_2}$, and *p*H, and the raw data corrected for patient–electrode temperature difference and tonometry factor.

Neon was measured by gas chromatography.[11] The identity of the two neon samples indicated that complete mixing had occurred, and FRC was calculated from the syringe volume and neon dilution, without

TABLE 2. Arterial Oxygen Tensions and Functional Residual Capacities with Various End-expiratory Pressures

| | FRC (Per Cent of Predicted Value)* | | | | $Pao_2$ (torr) | | | | Change in $Pao_2$ (torr)/cm $H_2O$ EEP |
|---|---|---|---|---|---|---|---|---|---|
| | 0 EEP | EEP 5 cm $H_2O$ | EEP 10 cm $H_2O$ | EEP 15 cm $H_2O$ | 0 EEP | EEP 5 cm $H_2O$ | EEP 10 cm $H_2O$ | EEP 15 cm $H_2O$ | |
| Patient 1 | 28.7 | 51.6 | 47.7 | 51.6 | 65.0 | 76.4 | 101.5 | 93.3 | 2.2 |
| Patient 2 | 13.4 | 19.4 | 25.8 | 34.4 | 59.3 | 54.4 | 60.1 | 63.6 | 0.4 |
| Patient 3 | 32.6 | 43.4 | 64.2 | 68.8 | 99.2 | 122.7 | 118.8 | 123.0 | 1.4 |
| Patient 4 | 75.3 | 79.7 | 116.9 | 143.6 | 95.2 | 87.8 | 99.0 | 106.3 | 0.9 |
| Patient 5 | 30.1 | 55.7 | 53.6 | 79.2 | 48.6 | 55.4 | 54.2 | 51.5 | 0.2 |
| Patient 6 | 91.3 | 110.4 | 123.3 | 140.3 | 86.7 | 107.4 | 94.8 | 102.9 | 0.7 |
| Patient 7 | 38.8 | 52.5 | 74.3 | 70.7 | 97.4 | 105.4 | 137.6 | 140.8 | 3.2 |
| Patient 8 | 88.7 | 122.7 | — | — | 93.6 | 111.5 | — | — | 3.6 |
| Patient 9 | 96.3 | 77.1 | 117.4 | 146.8 | 100 0 | 114.7 | 109.5 | 106.2 | 0.3 |
| Patient 10 | 72.8 | 93.1 | 117.7 | 110.9 | 72.8 | 74.4 | 89.3 | 97.9 | 1.8 |
| Patient 11 | 45.2 | 67.5 | 124.5 | 102.9 | 69.9 | 108.9 | 86.8 | 111.2 | 2.0 |
| Patient 12 | 101.4 | 117.4 | 113.9 | 128.5 | 76.1 | 101.4 | 128.0 | 114.5 | 2.8 |
| MEAN | 59.6 | 74.2 | 89.0 | 98.0 | 80.3 | 93.4 | 98.2 | 101.0 | 1.6 |
| SE | 9.0 | 9.2 | 11.0 | 11.9 | 5.0 | 6.7 | 7.8 | 7.6 | 0.33 |
| Patient 13 | 73.3 | 86.5 | 100.3 | 116.9 | 152.4 | 156.8 | 161.3 | 161.6 | 0.6 |
| Patient 14 | 69.2 | — | — | 150.6 | 149.4 | — | — | 147.4 | −0.1 |
| Patient 15 | 111.6 | 134.3 | 159.1 | 172.7 | 187.6 | 170.3 | 170.5 | 179.3 | −0.5 |
| Patient 16 | — | 121.7 | 143.9 | 183.6 | — | 165.1 | 161.3 | 150.6 | −1.5 |
| Patient 17 | 60.5 | 75.6 | 83.7 | 113.9 | 127.3 | 122.1 | 115.3 | 122.0 | −0.5 |
| Patient 18 | 53.6 | 109.7 | 125.6 | 158.5 | 159.5 | 163.9 | 149.4 | 155.1 | −0.6 |
| Patient 19 | 76.5 | 92.6 | 111.6 | 135.5 | 120.2 | 141.1 | 138.9 | 133.1 | 0.7 |
| Patient 20 | 55.7 | 65.4 | 82.9 | 100.0 | 158.1 | 164.4 | 164.4 | 158.4 | 0 |
| Patient 21 | 105.9 | 100.9 | 130.6 | 145.7 | 157.4 | 159.6 | 160.5 | 155.0 | −0.1 |
| Patient 22 | 71.5 | 99.2 | 104.9 | 138.4 | 145.7 | 132.5 | 147.9 | 146.8 | 0.4 |
| Patient 23 | 75.7 | — | 142.2 | — | 137.4 | — | 169.0 | — | 3.2 |
| Patient 24 | 76.7 | 102.1 | 124.3 | 131.9 | 140.8 | 139.6 | 157.5 | 149.4 | 0.9 |
| MEAN | 75.5 | 98.8 | 119.0 | 140.7 | 148.7 | 151.2 | 154.2 | 150.8 | 0.21 |
| SE | 5.6 | 6.5 | 7.5 | 7.6 | 5.5 | 5.2 | 4.8 | 4.5 | 0.33 |

