

## PRESIDENTIAL ADDRESS TO THE MIDWEST SURGICAL ASSOCIATION

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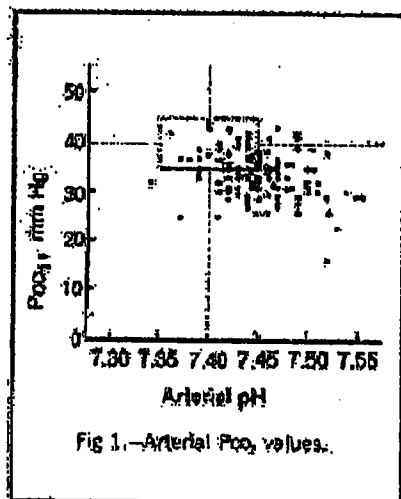
I cannot adequately express to you what a great honor it is to be President of the Midwest Surgical Association. I have enjoyed the members and the meetings of this group more than any other I have ever attended. I hope that we can all continue to work together to make this an association that will be increasingly attractive to other surgeons.

For my presidential address, I thought I would review some of the work that we have been doing relative to trauma, sepsis, and organ failure.

### 1. Changes after fractures.<sup>1</sup>

Because of our interest in the fat embolus syndrome and the respiratory and coagulation changes after fractures, we performed blood gas and coagulation studies on over 200 patients admitted to the Orthopedic Service of Detroit General Hospital between May 1970 and December 1972. Of these 200 patients, 107 were considered to have relatively uncomplicated extremity fractures and had no evidence of pre-existent respiratory or cardiac disease.

**Acid-Base Changes.** Respiratory alkalosis, with a  $PCO_2$  of less than 40 mm Hg and a pH greater than 7.40, was present during the first 48 hours in 83 (78%) of these patients (Fig. 1).



During the first 48 hours, only 43 (40%) of the patients had a "normal" arterial  $PO_2$  of 80 to 100 mm Hg. Thirty had a  $PO_2$  of 70 to 79 mm Hg, 23 had a  $PO_2$  of 60 to 69 mm Hg, and 11 had a  $PO_2$  of less than 60 mm Hg.

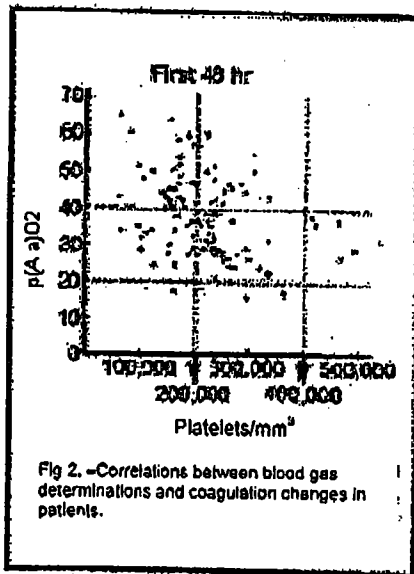
**Alveolar-Arterial Oxygen Differences  $p(A-a)O_2$ .** A  $p(A-a)O_2$  of 5 to 20 mm Hg while breathing room air is considered to be normal in our laboratory, but it was found initially in only five patients. The  $p(A-a)O_2$  was 21 to 39 mm Hg (slightly-to-moderately abnormal) in 56

patients; 40 to 49 mm Hg (moderately-to-severely abnormal) in 29 patients; and 50 mm Hg or more (severely abnormal) in 17 patients.

The platelet counts were initially normal (200,000 to 400,000/mm<sup>3</sup>) in 54 patients, greater than normal in five patients, and less than normal in 43 patients. In general, the more severe the fractures were, the lower the platelet count.

**Fibrinogen.** The initial fibrinogen levels were normal (200 to 300 mg/dl) in 38 patients, less than normal in three patients, and greater than normal in 60 patients. The fibrinogen levels tended to rise rapidly and progressively thereafter. After the third day, all except three patients had elevated fibrinogen levels.

