

COMPLICATIONS OF THE MANAGEMENT OF ACUTE RESPIRATORY FAILURE AND THEIR PREVENTION

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SUMMARY

We present an analysis of the complications of the management of Acute Respiratory Failure (ARF) in 1955 patients over a ten year period at Queens Hospital Center in New York City. Stress ulcer gastric bleeding, renal failure, nosocomial infections, pulmonary embolism, cardiac arrhythmias, atelectasis, barotrauma, laryngeal and tracheal complications are reviewed. Based on our experience recommendations regarding the avoidance of these complications are presented.

KEY WORDS:

Respiratory Intensive Care Unit — RICU
 Acute Respiratory Failure — ARF
 Flexible Fiberoptic Bronchoscopy — FFB
 Endotracheal Tube — ETT
 Positive End Expiratory Pressure — PEEP

INTRODUCTION

The Respiratory Intensive Care Unit (RICU) at Queens Hospital Center was established in May, 1968. Table 1 lists the total number of patients admitted and their mortality from May, 1968 to July, 1979. During the initial three years the average yearly mortality was 32%. As our experience enlarged, the mortality decreased to around 20%. However, some complications seem to occur with regularity. In January, 1974, a prospective study of the various complications of respiratory failure was started. This paper describes our experience with the various complications of respiratory failure, along with the pertinent literature review. The studies on gastric bleeding, renal failure, coagulation disorders, atelectasis were done at different times between 1974-

TABLE 1
RICU AT QUEENS HOSPITAL CENTER
May, 1968 to July, 1979

Total Admissions 1955
 Total Intubations 1370

Diagnosis	No. Admissions/Percent		Mortality %
C.O.P.D.	387	(19.6)	19
Post Operative	356	(18.2)	15
Pneumonia (complicated)	189	(9.6)	44
Bronchial Asthma	182	(9.3)	3
Multiple Trauma	98	(5.0)	17
Tuberculosis (complicated)	96	(5.0)	15
Drug Overdose	53	(2.7)	7
Respiratory Burn	26	(1.4)	38
*Miscellaneous (1.5% or less)	568	(29.06)	23

20%

*Sarcoidosis	Botulism	Upper Airway Obstruction
Myxedema	Old Polio	Myasthenia Gravis
Cong. Heart Failure	Pulmonary Fibrosis	CO Poisoning
Pulmonary Emboli	Kyphoscoliosis	Hodgkin's Disease
Rheumatoid Lung	Cardiomyopathy	Tetanus
Renal Tubular Acidosis	Steven Johnson Syndrome	Primary Pulmonary Vascular Disease
Guillain Barre Syndrome	Systemic Lupus Erythematosus	

1979, therefore the total number of patients in each study is different. The patho-physiologic changes and treatment of respiratory failure have been reviewed elsewhere^{1,2}.

GASTRIC BLEEDING

Massive gastric bleeding due to stress ulceration has been reported from RICU³. The presence of gastric acid with breakdown of the gastric mucosal barrier is necessary for the development of such stress ulcers.

From January, 74 to December, 75 we observed massive upper gastric bleeding necessitating more than 2 units of blood transfusion due to multiple gastric stress ulcers, in 40 of the 420 consecutive admissions (9.5%) to the RICU. Although the overall mortality was 18%, 25 of the 40 patients with gastric bleeding died (62.5%)⁴.

In July, 1976 we began an antacid neutralization protocol in which the gastric contents are aspirated and the first 10 to 15 cc discarded, as this material usually represents the aspirate from the nasogastric tube and may give a falsely high pH level from the antacid in the nasogastric tube. The pH of the gastric aspirate is measured, then antacid (between 10-120 cc) is added to increase pH above 5 units. The procedure is repeated every hour, 24 hours a day, until patient is extubated and resumes a regular diet. Since January, 1978 we have been using cimetidine, an H₂ Blocker, 30 mgm IV every 6 hours in patients with odd numbered birthdates while patients with even numbered birthdates continue to receive hourly antacids only.

Results of Gastric Neutralization

In the antacid group only three of the one hundred and seventy-five patients had massive gastric bleeding. Thus, the incidence of bleeding was reduced from 9.5% to 1.7%. These results are similar to the results of other investigators³.

In the 75 patients in the Cimetidine group, three bled massively. All three had large solitary bleeding duodenal ulcers, two requiring surgical therapy. In addition, seventeen patients were admitted with respiratory failure and massive gastric bleeding. All stopped bleeding within 24 to 48 hours after starting gastric neutralization with antacids in 24 patients and Cimetidine therapy in 3 patients.

