# Postoperative Respiratory Complications

## A Comparison of Arterial Gas Tensions, Radiographs and Physical Examination

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**A study was made on 27 patients undergoing elective surgery. Preoperative and postoperative chest roentgenograms and arterial blood-gas studies were compared. Temperature, pulse rate, respiratory frequency, and clinical observations were noted. A high incidence (19 of 27) of low oxygen tensions occurring with normal or increased ventilation was observed. No correlation could be established between roentgen-ray or clinical finding on the one hand and laboratory findings on the other.**

**The concept is presented that the findings represent "miliary" atelectasis occurring so diffusely as to escape detection by physical or radiographic examination. More severe reductions in ventilation-perfusion ratios and complete atelectasis are best considered as a continuum since they produce the same effects and may not be distinguishable. Postoperative patients present a pattern of near constant tidal volume without sighing, known to produce these changes.**

**That these changes occur without airway obstruction is suggested by improvement with deep breathing. Although appearing benign, decreases in oxygen tension may be of serious magnitude.**

A RECENT survey of postoperative atelectasis noted wide variation in incidence of this complication.[1] Partial reason for the varying incidence probably is due to difficulty in detecting minute alterations in pulmonary function. A useful method of detecting small areas of atelectasis is now available in the direct measurement of oxygen and carbon dioxide tension of arterial blood. This allows separation of hypoxia due to generalized hypoventilation from that due to decrease in pulmonary ventilation-perfusion ratios. No reports have been found concerning the relative value of blood-gas studies, physical examination and chest roentgenogram as diagnostic procedures. Exact mechanisms producing atelectasis and the role of various etiologic factors are not well defined. The present investigation was designed to compare clinical observation, roentgenograms and arterial blood-gas determinations in the study of postoperative pulmonary status of patients undergoing operation and anesthesia. This is a preliminary phase of a study to further elucidate mechanisms and causative factors of postoperative atelectasis.

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## Methods

Measurements on patients scheduled for elective surgery were made on the day preceding operation. Data were also collected during the first, twenty-fourth and forty-eighth hours postoperatively. These included oxygen and carbon dioxide tensions and $p$H of arterial blood while breathing air, portable chest roentgenograms, clinical notes written by surgical residents, and patient's temperature, pulse and respiration. In the first period (one hour postoperatively), no roentgenogram was taken. At 24 and 48 hours patients were subjected to a dead space rebreathing maneuver as described by Schwartz, Dale and Rahn.[2] Apparatus consisted of an anesthesia face piece to which was attached a corrugated breathing tube from an anesthesia circle system. The total dead space was 700 ml. This apparatus was applied to the patient's face long enough to produce definite hyperventilation, then removed; after a few minutes another arterial blood sample was obtained for analysis of oxygen and carbon dioxide tensions and $p$H. Oxygen tensions were determined using a modified Beckman electrode,[3] $CO_2$ tensions by a Severinghaus electrode,[4] $p$H using a Radiometer $p$H meter. House officers writing clinical

