"BRONCHOPNEUMONIA": THE ANESTHETIST'S RESPONSIBILITY? *

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A RELATIONSHIP between the treatment of pain and respiratory morbidity has long been recognized. It was expressed in the phrases "ether pneumonia," "postoperative pneumonia" and "postanesthetic pneumonia." In the light of present knowledge, it would be as logical to speak of "morphine pneumonia," "barbiturate pneumonia," "post-head-injury pneumonia" or "post-depression pneumonia." Realizing the non-specificity of the condition, the internist and the pathologist have come to write "Bronchopneumonia" in the records as a part of the clinical picture in nearly all illnesses following injury and surgery. The term is seldom missing from the terminal clinical record or autopsy report.

Two schools of thought have grown up to explain the appearance of pulmonary morbidity in patients subjected to surgery and injury. The first utilizes embolism as the blanket etiologic factor. Such a concept offers little encouragement that prevention can be accomplished other than through improvements in surgical technique and circulatory stimulation. The second explanation is based upon the belief that bronchial obstruction and alveolar inactivity is followed by absorption of the atmosphere in the affected area of lung, and bacterial growth follows in the resulting atelectatic air spaces. The latter concept serves as a rational basis for prophylaxis and treatment. Since atelectasis, massive collapse, and bronchopneumonia not infrequently follow the efforts of the anesthetist, he is interested in any analysis that can be brought to bear upon the subject which promises a reduction in the incidence and the mortality. If either drug administration or the care of drugged patients can be shown to affect the occurrence of pulmonary morbidity, consideration of the anesthetist's care of his patients may point the way to a reduction in such morbidity.

Efforts at the Wisconsin General Hospital during the past thirteen years have undoubtedly decreased the incidence of respiratory disease. The Department of Anesthesia was initiated in February, 1927. During the first six months of that year, the Department of Pathology reported six autopsies performed on patients who had died of "post-anesthetic bronchopneumonia" on one surgical ward of sixty beds. Adequate records were not available at that time to make a statistical report covering the whole surgical service. The records of these six

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cases contained notes of fever, rales, etc., being present before operation in four cases. The anesthetic drugs and techniques used covered the range of the available armamentarium at that time, including one minor infiltration by the surgeon. It was obvious that factors other than anesthetic drugs or techniques were etiological considerations. A strict ruling by the chief of surgery that patients must enter the hospital at least twenty-four hours previous to operation, that elective operations should not be performed upon patients with fever, acute upper respiratory disease, or abnormal chest findings, was a step in advance. Discontinuance of the custom of returning seriously ill patients from the operating rooms to large crowded wards, filled mainly with convalescent patients and visitors, was another valuable change.

MAJOR POSTOPERATIVE RESPIRATORY COMPLICATIONS

	1933	1934	1935	1936	1937	1938	1939	Total	Incidence
Bron. and hypo. pneumon.	33	18	44	35	27	21	42	223	0.752%
Lobar pneumonia	1	2	1	3	3	1	2	13	0.044%
Tuberculous pneumon.	1	1	0	1	1	1	4	9	0.030%
Partial atelectasis	15	7	17	20	27	24	33	143	0.482%
Massive collapse	7	6	5	8	10	2	4	42	0.142%
Lung abscess	0	0	0	0	0	1	0	1	0.003%
Empyema	0	0	0	0	0	1	1	2	0.007%
Others	0	0	0	0	0	0	4	4	0.013%
Total complications	60	34	67	67	68	51	90	437	
Total operations	3764	4064	4248	4167	4160	4461	4784	29648	
Incidence by years	1.59%	0.84%	1.58%	1.61%	1.65%	1.15%	1.81%	1.473%	1.473%

Note: Chest surgery excluded. Table shows actual number of cases.

A more efficient system of record keeping has developed since 1927. It has permitted analysis of our results in such a manner as to afford data on which to base changes in the manner and dosage of drug administration and anesthetic technique as well as data upon which to base changes in the nursing and professional supervision of patients depressed by pain relieving drugs and by injury, illness or surgical trauma. The incidence in recent years of major pulmonary morbidity and mortality is shown in the accompanying tables. It will be seen that the mortality among patients who acquire such major complications as atelectasis, massive collapse or bronchopneumonia after operations is one in four. Obviously, improvement lies along the line of prevention of major complications, not in treatment of bronchopneumonia after it is established. Perhaps it would be profitable to review the attitudes and procedures which, we believe, have tended to reduce the incidence of pulmonary complications and possibly have aided in the treatment of them. Such a review may offer food for thought to other groups, while at the same time aid us to see how we may further decrease our mistakes. An attempt to reduce to a common denominator the etiologic

factors involved in the production of pulmonary complications and thus indicate the direction which prophylactic measures might take, has led to the conclusion that, with rare exceptions, such morbidity *follows a period of interference with the normal functions of the respiratory mechanism whether due to drug action, trauma or illness.*

