MEDICAL GRAND ROUNDS

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PULMONARY COMPLICATIONS OF GENERAL SURGERY

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PULMONARY COMPLICATIONS OF GENERAL SURGERY

Patients required to undergo general surgery may experience a variety of pulmonary complications. Although the internist relinguishes his role of primary care physician to the anesthesiologist and general surgeon in the intraoperative period, he is frequently requested to assess the likelihood of postoperative complications in the preoperative patient and to assist in the therapy of postoperative complications should they occur. It seemed appropriate, therefore, to review this subject with this audience. The review does not include the specialized problems that are encountered following thoracic surgery or following surgery of the traumatized patient. The most frequent complications encountered in general surgery are indicated in Table I.

TABLE 1

PULMONARY COMPLICATIONS OF GENERAL SURGERY

Hypoxemia
Aspiration of Gastric Contents
Atelectasis with or without Infection
Ventilatory Failure
Thromboembolism

Hypoxemia while breathing room air is exceedingly frequent in the postoperative patient. Since hypoxemia may lead to serious extrapulmonary manifestations, especially in the presence of cardiac disease, its pathogenesis will be reviewed in detail.

The aspiration of gastric contents is a well recognized and serious complication of general anesthesia (1, 2). Regurgitation of gastric juice into the pharynx has been observed in from 14 to 26% of patients undergoing general anesthesia (3, 4). The regurgitated material has been observed to be aspirated into the tracheobronchial tree in from 7 to 16% of patients. Aspiration is frequently silent at the time of surgery and is diagnosed only retrospectively. The clinical consequences depend on the volume and the pH of the material aspirated (5). If a sufficient volume of juice with a pH of 2 or less is aspirated, the injury produced is a chemical lung burn. The clinical presentation is that of a bloody, non-cardiogenic pulmonary edema, which is frequently referred to as the adult respiratory distress syndrome. This complication results in approximately 11% of anesthetic deaths (6). In obstetrical deaths due to anesthesia, an even higher percentage is due to vomiting and aspiration. The prevention of this serious complication is primarily within the area of anesthesia technique, and the treatment of the adult respiratory distress syndrome is a separate subject requiring more time than available to develop fully. This review will not deal further with this problem.

Overt atelectesis with or without pulmonary infection or pulmonary infection alone is the most commonly recognized postoperative pulmonary problem leading to increased morbidity and mortality. Since atelectasis and infection frequently may not be separated either in the reports of series of patients or in a particular patient for whom one is caring, these complications will be considered as a single group subsequently.

Ventilatory failure with hypoventilation and worsening hypoxemia may be precipitated by general surgery in patients with preexisting serious pulmonary disease, primarily of the airways obstructive type. The assessment of the probability of this event by preoperative pulmonary function studies will be reviewed.

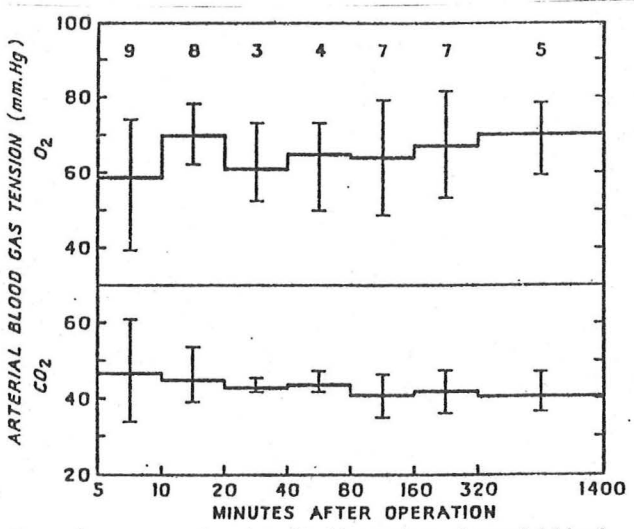
It has long been recognized that post-operative patients have a high incidence of venous thrombosis and pulmonary embolism (7). More recently, utilizing the ^{135}I labeled Fibrinogen technique, it has been demonstrated that up to 74% of post-operative patients have deep venous thrombosis (8-12). Deep venous thrombosis is especially prevalent among men, older patients, patients with varicose veins, those with malignant conditions, and especially those with surgery for fractured hips. Despite the obvious importance of thromboembolism to the surgical patients, its manifestations in the post-operative patient are similar to those in other patients and this broad field will not be included in this review.

HYPOXEMIA

Mechanism

That major abdominal surgery might be associated with significant hypoxemia postoperatively was suggested by several investigators during the 1950's (13-15). The association of general anesthesia per se with an increased A-a O_2 difference was suggested in 1958 (16) and confirmed in 1959 (17). The magnitude of hypoxemia in the post-operative patient was demonstrated by Nunn and Payne in 1962 (18), whose data are demonstrated in Figure 1.

FIGURE 1



Group 1: oxygen and carbon-dioxide tensions of arterial blood. Mean values and range are shown for each period. Figures in circles indicate the number of observations during each period.

They demonstrated potentially serious postoperative hypoxemia in a small group of previously normal adults who had undergone elective surgery of a non-specified type requiring general anesthesia. The mean arterial oxygen tension (PaO_2) while breathing room air immediately after surgery was 58 mm Hg with the lowest PaO_2 31 mm Hg. That this was not due to hypoventilation caused by residual anesthesia was indicated by the relatively mild increase in the arterial pCO_2 and hence the large A-a O_2 difference. Significant hypoxemia persisted during the 24 hours of study, although the hypercapnia abated more quickly. These patients had no recognized pulmonary complication such as atelectasis or infection; the results could be ascribed only to anesthesia and surgery. This degree of hypoxemia in the postoperative tive interval has been adequately confirmed by many investigators since that time. I am aware of no study that has carefully correlated postoperative hypoxemia with increased morbidity or mortality; nevertheless, it is reasonable to believe that such hypoxemia may be associated with serious extrapulmonary complications, especially in patients with cardiovascular disease.

Since the original observations of hypoxemia in the intra- and postoperative periods many studies have been carried out to determine the cause. The sometimes conflicting data that has been generated has been well summarized by Marshall and Whyche (19), and their conclusions regarding the intraoperative period are:

"1. General anesthesia is accompanied by increased venous admixture, but the extent of the change is variable.

2. Changes are minimal when tidal volumes exceed 8 ml/kg.

3. With tidal volume less than 8 ml/kg, venous admixture is always markedly increased compared with the awake values in those studies in which the mean age is less than 45 years. With ages exceeding 45 years, the awake values are already increased and subsequent increases during anesthesia are smaller.

4. Unlike the awake values, the value achieved during anesthesia does not seem to be a function of age (in this sense anesthesia seems to make all lungs function as if "aged" to the same extent)."

5. While increases in both mismatching of ventilation and blood flow and true right to left shunting are evident, the latter mechanism is the most important.

