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Atelectasis : a discussion and incidence at UNH

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ATELECTASIS:
A Discussion and Incidence at UNH

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TO THE WIZARD OF ID

With the practice of anesthesia advanced to the state of science that it is today, it is distressing to find that postoperative pulmonary complications are as common as they still are. The most common of these, accounting for around 90 percent of the pulmonary complications, is atelectasis.[1] Atelectasis itself can lead to a few of the more serious complications such as bronchopneumonia and pulmonary abscesses. Atelectasis itself has been reported to occur in from one percent to over 80 percent of all anesthetics. This figure varies so much on the basis of definition alone.

Atelectasis comes from the Greek "ateles"--imperfect and"ektosis"--expansion, which simply means imperfect expansion of the lungs.[2] Although most clinicians agree on the definition of atelectasis, few agree on the diagnosis.

In order to discuss the diagnosis, we must understand the mechanics of atelectasis. There are four major types of atelectasis: physiologic, compressive, contractive, and obstructive.

Physiologic atelectasis is that which occurs normally in the fetus prior to expansion of the lung at birth. A normal lung at the end of expiration contains many collapsed alveoli--atelectatic areas of the lung; but these are constantly being reinflated with the unconsious periodic deep breaths that we all take called sighs.[3]

Compressive atelectasis is incomplete expansion of the lung from forces external to the lung. Such things as pneumothorax, hemothorax, severe chest deformities, and neuromuscular disorders that limit the physical expansion of the lung can cause atelectasis.[4]

The third type, constrictive, is based on the finding of large amounts of smooth muscle lining the alveolus in people with chronic lung diseases. Under the proper autonomic stimulus, it is thought that these

fibers could cause a contraction of an alveoli and thereby cause atelectasis.[5]

The fourth type, obstructive, can be subdivided into:first, extra mural obstruction caused by a lymph node; second, intrinsic mural obstruction caused by an adenoma, stenosis of a bronchus due to tuberculosis, edema due to infection or trauma, and constriction of bronchiolar musculature as seen in asthma and emphysema; and thirdly, intramural obstruction caused by a foreign body or secretions.[6]

Postoperative atelectasis falls into the category of obstructive atelectasis secondary to secretions or miliary atelectasis, which has its mechanism in the failure to reexpand collapsed alveoli due to absence of sighs or coughs. More likely elements of both are involved.

One would think that if a terminal bronchiol developed a sudden obstruction that the alveoli beyond this would not collapse because of the idea that an alveolus is a balloon and the occluded bronchiol is the stem. This would be true if this were a closed system, but it is not. There is a constant flow of blood past this alveolus. This blood absorbs gases from the alveolus until it ultimately is collapsed.

Now the important factor in this mechanism is the gas contained in the alveoli. Breathing room air, an alveolus contains about 73 percent nitrogen, 16 percent oxygen, 6 percent water vapor, and 5 percent carbon dioxide. In a closed system with only blood flow past an obstructed alveolus, the gases are quickly absorbed except the nitrogen which is slowly dissolved into the blood. Thus, the normal alveolus even if obstructed for a short time does not become atelectatic instantaneously. However, if a patient has had an anesthetic and received 50 percent oxygen, 50 percent nitrous oxide and one percent halothane and then breathed only 100 percent oxygen for five or ten minutes postoperatively, the alveolus

then would contain only oxygen, carbon dioxide, and water vapor. If obstructed, the alveolus will have all the oxygen absorbed in a period of six to eight minutes resulting in a collapsed alveolus. Now if the blood flow past the alveolus is decreased or different gases used, it will take longer. If, for example, helium were used in a 75 percent mixture, it would take a day for the gas to be dissolved. So it is that the high concentrations of oxygen used during anesthesia may lend the lung more susceptible to atelectasis if a closed system develops.[7] The obstruction postoperatively can be due to aspirated blood and the thick secretions of the tracheo-bronchial tree. How blood can get into the trachea is easily understood. The real problem is the patient's own natural secretions.

During anesthesia, the cilia of the cells lining the tracheo-bronchial tree are slowed down both by the anesthetic agent directly and indirectly. They are slowed by the high flow rates of dried gases over a prolonged period of time. This stops the natural flow of secretions up to the trachea to be coughed out. The cough reflex is also depressed so that a patient does not automatically cough these secretions out. The premedications that are given to prevent the production of thin secretions so that only the thick mucoid ones are present causes these to be more tenacious and thereby more difficult to clear. But this is better than having such a proliferation of secretions which could completely clog a main stem bronchus or even the trachea.