* Predicted from table of values for seated subjects minus 600 ml for supine value.

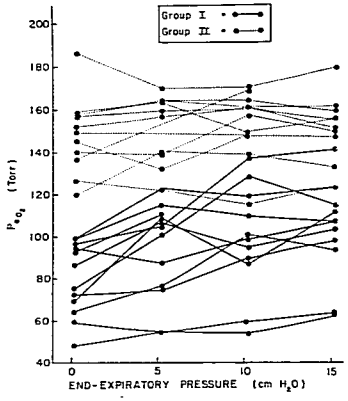


FIG. 2. Arterial oxygen tension as a function of graded increases in end-expiratory pressure for the 24 patients. The solid and dashed lines indicate the patients whose $Pa_{O_2}$'s at zero EEP were below and above 100 torr, respectively.

correction for oxygen uptake and $CO_2$ excretion during the rebreathing period.

## Results

The randomization of the different levels of EEP was critical to the design of this study. The uncontrolled effects of time and surgical manipulation are thereby confounded. They are manifested by the frequent irregularities in progression of the dependent variables, $Pa_{O_2}$ and FRC, for individual patients.

Experimental data are presented in table 2. The patients are separated arbitrarily into two groups: Group I, those with $Pa_{O_2}$'s below 100 torr with zero EEP, and Group II, those with $Pa_{O_2}$'s above 100 torr with zero EEP. The order of data within a row is that of increasing EEP, regardless of the experimental protocol for the individual.

FRC increased significantly ($P < 0.01$) with EEP. Although no statistically significant correlation between EEP and $Pa_{O_2}$ was found for the 24 patients collectively, there was a tendency toward improvement in $Pa_{O_2}$ among the 12 patients whose $Pa_{O_2}$'s did not exceed 100 torr at zero EEP (Group I). The effects of EEP on the patients with $Pa_{O_2}$'s above 100 torr at zero EEP were quite variable (Group II).

Figure 2 shows the change in $Pa_{O_2}$ with EEP for each patient. The slope ($\Delta Pa_{O_2}/\Delta EEP$) of each of these lines was calculated from the least-squares regression equation and the mean value for each group determined (table 2). There was a uniform improvement in $Pa_{O_2}$, averaging 1.6 torr/cm $H_2O$ EEP, among the patients of Group I. The patients in Group II had variable changes in $Pa_{O_2}$, averaging 0.2 torr/cm $H_2O$ EEP. Six patients in the latter group showed decreases in $Pa_{O_2}$ averaging 0.6 torr/cm $H_2O$ EEP, none of which was clinically significant.

Figure 3 plots the slopes for 23 patients (Patient 16 excluded for lack of measurement at zero EEP) as a function of $Pa_{O_2}$ at zero EEP. The dashed horizontal line extending from zero divides positive from negative slopes. Patients with low $Pa_{O_2}$'s at zero EEP generally developed increases in $Pa_{O_2}$. High $Pa_{O_2}$'s at zero EEP were minimally altered. Patients more than 50 years old were among the group with the lowest $Pa_{O_2}$'s at zero EEP and, therefore, showed the most clinically significant improvements in $Pa_{O_2}$. Only one of the three obese patients showed an increase in $Pa_{O_2}$ with EEP, although all had $Pa_{O_2}$'s of less than 100 torr at zero EEP.

The effects of CPPB on the cardiovascular system were not studied. Several transient hypotensive episodes occurred among the 24 patients; all were correctible with intravenous blood or fluid therapy. Hypotension usually coincided with surgical hemorrhage. Although the decrease in arterial blood pressure associated with hypovolemia seemed enhanced by CPPB, as much as 15 cm $H_2O$ EEP was well tolerated when circulating volume was maintained. Cessation of EEP was not associated with signs of fluid overload.