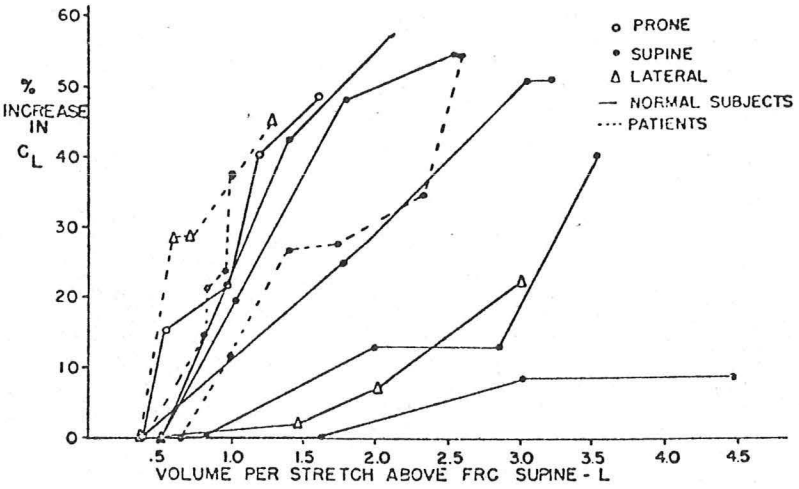
There are evidently two closely related but independent mechanisms leading to hypoxemia preceding, during and after surgery. The first of these is a diffuse microatelectesis consisting of alveolar closure spread widely throughout the lung. Each anatomical focus of alveolar closure is so small that the resulting lesions do not produce physical or radiographical abnormalities. The original concept of microatelectesis is ascribed to Mead and Collier (20) who observed that anesthetized dogs, whether breathing spontaneously or mechanically ventilated in a resting tidal volume range, had a

progressive fall in pulmonary compliance. That is, the dogs' lungs became progressively stiffer requiring higher inflation pressures to achieve the same tidal volume. These changes could be due to progressive atelectesis, since an atelectatic lung contains less volume that can be ventilated for any inflation pressure. These investigators noticed that the pulmonary compliance was rapidly reversed toward controlled values following forced inflations of the lung. They further noted that the appearance of the lungs at postmortem suggested that of closure of air spaces. They suggested that their findings could explain the decreased compliance observed by previous investigators in anesthetized compared to awake patients (21-24).

It was soon demonstrated by Ferris and Pollard (25) that normal unanesthetized humans develop a progressive fall in compliance when breathing at normal but monotonously regular tidal volumes. It also was demonstrated that the fall in compliance is reversed when the subjects are allowed to take larger tidal volumes. That is, the lung becomes less stiff following deep breaths. Their important results are indicated in Figure 2.

FIGURE 2

PULMONARY COMPLIANCE AFTER DEEP AND QUIET BREATHING



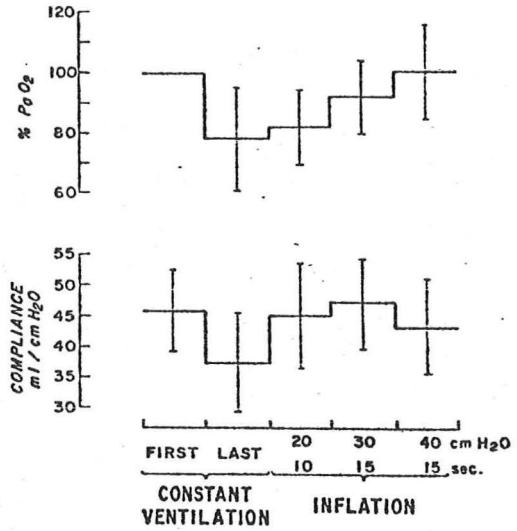
EFFECT OF PROGRESSIVELY DEEPER BREATHS ON PULMONARY COMPLIANCE (C_L)

On the vertical axis is plotted the increase in compliance caused by large tidal volumes, and on the horizontal axis is plotted the size of tidal volumes above the resting level. It is apparent that the increase in compliance caused by large tidal volumes is dependent on the size of the tidal volume above the resting level. That is, the more the lung is stretched the greater the compliance. It was demonstrated that for normal persons two large breaths cause maximal change. In patients with poliomyelitis who had been breathing at small tidal volumes for protracted intervals two breaths were insufficient to produce maximal increases in compliance. These patients

had progressive increases in compliance when the lung was stretched repeatedly. This study is interpreted to indicate closing of lung units even in normal persons when the lung is ventilated at tidal volumes adequate to excrete carbon dioxide but never allowed to be hyperinflated.

In a series of important investigations Bendixen and his co-workers at Massachusetts General Hospital demonstrated the importance of microatelectasis to the oxygenation of surgical patients (26-31). These workers related the changes in compliance caused by constant, small tidal volume ventilation to changes in arterial oxygenation. Their results, in part, are indicated in Figure 3.

FIGURE 3



Average Falls (with Standard Deviations) in Oxygen Tension (Expressed in per Cent) and in Total Lung Compliance as Difference between First and Last Measurements during Constant Ventilation.

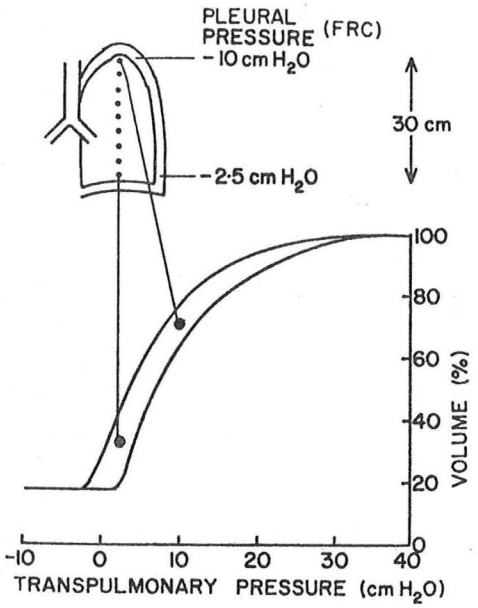
Three successive inflations restore previous levels of compliance (with the first inflation) and oxygen tension (in steps).

In this graph oxygenation is indicated as 100% of the control value on the verticle axis. In patients undergoing surgery with general anesthesia and controlled ventilation the anesthesiologists maintained normal CO_2 exchange but did not periodically deep-breathe the patients. The average period of study was 76 minutes. There was an average fall in compliance of 15% and an average fall in oxygen tension of 22%. At the end of surgery the patients were hyperinflated with three breaths; the first to 20 cm of water for 10 seconds, the second to 30 cm of water for 15 seconds, and the third to 40 cm of water for 15 seconds. With hyperinflation not only did compliance return to control values, but PaO_2 increased progressively with successive hyperinflation until it also returned to control values.

The concept of microatelectasis as a natural consequence of general anesthesia has not gained universal acceptance, and some data have been generated that minimize the concept (32-35). The most frequently quoted reference in this regard is the work of Panday and Nunn (35). In my opinion, their work supports the concept of microatelectasis in the anesthetized patient rather than refuting it. The A-a O_2 difference was large in all of their patients at the time of the initial analysis which was after induction of general anesthesia. This measurement increased in some patients at the time of the second measurement, although the interval was only about 20 minutes. That it did not increase in every patient is not surprising, since it has been demonstrated that there is considerable variability from patient to patient; moreover, there is no indication about the variability of tidal volume during anesthesia in these patients and whether some of them spontaneously took large tidal volumes. In addition, with repeated hyperinflation to 30 cm H_2O there was a statistically significant increase in PaO_2. Such occurred despite a relatively inefficient procedure for hyperinflation (inflation of the lungs to a pressure of 30 cm H_2O ten times in one minute).