POSTOPERATIVE RESPIRATORY MORTALITY

	1933	1934	1935	1936	1937	1938	1939	Total
Deaths.....	27	15	20	8	7	11	26	114
Operations....	3764	4064	4248	4167	4160	4461	4784	29648
Incidence.....	0.72%	0.37%	0.47%	0.19%	0.17%	0.25%	0.54%	0.38%

Note: Chest surgery excluded. Table shows actual number of cases.

Many of the signs and symptoms seen in patients depressed by illness or trauma are also observed as side effects of drugs administered to relieve pain. Likewise the treatment of pain is most frequently necessary in this group of patients; therefore a summation of drug effects and the accompaniments of illness and trauma is not infrequently encountered. The clinical anesthetist may say, "What you are driving at is simply the necessity of avoiding oxygen want and carbon dioxide excess. Why all the palaver? Prevent respiratory obstruction and avoid or treat respiratory depression, and no harm can come to your patients." Right! But an understanding of the many ways in which obstruction and depression can be brought about may be an aid toward recognizing the best methods of avoiding and treating them. Illness, injury and pain therapy may interfere with normal function of the breathing mechanism through interference with normal (1) innervation, (2) psychic activity, (3) muscle tone, (4) activity of the respiratory center, and (5) inhaled atmospheres.

(1) The *Nerve Supply* responsible for breathing consists of (*a*) the *phrenic and intercostal nerves.* Partial or complete paralysis from drug action, illness or injury demands specific management. The acute circumstance of temporary intercostal paralysis from ether in the operating room is frequently corrected by the anesthetist through decreasing the depth of anesthesia or amplification of inspiration by manual pressure on the breathing bag. Paralysis of innervation over long periods deserves attention.

A woman with a cervical injury to the spinal cord, paralyzing the musculature below the 7th cervical segment, recovered from a major pulmonary complication, by frequent negative pressure clearance of secretions from the tracheobronchial tree through a tracheotomy opening made solely for that purpose, in the absence of effectual cough.

A boy, completely paralyzed below the second cervical segment with poliomyelitis, has been kept in an artificial respirator for 2 years. Careful nursing management to avoid contamination of the larynx and trachea has prevented pulmonary disease.

(*b*) The *reflex mechanism* governing normal breathing, coughing, swallowing and vomiting is most intricate. It involves cyclical, perfectly timed activation and inhibition of each component reflex with nicely coordinated, alternating muscular activity and relaxation. Illness (e.g., intestinal obstruction) and injury (e.g., following skull fracture and brain surgery) cause an imbalance of this mechanism in such a manner as to obstruct the airway or to permit contamination of the air spaces with foreign material. We anesthetists are all too familiar with hyperactivity of the laryngeal reflex. Do we always bear in mind the hyperactive oculo-cardiac, tracheo-cardiac and carotid sinus reflexes, or the possibility of a hypoactive laryngeal reflex in conjunction with a hyperactive vomiting reflex or the opening of an abscess or a blood vessel in the upper respiratory passages?

A woman whose oculo-cardiac and carotid sinus reflexes were normal during ward examination showed such marked hyperactivity during second plane ether or cyclopropane anesthesia that pressure over the eyeballs or the carotid sinus resulted in a complete cardiac standstill. Illustrating the reverse effect, a man, on whom asystole could be produced by eyeball pressure during ward examination, had a completely normal oculo-cardiac reflex while he was anesthetized with ether for a hernia repair.

A woman anesthetized for cholecystectomy with spinal block and previous sedative medication regurgitated gastric contents and inhaled a small amount into the trachea. The occurrence was followed by a brief period of respiratory and, what appeared to be, cardiac arrest. Prompt inflation of the lungs a few times with oxygen restored respiratory and circulatory conditions to their previous state. The quantity of aspirated fluid was not sufficient to cause noisy breathing or other evidence of tracheal contamination and must have exerted its effect through irritation of the tracheal mucosa. The experimental injection of a small quantity of dilute hydrochloric acid into a dog's trachea caused a marked drop in blood pressure and disturbed respiratory rhythm.

