High Output Respiratory Failure: *

An Important Cause of Death Ascribed to Peritonitis or Ileus

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PERITONITIS and ileus lead to pathologic change not only in the peritoneal cavity, but throughout the body. These extraperitoneal effects are more extensive with peritonitis than with ileus. They usually have been considered to be the systemic manifestations of inflammatory disease so comparatively little attention has been paid to the function of specific organ systems and to the mechanisms of their decompensation during this period of intense metabolic activity. It is clear, however, that peritonitis or ileus is not a primary cause of death, since it is possible to establish several more basic pathological abnormalities. It also has been established that inadequate function of one or more specific organs may result in death.

A variable degree of interest has been manifested in these failing systems. The effects of serious abnormalities of the electrolytes, plasma or red blood cell mass, as well as local sepsis, have been studied thoroughly. On the other hand, important *target organs*[32] (as Moore has called them) include the kidneys, the cardiovascular system, the respiratory system and the intestine itself. As a group these viscera have received relatively little emphasis. This is probably because the normal respiratory, cardiovascular or renal systems have such great functional reserve that little thought is given to them in patients with peritonitis or ileus unless there has been serious preexisting disease involving these viscera.

In this paper the discussion will center on the respiratory system and upon a type of failure that occurs with initially normal or essentially normal lungs. We believe that this is a particularly dangerous and often unrecognized syndrome that merits careful attention.

It is clear that failure of any single organ is related intimately to failure of all other viscera. Nevertheless, failure of a single organ does not imply failure of all simultaneously. There is indeed a very interesting temporal relationship which can be outlined and in itself emphasizes the primary importance of decompensation of the respiratory system.

Thus, as shown in Figure 1, the type of respiratory failure discussed in this paper tends to appear in the very early phases of peritonitis. There is indeed a later stage of pulmonary failure that may occur a week or more after onset of peritonitis that is secondary to aspiration, pneumonitis or embolization, but in this paper we are not concerned with this late type. Cardiac failure in the early course of peritonitis is a sequel of hypoxia that is dependent on pulmonary failure and is manifested by cardiac arrest. Late cardiac failure is usually

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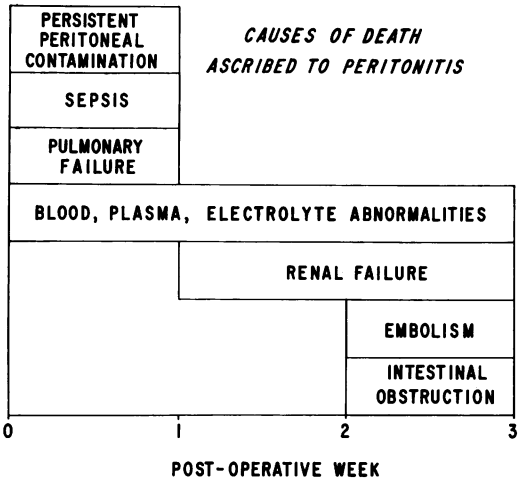

FIG. 1. Causes of death ascribed to peritonitis.

a result of pulmonary embolization. Renal failure, another late manifestation, has been documented fully, particularly by Hagen and Merrill.[21] Failure of the intestine itself is temporally one of the last of the types of organ failure. If it is not relieved, as shown in a previous communication,[39] it essentially leads to death from inanition.

The period of intraperitoneal sepsis most dangerous for the patient is the first week. Unless there is continued peritoneal soilage or undrained sepsis that leads to bacteremia, fatal sepsis originating from the peritoneal cavity is not common. On the other hand, careful attention to electrolytes, plasma and red cell mass is required throughout the entire illness.

It therefore appears that in the first few days after the onset of a severe peritoneal insult the surgeon must give priority to fluid and electrolyte replacement, to the control of sepsis and to the prevention of respiratory failure. On the other hand, successful treatment of one decompensated system may allow the patient to live to become vulnerable to failure of another *target organ* that characteristically is manifested at a later date.

The concept of early pulmonary failure has been considered by surgeons for many years. A basic theory has been outlined and a specific method of therapy has been considered in many centers. However, to test the validity of these concepts, it is highly desirable to study a series of patients with this specific lesion and to document their course.

Material

We have been interested in these patients for the past five years. During that period we have studied 21 patients with acute pulmonary failure due indirectly to peritonitis or ileus. Their course is outlined briefly in Table 1. In common with all developing studies, investigation of various parameters has been more complete with the passage of time and with the development of a special respiratory unit under the direction of one of us (H. P.).