More recently a separate but related mechanism of hypoxemia during surgery and the postoperative interval has been delineated. This mechanism relates to the differences in regional distribution of ventilation in the lung originally proposed by West and Dollery (36) and subsequently studied in detail by the group at McGill (37-39). The determinants of regional distribution of ventilation are, in part, indicated in Figure 4.

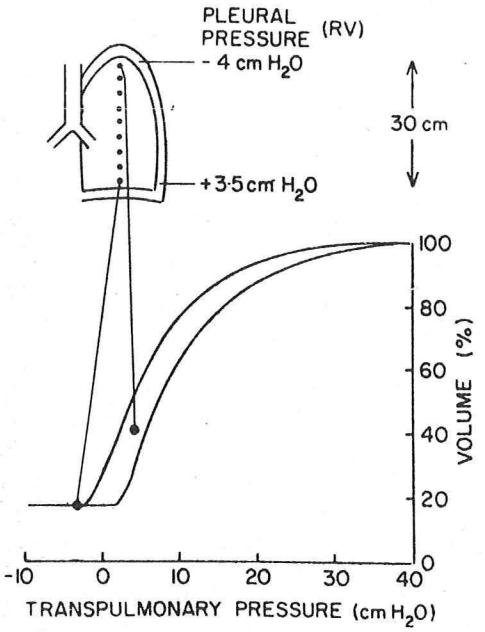
FIGURE 4



At FRC, because there is a 7.5 cm H_2O difference between the top and the bottom of the lung, alveoli in these regions are expanded to different degrees.

When the lung comes to rest at the end of a normal expiration the amount of gas remaining in the lungs is the functional residual capacity (FRC). This graph represents a lung at FRC with lung volume plotted as a per cent of the total possible lung volume (total lung capacity, TLC) on the vertical axis and the pressure difference from inside the alveolus to inside the pleural space on the horizontal axis. At FRC the apical lung regions are more expanded than the basilar regions. These regional differences in lung volume are explained by differences in pleural pressure from the top to the bottom of the lung. There is a gradient in pleural pressure down the lung with the more negative values towards the apex. The reason for this vertical pressure gradient is not entirely clear, but it is apparently gravity-dependent and is related to the weight of the lung. The pleural pressure increases at a rate of approximately 0.25 cm H_2O per centimeter down the lung. Thus, in this example, a lung with a vertical length of 30 cm has a difference in pleural pressure from top to bottom of approximately 7.5 cm H_2O. Thus, when the entire lung is at FRC the apical lung units are subjected to a more subatmospheric pleural pressure and are at approximately 60% of their total capacity while the lung units at the bottom of the lung are expanded to only 30% of their total capacity. If additional gas is breathed out of the lung from this resting position, the results are indicated in Figure 5.

FIGURE 5



At residual volume (full expiration), airways at the bottom of the lung have closed, whereas airways at the top are still open because the upper lung regions are still at 40 per cent of their maximal volume.

This plot is similar to the previous one excepting that the lung is now at full expiration, i.e., at residual volume (RV). At this low lung volume pleural pressure in dependent lung regions exceeds the airway pressure; that is, pleural pressure is above atmospheric pressure. This leads to closure of airways in dependent lung regions, and gas is trapped behind the closed airways. Airways at the top of the lung are still open, and the upper lung regions are still at 40% of their maximal volume. Similar changes occur in the recumbent patient, but the vertical pressure gradient is from anterior to posterior so that airway closure occurs in the posterior part of the lung.

The lung volume at which airways begin to close is referred to as the closing volume (40, 41). Airway closure (closing volume) occurs very near residual volume in young, healthy persons but occurs at larger and larger lung volumes with advancing age (42). Closing volume is also affected by position, obesity, and airways disease.

The relationship between closing volume and functional residual capacity is an important determinant of matching of ventilation and blood flow and hence an important determinant of arterial oxygenation. If airway closure occurs at a lung volume above the resting volume (FRC) the alveoli served by the closed airways do not receive normal ventilation. The gas trapped in alveoli with closed airways comes into equilibrium with pulmonary arterial blood as oxygen continues to be removed but not replenished. This contributes to venous admixture and a lowering of arterial oxygen tension. Only by an inspiration of at least 500 mls above closing volume is the oxygen tension of the trapped gas partially restored. Following such a large tidal volume the oxygen concentration falls again, and the cycle is repeated. Thus, a large tidal volume has only a transient effect on gas exchange, but it may help to prevent atelectasis in the parts of the lung where gas trapping occurs.

Functional residual capacity decreases as one shifts from an upright to a supine position, primarily due to an encroachment on the thorax by the diaphragm caused by the weight of abdominal viscera. This shift is more marked in obese persons (43) and is accentuated by surgical postures such as lithotomy or head down tilt (44). In addition, general anesthesia causes a significant reduction in FRC (45-48). It is not clear why FRC changes during anesthesia, although augmented expiratory activity of the abdominal muscles has been suggested (49). Whatever the cause, FRC decreases immediately after the induction of anesthesia, and the change is not progressive. The decrease in FRC is not accompanied by a decrease in closing volume, and hence airway closure with gas trapping and arterial hypoxemia may occur in the anesthetized patient ventilated with normal tidal volumes.

Thus, two interrelated mechanisms lead to hypoxemia in the perioperative period. It may be that the microatelectasis that occurs develops in areas of intermittent airways closure when the trapped gas is absorbed from the alveoli with closed airways. Alternately, alveolar collapse could occur without airway closure. Once alveoli have collapsed, considerable pressure is necessary to overcome surface forces and reopen them. High inspiratory pressure does not seem to be necessary to reopen closed airways.

Premedication

Patients undergoing general surgery are usually given preanesthetic medication in order to allay anxiety and to minimize respiratory secretions. Atropine or a similar drug is commonly administered for the latter purpose. Although such drugs cause several changes in pulmonary function (50), they do not lead to preoperative hypoxemia (50-52). Similarly, preanesthetic doses of barbituates do not alter PaO_2 (53). Although results have been variable, such is evidently not the case with narcotic drugs. Both meperidine and morphine have been demonstrated to significantly decrease PaO_2 in doses that are used for preanesthesia (31, 53). Normal persons at rest take deep breaths on average ten times an hour (29), but following morphine such deep breathing is suppressed (31). It has been suggested that the mechanisms of hypoxemia already described may ensue in the absence of deep breathing.

These data suggest preanesthetic regimens which include narcotics should be avoided in patients already known to be hypoxemic or that oxygen should be given from the time of preanesthetic administration until induction of anesthesia. In addition, the respiratory depressant effects of narcotics may be deliterious to patients with severe obstructive airways disease and hypercapnia.

Anesthesia

Arterial hypoxemia is least likely to occur during general anesthesia than at other times of the perioperative period. Although the mechanisms of airway closure and microatelectasis that have been described lead to excess intrapulmonary right to left shunting during the course of anesthesia, these changes are not usually so great that satisfactory arterial oxygenation cannot be maintained by administering high concentrations of oxygen in the inspired gas. Since the anesthetized patient is given high concentrations of inspired oxygen during the course of surgery with virtually all anesthetic techniques, there is little likelihood of hypoxemia during surgery.