TABLE 1. Arterial Blood-Gas Tensions and Chest Roentgenograms

| | $PaO_2$ mm. Hg | | | | $PaCO_2$ mm. Hg | | | | Roentgenogram |
|---|---|---|---|---|---|---|---|---|---|
| | Preoperative | Postoperative | | | Preoperative | Postoperative | | | |
| | | 1 hr. | 24 hr. | 48 hr. | | 1 hr. | 24 hr. | 48 hr. | |
| 1. | 80 | 78 | 66 | 83 | 36 | 39 | 36 | 35 | N |
| 2. | 82 | 76 | 78 | 74 | 40 | 42 | 45 | 36 | A |
| 3. | 78 | 76 | 61 | 57 | 31 | 32 | 31 | 29 | N |
| 4. | 82 | 82 | 63 | 60 | 36 | 31 | 34 | 36 | N |
| 5. | 80 | 52 | 62 | 76 | 30 | 32 | 27 | 26 | N |
| 6. | 80 | 76 | 78 | 82 | 37 | 38 | 37 | 36 | H |
| 7. | 74 | 71 | 63 | 72 | 36 | 34 | 38 | 34 | H |
| 8. | 88 | 68 | 86 | 88 | 37 | 42 | 38 | 28 | N |
| 9. | 68 | 71 | 59 | 70 | 42 | 37 | 35 | 33 | N |
| 10. | 78 | 72 | 55 | 76 | 36 | 47 | 34 | 35 | N |
| 11. | 80 | 75 | 67 | 77 | 35 | 42 | 38 | 36 | N |
| 12. | 82 | 68 | 68 | 72 | 42 | 45 | 38 | 36 | N |
| 13. | 86 | 65 | 62 | 76 | 38 | 40 | 34 | 37 | N |
| 14. | 82 | 76 | 75 | 80 | 36 | 33 | 33 | 33 | A |
| 15. | 80 | 79 | 84 | 78 | 33 | 35 | 39 | 35 | H |
| 16. | 80 | 78 | 64 | 76 | 30 | 30 | 34 | 32 | N |
| 17. | 76 | 66 | 58 | | 36 | 36 | 34 | | A |
| 18. | 85 | 62 | 64 | 70 | 37 | 32 | 36 | 37 | N |
| 19. | 68 | 59 | 76 | 75 | 37 | 41 | 39 | 31 | N |
| 20. | 72 | 64 | 74 | 61 | 34 | 31 | 35 | 35 | A |
| 21. | 62 | 66 | 67 | 68 | 35 | 37 | 43 | 34 | H |
| 22. | 56 | 48 | 63 | 59 | 32 | 40 | 41 | 27 | N |
| 23. | 78 | 60 | 58 | 54 | 35 | 31 | 30 | 34 | A |
| 24. | 76 | 62 | 64 | 60 | 35 | 31 | 33 | 29 | A |
| 25. | 75 | 78 | 60 | 68 | 39 | 32 | 34 | 37 | H |
| 26. | 67 | 54 | 64 | 66 | 37 | 39 | 35 | 32 | N |
| 27. | 70 | 69 | 60 | 58 | 44 | 24 | 38 | 42 | N |

* N = Normal; A = Atelectasis; H = Hypoventilation. See text for definition of these terms.

notes were aware that a study was being performed, that it concerned respiration but did not know the nature and purpose of the investigation.

Chest roentgen-ray examination was made at the bedside with a 20 ma. General Electric portable roentgen-ray machine. For each patient the same kilovoltage and milliamperage were used in the posterior-anterior projection with the patient erect whenever possible; if examination could not be performed erect, it was accomplished with the patient sitting in bed. Development and fixation were the same for all films. Kodak Blue brand films were used with high speed Radelin intensifying screens.

Chest radiographs were interpreted without prior knowledge of clinical status or blood-gas studies. The appearance of the lungs was classified as normal, showing hypoventilation or atelectasis. Hypoventilation was diagnosed when a full inspiratory effort was not made and the diaphragm was in a higher position than expected in healthy, asymptomatic individuals. Atelectasis was considered present when there was abnormal lung density with evidence of loss of lung volume. The density could be of the linear type, involve part or all of a lobe, or the entire lung. Evidence of over-expansion of adjacent lung tissue was also sought.

No patients were given special treatment to promote or prevent respiratory complications other than routines accepted as general postoperative measures. Anesthesia was selected and administered by personnel not participating in or aware of the nature of the study. Attempts to include 100 per cent oxygen breathing for more detailed analysis were abandoned because of respiratory changes apparently induced by the equipment employed.

## Results

The 27 patients (15 men, 12 women) ranged in age from 35–85 years, and weighed from 93 to 196 pounds. Twenty abdominal and seven inguinal operations were performed lasting from 75 to 480 minutes. Anesthesia included inhalation agents with and without relaxants, spinal or epidural blocks, and combinations of regional and general anesthesia.

*Arterial Blood Studies.* $Pa_{CO_2}$: Preoperatively no values were significantly abnormal. The lowest value was 30 mm. of mercury; the highest 42 mm. In only one instance in the postoperative period did arterial carbon dioxide exceed 45 mm. of mercury. Hyperventilation appeared to predominate in the 24 and 48 hour periods although changes were minimal.