It is obvious that criteria for inclusion in such a study must be sharp or all types of combined pulmonary disease and peritonitis could be included. The criteria used were as follows:

1. The patient had acute peritonitis or ileus secondary to an intraperitoneal lesion.
2. No patient had an acute pulmonary or cardio-vascular disease or injury.
3. Pulmonary failure was the only immediate threat to life.

The exact state of the patient's pulmonary function preceding his illness is of great importance. Since these patients were not considered to have significant respiratory problems prior to operation, no preliminary function studies were carried out on any of them. They could, however, be divided into three groups depending on the presence and extent of previous pulmonary disease, as follows:

Group 1. No previous chest disease (7 patients).

Group 2. No previous symptoms, but slight pulmonary changes on physical or x-ray examination (10 patients).

Group 3. Symptoms and signs of previous emphysema and chronic bronchitis (4 patients).

Clinical Data

The clinical data are summarized in Table 1. The 21 patients included 12 men and nine women. The ages ranged from 31 to 76 years.

Causes of Peritoneal Insult. Peritonitis and concomitant ileus existed prior to operation in 10 patients. Four of them had bacterial peritonitis (perforated duodenal ulcer—1; perforated diverticulitis—2; perforated herniated colon—1); one had pancreatitis and five had ruptured aortic aneurysms. Eleven developed postoperative peritonitis (following gastric resection for massive hemorrhage—4; following total cystectomy—2; following jejunal perforation—2; following aortic aneurysm resection—2; following nephrolithotomy—1).

The low incidence of perforated duodenal ulcers in this series is worthy of comment. There were indeed many patients seen in the hospital following perforation of a peptic ulcer with severe restriction of pulmonary function. However, with laparotomy and removal of large quantities of peritoneal exudate, particularly from the subdiaphragmatic space, there was dramatic improvement in pulmonary function almost at once. The only patient who failed to respond to this direct form of therapy was one with previous emphysema (S. H., Table 1) who later required tracheostomy and mechanical ventilation. It is of interest in this respect to quote Anscome: [3]

"Patients with a generalized peritonitis following perforation of a peptic ulcer showed a greater reduction in pulmonary mechanical power than any other group. The effect was so severe that the vital capacities were all less than one liter and in one patient it was 420 cc. and vital spirograms could therefore not be done. Measurements on the first day after operation showed an increase over the preoperative figure, a finding which did not occur in any other type of patient or condition. Despite this severe initial reduction and in the presence of an epigastric midline incision, there was a surprisingly rapid recovery, the maximal figures being reached in six days."

Our experience, as well as that of Anscombe, indicates the serious impairment of pulmonary function by peritoneal inflammatory disease. If, however, the inflammatory stimulus can be removed, as in the case with perforated peptic ulcer, and distention relieved before fatigue, pulmonary function returns to normal almost immediately following the relieving operative procedure.

It is also of interest that three of the four patients with peritonitis following gastric resection for massive hemorrhage were found in Group 3 patients. The association of severe ulcer disease with emphysema has been noted frequently in the past.[18–20, 28, 30, 36] Parenthetically it may be noted that massive gastro-intestinal hemorrhage has been a frequent major complication in patients under treatment in our respiratory unit in the past one and one-half years.

Examination of Patient

Pre-existing chronic bronchitis and emphysema, obesity, malnutrition or feeble muscles of respiration were important premonitory warning signals. A history of heavy cigarette smoking [9, 33] is also a danger sign.

During the development of respiratory failure following a severe peritoneal insult all patients were severely ill. They were usually extremely anxious, apprehensive and were at times thrashing. Respirations were rapid, shallow, labored and oftentimes grunting. They appeared exhausted and their faces were often flushed. All were severely dyspneic.

X-rays of the chest taken at this time showed elevated diaphragms and varying degrees of basal atelectasis. The absence of any striking changes in the lung fields may lead the attending surgeon into a false sense of security concerning the stability of respiratory compensation.