Another important factor to consider is any disease state of the individual patient which causes a hypersecretion of this matter such as chronic bronchitis, bronchiectasis, the common cold, and smoking. The morning cough of chronic bronchitis or "smoker's cough" is considered to be a sign that this patient will have an increased risk of postoperative

pulmonary complications.[8]

A final point to consider in the etiology of atelectasis is any interruption of the natural deep breath or sigh mechanism; because it is this mechanism which reexpands the ever-closing alveoli in the lung. This is why people on ventilators without occasional sighs develop atelectasis. This is also why people with brain damage to the respiratory center can develop atelectasis. This is also why postoperative patients, whom it hurts to take deep breaths, develop atelectasis.

Having discussed atelectasis on a pathophysiologic basis, we can now discuss it as a disease entity. In general there are three types of atelectasis: the first has no other name than atelectasis, second is progressive atelectasis, which is an invariably fatal disease process in which the lung progressively collapses despite vigorous treatment. It is thought that this disease process is due to some acute abnormality in the surfactant of the lung. Surfactant is a lipoprotein that lines the alveoli and has an ever-changing surface tension which is a function of the diameter of the alveolus. This allows the collapsed alveolus to be opened without the tremendous forces that the ordinary laws of physics state it would take to inflate a collapsed fluid-lined sphere. Little else is known about surfactant because it is so fastidious. It is known that hypoxia, manipulation, and some chemicals can change it.[9] In any case, progressive atelectasis is a malignant process which fortunately is very rare and is unresponsive to the most vigorous treatment.

The third type of atelectasis is called congestive atelectasis and is almost a post mortem diagnosis. These individuals have the history and every clinical finding to go along with hypovelemic shock except their cyanosis. They are cold, clammy, have tachycardia, hypotension or hypertension, dyspnea, tachypnea, and cyanosis. This disease is very frustrating

and treatment is very unsatisfactory. It is considered by some to be a complication of parenteral therapy in which the addition of blood trips these people into a progressive downhill course which is unresponsive to any amount of "tinkering." The only reported successful treatment is vigorous use of the positive pressure ventilators with 100 percent oxygen with very high flow rates, and very high pressures. Although there is no edema of the lung like in acute pulmonary edema, this disease must be treated like acute pulmonary edema if the diagnosis is made. This addition of parenteral fluids is contraindicated for it is apparently the "coup de gras" that causes these people's demise.[10]

The first type of atelectasis is the one which makes up 90 percent of the postoperative pulmonary complications. Its incidence is largely a function of how hard it is looked for and what the criteria for diagnosis are.

The clinical diagnosis of atelectasis is highly debatable. There are several schools of thought. There is the conservative group who insists that an x-ray is needed to diagnose the disease. This group believes the incidence is very low; others say that anyone who undergoes an anesthetic that affects the central nervous system must have an element of atelectasis. Most clinicians believe a clinical diagnosis can be made on a basis of tachypnea, tachycardia, and an increase in temperature with or without dyspnea during the first 48 hours postoperatively.

This statement must be qualified. If atelectasis is to occur, it will do so in the first 48 hours postoperatively. In fact, 95 percent of it will occur in the first 36 hours postoperatively. If a patient is on a ventilator, it can happen at any time.

The tachypnea is due to hypoxemia, the tachycardia is due to infection distal to the obstruction, and the temperature increase is due to a peripheral

automonic vasoconstriction causing heat retention.[11] The dyspnea depends on the extent of the atelectasis. The difference then, between atelectasis and another feared pulmonary complication, pulmonary embolism, is the rapidity of the onset of symptoms. A pulmonary embolism is usually acute in onset, can happen at any time, and often is accompanied by hemoptysis. Atelectasis is usually insidious and does occur over a relatively short period of time, but not with the abruptness of a pulmonary embolus. Another aspect in the differential diagnosis is the laboratory studies. A pulmonary embolus has typical enzyme changes, whereas atelectasis has none.

A new area of investigation has been the blood gas studies. It was thought that these could be quite helpful in the diagnosis of atelectasis, but they have not proven to be a panacea. The reason for this has been the unpredictable PaO_2 of the normal individual and its normal variation in the same individual. Atelectasis causes a shunting of blood subsequently a desaturation. But a certain amount of shunting occurs in normal individuals. The amount depends on many things. The normal range is not known. Studies have shown that so called "normal people" (at least asymptomatic) have PaO_2's anywhere from 50 millimeters of mercury to 98 millimeters of mercury breathing room air. The factors involved are innumerable i.e., age, activity, general health, amount of lung pathology, smoking, weight, sex, medications, state of anxiety, position in bed, etc. The indication is that few individuals have their PaO_2 at the level that the theoretical oxygen desaturation curve says it should be. Also the point on the curve when symptoms begin is also undefined.