We conclude that gastric neutralization is effective in both preventing and treating massive gastric bleeding in a RICU.

RENAL FAILURE

The occurrence of renal failure during respiratory failure is associated with a mortality of up to 80%⁵. In our study of 686 consecutive patients from January, 74 to August, 77 we identified 74 patients with renal failure. Renal failure was defined as a serum creatinine of 2 mgm/dl or more with a BUN to creatinine ratio of

less than 20 to 1, or a serum creatinine over 4 mg/dl regardless of the BUN level. The major etiological factors of renal failure were: 1) Massive gastric bleeding leading to hypovolemia with shock in 18 patients (24%), 2) Septic shock in 22 patients (30%), 3) Aminoglycoside nephrotoxicity in 9 patients (13%), 4) Cardiogenic shock in 8 patients (11%), 5) Hypotension in 8 patients (11%), 6) Pre-existing renal failure in 6 patients (7%), 7) Miscellaneous 3 patients (4%).

Whereas the overall mortality in the RICU was 17-20%, the mortality in this group of patients with renal failure was 80% (60/74). We do not feel the use of PEEP (24 patients), the type of respiratory failure—hypercapnic or predominantly hypoxic was responsible in the evolution of renal failure⁵. Five patients had hemodialysis and eleven had peritoneal dialysis. The mean creatinine level of all 16 patients at the start of the dialysis was 10 mgm/dl. Eight of the sixteen dialyzed patients survived. Of the 58 non-dialyzed patients, 52 died. We feel the early institution of dialysis in these critically-ill patients is helpful in improving the chances for survival.

Our conclusions from this study were: 1) Renal failure is prevalent in the RICU, 2) The main etiological factors precipitating renal failure are gastrointestinal bleeding leading to hypovolemia and shock, sepsis, drug nephrotoxicity and hypotension. These conditions should be anticipated and avoided where possible. Once renal failure occurs, early dialysis should be seriously considered.

PULMONARY EMBOLISM

Pulmonary thromboembolic disease has been reported to be the most common pathologic process in hospitalized patients. However, because of the lack of sensitivity and specificity of clinical manifestations of pulmonary embolism, its precise diagnosis is difficult. The problem is underscored in patients with acute respiratory failure, whereby, similar manifestations may already be present from underlying cardiopulmonary disease or from other varied complications of respiratory failure. One series reported an autopsy incidence of 27% of pulmonary embolism in 66 patients dying in RICU, half of whom were not diagnosed antemortem⁶. In an analysis of 100 consecutive autopsies of patients dying in our RICU, the incidence of pulmonary thromboembolism was 12%. Half the patients affected by the pulmonary embolism died as a result of it. This low incidence is attributed to the emphasis in our RICU placed on the prevention of pulmonary embolism by vigorous leg care and early ambulation.

It is likely that, a constant awareness, continuous clinical surveillance and, aggressive attempts to document pulmonary emboli, together with vigorous leg care, early ambulation and, possibly low dose

heparin therapy in selected cases, may further reduce the morbidity and/or mortality from pulmonary thromboembolism in patients being managed for respiratory failure.

COAGULOPATHIES

Laboratory abnormalities of coagulation studies consisting of minimal fluctuation in prothrombin time and partial thromboplastin time, increased fibrinolytic activity and reduced platelet count are often seen in ARF⁷. In a prospective and retrospective study of coagulation in 26 patients with stress ulcer gastric bleeding and 120 consecutive patients with ARF over a six month period we found: 1) One or two abnormalities of coagulation studies were not uncommon (27%). 2) A pattern of significantly abnormal coagulopathy was uncommon (5%). 3) Generalized bleeding due to coagulopathy was not observed. 4) Stress ulcer hemorrhage did not appear to result from a coagulopathy.

The minimal coagulation changes observed in our patients are similar to those reported⁷ and are attributed to vigorous treatment of underlying disease coupled with judicious cardiopulmonary support.

CARDIAC ARRHYTHMIAS:

Cardiac arrhythmias during ARF are intermittent and frequent, with an electrocardiographic incidence of 32 to 47%^{8,9} and an 89% incidence on continuous monitoring in whom 57% were severe enough to require treatment⁹. Although the supraventricular arrhythmias are more frequent than ventricular arrhythmias, the prognosis of patients with the latter is much worse than the former with mortality up to 70%^{8,9}.

A combination of factors responsible for cardiac arrhythmias during ARF include hypoxemia, associated hypocapnea, metabolic abnormalities that occur during the management of ARF, associated coronary artery disease, and possibly elevated right atrial pressure.

