Oxygen Tissue Saturation Is Lower in Nonsurvivors than in Survivors after Early Resuscitation of Septic Shock

Marc Leone, M.D., Ph.D.,† Sami Bledi, M.D.,‡ François Antonini, M.D.,† Bertrand Meyssignac, M.D.,† Sébastien Bordon, M.D.,‡ Frédéric Garcin, M.D.,‡ Aude Charvet, M.D.,‡ Valéry Blasco, M.D.,† Jacques Albanèse, M.D., Ph.D.,‡ Claude Martin, M.D.||

Background: Growing evidence suggests that the microvascular dysfunction is the key element of the pathogenesis of septic shock. This study's purpose was to explore whether the outcome of septic shock patients after early resuscitation using early goal-directed therapy is related to their muscle tissue oxygenation.

Methods: Tissue oxygen saturation (StO2) was monitored in septic shock patients using a tissue spectrometer (InSpectra Model 325; Hutchinson Technology, Hutchinson, MN). For the purpose of this retrospective study, the StO2 values were collected at the first measurement done after the macrohemodynamic variables (mean arterial pressure, urine output, central venous saturation in oxygen) were optimized.

Results: After the hemodynamic variables were corrected, no difference was observed between the nonsurvivors and survivors, with the exception of pulse oximetry saturation (94% [92–97%] vs. 97% [94–99%], P = 0.04). The StO2 values were significantly lower in the nonsurvivors than in the survivors (73% [68–82%] vs. 84% [81–90%], P = 0.02). No correlations were found between the StO2 and SpO2 (P = 0.7).

Conclusions: In septic shock patients, tissue oxygenation below 78% is associated with increased mortality at day 28. Further investigations are required to determine whether the correction of an impaired level of tissue oxygenation may improve the outcome of these patients.

Near-infrared spectroscopy is a noninvasive monitoring, providing real-time feedback.4 Near-infrared spectroscopy monitors only vessels with a diameter of less than 1 mm because the high concentration of blood in arteries and veins makes photon emergence unlikely. Near-infrared light (600–800 nm) easily crosses biologic tissues and is absorbed by hemoglobin, myoglobin, and oxidized cytochrome, as described elsewhere.5 This tool can quantify microvascular dysfunction in patients with septic shock.6

One can hypothesize that near-infrared spectroscopy can detect a potential microvascular dysfunction in the patients adequately resuscitated from a macrohemodynamic standpoint. The purpose of this study was to explore whether the outcome of the septic shock patients after early resuscitation using early goal-directed therapy was related to their muscle tissue oxygenation.

Materials and Methods

This study was retrospectively conducted in a 16-bed intensive care unit of an 800-bed university hospital (Hôpital Nord, Marseille, France). Informed consent and approval by the Ethics Committee were waived due to the observational nature of the study.

Patients

Septic shock was defined according to the criteria of the International Sepsis Definitions Conference.7 All patients received fluid expansion (crystalloids or 6% hydroxyethyl starch) and then required norepinephrine to raise mean arterial pressure to 65 mmHg or more.1 All patients received broad-spectrum antibiotic coverage, usually a β-lactam and a quinolone. Vancomycin was added when oxacillin-resistant staphylococci were suspected. All patients needed mechanical ventilation and sedatives because of acute respiratory failure. According to previously conducted studies, 50 patients with septic shock were expected within a 1-year period. Thus, all the patients with septic shock were included during a 1-year period (2007).