Vital Signs. Pulse and respiratory rates showed marked increases that were sustained until respiratory failure was cor-

TABLE 1

Group	Pt. Age Sex	Diagnosis	Pre-existing Pulmonary Disease	Before Specific Treatment							After Specific Treatment							Hospital Course
				Pulse	Resp	BP	O_2Sat	$PaCO_2$ mm. Hg	CO_2 mEq/L	pH	Pulse	Resp	BP	O_2Sat	$PaCO_2$ mm. Hg	CO_2 mEq/L	pH	
I	M.B. 49 F	perf. sig. diverticulitis	none	120	30	110/50	92%	78	20.3	6.98	90	20	100/50	100%	46	19	7.22	recovered
I	M.C. 74 F	mult. renal stones & perirenal sepsis	none	136	45	120/50	90%	31	25	7.52	100	16	120/80	95%	31	31	7.6	died: renal failure
I	E.D. 50 F	gastrojejuno colic fistula	none	155	40	120/80	cyan-otic	comatose			110	20	110/70	alert	37	30	7.43	recovered
I	J.D. 48 F	perf. incarc. umbilical hernia	none	120	45	130/80	cyan-otic		25		90	25	120/80			26		recovered
I	P.G. 76 M	ruptured aortic aneurysm	none	130	50	120/50	cyan-otic		27		100	24	110/70		37	27	7.42	recovered
I	M.P. 32 F	acute pancreatitis	none	145	50	140/90	cyan-otic		23		100	23	120/80			25		recovered
I	A.W. 70 M	total cystectomy	none	120	40	130/80	89%	28	25	7.56	120	18	130/80	98%	45			died: renal failure
II	S.A. 60 F	perf. sig. diverticulitis	rec. pulm. infection	140	44	105/70	cyan-otic		29		110	20	110/70	96%	35	28	7.51	recovered
II	L.B. 66 F	ruptured abd. aortic aneurysm	"mild" emphysema	130	40	120/80	82%	30	26	7.54	80	25	120/80	100%	37	24	7.43	recovered
II	R.D. 75 M	ruptured aortic aneurysm	"mild" emphysema	120	30	140/70	81%	62	34	7.39	100	20	130/70	97%	40	35	7.51	recovered

(O_2Sat) % arterial oxygen saturation, ($PaCO_2$) arterial CO_2 tension, (CO_2) plasma CO_2 content.

TABLE 1. (*Continued*)

Group	Pt. Age Sex	Diagnosis	Pre-existing Pulmonary Disease	Before Specific Treatment							After Specific Treatment							Hospital Course
				Pulse	Resp	BP	O_2Sat	$PaCO_2$ mm. Hg	CO_2 mEq/L	pH	Pulse	Resp	BP	O_2Sat	$PaCO_2$ mm. Hg	CO_2 mEq/L	pH	
II	J.D. 41 M	massive G.I. bleeding; Z.E. syndrome	"mild" emphysema	160	40	140/60	82%	67	31	7.26	110	25	120/80	97%	32	30	7.57	recovered
II	A.L. 65 M	ruptured aortic aneurysm	mild chronic bronchitis	140	35	110/70	93%	54	30.4	7.34	80	20	140/80	97%	51	33	7.41	died: renal failure
II	A.L. 67 M	total cystectomy	coronary heart disease	120	30	120/70	93%	53	25	7.22	100	20	130/70	97%	40	29	7.46	died: pulmonary embolus
II	J.L. 66 M	aortic and iliac aneurysms	"modest" emphysema	130	30	130/80	87%	40	27	7.43	100	20	150/90		44	34	7.44	recovered
II	J.M. 62 M	ruptured aortic aneurysm	"mild" emphysema	150	45	140/70	cyan-otic	60	cardiac arrest		80	25	120/70	97%	46	39	7.53	recovered
II	H.N. 53 F	jejunal fistula	"mild" emphysema	130	34	110/80	prev. cardiac arrest	48	32	7.4	90	22	140/80		46	30	7.4	recovered
II	H.T. 59 M	large abd. aortic aneurysm	"minimal" emphysema	120	35	170/100	cyan-otic	35			100	25	150/100	98%	44	29	7.41	recovered
III	S.H. 74 M	perforated ulcer	severe pulmonary emphysema	130	40	150/90	56%	52	45	7.53	90	15	110/60		49	35	7.47	recovered
III	J.S. 68 M	massive upper G.I. bleeding	chronic emphysema & bronchitis	110	25	150/70	91%	38	29	7.48	80	18	140/80	96%	26	24	7.6	recovered
III	E.T. 73 F	massive upper G.I. bleeding	chronic bronchitis	128	38	110/80	82%	39	28	7.44	105	26	110/80	96%	47	38	7.53	recovered
III	E.Y. 72 M	massive upper G.I. bleeding	chronic emphysema & bronchitis	100	35	110/60	90%	43	30	7.45	100	30	130/70	98%	40	23	7.38	died: cardiac arrythmia