**Platelets and p(A-a)O<sub>2</sub>.** The patients with the lowest platelet counts tended to have the lowest arterial PO<sub>2</sub> and the highest p(A-a)O<sub>2</sub>. Of 44 patients with a low platelet count initially, 27 (61%) had a p(A-a)O<sub>2</sub> of 40 mm Hg or higher. In contrast, of 63 patients with a normal or high platelet count, only 19 (30%) had an p(A-a)O<sub>2</sub> of 40 mm Hg or higher (Fig. 2).



| Fracture Severity Scale | No. of Patients (107) | Platelets, cu mm | PO <sub>2</sub> | P(A-a)O <sub>2</sub> |
|-------------------------|-----------------------|------------------|-----------------|----------------------|
| 17-23                   | 4                     | 134,000          | 66              | 50                   |
| 11-16                   | 14                    | 191,000          | 79              | 33                   |
| 8-10                    | 25                    | 229,000          | 76              | 39                   |
| 6-7                     | 29                    | 234,000          | 78              | 34                   |
| 3-5                     | 35                    | 214,000          | 74              | 43                   |
| Av value                |                       | 217,000          | 75              | 38                   |

Although none of these patients showed the classic fat embolism syndrome, many of them had severe reductions in the arterial  $PO_2$  and platelet count. These changes were similar to those found by E. Mammen, MD, and R. Henry, PhD (unpublished data) after experimental closed fractures of the femur in dogs. The platelet count and fibrinogen levels fell sharply almost immediately after the fracture; however, within hours, the fibrinogen levels returned to normal and continued to rise to supernormal levels for several days.

Our findings suggest that there is an important correlation between fat embolism and intravascular coagulation and that low or falling platelet counts may be of value in predicting which patients may need ventilatory support.

If the treatment of posttraumatic respiratory failure is to be successful, it must be begun early and pursued aggressively. Many of the physicians involved in this study were impressed with the lack of any clinical evidence of respiratory distress even when the arterial  $PO_2$  was less than 70 mm Hg or the  $p(A-a O_2)$  exceeded 40 Hg. If an abnormality is noted, frequent serial repeat determinations should be performed. If a progressive deterioration in blood gases and platelet count is found, the patient should probably be given ventilatory assistance. If the physiologic shunting in the lung can be measured, this may be an even better method of diagnosing impending respiratory failure.

## 2. Physiologic shunting in the lung.<sup>2</sup>

In another study (1967-1969), physiologic shunting in the lungs was calculated in 200 critically ill or injured patients. All of the deaths in these patients were directly attributable to or were associated with acute respiratory failure. A normal shunt is 3-5% of the cardiac output. As the amount of physiologic shunting in the lungs increased, the mortality rate rose. Of eight patients with "almost normal" shunts (0-19%), only one (13%) died. Of the 89 patients with "increased" shunts (20-39%), 32 (36%) died. Of the 77 with "significant" shunts (40-59%), 45 (58%) died; and of the 26 patients with "lethal" shunts (60% or more), 23 (88%) died. Within each of these groups, however, other factors, especially abnormal pH, shock, sepsis, and liver disease, seemed to correlate with increased shunting and a higher mortality rate.

The 85 septic patients in this study had an average shunt of 46% and mortality rate of 68%. This could be contrasted with 115 other patients without sepsis who had an average shunt of 37% and a mortality rate of 37%.

## 3. Oxygen consumption.<sup>3</sup>

In another study, oxygen consumption was measured directly with the Guyton continuous oxygen analyzer in 100 patients. A theoretical "normal" oxygen consumption was determined for each patient from a standard nomogram and was based upon the patient's age, sex, height and weight.

The initial oxygen consumption in these patients ranged from 65% to 205% of the theoretical basal values with an average of  $109 \pm 21\%$  (SD) or  $160 \text{ ml/M}^2$ . These patients were divided into

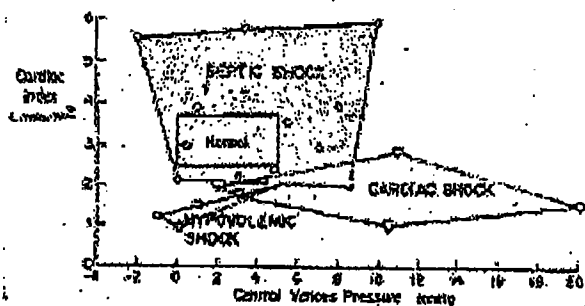
three groups: 20 patients with a reduced oxygen consumption (less than 90% of the theoretical basal value), 34 patients with normal oxygen consumption (90-110%), and 46 patients with increased oxygen consumption.

The patients in shock were much more likely to have a reduced oxygen consumption and a metabolic acidosis than those who were not in shock.