Measurements

Heart rate, mean arterial pressure, oxygen plethysmography, and end-tidal carbon dioxide were continuously monitored (Moniteur Patient Intellivue MP 70; Philips, Andover, MA). All patients had an arterial catheter and a central venous catheter placed through the subclavian vein. The arterial and venous catheters were connected...
to a Picco-Plus monitor (Pulsion Medical Systems, Munich, Germany) in the patients in whom vascular accesses made it possible to insert this monitoring system. This monitoring is based on the transpulmonary thermodilution technique and arterial pulse contour analysis. An indwelling urinary catheter was inserted in each patient. Urine was collected via a urinometer (Curity 0123; Kendall, Hands, United Kingdom). The following variables were prospectively collected: heart rate, mean arterial pressure, central venous saturation in oxygen (ScvO₂), pulse pressure variations if its measure seemed relevant to the attending physician (sinus rhythm, no right heart failure, controlled ventilation), cardiac index if available, lactate plasma level, pulse oximetry saturation (SpO₂), hemoglobin, creatinine plasma concentration and urine output. Demographic data, severity score (simplified acute physiology score II), and sedation score (Ramsay score) were retrospectively collected. Our local protocol was aimed at correcting macrohemodynamic variables (mean arterial pressure, urine output, ScvO₂). Initial resuscitation consisted of intravenous fluid targeted to achieve pulse pressure variations below 13% in the patients with equipment. If the patients were not equipped, echocardiography (respiratory variation in inferior vena cava diameter) analysis and passive leg raising (pulse pressure variations below 10%) were used. The objectives of mean arterial pressure, urine output, and ScvO₂ were achieved in 42 (100%), 36 (86%), and 35 (84%) patients, respectively. After the hemodynamic variables seemed optimal according to the decision of the attending physician. Briefly, preload was optimized (pulse pressure variations below 13% or lack of response to passive leg raising or no respiratory variations of the inferior vena cava diameter or pulmonary edema), mean arterial pressure was above 65 mmHg, urine output was above 0.5 ml/kg of body weight (except in the patients with acute renal failure), and the ScvO₂ was at 70% or more. At the measurement time, at least three of these aims had to be achieved in all patients. The near-infrared spectroscopy probe was placed on the clean skin of the thanar eminence. After a 3-min period to stabilize the near-infrared spectroscopy signal, the value of StO₂ was recorded. A brief clinical assessment was performed by assessing the capillary refill time (less than 2 s) and the temperature at the level of the big toe (cold or hot). This assessment was done by two investigators (Drs. Blidi and Meyssignac). To ensure that the data are not unique to the thanar eminence and not representative of the rest of the body, we included a second cohort of patients with septic shock after the macrohemodynamic variables were optimized as previously described. In this cohort, the values of StO₂ were successively recorded at three sites: thanar, deltoid, and masseter.

Statistics Analysis

Statistical calculations were performed using the software package SPSS 15.0 (SPSS Inc., Chicago, IL). For continuous and ordinal variables, data were expressed as median with interquartile range (25–75 quartiles). Data were performed using the Mann–Whitney U test. Comparisons of percentages were performed with the Fisher exact test. Comparisons of two continuous variables were performed using a linear regression. Comparisons between the three groups of the second cohort were made using the Kruskal–Wallis test. Discrimination of values was assessed with the receiver operating characteristic analysis. Sensitivity and specificity were also computed. All comparisons were two-tailed, and P < 0.05 was required to exclude the null hypothesis.

Results

Forty-two consecutive patients with septic shock were included in the study. Their characteristics are shown in table 1. Mortality at day 28 occurred in 13 (31%) patients (table 1). Three patients (23%) did not survive on day 3. The objectives of mean arterial pressure, urine output, and Scvo₂ were achieved in 42 (100%), 36 (86%), and 35 (84%) patients, respectively. After the hemodynamic variables were optimized, no difference was observed between the survivors and nonsurvivors, with the exception of SpO₂, which was significantly lower in the nonsurvivors than in the survivors (94% [92–97%] vs. 84% [78–89%]).
ingly, the StO2 values were significantly lower in the survivors and nonsurvivors than in the survivors (73% [68–82%] vs. 84% [81–90%], P = 0.02) (fig. 1). A receiver operating characteristic analysis confirmed that StO2 was significantly associated mortality with an area under the curve at 71% (52–91%, P = 0.03), hemoglobin (P = 0.8, R² = 0.02), Ramsay score (P = 0.2, R² = 0.02) and norepinephrine dosage (P = 0.9, R² = 0.02). In contrast, the lactate plasma level was correlated with StO2 (fig. 3D).

Finally, in a new cohort of nine patients, we tested three distinct sites: thenar, masseter, and deltoid. Briefly, the two cohorts were similar in age (54 [40–59] vs. 59 [51–69] yrs, P = 0.3), sex ratio (33% vs. 33% vs. 33% vs. 33%), sex ratio (33% vs. 33% vs. 33% vs. 33%), SAPS II (50 [45–55] vs. 47 [38–58], P = 0.8), mean arterial pressure (84 [71–85] vs. 80 [72–85] mmHg, P = 0.9), urine output (50 [45–120] vs. 60 [40–100] ml·kg⁻¹·h⁻¹, P = 0.5), and ScvO₂ (76 [73–82] vs. 78 [71–84]%, P = 0.1), and