The x-ray diagnosis is legitimate for large areas of atelectasis only and thereby missing the smaller yet clinically significant cases of atelectasis.

The best method yet of diagnosing the disease is the clinical impression. Any rise in temperature to a level over 100 degrees Farenheit accompanied

by a tachycardia and tachypnea within the first 24 to 36 hours postoperatively whether or not lung findings can be demonstrated must be considered atelectasis until proven otherwise.[12] A large area of atelectasis can be detected with a stethescope, but again smaller yet significant diseases will be missed. The usual ascultatory findings will be decreased fremitus, voice sounds, dullness to percussion, and possibly some rales; but again these gross appraisals will miss many significant cases of atelectasis.

Why is it so important to diagnose atelectasis? It is important because atelectasis sets up a vicious cycle which contributes to the patients morbidity and even to his mortality.

The treatment of atelectasis can be viewed from two vantage points: first, prevention, second, treatment of the disease. Most people will agree that it is better to prevent a disease than it is to cure it. This is very true in the case of atelectasis.

The pathogenesis and pathophysiology of atelectasis is fairly well understood, and it is easier to prevent than to cure. We are then faced with the problem of preventing a disease that has an opportunity to occur every time a patient goes into the operating room. Most of the people do well, and most of those that develop the disease are not too ill for a brief period of time. The important thing then is to try and find the susceptibles. These are the patients who are more likely to develop the disease and those who would do very poorly if it were contracted. Once the susceptibles are found, then they must be treated vigorously trying to prevent the disease pre, intra, and postoperatively.[13]

I would like to first discuss prevention and treatment and the definition of a susceptible. There are two types of preventive therapy. The first is that therapy given to every preoperative patient and second that which the susceptible patient should receive. Every preoperative candidate should

be instructed on deep breathing exercises i.e., yawns, deep breaths, and coughs, and the importance of these in his postoperative course. Most patients for an elective procedure will not think this is important, so it is up to the anesthesiologists, surgeons, and the nursing personnel to reinforce these instructions and observe the patient practicing his breathing exercises. The susceptible patient, on the other hand, gets a more vigorous training course. Depending on the extent of disease present, he may learn postural drainage, IPPB, he may receive prophylactic drugs, or even tracheal suction.[14]

Intraoperative prevention is a chance par excellance for the anesthesiologist to make sure secretions are properly suctioned, that the lung doesn't get compressed, and that the patient is sighed. In some cases a patient's respiratory system may actually be in better condition leaving the operating rooms than when he entered it.

Postoperative prevention is centered around early ambulation and breathing exercises. The most successful exercises for expanding collapsed alveoli is a yawn because of the more prolonged pressure build up while the most successful measure for unclogging an obstructed alveolus is a good brisk cough because of the sudden pressure.[15] Vigorous use of these exercises prevents most atelectasis. Also included in prevention is position in bed. A slight Trendelenburg in the prone position is not really too uncomfortable even for abdominal incisions once it is attained and can do wonders for the drainage of secretions. The proper suctioning of endotracheal tubes left in tracheotomies is also very important in preventing atelectasis and pneumonia because this removes potential plugs.

Adjuncts to prevention can be intercostal nerve blocks to decrease pain and encourage deep breaths, etc. Most people, if they have ambulated early and do their exercises properly, will have little difficulty postoperatively. Also important is the right dosage of narcotics postoperatively--

enough to relieve discomfort yet not enough to take away the normal urge to sigh. The key to prevention postoperatively is the nursing personnel. For it is these people who must make sure that the breathing exercises are performed correctly and often enough. It is important that they themselves be instructed by the physicians both in the technique and the rational of these procedures.

The treatment of atelectasis once it is diagnosed is varied. It ranges from increased insistance of breathing exercises, to more ambulation, to vigorous chest pounding, postural drainage, intratracheal suction by catheter, and even transtracheal suction. All of these stimulate a healthy cough unrestricted by pain.

There are drugs which can be used to make a person take deep breaths i.e., doxapram and even intravenous paraldehyde in doses of one to three cubic centimeters in a person who is not too drowsy.[17] These should not be relied upon because they are not better than proper deep breathing techniques. Antibiotics can also be added, but they are also only adjuvants. High humidity is also helpful, as is IPPB with mucolytic agents. At times even a respirator will be necessary. These are all accepted means of treating atelectasis.[18]

The big problem in prevention of atelectasis is determining who is susceptible. This has been the object of many investigators. First of all, it must be remembered that anyone undergoing an operation is susceptible; but there are many facets to be considered that increase the risk. Among these are age, sex, pain tolerance, anesthetic method, incision, length of procedure, preexisting lung disease, smoking, intubation, mental attitude of patient, affluence, dental hygiene, productive cough, self-propagating cough, obesity, malformation of the chest, recent upper respiratory infections, premedication, pain medication, ambulation, muscular diseases, etc.