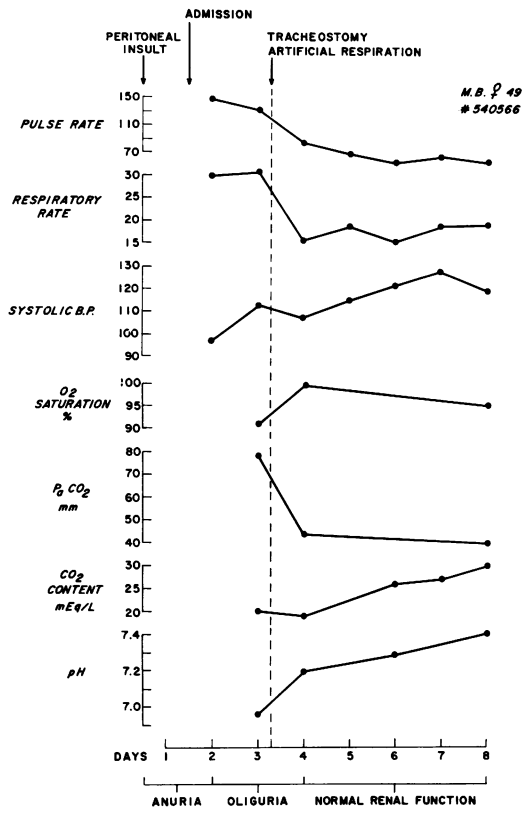

FIG. 2. Case 1. Vital signs and arterial blood studies.

rected. The blood pressure, on the other hand, remained in a normal range before and at the time of early respiratory failure. Hypotensive levels occurred only when the patient became moribund. In some patients a transient rise in blood pressure may be observed during the early stages of respiratory decompensation.

Blood Gas and pH Studies. The pH of arterial blood varied, as expected, with the adequacy of CO_2 elimination. In addition, it was also affected by the presence or absence of a metabolic acidosis caused by such abnormalities as tissue hypoxia,[29] and acidosis of renal origin.[21] The adequacy of CO_2 excretion does not follow the adequacy of arterial oxygenation. Arterial oxygen desaturation almost always precedes insufficient CO_2 elimination. In fact, in the early stages of this syndrome respiratory alkalosis was seen not infrequently as the result of a relative hyperventilation, probably because the patient attempted to oxygenate the arterial blood adequately. In the later stages CO_2 excretion also failed and a respiratory acidosis was the rule.

The arterial blood gases showed a consistent pattern only in oxygen unsaturation. The arterial CO_2 tension ($PaCO_2$) varied according to the adequacy of CO_2 excretion as described above. The plasma CO_2 content changed with other variables such as renal function and arterial CO_2 tension ($PaCO_2$).[5] Nevertheless the information gained by these four determinations was of great value in the minute to minute care of the patient.

Treatment. An initial attempt to establish adequate respiratory function was made by relief of intra-abdominal tension (by gastro-intestinal intubation or by laparotomy with decompression or drainage where indicated). In addition, tracheal suction and chest physiotherapy was used in all cases. Bronchoscopy was done rarely. These methods were ineffective in 20 of the 21 patients in this study, a fact that does not minimize the value of these maneuvers but indicates the severity of the disease in our cases. Effective therapy was instituted with a tracheostomy and insertion of a cuffed silver tracheostomy tube in conjunction with an intermittent positive pressure mechanical respirator.

Complications and Mortality. Five of the 21 patients died. One succumbed to a fatal cardiac arrhythmia during tracheal suction two weeks following tracheostomy. Two cardiac arrests occurred just prior to tracheostomy. One patient lived and the other died of pulmonary embolus three weeks later. The other three patients died of late renal failure two weeks to two months after the date of tracheostomy.

Case Reports

Three cases are included to demonstrate specific points. Patient 1 is typical, illus-

trating this type of respiratory failure. She was salvaged by the treatment outlined. Patient 2 is an example of the effects of fatigue. Respiratory decompensation appeared ten days after operation and two days after onset of peritonitis. Patient 3 is a representative patient in whom the implications of this syndrome were not recognized because of the maintenance of a normal blood pressure until cardiac arrest occurred.