Table 1. Characteristics of the 42 Patients Included in the Study

<table>
<thead>
<tr>
<th>Variables</th>
<th>Survivors (n = 29)</th>
<th>Nonsurvivors (n = 13)</th>
<th>Entire Cohort (n = 42)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>9 (31)</td>
<td>4 (30)</td>
<td>13 (31)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>59 (40–67)</td>
<td>60 (55–73)</td>
<td>59 (52–67)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>24 (21–29)</td>
<td>23 (22–24)</td>
<td>24 (22–28)</td>
</tr>
<tr>
<td>Simplified acute physiology score II</td>
<td>47 (37–54)</td>
<td>58 (42–63)</td>
<td>47 (38–58)</td>
</tr>
<tr>
<td>Creatinine plasma levels, μmol/l</td>
<td>96 (66–175)</td>
<td>111 (62–144)</td>
<td>96 (66–172)</td>
</tr>
<tr>
<td>Ramsay score</td>
<td>5 (3–6)</td>
<td>5 (4–6)</td>
<td>5 (3–6)</td>
</tr>
<tr>
<td>Source of septic shock</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lungs</td>
<td>13 (45)</td>
<td>7 (54)</td>
<td>20 (48)</td>
</tr>
<tr>
<td>Intraabdominal</td>
<td>13 (45)</td>
<td>5 (38)</td>
<td>18 (43)</td>
</tr>
<tr>
<td>Urinary tract</td>
<td>2 (7)</td>
<td>0 (0)</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Skin</td>
<td>0 (0)</td>
<td>1 (8)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

No difference was observed between the survivors and the nonsurvivors. Data are presented as median (interquartile range) and number of patients (percentage).

97% [94–99%], P = 0.04) (table 2). Clinically, 22 (77%) of the survivors had a capillary refill time of less than 2 s, as compared with 13 (77%) of the nonsurvivors (P = 1.00). The big toe temperature was hot in 18 (63%) survivors and 8 (61%) nonsurvivors (P = 0.7). Interestingly, the StO2 values were significantly lower in the nonsurvivors than in the survivors (73% [68–82%] vs. 84% [81–90%], P = 0.02) (fig. 1).

A receiver operating characteristic analysis confirmed that StO2 was significantly associated mortality with an area under the curve at 71% (52–91%, P = 0.03), hemoglobin (P = 0.8, R² = 0.02), Ramsay score (P = 0.2, R² = 0.02) and norepinephrine dosage (P = 0.9, R² = 0.02). In contrast, the lactate plasma level was correlated with StO2 (fig. 3D).

Finally, in a new cohort of nine patients, we tested three distinct sites: thenar, masseter, and deltoid. Briefly, the two cohorts were similar in age (54 [40–59] vs. 59 [51–69] yrs, P = 0.3), sex ratio (33% vs. 33% vs. 33% vs. 33%), SAPS II (50 [45–55] vs. 47 [38–58], P = 0.8), mean arterial pressure (84 [71–85] vs. 80 [72–85] mmHg, P = 0.9), urine output (50 [45–120] vs. 60 [40–100] ml·kg⁻¹·h⁻¹, P = 0.5), and ScvO₂ (76 [73–82] vs. 78 [71–84]%, P = 0.1), and

Table 2. Hemodynamics of the Patients According to Their Survival

<table>
<thead>
<tr>
<th>Variables</th>
<th>Survivors (n = 29)</th>
<th>Nonsurvivors (n = 13)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td>100 (85–114)</td>
<td>94 (88–115)</td>
<td>0.4</td>
</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>79 (72–87)</td>
<td>80 (71–84)</td>
<td>0.5</td>
</tr>
<tr>
<td>Urine output, ml/h</td>
<td>80 (40–100)</td>
<td>50 (32–80)</td>
<td>0.3</td>
</tr>
<tr>
<td>Pulse oxygen saturation, percentage</td>
<td>97 (94–99)</td>
<td>94 (92–97)</td>
<td>0.04</td>
</tr>
<tr>
<td>Pulse pressure variations,* percentage</td>
<td>9 (7–12)</td>
<td>8 (4–9)</td>
<td>0.3</td>
</tr>
<tr>
<td>Cardiac index,* (l/m²)</td>
<td>3.8 (3.1–4.2)</td>
<td>4.1 (3.4–4.3)</td>
<td>0.9</td>
</tr>
<tr>
<td>Oxygen central venous saturation, percentage</td>
<td>78 (72–84)</td>
<td>79 (71–85)</td>
<td>0.9</td>
</tr>
<tr>
<td>Lactate plasma level, mmol/l</td>
<td>2.3 (1.4–2.9)</td>
<td>2.5 (1.5–4.7)</td>
<td>0.1</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>8.1 (8.6–11)</td>
<td>9.1 (8.6–11)</td>
<td>0.9</td>
</tr>
<tr>
<td>Norepinephrine, μg·kg⁻¹·min⁻¹</td>
<td>0.4 (0.1–1.1)</td>
<td>1.0 (0.5–2.0)</td>
<td>0.2</td>
</tr>
<tr>
<td>Norepinephrine duration, h</td>
<td>36 (18–56)</td>
<td>24 (14–61)</td>
<td>0.5</td>
</tr>
<tr>
<td>Dobutamine, percentage of patients</td>
<td>3 (10)</td>
<td>1 (10)</td>
<td>1</td>
</tr>
</tbody>
</table>