With this many things to consider, one cannot have a straight formula for determining a susceptible. To determine a susceptible, one must use his own clinical judgment at the bedside. There are, however, several key factors to be considered in discovering the susceptible. These will be discussed in the following presention.

Since the reported incidence of atelectasis varies according to the diligence of the inspector, it seemed appropriate to find out what the incidence of atelectasis was at the University of Nebraska Hospital. This study was undertaken in the hopes of finding out the incidence of clinically significant atelectasis and also to evaluate a method of defining the "susceptible."

The most crucial and debatible aspect of this study is the definition of atelectasis. With the thought in mind of making this a practical way of evaluating patients, the diagnosis was based solely on clinical findings which were: 1. abrupt increase in temperature, 2. increase in heart rate, 3. increase in respiration. Arbitrarily an oral temperature of 100 degrees Farenheit or greater was considered to be abnormally high in a person whose temperature was normal preoperatively. An increase in resting heart rate to over 100 was considered significant and a resting respiratory rate of 25 was considered pathologically elevated. All of these must occur within the first 36 hours postoperatively before a clinical diagnosis of atelectasis could be made. If any rales or rhonchi were heard, these too were sufficient to be called atelectasis. This undoubtedly may have included some lung pathology other than atelectasis. However, the purpose of this study was to find out what the incidence of clinically significant lung disease was postoperatively. These requirements are subject to debate, but I believe these to be the most useful clinical tools in evaluating this disease.

This survey was a prospective study of all scheduled general anesthetics given to patients over 19 years of age not including thoractomies and craniotomies over a five-week period in the summer of 1968. Thoracotomy and craniotomy were omitted because of the special problems they inherently have. All patients were seen the evening before the operation, the evening following the operation, and the day after the procedure. This time period covered 36 hours postoperatively.

All visits were made by the same individual independent of all preoperative and postoperative visits by the anesthesia or surgery staff. There was no attempt made to alter any positive or negative effect any preanesthetic visit might have had. The visit consisted of first reading the chart and copying data such as name, age, sex, diagnosis, procedure planned, and any complicating disease. The patient was then interviewed and asked about any history of pulmonary disease, such as asthma, bronchitis, recent upper respiratory infection, etc. Also, an inquiry was made in smoking history and a test cough was elicited. The patient's chest was oscultated and percussed and all findings recorded on a special sheet. Then, if the blood gas analyzer was operational, a sample of arterial blood was drawn if this could be done relatively atraumatically. If it could not, then the blood gas study was not done on this patient.

During the first postoperative visit, the chart again was purused and data about the actual anesthetic was recorded including agents, duration, position, intubation, and any complications of surgery. The patient was then examined for site of incision and the chest was again examined. If the patient had preoperative arterial gas studies, a followup tap was attempted. If the preoperative attempt was unsuccessful, no attempt was made postoperatively. The patient's temperature pulse and respiration curves were then studied and noted.

On the day after the operation, the patient's chest was examined again along with his TPR curves; and at this time, a diagnosis of atelectasis was or was not made and recorded on the patient's own sheet. At the end of the five-week period, the information was then tabulated and interpreted.

The series contained 42 patients all of who received halothane anesthesia with nitrous oxide. In this group, there was only one death within the first few days of the procedure. This was a 61 year-old man with obvious terminal carcinoma of the pancreas. In this group, there were 14 cases of clinical atelectasis giving a rate of 14 out of 42 patients or 33 percent; but when the subdivision of this group is examined, some interesting facts are uncovered.

Now there are other factors to be considered when examining these figures. First, since there are only 42 subjects in this series, the deductions are not very significant, but the implications are. The data here is not presented as proven fact but only as interesting happenings which can be observed if looked for. For instance, because of the outcome it can be said that certain people are more likely to get atelectasis; they are the so called "susceptibles." In hopes of finding these people, certain particular factors were investigated. The relative incidence of atelectasis in relation to the point of incision, smoking, or the presence or absence of a wet, dry, or self-propagating cough was considered. Also included were intubation, duration, and position.

The sites of incision were classified as: upper abdominal, i.e., above the umbilicus; lower abdominal, i.e., below the umbilicus; abdominal, i.e., any incision that crossed the transverse at the level of the umbilicus; perineal, i.e., D and C, cystoscopy; the peripheral including hand, leg, back, and breast; and finally the head and neck surgery including tooth extraction, etc., excepting craniotomy and any surgery directly involving

the trachea or larynx. In the analysis of the data, any abdominal incision was called abdominal although it was originally classified as upper or lower.