With the exception of the asthmatic patient, there is no apparent advantage to any particular anesthetic technique in patients with or without lung disease either during surgery or in the postoperative interval (54-60). In the patient with asthma, it has been suggested that halothane or ketamine anesthesia may have some advantage, but there is insufficient evidence to be clear in this judgement (61, 62).

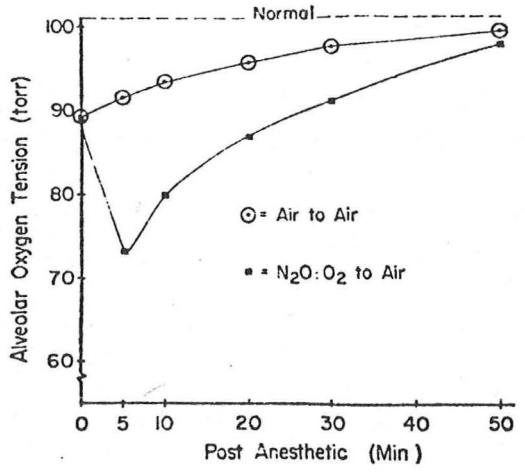
Postoperative

Although premedication with narcotics may lead to mild hypoxemia, and although the potential for intraoperative hypoxemia exists if the patient is not carried on high concentrations of inspired oxygen, it is the postoperative period in which the potential for serious hypoxemia is most marked. Since the original work of Nunn and Payne (18) many studies have documented the frequent

occurrence of severe hypoxemia in the postoperative patient breathing room air (63-70). It is clear that serious hypoxemia may exist in persons without antecedent lung disease. Hypoxemia is minimal and transitory in patients undergoing minor surgery lasting less than thirty minutes (71, 72). Hypoxemia is more likely to occur in older patients, patients with preexisting lung disease, obese patients, and patients undergoing abdominal surgery.

Immediately at the end of surgery two factors may lead to arterial hypoxemia in addition to the microatelectesis and airway closure already discussed. These are post-hyperventilation and diffusion hypoxemia and are indicated in Figure 6 taken from the paper by Marshall and Wyche (19).

FIGURE 6



Post-hyperventilation hypoxia and diffusion hypoxia. The upper (dashed) line represents normal $P_{A_{O_2}}$ during breathing of air. Post-hyperventilation hypoxia is indicated by the upper solid line, calculated from Sullivan, assuming that the patient hyperventilated for an hour and then was allowed to accumulate CO_2 for 10 min before zero time. Diffusion hypoxia (following equilibration with 79 per cent $N_2O:O_2$) was added to post-hyperventilation hypoxia as above, except that the inspired gas was changed to air at zero time. (N_2O excretion from Salanitre.[317])

Following surgery there may be residual depression of the respiratory center by the anesthetic leading to mild degrees of hypoventilation causing a moderate increase in $PaCO_2$ and decrease in PaO_2 (73). Even without hypercapnia, hypoventilation may cause moderate reductions in alveolar and hence arterial PO_2 (74, 75). If the anesthetic technique has employed hyperventilation, body stores of carbon dioxide are depleted. These stores of CO_2 are refilled from endogenous CO_2 production causing ventilation and therefore P_AO_2 to be reduced. The effect of this is seen in the top solid line of the figure. Many anesthetic techniques employ the use of nitrous oxide : oxygen mixtures for maintenance of sleep. When nitrous oxide : oxygen is abruptly replaced by air at the end of anesthesia, nitrogen replaces nitrous oxide in the blood. Because nitrous oxide is thirty-one times more soluble than nitrogen, there is a

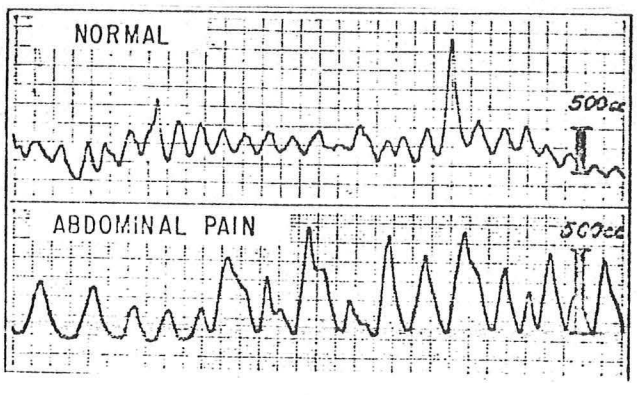
dilution of the inspired gas with the excess volume of nitrous oxide as it escapes from the tissues to the lungs; hence P_AO_2 decreases. These results are indicated by the lower solid line in the figure. Both post-hyperventilation and diffusion hypoxemia should be of relatively brief duration, and hypoxemia may be prevented by administration of high concentrations of inspired oxygen during the immediate postoperative interval.

More serious and more lasting hypoxemia postoperatively is in large part determined by the site of operation. The likelihood of hypoxemia lessens as the distance of surgery from the upper abdomen increases. It was first noted in 1927 that vital capacity is reduced by 75% in the first twenty-four hours after upper abdominal operations, by approximately 50% following lower abdominal operations, but that there was no change in vital capacity after non-abdominal procedures (76). Subsequently, several investigators have documented the reduction of all lung volumes save residual volume in patients undergoing laparotomies but not in patients with extra abdominal operations (77-83). These reductions in lung volume are maximal on the first postoperative day and return to preoperative values over the course of approximately two weeks. These changes are ascribed to postoperative pain. There is also a reduction in motion in the diaphragm following laparotomy (84, 85); whether this is due to pain or is an independent change is not clear. These changes lead to a rapid, shallow pattern of breathing in the postoperative patient and to a reluctance to breathe deeply or cough (86, 87). The result of these changes is a tendency toward hypoxemia from airway closure and microatelectasis (88-90).

The universal occurrence of hypoxemia following abdominal surgery indicates the need for oxygen administration to all such patients in the postoperative period. The potential for postoperative hypoxemia in patients undergoing general surgery other than laparotomy suggests that older patients and patients with diseases adversely affected by hypoxemia, such as arteriosclerotic heart disease, should have oxygen administered postoperatively at least until it can be determined whether the patient will experience hypoxemia. The means of oxygen administration is not critical in most patients; since nasal cannullae gain patient acceptance better than face masks, their use seems reasonable. In patients with severe lung disease creating the potential of hypoventilation with oxygen administration more precise means of delivering accurate inspired oxygen concentrations may be necessary.

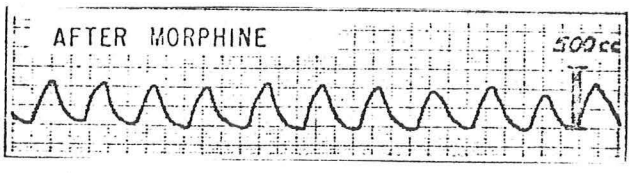
Oxygen administration merely alleviates the hypoxemia but does not correct the underlying airway and alveolar closure. Periodic deep breathing is necessary for that purpose. Although investigative results are not uniform, it is unlikely that narcotic agents can be relied on to sufficiently relive pain to allow deep breathing. The results of morphine on the breathing pattern of postoperative patients as reported by Egbert and Bendixen (31) is indicated in Figure 7.