$Pa_{O_2}$: Values for each period of analysis are listed in table 1. Nineteen of 27 patients had decreases (at least 10 per cent of preoperative value) in arterial oxygen tension not due to hypoventilation (increased $Pa_{CO_2}$). Nine of these had definite decreases in oxygen tension in the first hour postoperatively. Eleven exhibited minimal decrease at one hour with further decrease at the end of the day. Six of the 19 demonstrated the lowest arterial oxygen tensions at 48 hours. Only five patients exhibited no change (2, 6, 14, 15, 21). Three (8, 19, 22) had changes compatible with mild hypoventilation. ($Pa_{O_2}$ decreased, $Pa_{CO_2}$ increased.)

A beneficial effect of hyperventilation produced by the rebreathing maneuver was consistently noted. In all patients in whom a decrease in arterial oxygen tension had been observed postoperatively, rebreathing was followed by an elevation in $Pa_{O_2}$. The rise ranged up to 35 mm. of mercury and averaged 11 mm. at both 24 and 48 hour periods.

Preoperative values were all well below the "textbook" normal of 100 mm. of mercury. However, they were not below the values noted in a hospital population where arterial blood is obtained in the supine position.

Patients 21 and 22 had unusually low preoperative oxygen tensions. The reason for this was not apparent. Some degree of pulmonary disease may have been present. The values reported represent results of repeated determinations and it is believed these are not laboratory errors.

*p*H: Preoperative *p*H values were within normal limits. A low of 7.33 and a high of 7.47 represented extremes. Postoperatively only two patients (8 and 25) developed acidosis. Patients 1, 3, 8, 12, 13 and 15 had definite increases in *p*H of which 8 and 12 were associated with decreased $CO_2$ tensions.

*Temperature, Pulse and Respiration.* No correlations could be established between these observations and blood-gas figures. A graphic representation of the findings in patient 4 (fig. 1) indicates that decreased oxygen tension was not due to hypoventilation (same patient as figure 2) and showing no change in the temperature, pulse or respiration.

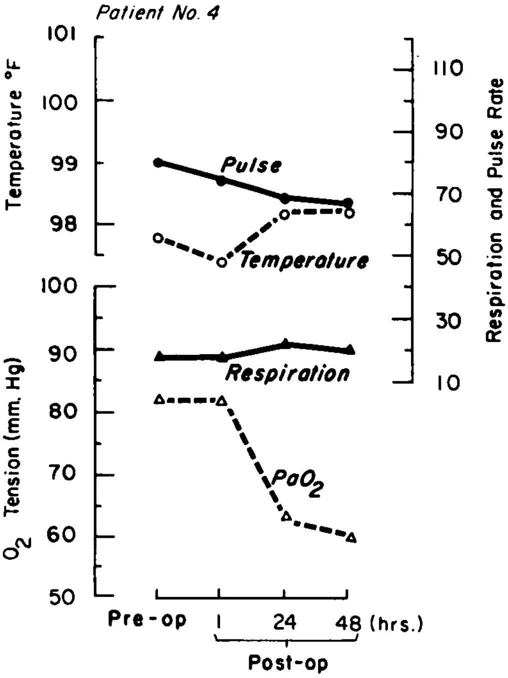


FIG. 1. Changes in $Pa_{O_2}$ with no corresponding change in temperature, pulse or respiration.

*Roentgenograms.* Radiographic impressions of the 27 patients examined are given in table 1. Blood-gas determinations in 19 patients indicated the changes described above and yet 15 of these chest roentgenograms were interpreted as normal. In only four did the chest