The prevention and treatment of cardiac arrhythmias is dependent upon 1) early recognition by continuous monitoring, 2) the correction of hypoxemia, inappropriate ventilation, metabolic disturbances and treatment of associated underlying cardiac disease.

NOSOCOMIAL INFECTIONS

Nosocomial infections are a major cause of morbidity and/or mortality in patients being treated for ARF. Impaired host defenses, mechanical or immune, contaminated support equipment — especially reservoir nebulizers and, colonization of the respiratory tract are important predisposing factors. Nosocomial infections account for 0.5% to 5% of all hospitalized patients. However, the incidence is greater in critical care units with a range between 12%

to 21%¹⁰. With the widespread use of antibiotics, and the technological advances in medical care, the spectrum of nosocomial infections over the past three decades has changed. Currently, gram-negative bacilli including *Pseudomonas aeruginosa*, *Serratia marcescens*, *Klebsiella* and *Enterobacter* species are mainly responsible for nosocomial infections. Furthermore, fungal superinfections, especially with *Candida* species are also common. Pneumonias due to these organisms are associated with a high mortality, especially *Pseudomonas pneumonia*¹¹, and are often complicated by slow resolution, cavitation and empyema.

The incidence of bacterial or fungal colonization increases during or after antimicrobial therapy. A direct relationship seems to exist between colonization with gram-negative organisms and the development of pneumonia. In one series, colonization occurred in 45% of patients — 22% on the first day. Pneumonia developed in 25% of colonized patients compared to 3.3% of non-colonized patients¹².

Appropriate measures reduce the incidence of colonization and nosocomial pneumonia. A surveillance program with regular cultures of the affluent air flow, decontamination of the respiratory support equipment and sinks, meticulous care of the endotracheal or tracheostomy tube and in-dwelling catheters, reverse isolation of most susceptible hosts and, control of human carriers with frequent hand washings are the preventive measures recommended. Suggestions have been made for nebulization of antimicrobial agents into the trachea and large airways in an attempt to prevent or delay gram-negative colonization. However, the data are inconclusive¹⁰.

ATELECTASIS AND ROLE OF FLEXIBLE FIBEROPTIC BRONCHOSCOPY (F.F.B.)

The left main bronchus (LMB) has a 45° take off angle from the trachea and is smaller than the right main bronchus (RMB). Therefore, LMB is less accessible to suctioning resulting in a pooling of secretions, segmental, lobar or total atelectasis. With flexible fiberoptic bronchoscopy (FFB) this problem has been greatly alleviated¹³. Some complications associated with bedside FFB include: 1) development of a PEEP effect, 2) increased incidence of barotrauma, 3) decreased tidal volume and minute ventilation, 4) increased PCO₂ and decreased PaO₂ particularly during prolonged suctioning, 5) increased cardiac output. In order to minimize these complications we give 100% inspired oxygen for a few minutes before, during and a few minutes after completion of the procedure, readjust and monitor the tidal volume to overcome the small air leak which invariably develops, minimize the duration of

suctioning, check chest x-ray after the procedure, and use a size 8.0 endotracheal tube or larger.

In a series of 448 consecutive admissions, we found 33 (7.3%) with persistent unresponsive segmental and/or lobar atelectasis. After bedside FFB 26 (79%) showed immediate significant radiologic and physiological improvement. In the patients who do not improve with FFB we treat with sequential segmental and/or lobar lavage using up to 300 cc of normal saline as lavage fluid.

LARYNGEAL AND TRACHEAL COMPLICATIONS

The problems resulting from the use of an endotracheal tube (ETT) can be immediate or delayed. Immediate problems include malpositioning of the ETT, trauma to the tongue, epiglottis, larynx, trachea with secondary infection at the site of the trauma¹⁴.

LARYNGEAL COMPLICATIONS

Laryngeal complications consist of arytenoid damage, vocal cord immobility, subglottic granulation leading to stenosis and upper airway obstruction. In a study of 65 orally intubated patients we found three female patients with bilateral vocal cord immobility and subglottic stenosis. Two required permanent tracheostomy and one had multiple surgical procedures over a two-year period including attempted dilatation of subglottic stenosis, placement of a stent and eventual excision of part of the larynx and anastomosis of the trachea to the larynx with an excellent functional recovery.

From our study we concluded that laryngeal damage can be minimized if (a) proper size ETT is selected, particularly in women as they have a smaller larynx than men, (b) the ETT is firmly anchored and movement of patient's head minimized, (c) immediate post extubation hoarseness is the rule but improves with time. We leave oral ETT for up to three weeks.