FIGURE 7



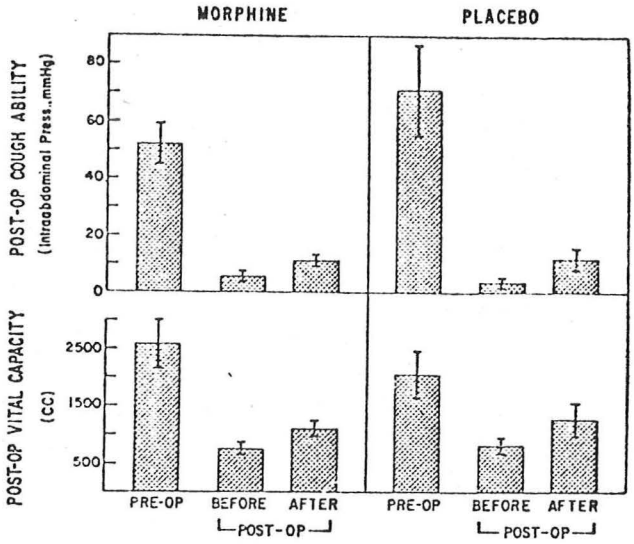
BREATHING PATTERNS

—Pneumograph tracing. After morphine, volume of each breath tends to become constant.



These investigators found that abdominal pain suppressed the very large tidal volumes taken, on average, ten times an hour by normal resting persons. When persons with abdominal pain were given morphine it was found that tidal volumes tended to become monotonously constant and even smaller than before morphine. Moreover, large sighs were virtually eliminated.

FIGURE 8



Vital capacity and coughing after abdominal operations. Vital capacity and ability to cough were depressed after operation ($P < 0.01$). Both improved after treatment with medicine ($P < 0.01$); differences between morphine and placebo were not statistically significant.

They further found that not only vital capacity but coughing ability as measured by intrarectal pressure was markedly decreased in the postoperative patient. Although there was a slight improvement in both after administration of medicine to the postoperative patient, there was no difference whether the medicine was morphine or a placebo.

Better postoperative analgesia may be obtained with continuous extradural nerve block (91). However, such is not practical on a routine basis. Other means utilized to promote deep breathing and coughing in the postoperative patient are considered in the section on prevention and treatment of atelectasis and infection.

ATELECTASIS AND INFECTION

Incidence

The reported incidence of postoperative atelectasis varies widely depending on the criteria for diagnosis. As suggested in the previous discussion, microatelectasis during and following surgery is almost universal, so that series utilizing postoperative hypoxemia as the definition of atelectasis

report an exceedingly large incidence (92). If one accepts subtle radiographic changes without clinical symptoms as diagnostic, the incidence is somewhat less but still high (3). Abnormal physical findings in the chest such as inspiratory rales and diminished breath sounds in the bases are also frequent and may (94) or may not (95) correlate with hypoxemia and radiographic abnormalities. It has also been suggested that any fever unexplained by wound infection indicates a respiratory complication (96).

If one realizes that microatelectasis, and hence postoperative hypoxemia, is exceedingly frequent in the postoperative interval, there are nevertheless patients who develop even more overt pulmonary changes which might be termed macroatelectasis. These patients tend to have even worse hypoxemia and more complicated postoperative courses. They also are more likely to develop overt pulmonary infection. The criteria that have been utilized for the diagnosis of this type of pulmonary complication have been fever, cough, increase in sputum production, and leukocytosis. The fever and leukocytosis have been demonstrated to be due to growth of bacteria distal to an obstructed bronchus (97), but since all symptoms may regress with the removal of bronchial obstruction and without antibiotics, at least in the early phases, this syndrome is called atelectasis. Utilizing these criteria the reported incidence of postoperative pulmonary complications is indicated in Table 2 (98-100).

TABLE 2

POSTOPERATIVE PULMONARY COMPLICATIONS

Author	Year	Total	Incidence, Per Cent		
			Nonabdominal	Abdominal	Upper Abdominal
King	1933	8.9	1.2	14	27
Pooler	1949	4.0	0.7	13	19
Wightman	1968	6.2	0.6	10	21

From these data it is apparent that clinically significant postoperative pulmonary complications (in addition to hypoxemia) occur in a significant fraction of patients. Moreover, it is apparent that the incidence has not changed much in the last forty years. However, this may not represent a lack of progress in the management of postoperative patients. It is reasonable to believe that older and more seriously ill patients are considered candidates for surgery more frequently now than in former years. That these complications are not inconsequental is reflected by the report that 13.1% of 1,736 postoperative deaths were caused by lung complications (101). Phrased differently, among 22,000 surgical patients, 1.1% died postoperatively of respiratory difficulties.

Risk Factors

The data in Table 2 also indicate the importance of the type of operation on the likelihood of postoperative pulmonary complications. Operations that do not violate the peritoneal cavity are associated with a very low incidence of pulmonary complications. Such operations include not only head and neck and extremity surgery, but also breast surgery, transurethal prostatic resection, and anal surgery (100). These data do not apply, however, to thoracic surgery. It also is apparent that upper abdominal surgery is more likely to lead to postoperative complications than is lower abdominal surgery. Insofar as I can tell, the type of upper abdominal procedure is not critical in this regard.

Additional factors increasing the incidence of pulmonary complications are indicated in Table 3.

TABLE 3

POSTOPERATIVE PULMONARY COMPLICATIONS

ADDITIONAL RISK FACTORS

Men	Smoking
Obesity	Recent URI
Excess Narcotics	Abnormal PFT's
Advancing Age	

Although there is a higher incidence of postoperative complication among men than among women, this evidently relates to the higher prevalence of smoking among men. If only men and women with similar smoking histories are included, complications are as great among women as men (100).

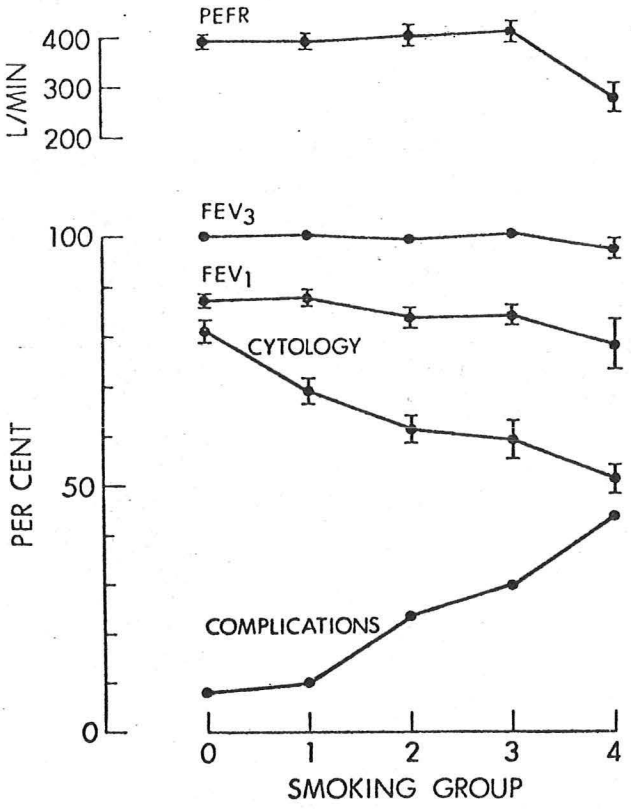
Although not all series find an increased incidence of postoperative complications in the overweight patient, others have found that obesity is associated with an increased incidence of postoperative complications (102-105). Since extreme obesity causes small lung volumes and increased hypoxemia in the absence of surgical procedures, the data suggesting increased postoperative complication in obese patients are more reasonable.