During the drainage of a lung abscess, it was accidentally ruptured into a bronchus and the air passages flooded with pus. Instantaneous utilization of gravity drainage (head low), prompt intubation and cleansing of the tracheobronchial tree with suction through the tube, rapid restoration of cough reflex, meanwhile rolling the patient from side to side, followed by intelligent nursing management prevented an exacerbation of the pulmonary disease.

Similar or less drastic measures are indicated when opiates, barbiturates, tribromethanol, or anesthetic gases and vapors produce depression of the cough reflex and ciliary activity. Excessive secretions, thick and tenacious from various drug effects, are difficult for the patient to remove, even when action of cilia and cough are normal. Not infrequently aspiration of mucus from the tracheobronchial tree under local anesthesia, in cases subject to continued pain or sedative therapy, is life saving. Such a procedure should be instituted promptly, and not reserved solely as a treatment for atelectasis or pneumonia.

A woman suffered multiple fractures and six weeks' hospitalization, with need for prolonged administration of narcotics and hypnotics. She entered the psy-

chiatric service uncooperative and "crazy." To keep her in bed the night of her arrival she was made to drink four drams of paraldehyde. Next morning she was in extremis with noisy, ineffectual breathing, resulting in acute oxygen want. A thorough tracheobronchial toilet with interspersed oxygen inflation of the lungs, followed by a "stir-up regime" every hour and oxygen therapy, permitted recovery. A cumulative effect of sedative drugs had disturbed the reflex protection of the larynx as well as the psychic poise of the patient. Some of the paraldehyde solution was aspirated. In a busy ward, this accident was not noticed until hours later.

A man of 36 years came to the hospital for appendectomy six weeks after recovery from pneumonia at the time of the rupture of his appendix. Appendectomy was performed under open drop ether. For the following 30 hours, pain alone (treated with morphine) disturbed his normal recovery.

36 hrs. postop. T. 100.2°, P. 88, R. 24.
41 " " T. 101. °, P. 120, R. 24.
44 " " T. 101.8°, P. 132, R. 26. Pneumonia, right side, diagnosed.
48 " " T. 103. °, P. 148, R. 40. Senior house staff called.
50 " " X-ray diagnosis of massive collapse.
52½ " " Bronchoscopic aspiration of tracheobronchial tree.
53 " " T. 101.6°, P. 96, R. 24, following which hourly change of position, deep breathing and forced cough were instituted.

Temperature, pulse and respiration normal the next day. Satisfactory recovery. How much better to have treated this man immediately following operation with the "stir-up regime". Certainly the temperature, pulse and respiration record at 36 hours demanded aid in clearing the air passages of excessive secretions which were dried from the effect of morphine and difficult to expel because of depressed ciliary and cough action.

(2) *Deviation From Normal Psychic Activity* not infrequently leads to gross obstruction of the respiratory passages and their contamination with nasal secretions, vomitus, etc. Patients psychically stimulated have been seen to refuse to turn the head or to expectorate when vomitus was in the pharynx. Psychic depression may also lead to disaster.

A patient with a papilloma of the larynx, somnolent from the hypodermic administration of an eighth of a grain of morphine, turned to the left lateral position (a position recognized by her and by her family as impossible for her during normal sleep). The attendant did not enter the patient's room for a period of a half hour at which time she was found dead from complete respiratory obstruction.

(3) *Muscle Tone:* Respiratory obstruction secondary to the excessive muscle tone produced by acute extreme oxygen want or carbon dioxide excess is familiar to every anesthetist and is properly treated. The obstruction resulting from a relaxed tongue partially covering the glottic opening when the patient is in the dorsal position varies in severity from unpleasant snoring and mild oxygen lack to complete obstruction and death.

A woman of sixty was anesthetized with ether and intubated in preparation for a gasserian ganglion operation. She recovered laryngeal reflex activity at the end of operation and coughed. The laryngeal tube was removed and an orderly was permitted to transport her, without a pharyngeal airway, to her room. Muscle relaxation was still present when she was placed in bed in the dorsal position without care as to the relative position of the head to the trunk or the mandible to the cranium. A few minutes later, she was pronounced dead by an ill-trained house surgeon and her family physician. Insertion of a pharyngeal airway and mouth to mouth inflation of her lungs until oxygen and a mask and bag could be secured, restored her and she walked out of the hospital in four days.

(4) The *Respiratory Center:* We all recognize that opiates, barbiturates and other sedatives and narcotics including gases and vapors decrease the sensitivity of the respiratory center to a normal stimulus and hence reduce pulmonary ventilation. Two unphysiologic practices have become common in the management of patients in respiratory depression. One is the administration of high tension oxygen (a very desirable therapeutic maneuver) with the neglect of the carbon dioxide factor. Because oxygen restores the appearance of the patient to normal, the remainder of the picture, high carbon dioxide, is forgotten. If depression of respiration is extreme, artificial aid to pulmonary ventilation is often indicated although a pink color can be maintained with oxygen therapy.