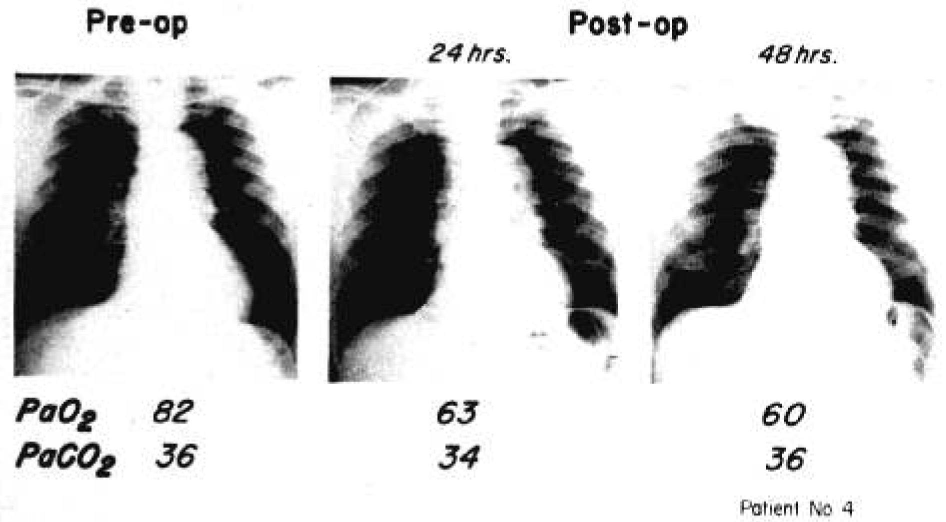


FIG. 2. An example of changes in blood-gas values occurring without evidence of atelectasis in chest radiographs.

roentgenogram agree with blood-gas values by revealing atelectasis. Of the remainder three (17, 20 and 23) had blood-gas studies and chest roentgenograms both interpreted as normal. In three others (8, 19 and 22) blood-gas studies revealed minor degrees of hypoventilation while chest films were thought normal. In two patients (2 and 19) the diagnosis of atelectasis was made by roengen-ray when blood-gas determinations showed only minor alterations. Figure 2 illustrates normal roentgenographic findings in a patient (4) with decreased arterial oxygen and normal total ventilation on the first and second postoperative day.

*Clinical Observations.* A pulmonary complication was suspected only in patient 17. In the clinical notes no mention of cyanosis was made even with the lowest oxygen tensions. The number of patients studied does not provide sufficient data to answer specific questions concerning causative factors.

We found no evidence that anesthetic agent or technique had any effect on results. The five patients with minimal changes in blood-gas values each had major upper abdominal surgery. The number of doses of postoperative analgetic did not seem related to occurrence of abnormal findings.

## Discussion

To establish the exact incidence of atelectasis one must define the condition. For purposes of this study postoperative atelectasis will be considered as a condition in which arterial oxygen tension definitely falls from preoperative values without evidence of generalized hypoventilation (increased $Pa_{CO_2}$). This concept does not require that *complete* collapse of lung units has occurred. We believe atelectasis may be defined by and really represents a severe reduction in ventilation-perfusion ratios ($\dot{V}_A\dot{Q}_c$), in which ventilation is reduced to zero or nearly zero with persistence of some pulmonary capillary blood flow. We are aware that complete collapse is usually implied but believe our definition justified because we are unable to tell when the last molecule or molecules of gas leave an alveolus or the alveolar complex. Therefore, it is necessary to accept some degree of variation in degree of collapse. Poorly ventilated alveoli and totally collapsed alveoli, if perfused, both contribute to hypoxemia, to decreased compliance, and perhaps increased susceptibility to infection. It thus seems reasonable to consider this type of change in ventilation-perfusion ratio as a continuum rather than composed of separate entities.

As herein defined the incidence of atelectasis is high. This incidence is in agreement with studies reported by Beecher,[5] Nunn and Payne[6] and Siebecker.[7] Correlation with other reports has not been possible.

Physical examination and roentgenograms cannot be expected to detect small areas of atelectasis, especially if diffuse, precluding unilateral or regional differences in density or displacement of structures. Variations in normal lung density prevent recognition of many small and scattered areas of atelectasis surrounded by normally aerated lung, yet this is the type of condition which probably accounts for the altered blood-gas values. Nunn and Payne[6] assume that there should be radiological evidence of collapse. However, our studies and theirs clearly show these changes without radiographic evidence of atelectasis and we share their opinion that no other factor explains this occurrence.

Examination by portable apparatus at the bedside is admittedly not an optimum radiographic technique, but evaluation of early postoperative atelectasis is most frequently made in this fashion.