It already has been indicated that postoperative narcotics do not cause sufficient relief of peritoneal pain to allow patients to deep breathe and cough (31). Further, by suppressing the normal sighing mechanism narcotics tend to promote microatelectasis and hypoxemia. The data of Bunder, et al, (106) suggest that excess narcotic administration leads to an increased incidence of macroatelectasis as well. They found that men given more than four doses of narcotic following upper abdominal surgery were more likely to develop atelectasis than men given four or less doses.

Aging has been found by many investigators to effect postoperative complications (63, 72, 73, 100, 107). It has been suggested that this is due to the increased prevalence of obstructive airways disease in older patients, but it is reasonable to believe that older patients experience greater difficulties even in the absence of overt airways disease or smoking.

The increased incidence of postoperative pulmonary complications among smokers has been noted for many years. The importance of the amount of smoking is indicated by the data of Chalon, et al, (108) indicated in Figure 9.

FIGURE 9



Mean variations in percentages of normal cellular features, FEV_1 and FEV_3 (timed vital capacities expressed as percentage of total vital capacity forcefully expired in one and three seconds), PEFR (liters/min), and percentage of complication rate after operation in various smoking categories. O=nonsmoker (N-24), 1=light smokers (N=31), 2=moderate smokers (N=28), 3=heavy smokers (N=16), and 4=very heavy smokers (N=12). Brackets indicate ±1 SE.

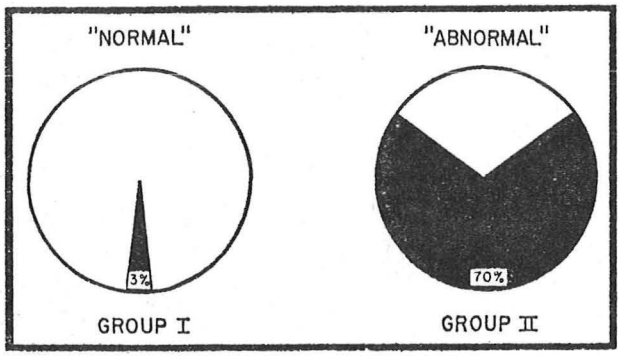
When patients were graded from non-smokers (0) to very heavy smokers (4), it was found that there was a steady rise in the number of complications after operation as cigarette consumption increased. Thus, non-smokers had a morbidity rate of 7.9% while very heavy smokers had a rate of 43%. Unfortunately, the

article does not contain sufficient data to determine the types of operations among the various groups. It is interesting to note, also, that smoking bore a strong correlation with postoperative complications even though chronic bronchitis was not severe enough to cause a reduction in the forced expiratory volume in one second (FEV_1).

It has been demonstrated that persons operated in the presence of an upper respiratory infection, including only mild pharyngitis, are more likely to experience postoperative pulmonary complications (109, 110). The data are sufficient to indicate that elective surgery should be postponed if a patient develops an acute respiratory infection.

The most important single factor leading to postoperative pulmonary complications is the presence of chronic obstructive lung disease (111-116). The predictive value of pulmonary function testing for postoperative pulmonary complications has been demonstrated by Stein, et al (112). These investigators studied a group of patients with a battery of pulmonary function tests including the maximal expiratory flow rate (MEFR), forced vital capacity (FVC), one second timed vital capacity (FEV_1), the nitrogen single breath test, the residual volume to total lung capacity ratio (RV/TLC), and an index of $PaCO_2$. Their overall results are indicated in Figure 10.

FIGURE 10



—Per cent of complications in Group 1 ("normal") and Group 2 ("abnormal") patients.

Among thirty-three patients whose preoperative pulmonary function tests were normal, there was one postoperative respiratory complication. Of the thirty patients with one or more abnormal function tests, twenty-one developed postoperative pulmonary complications. The single most sensitive test was the MEFR. The FEV_1 was not as sensitive as the MEFR but tended to give the same information. Among the five patients with an elevated $PaCO_2$, all had serious respiratory difficulties. Thus, Stein concluded that CO_2 retention should be considered as a contraindication to all but life saving operations.

Prevention and Treatment

Many different regimens have been advocated for the prevention of postoperative complications. Some regimens have been enthusiastically supported by some and rejected by others. Such variable results frequently have been due to difference in selection of patients; large well controlled series are not often reported. Variability in results also may be due to differences in technical proficiency with which treatments are performed. It is highly likely that several techniques decrease the incidence of postoperative pulmonary complications if carried out effectively. Enthusiastic attention to the postoperative patient by an interested group of investigators might yield gratifying results in one institution, while the routine use of the same procedure in a different hospital might be less efficacious. The principles, however, seem clear. As outlined by Dripps and Waters in their description of the "stir-up" regimen in 1941 (117), the patient must inflate his lungs adequately with deep breaths, and the tracheobronchial tree should be kept as clear of secretions as possible.

Considering the decrease in lung volumes, airway closure, and microatelectasis that exist in the postoperative patient, a part of the postoperative regimen should include maximal inspiratory maneuvers. Forceful expiratory maneuvers, with the exception of removal of secretions from airways by coughing, are not reasonable. Blow bottles or blowing out against resistance fall in this latter category (118). In order to generate the pressure at the mouth necessary to blow out against resistance, it is necessary to generate an even higher pressure in the pleural space surrounding the airways. Thus, the net effect is to reduce the caliber of airways rather than to enlarge them. If the patient inhales to a large lung volume before the forceful exhalation, some benefit is likely to accrue. However, it seems more likely that deep inspiration would result from a more direct approach. I am aware of no controlled study on the efficacy of blow bottles.

Scott and Cutler (119) initially suggested the prophalactic value of postoperative inhalation of 5% carbon dioxide in order to encourage deep breathing. Their method was modified by Schwartz and Dale (120) to utilize the patient's own endogenously produced carbon dioxide by the use of a rebreathing tube. Owing to the inconvenience of a tube 3.2 cm in diameter and 125 cm in length, Adler introduced a more compact rebreathing device for the same purpose (121). Investigations of CO_2 breathing in the postoperative patient have indicated a decrease in radiological (122) but not physiological (123) postoperative atelectasis. Since rebreathing is erratic in production of very large tidal volumes, it is reasonable to believe that it might be beneficial in some but not all patients. If utilized, the patient should be concurrently given oxygen, since hypoxemia may be precipitated by the rebreathing maneuver. In addition, it should clearly be avoided in patients with significant chronic obstructive lung disease and the potential for carbon dioxide retention. From available evidence, enthusiasm for this procedure does not seem justified.