Case 1. M. B. Figure 2 gives a detailed picture of the vital signs, blood gas and pH studies of a 49-year-old woman without pre-existing pulmonary disease who was admitted 18 hours after the onset of peritonitis resulting from perforated sigmoid diverticulitis. The chart illustrates the evolution of the vital signs and blood gases as observed during the first eight days of illness. On admission the patient was suffering from a severe electrolye and plasma disturbance. This was rapidly corrected with an improvement in the blood pressure to a normal level, but the rapid pulse and respirations persisted. X-ray examinations of the chest and abdomen on the day of admission (Fig. 3a, 3b) showed marked elevation of the diaphragm and basilar atelectasis without other pulmonary disease. The abdominal film shows considerable small bowel distention. Blood gases obtained on the day following admission with the patient breathing room air showed a reduced oxygen saturation (92%), a markedly elevated $PaCO_2$ (78 mm. Hg), a pH of 6.95 resulting from a mixed metabolic and respiratory acidosis. The BUN at this time was 111 mg.%. The patient had been anuric on admission and was oliguric at the time the arterial blood sample was drawn. She was anxious, dyspneic and appeared exhausted, with rapid, shallow respirations.

A tracheostomy was performed immediately and a cuffed silver tracheostomy tube inserted into the trachea. The patient was placed on artificial ventilation using a mechanical respirator. The pulse and respirations promptly dropped to normal rates. Arterial blood studied at this time with the patient breathing oxygen showed an oxygen saturation of 100%, $PaCO_2$ in the normal range (46 mm. Hg) with a CO_2 content (19 mEq./L.) and pH (7.2) indicating a metabolic acidosis secondary to renal failure. In the following four days normal renal function resumed and the metabolic acidosis was corrected. On the eighth day of illness, with the patient off the respirator and breathing room air, all studies were within normal limits. (Oxygen saturation 96%, $PaCO_2$—39 mm. Hg, pH—7.43, CO_2—30 mEq./L., BUN—9 mg.%.)

Case 1 serves to demonstrate the rapidity and severity of respiratory failure developing in a patient whose pulmonary function had been normal before the onset of the peritoneal insult. The case also demonstrates the rapid and complete correction of the abnormalities caused by respiratory failure by adequate ventilation as accomplished by the mechanical respirator.[7, 8, 10, 24, 35, 37]

Case 2. A. W. An examination of the case history shown in Figure 4 illustrates, in our opinion,

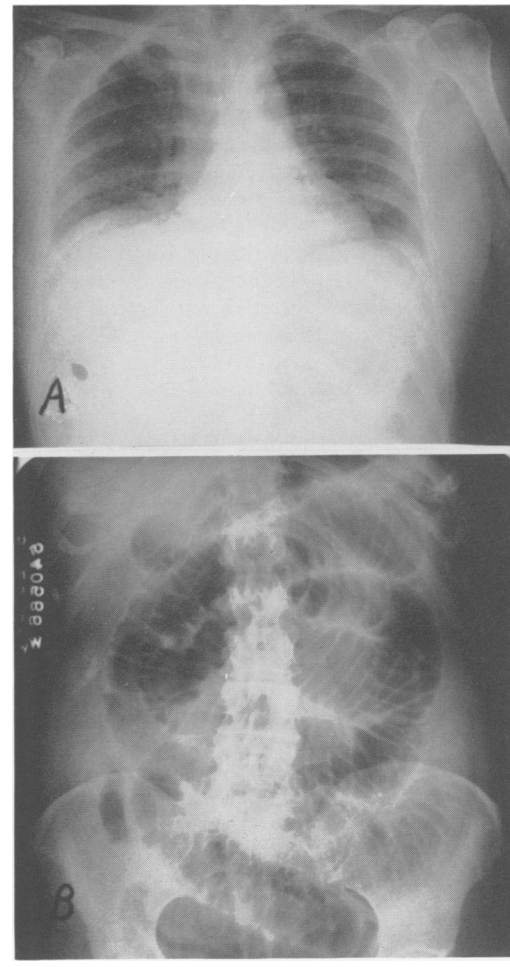

FIG. 3. Case 1. X-ray studies on admission. A. Chest x-ray showing minimal atelectasis. B. Scout abdominal film showing dilated bowel.