* Data were available in 30 (70%) patients. Data are presented as median (interquartile range) and number of patients (percentage).

Fig. 1. Individual values of oxygen tissue saturation (StO₂) are given for the survivors and nonsurvivors at day 28. Horizontal lines = median.

Fig. 2. Receiver operating characteristic curves. Tissue oxygen saturation (StO₂), central venous saturation in oxygen (ScvO₂), lactate plasma level, and norepinephrine dosage according to survival. The areas under the curves are 71% (52–91%, P = 0.03), 56% (31–70%, P = 0.9), 38% (19–58%, P = 0.1), and 39% (22–56%, P = 0.2), respectively.
0.9). StO₂ measured at the thenar eminence did not differ between the two cohorts (84 [80–89] vs. 78 [71–84] %, P = 0.4). No difference was found among the three sites of measurement (84 [80–89] vs. 89 [81–90] vs. 85 [78–90] %, P = 0.7).

**Discussion**

Our results show that, in a real-life study, after the macrohemodynamic variables were optimized, the StO₂ of the nonsurvivors at day 28 was significantly lower than that of the survivors. A value of StO₂ below 78% was associated with an increased risk of mortality. With the exception of plasma lactate levels, this variable was not correlated with the other markers of arterial oxygenation. As a result, this monitoring can provide an easy and direct tool to assess the risk of mortality in septic shock patients. In contrast, the impact of treatment aimed at correcting the StO₂ should be investigated in future studies.

At the bedside, it is challenging to identify the microcirculation dysfunction. In the current study, the clinical assessment using capillary refill time and big toe temperature did not provide relevant information. Several methods have been reported to assess the microvascular blood flow. Intravital microscopy is considered the standard for *in vivo* investigation of the microcirculation. This technique can be used for investigation of thin tissues that allow transillumination, whereas fluorescent dyes must be used to allow epillumination of thicker organ surfaces. Unfortunately, the use of dyes in humans is hindered by safety concerns. Thus, intravital microscopy studies have been limited to observation of nail fold capillaries that can be observed without using dyes. Other techniques like laser-Doppler flowmetry, orthogonal polarization spectral imaging, and the sidestream dark field imaging are of major interest but remain difficult to use in real-life conditions. Our approach was to give priority to being noninvasive, being user-friendly, providing real-time feedback, and influencing mortality. The use of near-infrared spectroscopy responded to these specifications.

In a previously published study, the same monitoring detected altered recovery after an ischemic challenge in patients with septic shock. The presence and persistence of such alterations in the first 24 h of sepsis were associated with impaired outcome. However, this technique consists on stopping arterial blood flow by inflating the cuff to 50 mmHg above the systolic arterial pressure. After 3 min of ischemia, cuff pressure is released, and StO₂ is recorded continuously for another 3-min period. The slope of the increase in StO₂ and the difference between the maximum StO₂ value during hyperemic phase and the baseline StO₂ are calculated. Although this technique provides interesting results, our approach offers a direct and continuous measurement available at the bedside. In addition, different sites of measurements seem available without affecting the significance of results.

The association between StO₂ and outcome has already been reported in trauma patients. In the emergency room, the information about StO₂ allows for discriminating the patients who would later go on to develop multiorgan dysfunction syndrome or die. In the current study, the StO₂ also discriminates the patients with
poor outcome. The univariate analysis shows that the 
$\text{SpO}_2$ is lower in the nonsurvivors than in the survivors. 
One may hypothesize that $\text{StO}_2$ is mainly dependent on 
$\text{SpO}_2$. However, no correlation was found between the 
$\text{StO}_2$ and $\text{SpO}_2$. This result is in agreement with an experi-
mental study, concluding that muscle tissue does not 
show changes reflecting a greater deoxygenation during 
acute hypoxia.\textsuperscript{19} As a result, the monitoring of $\text{StO}_2$ 
provides information about tissue oxygenation, which is 
independent of arterial oxygenation.