The routine use of intermittent positive pressure breathing (IPPB) for postoperative patients was suggested in 1958 (124). Since that time four controlled studies have been reported utilizing IPPB (125-128). Three of the

studies (125-127) reported no benefit. The fourth (128) reported a decrease in the development of postoperative temperature elevation, pulse rate elevation, cough or chest pain, or any combination of these accompanied by radiographic demonstration of atelectasis from 19.5% of the control group to 2.5% of the treated group. In the latter study, these results were obtained despite the fact that the treated group, on average, had worse pulmonary function. In none of these studies is the most critical information available; namely, there is no indication of the size of tidal volume achieved by IPPB. In each study a pressure limited IPPB device was used. With such a device the tidal volume achieved for any particular inflation pressure is markedly dependent not only on the mechanical properties of the patient's lungs and thorax but also on the ability of the therapist to invoke patient cooperation in allowing passive inflation. Viewed with these limitations, possible differences between the studies that found no benefit and the study that found benefit emerge. In the study demonstrating benefit of IPPB the patients were instructed in use of the equipment preoperatively; higher inflation pressures were used (20 cm H_2O); and a larger volume of bronchodilator was used. Whether these differences account for the varying findings is not clear.

It is reasonable to believe that the routine use of IPPB does not decrease the incidence of postoperative pulmonary complications. This judgement is based not on the theoretical benefits that might accrue from large tidal volumes generated by IPPB but from a realization that such large tidal volumes are not achieved when therapy is delivered in a routine manner. If in an individual patient it can be demonstrated that IPPB causes large tidal volumes (at least three times a resting tidal volume or approaching 50% of the patient's vital capacity), and if the patient cannot be encouraged to spontaneously take such tidal volumes, IPPB may be beneficial. To achieve this goal IPPB should be ordered by the tidal volume desired rather than by a fixed inflation pressure. The therapist must then be allowed to regulate the controls of the machine in an attempt to achieve the desired tidal volume. In order to accomplish this, it is likely that treatments must be given preoperatively as well as postoperatively.

Recently a device intended to encourage patients in taking maximal voluntary inspirations, an incentive spirometer, has been reported (129-131). With this device the patient is rewarded by a light when inspiration of a predetermined tidal volume has been achieved. It has been reported that patient cooperation is more easily elicited with less personnel time than with other means of physiotherapy or IPPB. A decrease in postoperative complications utilizing this device was reported (130), but insufficient data were tabulated to insure the reliability of the observation. In a small group of patients it also was reported that this device was more successful than IPPB (131); however, the number of patients involved was too small for statistical validity. If it is confirmed that this device assists in spontaneous deep breathing while minimizing personnel time, its use seems rational.

An entirely different approach to prevention of postoperative atelectasis was suggested in 1960 by Radigan and King (132). The procedure consists of a percutaneous plastic endotracheal catheter placed at the time of surgery for

subsequent instillation of solutions in order to stimulate involuntary coughs postoperatively. Instillations of 0.5 ml of detergent every four hours was reported to have virtually eliminated postoperative atelectasis in their patients. However, insufficient data was reported to fully evaluate the procedure. Subsequently, two additional series have reported on use of transtracheal catheters (133, 134). One series reported no statistical difference in treated and control patients whereas the other found a highly significant difference. It is not possible to sort out the cause of differences in these results. However, it should be noted that this procedure may provoke bronchospasm in patients with asthma, and hence it should be used only with caution in such patients.

The beneficial effects that can be obtained with physiotherapy have been well demonstrated by Thoren (93) whose data are indicated in Figure 11.

FIGURE 11

The influence of chest physical therapy on postoperative respiratory complications*

	Chest physical therapy		
	Both before and after surgery	*Only after surgery*	*No chest physical therapy*
Number of patients	101	70	172
Atelectasis	11	18	68
Pleurisy	9	6	13
Pneumonia	1	2	7
Postoperative temperature elevation above 38.5° C.	19	20	63
Frequency of atelectasis or pneumonia	12%	27.1%	41.9%

Thoren studied 343 patients undergoing gallbladder operations. He based the diagnosis of atelectasis on radiographic findings only. Physiotherapy was defined as instructing the patient in deep breathing, encouragement of coughing while the wound was supported with the hands, and postural drainage. He found that if such physical therapy was administered twice daily after surgery there was a significant decrease in the frequency of atelectasis and pneumonia. However, the results were much more striking if chest physical therapy was begun before surgery and continued in the postoperative interval. These results indicate that procedures relying on patient cooperation are best taught to the patient before he is already experiencing pain and narcosis postoperatively. It is likely that these same admonitions apply to the use of IPPB and perhaps incentive spirometry.

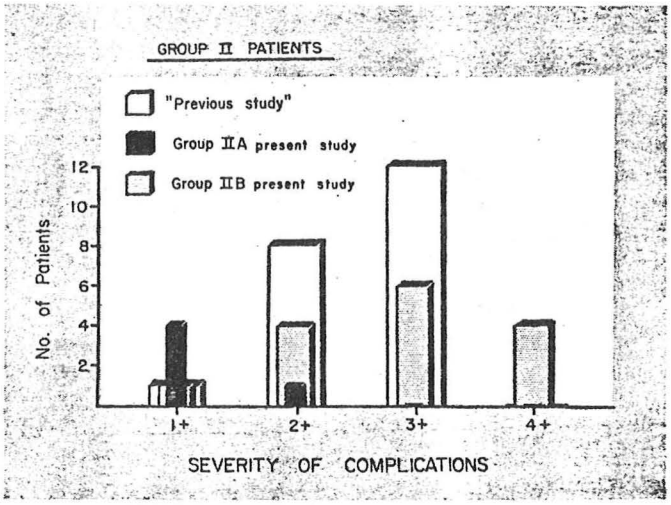
The work of Palmer and Sellick (135) indicates an additional advantage of inhaled bronchodilators. In a consecutive series of 180 patients admitted for repair of inguinal hernia or partial gastrectomy; the patients were randomly divided between two treatment groups. Half received inhaled bronchodilators by a hand bulb nebulizer three times a day followed by 15 to 20 minutes of postural

drainage and chest percussion. Such treatment was continued preoperatively until cessation of sputum production. The treatment was then continued postoperatively. The other group of patients received breathing exercises before and after operation. Based on x-ray findings it was found that only 9% of the group treated with bronchodilators and postural drainage showed atelectasis compared with 43% of the breathing training group. They then extended the study to fractionate therapy into bronchodilators alone, postural drainage and chest clapping alone, breathing exercises alone, and bronchodilators and postural drainage in combination. They demonstrated that the combination of postural drainage with chest percussion and inhaled bronchodilators was necessary for the prevention of postoperative atelectasis. Either procedure used alone was not sufficient.

The more recent study by Stein and Cassara (136) similarly demonstrates the benefit of a combined approach to therapy. Based on his previous study (112) Stein studied the postoperative complications of patients with abnormal pulmonary function tests who were considered to be at high risk for developing postoperative complications. The major results are demonstrated in Figure 12.

FIGURE 12

Severity of complications in previous study, group IIA (treated); and group IIB (nontreated) patients.



High risk patients in Group IIA received pre- and postoperative therapy including cessation of smoking, antibiotics when indicated, bronchodilator drugs, inhalation of humidified gases with an ultrasonic nebulizer, segmental postural drainage, and chest physiotherapy administered one to three times daily. The Group IIB patients were treated by whatever means desired by their surgeons. Among the twenty-three patients in the treated group there were five pulmonary complicaitons. Fifteen of the twenty-five patients in the randomly treated group had postoperative pulmonary complications. It is apparent from the figure

that the untreated patients had more severe complications as well as more frequent complications. These results support reports of earlier workers in regard to inhaled bronchodilators and physiotherapy.