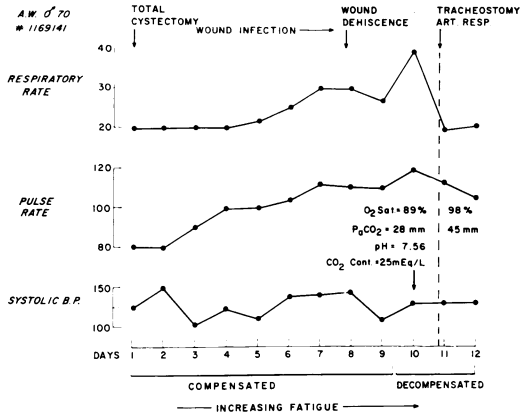

FIG. 4. Case 2.

the fatiguing effect of an extensive operative procedure followed by multiple complications. The patient was a 70-year-old man with no history of previous pulmonary or cardiovascular disease. Admission physical examinations showed no abnormalities in these systems. Following total cystectomy the patient did well for the first three postoperative days. A wound infection developed and was drained. He became distended and the wound dehisced and was resutured on the seventh postoperative day. The following day his clinical condition was improved but the second day following dehiscence (9th postoperative day) he was restless and anxious with a respiratory rate of 40/min. Arterial blood studies with the patient breathing room air showed O_2 saturation 89%, $PaCO_2$ 28 mm. Hg, CO_2 25 mEq./L. and a pH of 7.56, representing an early phase of respiratory failure (mild, uncompensated respiratory alkalosis). In an attempt to maintain an adequate arterial O_2 saturation the patient was hyperventilating and *blowing off* CO_2. Tracheostomy and artificial ventilation was begun using a mechanical respirator. The patient's clinical condition improved and blood gas studies and pH returned to normal (O_2 saturation 98%, $PaCO_2$ 45 mm. Hg). Although this patient's clinical course before respiratory failure was complicated, all of the complicating factors except the increasing fatigue of a long illness were controlled before the onset of respiratory failure. It seems clear that exhaustion played a major role in creating an intolerable respiratory work load.

Case 3. J. M. The ultimate effects of respiratory failure lead to cardiovascular failure and cardiac arrest. Figure 5 illustrates this chain of events in a 62-year-old man who was admitted with a ruptured abdominal aortic aneurysm. In retrospect, he had *mild emphysema* on admission. Following operative repair he did well and was in excellent clinical condition on the first postoperative day. The pulse and respiratory rate rose steadily on the second postoperative day and massive distention was noted. On the evening of the second postoperative day the patient was disoriented and cyanotic. On the third day pulse and respirations remained elevated but the blood pressure continued at a normal level. The patient was flushed, cyanotic, restless, confused and thrashing about in bed. Cardiac arrest occurred 72 hours postoperatively. Open cardiac massage and artificial ventilation were immediately begun. A $PaCO_2$ immediately following tracheostomy was 60 mm. Hg. The patient was resuscitated. Following mechanical respiration the arterial O_2 saturation was 97% and the $PaCO_2$ was 35 mm. Hg. Adequate oxygen saturation and CO_2 elimination were maintained by a period of continued artificial ventilation and the patient left the hospital well on the 24th postoperative day. The vital signs as plotted illustrate that the patient was able to compensate for the increased energy demanded by the severe peritoneal insult and the decreased efficiency of the mechanics of respiration on the first postoperative day. However, as distention increased, the mechanics of respiration became more inefficient and the patient became exhausted. He was not able to maintain adequate gas exchanges and cardiac arrest followed.

Discussion

In order to understand the genesis of this relative respiratory failure, it is necessary to examine the demands made on the pa-

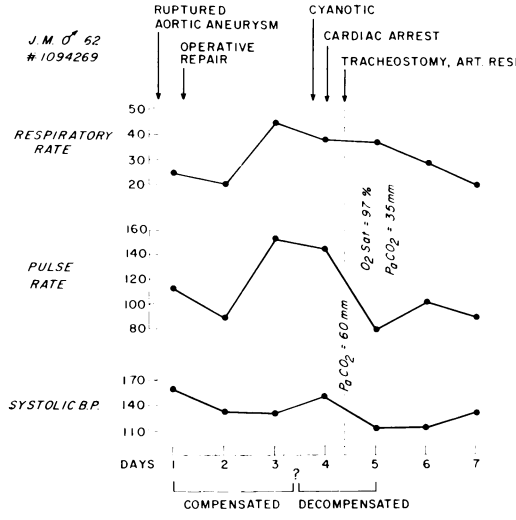

FIG. 5. Case 3.