## Discussion

The FRC of almost every patient was below the predicted value [12] at zero EEP, confirming previous reports.[6, 7] Undoubtedly, the limitations of diaphragmatic motion by packs, retractors, and manipulations contributed to the

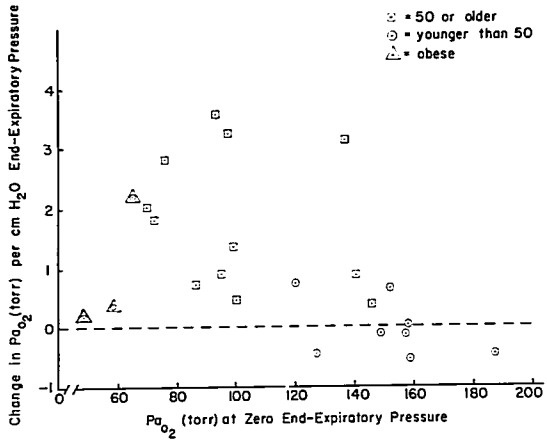


FIG. 3. Mean rate of change of $Pa_{O_2}$ (slope, $\Delta Pa_{O_2}/\Delta EEP$) as a function of $Pa_{O_2}$'s at zero EEP for 23 patients. Patient 16 was omitted for lack of measurement of $Pa_{O_2}$ at zero EEP. The dashed horizontal line extending from zero divides positive from negative rates of change in $Pa_{O_2}$ with increasing EEP.

reductions in FRC. The mean values of FRC (per cent of predicted) for patients of Group I were smaller than those of Group II patients for all values of EEP. Although this difference was not statistically significant at zero EEP, the former group did contain three very obese patients (1, 2, and 5) and a larger number of upper abdominal operations, both of which may enhance reduction of FRC. This, together with the less compliant chest walls of the obese patients and the effects of surgical manipulation, especially during upper abdominal surgery, explains, in part, the larger spread of mean FRC's between the two groups at 5, 10, and 15 cm $H_2O$ EEP.

That EEP increases the volume of gas within the lung is shown nicely by the present data. Most important, however, is the improvement in oxygenation in certain patients. Patients more than 50 years old had the lowest $Pa_{O_2}$'s at zero EEP, and most had clinically significant increases in $Pa_{O_2}$. In contrast, the younger patients generally did not follow this pattern of improvement, since their $Pa_{O_2}$'s were much higher at zero EEP. Factors contributing to the lower $Pa_{O_2}$'s of the patients of Group I are probably age, body weight, and site of operation.

Recent investigations have suggested a causative relationship between reductions in FRC and the closure of small airways in the lung.[12, 14] The functional significance of airway closure is thought to be impairment of ventilation in dependent areas of the lung, resulting in alterations in ventilation–perfusion ($\dot{V}/\dot{Q}$) ratios and, in some circumstances, atelectasis. Thus, the reductions in lung volume that occur in the supine position,[4, 5] during anesthesia,[6, 7] and in obesity [7-9] could be important determinants of the state of oxygenation in the anesthetized patient. Furthermore, airway closure has been shown to occur at much higher lung volumes in older patients,[15, 16] and may explain, in part, our findings of lower $Pa_{O_2}$'s in patients more than 50 years old. With the maintenance of positive pressure at end-expiration, lung volume is increased, the effect of which may be the prevention of airway closure and/or atelectasis. In obese patients, however, 15 cm $H_2O$ EEP may minimally affect FRC because of the increased weight of the chest wall and abdomen.

The optimal level of EEP for any patient is best determined by increasing it until an acceptable level of oxygenation is achieved. For some individuals, *i.e.*, obese, elderly, or hypo-

volemic patients, the cardiovascular effects of CPPB might outweigh any improvement in gas exchange.

Like Frumin and associates,[17] we found that CPPB can alter gas exchange and improve oxygenation in select patients. These results differ from those of Colgen *et al.*, who found in dogs no significant change in A-a$D_{O_2}$ with CPPB.[18] However, the present study was performed during abdominal operations, where limitation of diaphragmatic motion may have contributed to the observed reductions in FRC and $Pa_{O_2}$. Also, we included patients in whom regional $\dot{V}/\dot{Q}$ relations may be significantly altered, as evident from the lower $Pa_{O_2}$'s observed in the older and obese patients. Thus, CPPB, under the conditions described in this report, may be instrumental in improving $Pa_{O_2}$. We do not, however, recommend the use of CPPB in the absence of analysis of arterial blood gases.

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