Septic patients had a greater tendency to have a reduced oxygen consumption and a metabolic acidosis than the patients who were not septic. Of the 61 septic patients, 16 (26%) had a reduced oxygen consumption and ten (16%) had a metabolic acidosis. Of the 39 patients who were not septic, only four (10%) had a reduced oxygen consumption and two (5%) had metabolic acidosis. Of the 33 patients with both sepsis and shock, twelve (36%) had reduced oxygen consumption and seven (21%) had metabolic acidosis.

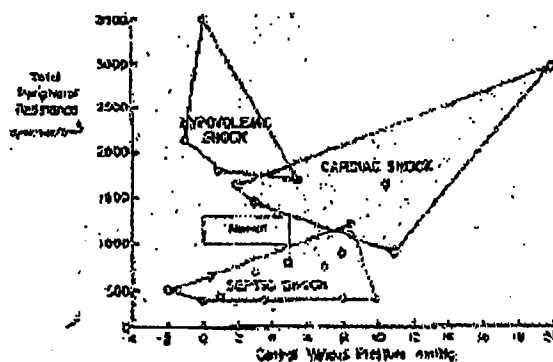
#### 4. Hemodynamic changes in sepsis.<sup>4</sup>

Up until 1965, there had been a tendency to equate shock, regardless of its origin, with a low cardiac output and a high total peripheral resistance. While our experience suggested that this was true of hypovolemic and cardiac shock, the same could not be said of septic shock.



*In fig. 3 it can be seen that patients with cardiac and hypovolemic shock tended to have a low cardiac index. Those with cardiac shock, however, usually had an elevated central venous pressure. Patients with septic shock had cardiac output levels that were often normal, or elevated with a central venous pressure that was usually in the normal range.*

*In fig. 4 the three types of shock could usually be well delineated by the central venous pressures and total peripheral resistance. Patients with cardiac and hypovolemic shock had an elevated total peripheral resistance, but in the patients with septic shock it was usually below normal.*



In studying 12 patients with relatively pure septic shock, the cardiac output was higher and total peripheral resistance was lower than in those with hypovolemic or cardiac shock. Of 16 patients with a total peripheral resistance less than 1,000 dyne-sec/cm<sup>5</sup>, 15 had some element of sepsis,

and in 12 of these, sepsis was the dominant feature of the illness. On the other hand, in nine patients exhibiting relatively pure forms of hypovolemic or cardiac shock, the total peripheral resistance was invariably greater than 1,300 dyne-sec/cm<sup>5</sup> and, in almost half the cases, was over 1,800.

#### 5. Arterial-Central Venous Oxygen Differences.<sup>5</sup>

From 1967 through 30 June 1973, 328 patients had simultaneous determinations of arterial and central venous blood gases. Although many of these patients had multiple shunt and acid-base determinations performed, only those values recorded during the first determinations were used in this study.

The average arterial-central venous oxygen-content difference was  $3.5 \pm 1.6$  vol/dl (S.D.) so that with 10.0 grams of hemoglobin and an arterial oxyhemoglobin saturation of 95%, the central venous oxygen saturation (ScvO<sub>2</sub>) was 60%. The patients with the largest physiologic pulmonary shunts and the lowest arterial oxygen contents had the lowest A-V oxygen differences. The patients with a shunt of 60-89% had an average A-V oxygen difference of 1.0 vol/dl, with an SaO<sub>2</sub> of 93% and ScvO<sub>2</sub> of 80%. Those with a shunt of 4-19% had an A-V difference of 5.6 vol/dl, with an SaO<sub>2</sub> of 100% and ScvO<sub>2</sub> of 53%, implying an inadequate O<sub>2</sub> delivery.

The patients with an arterial oxygen content less than 12.0 vol/dl had an average A-V oxygen difference of only 2.5 vol/dl, while those with an arterial oxygen content of 20.0 vol/dl or more had an average A-V oxygen difference of 5.5 vol/dl.

The mortality rates were highest in the patients with the lowest A-V oxygen content differences and the lowest arterial oxygen contents. Of the 76 patients who had an A-V O<sub>2</sub> difference of less than 2.0 vol/dl, 51 (67%) died. In contrast, of the 30 patients who had an A-V O<sub>2</sub> difference of 6.0 vol/dl or more with an ScvO<sub>2</sub> of 70%, only 14 (36%) died. Of 86 patients with an arterial oxygen content less than 12.0 vol/dl, 66 (77%) died. In contrast, of the other 415 patients with a higher arterial O<sub>2</sub> content, only 200 (48%) died.