The cough was classified as dry, wet (productive), and self-propagating. Also noted was position: prone, supine, lithotomy, and lateral decubitus. Whether or not the patient was intubated was noted without regard to oro-tracheal or nasotracheal.

The data for males in the age group 20-49 years of age is that 3 of 4 developed atelectasis. In the 50 years and older age group, 5 of 9 developed atelectasis. Of those who developed atelectasis, 6 patients of 8 were active smokers; 5 of 8 patients were abdominal procedures (including upper and lower); 2 of 8 were head and neck surgeries; and 1 of 8 was peripheral. The supine position was predominate with 6 of 8 patients, 1 of 8 patients was prone and 1 of 8 was in the lateral decubitus position. None of the patients had any previous history of lung disease, 3 of 8 had a wet cough and all were intubated, but every man in this series was intubated. All had clear chests on preoperative examination. The length of the procedure in the atelectatics varied from 1½ hours to 4½ hours. In those who did not develop atelectasis, the range was 1-3/4 to 3½ hours. The mean in both groups being 2¼ hours. From the viewpoint of incision, 5 of 8 of the abdominal procedures, whereas 2 of the 3 patients with the head and neck procedures developed atelectasis; and the single peripheral procedure developed atelectasis.

From this group, we can surmise that any male is susceptible to atelectasis, especially if the incision is in the abdomen. The effect of smoking could not be justly evaluated because of the lack of non-smokers in the group. The effect of intubation cannot be judged because of the lack of non-intubated cases. The only conclusion that can be drawn is that any male subjected to major surgery, especially abdominal surgery,

is susceptible to atelectasis. Smoking probably aggravates this, but this cannot be proven here. Also the presence of a wet or dry cough did not seem significant in this group.

Among the older women, i.e., 50 years of age and older, 4 out of 14 patients developed atelectasis; of these 4, none were smokers; all had dry coughs; only 1 had any chest findings preoperatively; all were intubated; the duration of the procedure ranged from 2½ to 4 hours, the mean being 2½ hours. There were 2 abdominal and 2 peripheral procedures. Among this age group in general, there were only 2 smokers, and they did not develop atelectasis. Two of the four of the abdominal procedures developed atelectasis, while three perineal procedures did not. Two of the six of the peripheral procedures developed atelectasis. Only 3 of 14 patients were not intubated and these 3 did not develop atelectasis. The duration of all the cases ranged from 1 to 4½ hours. The mean being 2 hours. All of these women were in a supine position except 3 patients in the lithotomy position and one patient in the prone position . This patient in the prone position developed atelectasis. Two of the total of 14 ladies had previous history of lung pathology, one of these developed atelectasis. There were no wet coughs in this group. One lady had preoperative bronchitis, but she did not develop atelectasis.

From this group, we see a similar relation of site of incision to atelectasis. The effect of smoking again could not be correlated but this time because of the lack of smokers in the group.

Among the young ladies, i.e., 20-49 years of age, 2 of 15 developed atelectasis. One of these 2 underwent abdominal surgery and the other underwent peripheral surgery. One had a history of pulmonary disease. One patient was a smoker; and both had dry coughs, clear chest, were prone and intubated. One of the two patients was operated on for five

hours and the other was operated on for four hours. One patient was obese. A breakdown of the cases reveals that one of three of the abdominal procedures developed atelectasis, and one of these was a post partum tubal ligation. One of 3 of the peripheral procedures developed atelectasis. None of the three head and neck procedures and none of the six perineal procedures developed atelectasis. The duration of the procedures in this group ranged from 1 to 8 hours. The mean being 2 hours. Eight of 15 patients were intubated.

Three of the fifteen patient's coughs were wet, and none of these developed atelectasis. Only 2 out of 15 had a history of pulmonary diseases, and all 15 had clear lungs on preoperative examination. Also, 10 out of 15 were smokers and only one of these developed atelectasis. So in this group, we again see an increase in atelectasis in both abdominal and lengthly procedures. Also smoking seemed to have no effect in this group.

If we consider all 42 patients together, we find 8 of 16 abdominal cases developed atelectasis, 10 out of 16 patients were smokers, 5 patients out of 8 with atelectasis were smokers. Surprisingly both of the procedures done with the patient prone, i.e., laminectomy, developed atelectasis. No perineal case developed atelectasis. Four out of 10 peripheral procedures developed atelectasis. Two out of 7 head and neck cases developed atelectasis. The mean duration of the atelectatic cases was 2½ hours. The mean of the non-atelectatics cases was also 2½ hours. The preoperative test cough showed no correlation to atelectasis. Twenty-one out of forty-two patients smoked and seven out of fourteen atelectatics smoked.