tient's respiratory system following a severe peritoneal injury and the patient's ability to adapt to these demands. The immediate problem posed to the patient arises from two major trends, both working to the patient's disadvantage. First, the complex process of inflammation, including fever,[27] requires a considerable increase in energy expenditure,[4, 15, 17, 26] forcing the patient to compensate, using a proportional amount of his functional respiratory reserve; and second, the patient's total ability to compensate, that is, the normal extent of his reserve, is decreased by the intraperitoneal insult.[3, 22, 31] Satisfactory compensation is therefore possible only if the decrease in reserve does not lower the patient's total respiratory ability below the level required by the increased energy demands. A third, and important, factor of time must be introduced. The loss of reserve tends to be progressive as the patient becomes exhausted and it is obvious that the energy demands may also be progressive. Thus, the patient may be able to compensate during the first days following peritoneal insult only to succumb to respiratory failure at four or five days because the relationships of reserve and energy demand shift as time passes.

Figure 6 is a schematic diagram illustrating the relation between the total respiratory ability and the required work load under three different circumstances. In normal activity the required work load takes up only a portion of the total respiratory ability, leaving a large reserve. In vigorous exercise the required work load increases but the total respiratory ability is unchanged, leaving a smaller but significant reserve. Following severe peritoneal insult the required work load increases and, in addition, the total respiratory ability decreases. Decompensation takes place when the required work load is greater than the total respiratory ability. This diagram illustrates two important points: 1) that this type of respiratory failure is a combination

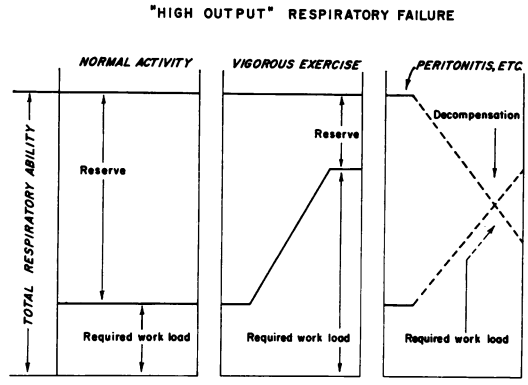

FIG. 6. Schematic diagram of *high output* respiratory failure.

of an increased demand and a decreased total respiratory ability; and 2) that the respiratory mechanisms, even though failing to arterialize the blood adequately, are functioning at a higher than normal level, i.e., a *high output* failure. *High output* respiratory failure, in brief, is the inability to satisfy the increased requirements for tissue oxygenation and later CO_2 excretion despite an initially increased respiratory effort.

A flow-sheet outlining the relationship of some of the major factors leading to what we have termed *high output* respiratory failure is given in Figure 7. The chain of events as outlined in the flow-sheet is as follows. As a result of a severe peritoneal insult a widespread inflammatory response takes place. The effects of this inflammatory response may be divided into biochemical and mechanical factors, both tending to press the patient toward respiratory failure. Mechanically, the inflammatory reaction produces high, relatively fixed diaphragms and considerable pain on respiration. Atelectasis,[23] particularly in the lower lobes, soon follows. Mechanical inefficiency is increased by intrapulmonary shunting caused by the mechanical collapse secondary to long periods without a deep breath.[6] In all, this amounts to considerable inefficiency.[3, 12] In addition to these effects, biochemically the wide-

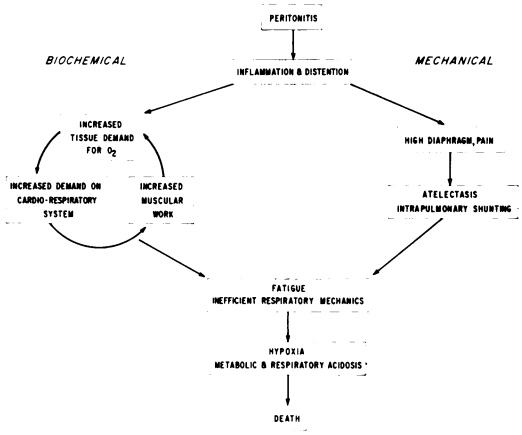

FIG. 7. Flow-sheet showing evolution of pathologic events in *high output* respiratory failure.

spread inflammatory reaction leads to an increased tissue utilization of oxygen.[4, 15, 17, 26] This increased utilization leads to an increased demand on the cardiorespiratory system [13, 25] and an increased demand for muscular work.[16] Increased muscular work leads to a further increase in oxygen utilization,[11, 34] thus establishing a vicious circle requiring more and more oxygen transport and CO_2 elimination. In time this work load leads to fatigue, and fatigue, exaggerated by the inefficient mechanisms of respiration, creates a situation where the ability to ventilate is not sufficient to provide the necessary amount of gas exchange for the greatly increased amount of energy expended. This decompensation causes tissue hypoxia and metabolic and respiratory acidosis. If these are not promptly reversed death results.