#### 6. Blood Volume Determinations.<sup>6</sup>

Blood volume determinations were made with radioiodinated serum albumin in 86 patients and with <sup>51</sup>Cr tagged red blood cells in 38 patients. Although one sample was always taken ten minutes after the radioiodinated serum albumin or tagged red blood cells were injected, serial samples were also obtained every ten minutes for one hour after the injection of radioiodinated serum albumin in 32 patients and <sup>51</sup>Cr in 25 patients.

There was no correlation between the initial central venous pressure and the blood volume. Of 57 patients with a low measured blood volume, 28 (49%) had an elevated central venous pressure. At the other extreme, of 29 patients with an increased blood volume, eight (30%) had a low central venous pressure (< 5.0 mm Hg).

Patients with cardiac problems tended to have an elevated central venous pressure (> 15 mm Hg) and a low or normal blood volume.

Central venous pressure responses to fluid were much more reliable than isolated central venous pressure readings for determining the amount of fluid needed. None of the single parameters studied—blood volume, central venous pressure, or clinical impression—correlated consistently with the amount of fluid required to maintain a normal BP and urine output. Central venous pressure or blood volume studies could not be relied upon to safely and consistently determine the amount of fluids required by critically ill patients.

#### 7. Alkalosis in critically ill patients.<sup>7</sup>

The deleterious effects and increased mortality associated with severe acidosis in critically ill or injured patients are well-known. Less well-recognized, however, are the prognostic implications of severe alkalosis. In the past two years we have noticed an increasing incidence of alkalosis in critically ill patients who have severe sepsis and/or trauma. This acid-base abnormality has often been difficult to correct, and many of these patients have died. Of 1,415 critically ill patients studied by the Shock Unit at Detroit General Hospital from 1962-1971, 177 (12.5%) were found to have an arterial pH of 7.55 or higher.

There was a strong correlation between the severity of the alkalosis and the mortality. Of 61 patients with a pH value of 7.55 to 7.56, 25 (41%) died; of 61 with a pH of 7.57 to 7.59, 29 (47%) died; of 40 with a pH value of 7.60 to 7.64, 26 (65%) died; and of 15 with a pH of 7.65 or higher, 12 (80%) died.

The highest mortality (60/90 = 66%) occurred in patients with a combined respiratory and metabolic alkalosis, followed by those with isolated metabolic alkalosis (12/27 = 44%), and isolated respiratory alkalosis (12/27 = 35%).

The severe alkalosis was usually not present at the time of admission; the majority of cases occurred several days later when these patients had developed increasing sepsis and respiratory difficulty.

#### 8. Massive blood transfusions.<sup>8</sup>

Many patients requiring emergency operations and massive blood transfusions in civilian practice also suffer from anemia, malnutrition, and cardiac, renal or liver impairment, which adversely influence their responses to shock and trauma.

Between 1959 and 1966, 402 patients were given massive blood transfusions of at least 10 units within a 24-hour period. The mortality rate increased roughly parallel to the amount of blood given. Of 45 patients receiving 25 or more units of blood, only three left the hospital alive.

Massive transfusion patients with infections, cirrhosis, malignancy, aortic aneurysms, or pancreatitis had a very poor prognosis with any type of stress or shock. The mortality rate in 158 massive transfusion patients with these complicating medical conditions was 72%. This contrasts with the 36% mortality rate in similarly transfused patients who had none of these comorbidities.

In 102 massive transfusion patients in whom shock was prevented or limited to less than 15 min duration, the mortality was only 19%. In contrast, in 273 similarly transfused patients in whom the clinical features of shock persisted for longer than 15 min, the mortality was 62%. Thus, massive transfusions are not necessarily harmful in themselves, but associated prolonged shock is a much greater factor in causing a high mortality rate.

The incidence of wound complications in the 241 massive transfusion patients surviving 7 or more days was 30% (71/241), or about six times the average seen on the general surgical services at this hospital.

Our experience seems to indicate that most massive transfusion patients who are not in shock do not need calcium, regardless of the amount of blood they are given. On the other hand, patients in shock receiving massive transfusions often are unable to mobilize sufficient amounts of calcium for optimal cardiovascular function.

#### Summary and Conclusions:

Thus, critically ill and injured patients can have a wide variety of problems. Prompt recognition and correction of these abnormalities appears to be the best way of reducing mortality rates.

I am honored to be able to give this address, and I wish all of you a great time at this meeting.

## References:

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