There were only five cases in which both preoperative and postoperative arterial taps were successfully drawn. These blood gases were analyzed on a Bechman gas analyzer operated by the same individual who made the visits.

Case No. 2 was a 47 year-old male for head and neck surgery. He had a submandibular gland removed, had no preoperative chest disease, did not smoke, had a clear chest, and a dry cough. Preoperatively, his PaO_2 was 84, and his $PaCO_2$ was 29. Postoperatively, his PaO_2 was 45, and his $PaCO_2$ was 29. He was clinically diagnosed as having atelectasis. Case No. 4 was a 47 year-old male for gastrectomy. He did not smoke and had no preoperative disease. His preoperative PaO_2 was 55 and his $PaCO_2$ was 36. Postoperatively, his PaO_2 was 70 and his $PaCO_2$ was 30. He was diagnosed clinically as having atelectasis. Case No. 9 was a 68 year-old man for parotid gland removal. He had no preoperative chest disease, had a wet cough, smoked, and had clear lungs. His preoperative PaO_2 was 55 and his $PaCO_2$ was 34. Postoperatively, his PaO_2 was 48 and $PaCO_2$ 36. Case No. 34 was a 38 year-old female for tooth extraction. She smoked, had a dry cough and clear lungs. Her preoperative PaO_2 98 and $PaCO_2$ was 24. Postoperatively, her PaO_2 was 62 and $PaCO_2$ was 35. She did not have clinical atelectasis. Case No. 27 was a 78 year-old woman for colycystectomy. She had no previous lung pathology, did not smoke, had clear lungs and a dry cough. Her preoperative PaO_2 was 92 and her $PaCO_2$ was 28. Postoperatively she had a PaO_2 of 50 and a $PaCO_2$ of 30. She had clinical atelectasis.

Four of the above five patients had atelectasis. Three had marked reduction of PaO_2 but one of these did not have atelectasis. One had essentially no change; and one had a better PaO_2 postoperatively than preoperatively, and she still developed atelectasis. This may be due to several deep breaths just prior to drawing the sample.

It is impossible to draw any conclusions here except that arterial gas studies may not be the perfect test for diagnosing atelectasis.

There were 8 other preoperative PaO_2 and $PaCO_2$'s drawn; these were

from apparently healthy (at least asymptomatic) people. These values ranged from PaO_2's of 48 to 92; the mean being 64 mm. hg. The mean of all preoperative PaO_2's which were drawn from supposedly healthy (pulmonary function wise) patients for elective procedures was 64 mm.hg. with a range of 48 to 98. The three 90+ readings were taken from people who had just walked down the hall a distance of 75 to 80 feet and then climbed into bed. The other readings were taken from patients already lying in bed for some time. Of the postoperative readings, all taken from people who had not been out of bed all day, the range was 46 to 70 with a mean of 50. We see that there was a drop in the oxygen saturation postoperatively.

The most interesting finding is, however, that very few people unless they are made to breathe deeply have oxygen partial pressure anywhere near the point where the theoretical oxygen desaturation curve says their PaO_2 should be. Most people lying down and even standing around are circulating desaturated blood. This is a little appreciated fact that becomes important when a physician draws an isolated PaO_2 from a patient and then hyperventilates a patient in the effort to get a normal saturation of 98 percent. A PaO_2 of 70 in a patient resting quietly in bed is not too bad especially if the $PaCO_2$ is not elevated.

In the quest of being able to define the patient most likely to develop atelectasis and on the basis of this study, we can say that any male undergoing abdominal surgery and is a smoker is almost a sure bet to develop atelectasis. (4 of 5) Any person undergoing a prolonged procedure in the prone position and is a smoker will develop atelectasis. (2 of 2) Also any woman undergoing major abdominal surgery is likely to develop atelectasis.(3 of 6) No other factors are considered to be significant or tested by this study. Factors not considered in this

study were obesity, effect of the time of the year upon atelectasis, and whether the preoperative visit by the anesthesia staff helped cut down the incidence of atelectasis or whether it affected it at all; or whether the postoperative nursing care was sufficient to prevent some atelectasis.

In summary, 42 consecutive elective operations under general anesthesia on adults not including thoracotomies and craniotomies were studied and an incidence of 14 of 42 patients or 33 percent was found. This was based on the clinical diagnosis of increase in temperature, increase in heart rate, and increase in respiratory rate. Also, arterial blood gas studies were attempted pre and postoperatively. This was found to be of much technical difficulty and extremely rewarding in findings.