A man, having undergone thyroidectomy under inhalation anesthesia, became very restless after operation. To keep him in bed, a fortieth of a grain of apomorphine was ordered and four-tenths of a grain was given. Artificial respiration, manual at first followed by the use of a mechanical respirator for a few hours until the drug was detoxified, plus oxygen therapy, left him little the worse for the experience.

The other reprehensible practice is to administer oxygen-carbon dioxide mixtures over long periods of time to patients whose respiratory centers are depressed by drug action or injury. Such centers may be insensitive to or depressed by the carbon dioxide tension chosen. Carbon dioxide administration serves no useful purpose other than the production of hyperpnea. Where respiratory exchange is depressed other than temporarily, there exists a marked accumulation of carbon dioxide in the blood and tissues. Addition of carbon dioxide to the inspired atmosphere may further depress the breathing as well as further tax the patient's reserves of available base which can combine with the excess carbon dioxide.

A baby born with opiates and barbiturates, received from his mother during delivery, was given a mixture of 5 per cent. carbon dioxide and 95 per cent. oxygen, blown into his semi-closed bassinet, over a period of hours. He had a gasping, slow, jerky, and ineffectual type of breathing and was manifestly about to die. The mixture was discontinued and the bassinet flushed out with pure oxygen and kept well ventilated by that means. In a half hour, this baby's condition was restored to normal.

Depressed respiratory exchange caused by too liberal premedication is often enhanced by the effect of high concentrations of ether or cyclopropane. To combat such depression, carbon dioxide is often added to the respired atmosphere, either from a cylinder or by means of rebreathing. This technic increases the depth of breathing but is not physiologically sound. It is more logical to augment each inspiratory effort with manual pressure on the breathing bag as long as the depressive concentration of the agent must be maintained.

(5) *Modifications of Inhaled Atmospheres:* The effects of irritants are obvious and need not be mentioned here. Atmospheres containing subnormal tensions of oxygen may, if slowly produced, increase respiratory rate and possibly depth. However, when the reduction of oxygen tension is marked, depression of the respiratory center takes place. No stimulant drug, even carbon dioxide, will have the slightest effect on a respiratory center depressed by severe oxygen lack, until oxygen is restored.

A high concentration of carbon dioxide (30 per cent.) in oxygen was administered to a dog. Having produced a condition simulating anesthesia, the carbon dioxide concentration was increased and the oxygen tension correspondingly decreased through a period of convulsions followed later by respiratory depression and eventually respiratory arrest. Sodium cyanide was administered intravenously in adequate dosage but no respiratory activity took place until the lungs were ventilated with pure oxygen, when respiration was immediately resumed. The respiratory center remains inactive, regardless of the stimulus, until oxygen is restored. Excess carbon dioxide is a respiratory depressant.

It has been shown that gases differ in the rate at which they are absorbed from lobes or lobules fed by an obstructed bronchus or bronchiole. It may also be observed that the resulting atelectasis becomes complete (liver-like) more rapidly when oxygen or nitrous oxide is obstructed in a closed portion of lung than when nitrogen is similarly treated. For this reason, it is not desirable to leave the alveolar spaces filled with a readily absorbable gas at the end of anesthesia if depressed exchange or obstructed air passages are anticipated.

A woman was anesthetized with nitrous oxide-oxygen-ether atmosphere, intubated and the tube passed into the right bronchus, blocking the left bronchus. In one-half hour, an x-ray film showed the left lung to be completely atelectatic. The tube was withdrawn two inches, plane of anesthesia decreased, the bag distended and cough stimulated. The massive collapse had disappeared in a period of five minutes. Had the relief of obstruction been delayed a few hours, the cure would not have been so dramatic. "Bronchopneumonia" might have been the diagnosis. Bacteria grow readily in inactive alveoli.

The normal transport of oxygen and carbon dioxide in blood and tissues is dependent upon a certain biochemical balance which must undergo a shift to accommodate excess tensions of oxygen and carbon dioxide. Such shifts must be reversed to normal when room atmosphere is restored. If there is no special indication to the contrary,

anesthetic atmospheres should contain tensions of oxygen and nitrogen as nearly normal as is consistent with adequate oxygenation. Patients in biochemical imbalance tolerate badly sudden changes in oxygen and carbon dioxide tensions.