When one reviews the many approaches that have been utilized in the treatment of postoperative patients and the variable results reported, one must agree with the evaluation of Hamilton (137): "With a complication such as atelectasis which appears dependent on immobility, hypoventilation, and lack of cleansing of the lower respiratory tract, any therapy which increases attention to the patient as a whole may effect cures in atelectasis." A reasonable regimen includes the following:

1. Patients at high risk for postoperative complications should be identified according to the risk factors indicated previously, especially simple pulmonary functional evaluation such as spirometry.

2. Patients in a low risk category probably need only careful instruction concerning what to expect in the postoperative period (138), instructions in deep breathing pre- and postoperatively, and supplemental oxygen administration postpoperatively until it can be demonstrated whether such is necessary.

3. Patients who are at high risk should be as free of respiratory secretions as possible before surgery. A reasonable regimen to achieve this includes cessation of smoking, inhaled bronchodilators four times a day, postural drainage and chest percussion following bronchodilators.

4. High risk patients should be instructed in deep breathing preoperatively. A mechanical device such as an incentive spirometer may be of benefit in this regard. If it is not possible to achieve spontaneous deep breathing, an attempt to accomplish this by IPPB may be undertaken. IPPB should be ordered by the tidal volume desired. If it is not possible to achieve large tidal volumes, there is little reason to persist in IPPB.

5. High risk patients should be instructed in coughing while the site of operation is supported.

6. The deep breathing procedure found to be most successful preoperatively should be continued postoperatively along with the other measures.

7. The patient should be as mobile as possible while in bed, and should be forced into ambulation at the earliest possible date.

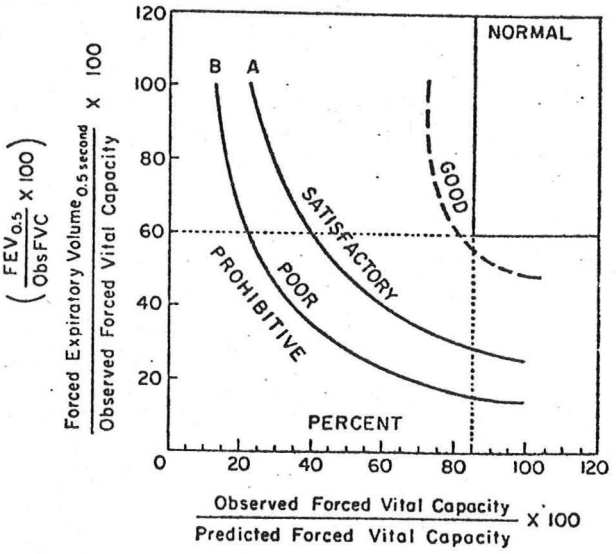
VENTILATORY FAILURE

Patients with severe chronic obstructive lung disease not only have a great risk of postoperative atelectasis and pneumonia, but they also may be precipitated into overt ventilatory failure by surgical procedures. In addition to the various pulmonary changes described following surgery that make lung function less efficient, there is an increase in metabolism owing to the trauma to tissues (139, 140).

This increased energy expenditure necessitates an increase in ventilation which is, on average, 25% above the preoperative values. The narcotics necessary for pain relief may also suppress the respiratory center and lead to ventilatory failure in patients with severe chronic obstructive lung disease. It is not possible to determine the incidence of postoperative ventilatory failure from current literature. However, in at least some hospitals the incidence is apparently increasing owing to a willingness to operate on patients with severe disease and electively artificially ventilate the patients in the postoperative period (141). Although extensive data are not available, Boutros and Weisel (58) found that patients with severe pulmonary disability undergoing major abdominal procedures who were managed carefully did not differ in outcome from patients with no known pulmonary disease receiving standard postoperative care. Similar results were suggested in the study by Ravin (59).

If surgery in patients with severe lung disease is anticipated, it is important to identify those patients most likely to need mechanically assisted ventilation in the postoperative period. Boutros and Weisel (58) found that four of their seven patients (57%) with a MEFR less than 20 L/min required mechanically assisted ventilation while only one of twenty-one patients (4.7%) with higher values required assisted ventilation. This level of expiratory flow is approximately 10-15% of normal. The data from Stein, et al, (112) suggest that patients with an elevated $PaCO_2$ fall in this same category. Perhaps the most useful means of predicting the need for postoperative mechanical ventilation remains the quadrant diagram suggested from this institution by Miller, Wu, and Johnson in 1956 (142). Their diagram is presented in Figure 13.

FIGURE 13



These investigators assessed preoperative lung function in a group of patients undergoing general surgery and determined the complication rate postoperatively. Their data are displayed in a diagram in which an index of expiratory flow, forced expiratory volume in one-half second, $FEV_{0.5}$, is plotted

on the vertical axis as a percentage of the patient's vital capacity; on the horizontal axis is plotted the patient's vital capacity as a percentage of normal. Patients with normal expiratory velocity and normal vital capacity fall to the upper right side of the diagram, and patients with impairment in both velocity and volume fall to the lower left. Working in the 1950's before the advent of current therapy, they found that patients with extremely impaired pulmonary functions were prohibitive operative risks. In contemporary practice, the diagram is useful to predict which patients should be considered for mechanically assisted ventilation in the postoperative interval.

Having identified patients preoperatively with such severe pulmonary impairment, the patient is treated by the means indicated in the previous section. Surgery is delayed, if possible, until expectorated secretions are at a minimum. Preanesthetic regimens probably should not include narcotics. The type of general anesthesia is not important (57-59). Our service prefers for the patient to have an anesthetic regimen that includes an endotracheal tube, so that control of the airway for mechanical ventilation is possible in the postoperative period. Following surgery the patient is maintained on mechanical ventilation in the recovery room until fully awake and alert. At that time an assessment is made of the patient's ventilatory capacity, and if such is thought to be adequate the patient is taken off the ventilator and observed. If respiratory and heart rate remain low, and the patient is capable of attempting to cough, he is observed for several minutes, and arterial blood gases are obtained. If the clinical status and the blood gases are determined to be satisfactory, the patient may be extubated; if not, mechanical ventilation is continued until such determinations are satisfactory. It is preferrable to transfer either type of patient from the recovery room to an intensive care unit where such parameters may be carefully monitored and good ventilatory therapy is possible.

PULMONARY HYPERTENSION

Although good data are not at hand, it is highly likely that severe pulmonary hypertension is a strong contraindication to general surgery. This is suggested by the high maternal mortality at time of delivery in patients with the Eisenmenger syndrome (27%) and with primary pulmonary hypertension (53%) (143). It is further suggested by the work of Skinner and Pearce (144) who found a 50% mortality for intrathoracic or intraabdominal surgery in patients with chronic pulmonary disease and cor pulmonale. It is likely that the mortality of these patients is intra- rather than postoperative. The peripheral vasodilating effect of general anesthesia coupled with the high metabolic demands of surgery necessitate a cardiac output that the patient with severe pulmonary hypertension may not be able to generate.

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