The clinical features associated with *high output* respiratory failure may be divided into *premonitory features, early signs* and *signs of frank decompensation.* The premonitory features are those factors that reduce the total respiratory ability of the patient before the onset of peritoneal insult. These include 1) previous pulmonary disease; 2) obesity; 3) heavy cigarette smoking; and 4) severe debility. The early signs of developing respiratory failure are 1) persistent rapid pulse and respiratory rate; 2) normal or slightly elevated blood pressure; 3) increased arterio-venous O_2 difference; and 4) decreased $PaCO_2$ due to hyperventilation in an attempt to maintain adequate arterial O_2 saturation. Signs of frank decompensation are 1) arterial oxygen saturation below that usual for the patient studied (since an occasional patient, as for example one with severe emphysema, may function with a lower than normal O_2 saturation, an O_2 saturation below normal alone does not confirm a diagnosis of this type of respiratory failure); 2) metabolic acidosis where causes other than tissue hypoxia of respiratory origin have been ruled out; and 3) respiratory acidosis. In addition to these signs, which may be documented by precise measurement, the clinical appearance and progressive course of the patient are of utmost importance. As outlined in the above flow-sheet, fatigue is of considerable moment in the development of this syndrome. Although respiratory failure should be documented by the determinations listed above, a high degree of suspicion of impending decompensation should be generated by the patient who has had a severe peritoneal insult, a persistent rapid pulse and respiratory rate in the face of a normal or slightly elevated blood pressure and who is obviously tiring. His face may be flushed, he is anxious, restless and may be thrashing. Respirations are rapid, shallow, labored and, at times, grunting.

Decompensation via *high output* respiratory failure seldom occurs before the second day following peritoneal insult and most often is seen on the fourth or fifth day. In order to prevent decompensation these patients must be followed with frequent evaluation of the adequacy of arterial oxygenation and CO_2 elimination. The most exact methods [1, 2] involve the examination of arterial blood. $PaCO_2$ alone can be conveniently measured by the rebreathing technic.[14, 38] It is necessary to

emphasize that the standard blood gas studies (O_2 saturation, $PaCO_2$, CO_2 content or standard bicarbonate and pH) are needed to confirm the presence and determine the extent of decompensation.

When decompensation is imminent its progression almost always can be interrupted by measures that improve the mechanics of respiration, i.e., relief of intra-abdominal tension by gastro-intestinal intubation or operative drainage and decompression of the gastro-intestinal tract or peritoneal cavity, and with efficient pulmonary physiotherapy. Nasal O_2 improves arterial oxygen saturation but the resulting absence of cyanosis may mask a progressive respiratory acidosis. If these measures fail the intolerable work load of respiration must be relieved by tracheostomy and artificial ventilation using a mechanical respirator. In addition to these essentials of specific therapy, it is important to maintain other adjuvant therapy that must include antibiotics, blood, plasma and electrolytes.

Summary

High output respiratory failure is defined as that type of respiratory failure characterized by inability to provide adequate tissue oxygenation and later carbon dioxide excretion despite an initially increased gas exchange. In the past few years, 21 patients have been studied and treated in the Massachusetts General Hospital with this type of failure associated with severe peritonitis or ileus. There were five deaths in this series including three due to renal failure, one to pulmonary embolism, one to ventricular arrhythmia. No death was due to *high output* respiratory failure since all deaths occurred from two weeks to two months after it had been relieved.

It is vitally important that incipient respiratory failure be recognized by the surgeon who is treating a patient with peritonitis or ileus. He should be alerted preoperatively by any previous lung disease, obesity, heavy smoking or severe debility. The syndrome of *high output* respiratory failure may be suspected by persistent elevation of pulse and respiration with a normal blood pressure in an anxious, exhausted patient. It is confirmed by a lower arterial oxygen saturation than is normal for the patient. (Thus, in the presence of normal lungs an arterial oxygen saturation of 95 per cent or less must be regarded as evidence of decompensation.) Prevention of decompensation often can be effected by abdominal decompression or drainage, as well as chest physiotherapy. If these fail immediate institution of tracheostomy and assisted respiration with a mechanical respirator will result in normal gas exchange and relief of this type of failure.

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