In conclusion, the topic of atelectasis was discussed concerning definition, classification, pathophysiology, pathogenesis, prevention, treatment, and finally the incidence at the University of Nebraska Hospital was determined along with an attempt to define the "susceptible."

We can now see that atelectasis is an important and common cause of postoperative morbidity. It is often slighted, but none the less, it is the chief offender of postoperative complications that should be looked for and treated vigorously both by prevention and by specific therapy by all those responsible for the care of the surgical patient.

Pt. #	Sex	Age	Incision	Operation	Preexisting Diseases	Op. Complications	History of Pul.Disease	Smoker	Cough	PE Pre. Op	Duration (Hrs.)	Position	Intubation	Atelectasis	Blood Gases (if any)
1	M	42	U. Ab.	Cholecystomy	Cirrhosis	None	TBC	yes	Dry	Clear	2½	Supine	Yes	No	pre.op. 68 35
2	M	47	H + N	Submandibular Glandectomy	None	None	None	no	Dry	Clear	2½	Supine	Yes	Yes	pre.op. 88 28
3	M	44	Peripheral	Laminectomy	AS + AI Severe	None	None	yes	Dry	Clear	4½	Prone	Yes	Yes	post.op. 46 29
4	M	47	AB.	Gastrectomy	None	None	None	in past	Dry	Clear	2½	Supine	Yes	Yes	pre.op. 55 36
5	M	61	U. Ab.	Coeliotomy	Pancreas	None	None	yes	Wet	Rales	1 3/4	Supine	Yes	No	post.op. 70 36 pre.op. 64 31
6	M	58	U. Ab.	Coeliotomy	Hodgkins	None	Asthma	no	Dry	Clear	3½	Supine	Yes	No	
7	M	61	H + N	Tooth Extraction	Alcoholism	None	None	yes	Wet	Rales	1	Supine	Yes	No	
8	M	68	L. Ab.	Hernia	BPH + Syphilis	None	None	yes	Dry	Clear	2½	Supine	Yes	No	
9	M	68	H + N	Neurotomy for tic Douloureux	Pitting Edema of Ankle	None	None	yes	Wet	Clear	2½	Supine	Yes	Yes	pre.op. 57 33
10	M	57	U. Ab.	Nephrectomy	Ca of Kidney	None	None	yes	Dry	Clear	2½	Lld.	Yes	Yes	post.op. 50 36 pre.op. 48 36
11	M	63	L. Ab.	Epicystotomy	Paraplegia	None	None	yes	Dry	Clear	3½	Supine	Yes	Yes	
12	M	70	Ab.	Ventral Hernia	Diarrhea	None	None	yes	Wet	Clear	1½	Supine	Yes	Yes	
13	M	60	Ab.	Ulcer Repair	ASHD	None	None	yes	Wet	Clear	2	Supine	Yes	Yes	
14	F	81	Peripheral	Hip Pinning	Senility	?	?	?	?	Bronchitis	3½	Supine	Yes	No	
15	F	61	L. Ab.	TAH	None	None	None	No	Dry	Clear	2½	Supine	Yes	No	pre.op. 92 22
16	F	60	H + N	Cervical Spondylolisthesis	Hypertension	None	None	No	Dry	Clear	2	Supine	Yes	No	pre.op. 48 35
17	F	69	Peripheral	Mastectomy	Diabetes	None	None	Yes	Dry	Clear	4½	Supine	Yes	No	
18	F	80	Peripheral	Mastectomy	Senility	None	?	No	?	Clear	4½	Supine	Yes	No	
19	F	84	Peripheral	A.K. Amputation	Obtunded	None	?	?	?	Clear	1	Supine	Yes	No	
20	F	70	U. Ab.	G. B.	None	None	C.Bronchitis	No	Dry	Clear	4	Supine	Yes	No	
21	F	62	Perineal	D + C	Chronic brain syndrome	None	None	No	Dry	Clear	1	Lld.	No	No	
22	F	62	Perineal	D + C	Obesity	None	None	Yes	Dry	Clear	1	Lld.	No	No	
23	F	70	Perineal	D + C	None	None	None	No	Dry	Clear	1	Lld.	No	No	
24	F	63	Ab.	Incisional Hernia	Hypertension	None	asthma bronchitis	No	Dry	Wheeze	4	Supine	Yes	Yes	
25	F	88	Peripheral	Hip Pinning	?	?	?	?	?	Clear	2½	Supine	Yes	Yes	
26	F	63	Peripheral	Laminectomy	None	None	None	No	Dry	Clear	2½	Prone	Yes	Yes	pre.op. 62 38
27	F	78	U. Ab.	G. B.	None	None	None	No	Dry	Clear	2½	Supine	Yes	Yes	pre.op. 92 28
28	F	22	U. Ab.	Splenectomy	I.T.P.	None	None	Yes	Dry	Clear	2 3/4	Supine	Yes	No	post.op. 50 30
29	F	28	L. Ab.	Tubal Ligation	None	None	None	Yes	Dry	Clear	1	Supine	No	No	
30	F	36	Ab.	Ventral Hernia	None	None	Bronchitis	Yes	Dry	Clear	4	Supine	Yes	Yes	
31	F	36	Peripheral	Vein Stripping	Obesity	None	None	No	Dry	Clear	5	Supine	Yes	Yes	
32	F	48	Peripheral	A.K. Amputation	General Vascular Disease	None	None	Yes	Dry	Clear	1 3/4	Supine	Yes	No	
33	F	43	Peripheral	Breast Biopsy	Urinary Tract Infection	None	None	Yes	Dry	Clear	1½	Supine	No	No	pre.op. 63 34
34	F	38	H + N	Extraction	None	None	None	Yes	Dry	Clear	1	Supine	Yes	No	pre.op. 98 29
35	F	36	H + N	Extraction	None	None	None	Yes	Dry	Clear	1½	Supine	Yes	No	post.op. 62 35
36	F	42	H + N	Radical Neck	None	None	None	Yes	Dry	Clear	8	Supine	Yes	No	
37	F	45	Perineal	Radium Implant	None	None	None	Yes	Wet	Clear	1	Lld.	Yes	No	
38	F	36	Perineal	D + C	None	None	None	No	Dry	Clear	½	Lld.	No	No	
39	F	32	Perineal	Vag. Hys.	None	None	None	No	Wet	Clear	1½	Lld.	No	No	
40	F	28	Perineal	Vag. Hys.	None	None	None	No	Dry	Clear	2	Lld.	No	No	
41	F	27	Perineal	D + C	None	None	None	No	Dry	Clear	1	Lld.	No	No	
42	F	47	Perineal	Retrograde	Ca of Cervix	None	TBC	Yes	Wet	Clear	1	Lld.	No	No	pre.op. 68 34