Two aspects of this study may shed some light on the mechanism of development of atelectasis. First, the use of dead space rebreathing resulted in definite improvement in all cases. This is in keeping with the concept that atelectasis may at least begin without airway obstruction.[1] Our findings are in rather complete agreement with previous reports indicating onset of atelectatic-like findings when occasional deep breathing or sighing is prevented.[8-11] Postoperative sedation and incisional pain can lead to constant tidal volume respiration or at least minimize effective sighing. In support of the latter are the findings in one patient (15) who did not exhibit a decreased arterial oxygen tension. This prevailed in spite of a six hour Whipple procedure for carcinoma of the ampulla of Vater. This patient was anesthetized with a continuous epidural technique and the epidural catheter was retained in place to provide postoperative analgesia with dilute tetracaine solution. He received no analgetic drugs in the postoperative period. Through only one instance, the suggestion is strong that a situation allowing comfortable respiration without respiratory depressant drugs as suggested by Gius is worthy of further study.[12]

The absence of elevated $CO_2$ tensions in the immediate postoperative period is contrary to an earlier report from this hospital.[13] Reasons for this difference are not apparent.

Most changes noted here are not of a major degree especially when considered in terms of arterial oxygen saturation. Whether they are of clinical significance is open to question. None of these patients developed serious, and only one a clinically manifest, pulmonary complication. We are therefore unable to make predictions regarding progress of the changes observed. It seems reasonable that the changes recorded here might on occasion be precursors of more serious complications.

The degree of hypoxemia noted, though not severe, is sufficient to give concern. No patient was suspected of being hypoxic during this study. This is confirmatory of the report by Comroe[14] and further emphasizes the fallacy of relying upon clinical observation to detect moderate degrees of hypoxemia. It does not seem wholly justified to point to the absence of complications as indicating lack of importance of the changes in oxygen tension. Rather the burden of proof would be on any who believe this situation harmless. We must at least recognize that changes occur without the expected alterations in temperature, pulse and respiration and without pulmonary roentgenographic evidence.

## Summary and Conclusion

A high incidence of decreases in arterial blood oxygen tension not the result of hypoventilation has been found following abdominal and inguinal operations. This abnormality was not accompanied by radiographic evidence of atelectasis, increase in temperature, pulse or respiratory rate, or abnormal physical findings. It is believed that these changes are due to miliary atelectasis. The condition may result from prolonged constant tidal volume respiration fostered by incisional pain or depressant drugs. It is suggested that decreased ventila-

tion-perfusion ratios and atelectasis be considered as a continuum, not as distinct and separate entities.

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**LOCAL ANESTHESIA** Lidocaine was used in dental surgery as: 2 per cent solution; 2 per cent solution with 0.001 per cent epinephrine; and 2 per cent solution with 0.02 per cent norepinephrine. The combination with norepinephrine gave the strongest and most lasting effect and showed far fewer cardiovascular symptoms than the combination with epinephrine. Secondary hemorrhage was observed only with the combination using epinephrine. Analgesia by lidocaine without vasoconstrictor was far less satisfactory. (*Inovav, J., Jr.: Value of Epinephrine and Norepinephrine in Local Anesthesia, Der Anaesthesist 13: 49 (Feb.) 1964.*)

**HOSPITAL PLANNING** For all general purposes, an operating theater of size 22 × 22 feet, or the equivalent area, is desirable. Every large general hospital should have at least one large theater (*e.g.*, 25 × 25 feet) and no fewer than two operating rooms of this size should be provided in general teaching hospitals. The height of theaters is commonly determined by the type of central light employed, but 10 feet is probably sufficient. The central sterile supply department should be close to the surgical suite, either on the same floor or, if on another floor, connected directly by an elevator used for no other purpose. Thus it should be possible to deliver sterile packages to the suite without their needing to go outside the clean area. Terrazzo when laid on copper grid is the floor covering which provides optimal electrical conduction. Screening must be provided for any operating room in which sensitive electrical equipment will be used, especially for monitoring purposes. Screening usually involves chicken-wire netting within the ceiling and wall structure, doors, windows, and other inlets. (*Brock, Sir Russel, and others: Design of Operating Theatre Suites, Ann. Roy. Coll. Surg. Eng. 34: 219 (April) 1964.*)