TRACHEAL COMPLICATIONS

The tracheal complications predominantly occur at the site of the cuff and at the stoma of the tracheostomy. The tracheal stomal lesions are unrelated to previous use of artificial ventilation and fibrosis is characteristically localized anteriorly, stomal lesions may be caused by too large size stoma and tube, excessive tube movement, and local infection. Stomal trauma can be minimized by using a swivel system of connectors at the tracheostomy.

Tracheal cuff complications are caused by the high intracuff pressures which impair capillary circulation, cause ischemic necrosis, exposure of tracheal cartilage leading to tracheoesophageal fistula (TEF), tracheomalacia or tracheal stenosis. Amongst 1370 intubated patients, we have seen 5 patients with tracheal stenoses, 4 patients with TEF and 2 with

tracheomalacia, an overall incidence of less than 1% (11/1370). We also found an excellent correlation between an enlarging tracheal cuff size on chest roentgenogram and progressive tracheal damage. A ratio of cuff size to trachea size (C/T) over 1.5 is a reliable early predictor of serious early tracheal damage¹⁵. Tracheal damage can be minimized by (a) the use of *only* high compliance cuffs, (b) the maintenance of cuff pressure less than 30 mm/Hg, (c) the use of minimal air leak technique, (d) monitoring C/T ratio.

PULMONARY BAROTRAUMA

Pulmonary barotrauma (i.e., pneumothorax, pneumomediastinum, subcutaneous emphysema or pneumoperitoneum) is a recognized complication in patients on assisted ventilation. Large tidal volumes, high peak inspiratory pressures, high levels of PEEP and the diseased state of the underlying lung are important predisposing factors in the development of pulmonary barotrauma. The reported incidence of pulmonary barotrauma has varied from 40%¹⁶ to as low as 0.5%¹⁷. The low incidence has been speculated in part to be due to refinement of ventilatory practices, effective sedation and/or relaxation of patients to facilitate mechanical ventilation and, the use of intermittent mandatory ventilation.

The diagnosis of pulmonary barotrauma is based on clinical suspicion, radiological evaluation and pulmonary function alterations. Alterations in static compliance curves i.e. large increase in pressure for a small gain in volume¹⁷ and sudden unexplained rapid elevation of pulmonary artery pressure are important indications of pulmonary barotrauma. Pneumoperitoneum due to pulmonary barotrauma is difficult to differentiate from pneumoperitoneum caused by a perforated abdominal viscus.

The prevention of pulmonary barotrauma lies in the judicious evaluation of the pulmonary function and hemodynamic alterations in a patient receiving assisted ventilation¹⁷.

MISCELLANEOUS COMPLICATIONS

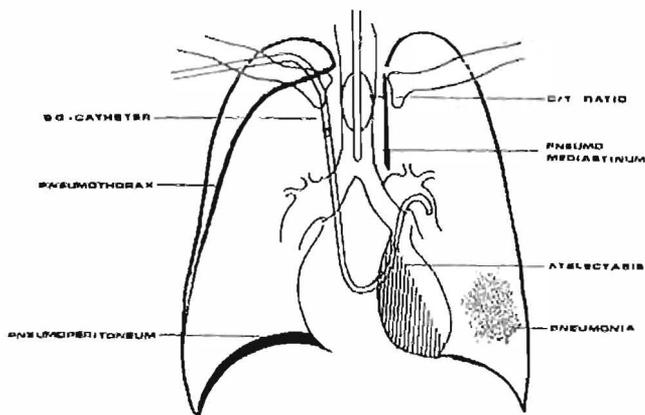
Some of the complications reported in the literature, but not observed with any regularity in our series include (a) gastric distension (b) intensive care delirium (c) pulmonary water retention (d) abnormalities of liver function and jaundice (e) pulmonary arteriovenous fistula (f) complications resulting from malfunctioning of the ventilator, machine failure, alarm failure, inadequate humidification, nebulization¹⁴.

In conclusion during management of ARF, several complications involving multiple organs are frequently encountered. Many of these complications can be avoided by judicious prophylaxis as outlined

above. We find evaluation of serial chest roentgenograms vital for the early detection of barotrauma, impending tracheal damage by evaluating cuff/trachea ratio, recognizing nosocomial infections, atelectasis and for evaluation of the Swanz-Ganz Catheter position. Effective modern

management of ARF requires a multidisciplinary approach involving trained and motivated nurses, therapists and physicians. As the experience of these personnel increases, the mortality from these complications decreases with resulting increased survival.

FIGURE 1



This drawing demonstrates the importance of chest roentgenogram in localizing the position of ETT, S.G. Catheter, calculating C/T ratio, and recognizing the complications of atelectasis, pneumonia and barotrauma.

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