FOOTNOTES

1. J.H. Modell and F. Moya, "Postoperative Pulmonary Complications," ANESTHESIA AND ANALGESIA, Vol. 45 #4, p. 432-39

2. J.R. Jones and W.W. Fieber, "Current Concepts in Atelectasis," MEDICAL TIMES, Vol. 94 #1, p. 48-50

3. Op. sit.

4. Op. sit.

5. G. Corssen, "Changing Concepts of the Mechanism of Pulmonary Atelectasis," JOURNAL OF THE AMERICAN MEDICAL SOCIETY, Vol. 183 #5, p. 314-17

6. J.R. Jones and W.W. Fieber, "Current Concepts in Atelectasis," MEDICAL TIMES, Vol. 94 #1, p. 48-50

7. Op. sit.

8. B.A. Greene and S. Berkowitz, "The Prevention of Atelectasis or Pneumonia Following Abdominal Operations," ANESTHESIOLOGY, Vol. 14 #2, p. 166-79

9. R.J. Ward, F. Danziger, J.J. Bonica, S.D. Allen, and J. Bowes, "An Evaluation of Postoperative Respiratory Maneuvers," SURGERY, GYNECOLOGY, AND OBSTETRICS, Vol. 123 #1, p. 51-54

10. R.E.L. Berry, C.A. Sanislow, "Clinical Manifestations and Treatment of Congestive Atelectasis," ARCHIVES OF SURGERY, Vol. 87 #1, p. 153-167

11. A.M. Lansing and W.G. Jamieson, "Mechanisms of Fever in Pulmonary Atelectasis," ARCHIVES OF SURGERY, Vol. 87 #1, p. 168-75

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13. Op. sit.

14. R.A. De Wall and G.L. Snider, "Postoperative Atelectasis," THE CHICAGO MEDICAL SCHOOL QUARTERLY, Vol. 29 #1, p. 7-10

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16. F.T. Kurzweg, "Pulmonary Complications Following Upper Abdominal Surgery," THE AMERICAN SURGEON, Vol. 19 #10, p. 967-74

17. B.A. Greene and S. Berkowitz, "The prevention of Atelectasis or Pneumonia Following Abdominal Operations," ANESTHESIOLOGY, Vol. 14 #2, p. 166-79

18. R.A. De Wall and G.L. Snider, "Postoperative Atelectasis" THE CHICAGO MEDICAL SCHOOL QUARTERLY, Vol. 29 #1, p. 7-10

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