A woman of 45 years was given ten grains of barbital at bedtime and a quarter of a grain of morphine and one-hundredth of a grain of scopolamine at 8 the next morning. She was anesthetized from 9:30 until 11:30 with an ether-oxygen atmosphere by the carbon dioxide absorption technic for a complete perineal repair. A large face mask permitted 300 cc. of dead space (rebreathing) between the face and soda lime. Respirations were slow and shallow. Obviously under such conditions, the blood and tissues contained a tension of both oxygen and carbon dioxide much higher than normal. Blood pressure and pulse rate were normal throughout the operation. When the operation was finished, the mask was removed and the patient sent to the ward in good condition. One-half hour later, the ward attendant reported that the patient was in "shock." The systolic blood pressure was 60 mm. mercury, the diastolic 35, and the pulse rate 80. The skin was pale, but the patient was conscious without the anxious look of a shocked patient. Without therapy directed at the circulatory system, a perfectly normal circulatory condition had been restored 3 hours later. We interpret such a circumstance as a reaction to the *sudden* change in gas tensions of the respired atmosphere of a depressed patient. An atmosphere consisting of air with an adequate addition of oxygen only, would have been a better vehicle for the ether vapor. Less premedication would have permitted more adequate ventilation. A smaller mask would have reduced the amount of rebreathing. By these means, there would have been a more normal oxygen and carbon dioxide tension in the tissues during anesthesia, requiring less biochemical adjustment. Perhaps in any case a gradual dilution, by ventilation of the breathing bag and mask with room air for the terminal minutes of anesthesia, would have prevented the "pseudo-shock" with slow pulse seen in this patient.

DISCUSSION

Try as we may, we have so far failed to prevent the occasional occurrence before, during and following anesthesia, of a period of interference with the normal functions of the respiratory mechanism. We still encounter patients after operation, after sedative and narcotic drug administration, after head injuries or even during debilitating illness who have atelectasis, massive collapse, bronchopneumonia and other major respiratory disease. What are we doing about it? Delay in the care of abnormally functioning respiration is fatal. Changes in pulse rate, respiration, blood pressures and body temperature afford earlier evidence of the onset of trouble than do physical signs such as cyanosis and auscultatory or percussion evidence. Such changes, however, can be masked by oxygen therapy. Careful inspection of the respiratory movements viewed from the foot of the bed, laterally and from the head may make evident the asymmetery which is the cardinal sign of atelectasis.

Similar plans of management have been found useful for prophylaxis and therapy. All patients markedly depressed from any cause

should be treated by what has been called the "stir-up regime." This means the hourly insistence upon meticulous attention to three details. The attendant must conscientiously attempt to make the patient (1) take several deep breaths; (2) cough effectively, and (3) change his position radically. Active aid and encouragement by the attendant is required. It is surprising how frequently a depressed patient with inactive breathing and inefficient or absent cough, can be made to expel large quantities of secretion from the air passages. The unconscious or completely uncooperative patient may be made to take several deep breaths by pouring pure carbon dioxide, as one would pour water, from a rubber tube held over the face. Such a flow of gas added to the inspired air may force the desired ventilation of inactive alveoli. For this specific purpose, rarely otherwise, do we find carbon dioxide of therapeutic value.

Where there is the slightest evidence of obstruction or of atelectasis, we have come to feel that the tracheobronchial toilet, described elsewhere, is justified. We feel confident that this apparently drastic regime, when instituted early and faithfully carried out, has reduced the incidence of major respiratory complications in our hospital and has reduced the severity of many of those which we still encounter. Focusing the attention of the house staff upon the extraneous pharmacologic effects of pain-relieving drugs has aided in our efforts to avoid the injudicious use of sedatives and to promote individualization of dosage of such agents.

CONCLUSIONS

It is our opinion that a period of interference with the normal functions of the respiratory mechanism precedes atelectasis and bronchopneumonia, and that it is one of the anesthetist's functions to try to prevent or at least minimize such periods of interference.

Careful selection of drugs and combinations of drugs for pain relief, and individualization of their dosage, further this aim.

Prompt and efficient application of means for the correction of the abnormally functioning respiratory mechanism is essential.

1. Obstructed airways must promptly be made patent.
2. Oxygen therapy must be utilized early.
3. Markedly depressed breathing must be made adequate either manually or mechanically. (Carbon dioxide therapy is not a physiologic remedy for depressed breathing. Oxygen therapy is logical for one-half the picture only, and may require supplemental augmentation of respiratory exchange to restore normal conditions.)
4. The "stir-up regime" and "tracheobronchial toilet" are valuable both prophylactically and therapeutically for many depressed patients.