The patients with $\text{StO}_2$ below 78\% are at increased risk 
of mortality. No specific intervention was conducted 
regarding the correction of the low values of $\text{StO}_2$. The 
goal of our protocol was aimed at correcting low $\text{SvO}_2$. The 
$\text{SvO}_2$ was above 65\% in all patients but one. Six 
patients had values ranged from 65\% to 70\%. The prog-
nosis value of this variable has been shown in the early 
phase of the management of patients with severe sep-
sis.\textsuperscript{20,21} However, its relevance remains unknown after 
the patients are resuscitated. According to our study, 
with the exception of a type 2 error, the accuracy of $\text{StO}_2$ 
to predict mortality seems superior to that of $\text{SvO}_2$. In 
addition, our findings confirm previous studies in which 
the correlation between the two variables is poor or 
lacking.\textsuperscript{22,23} However, we cannot conclude that the cor-
rection of $\text{StO}_2$ above 78\% would be an efficient measure 
to improve outcome.

Several limitations should be acknowledged. The study 
is of a small number of subjects at a single timepoint. The 
interpretation of nonsignificant results can result from a 
lack of power. Thus, it is difficult to discern between 
meaningful differences existing versus lack of power 
to detect them. Use of eminence thenar may raise some 
issues. Indeed, the measurements may reflect changes in 
skin circulation at a depth of 7.5 mm. One can argue that 
this circulation is not representative of other circulations 
in our patients. To reduce this uncertainty, we tested 
three distinct sites in nine other patients: thenar, mass-
ter, and deltoid. No difference was observed among the 
three sites, indicating that our results may be replicated 
by using other sites of measurement. However, in healthy 
volunteers, $\text{StO}_2$ determined from deep muscle, not thenar 
eminence, was an indicator of central hypovolemia.\textsuperscript{24} 
Thus, new technologies can improve the relevance of mea-
surements. The use of 25-mm probes, instead of 15 mm, 
can also affect the results because the presumed depth 
of penetration is half of the path length.\textsuperscript{18} This specific 
point requires future investigations. Finally, we did not 
explore $\text{StO}_2$ change over time because the study was 
aimed at assessing the microcirculation at a specific 
time. One should admit that this did not improve out-
come even though resuscitation increased $\text{StO}_2$. How-

ever, the dynamic changes of $\text{StO}_2$ over time are probably 
an interesting field of experimentation.

In conclusion, after the macrohemodynamic variables 
were optimized, the monitoring of $\text{StO}_2$ can discriminate 
the patients at high risk of mortality. We determined that 
a $\text{StO}_2$ below 78\% is associated with an increased mor-
tality. The impact of possible therapeutic aimed at in-
creasing the $\text{StO}_2$ was not explored in the current study. 
There is a need of further investigations to test such 
hypothesis.

References

1. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, Reinhart 
K, Angus DC, Brun-Buisson C, Beale R, Calandra T, Dhainaut JF, Gerlach H, 
Harvey M, Marini JJ, Marshall J, Ranieri M, Ramsay G, Sevransky J, Thompson BT, 
Campaign Guidelines Committee; American Association of Critical-Care Nurses; 
American College of Chest Physicians; American College of Emergency Physi-
cians; Canadian Critical Care Society; European Society of Clinical Microbiology 
and Infectious Diseases; European Society of Intensive Care Medicine; European 
Respiratory Society; International Sepsis Forum; Japanese Association for Acute 
Medicine; Japanese Society of Intensive Care Medicine; Society of Critical Care 
Medicine; Society of Hospital Medicine; Surgical Infection Society; World Feder-
aton of Societies of Intensive and Critical Care Medicine: Surviving Sepsis 
Campaign: International guidelines for management of severe sepsis and septic 
2. Spronk PE, Zandstra DF, Ince C, Bench to bedside review: Sepsis is a disease of 
the microcirculation. Crit Care 2004; 8:462–8
3. Sakr Y, Dubois MJ, De Backer D, Creteur J, Vincent JL. Persistent microcircu-
tulatory alterations are associated with organ failure and death in patients with 
14:561–6
prognostic value of muscle $\text{StO}_2$ in septic patients. Intensive Care Med 2007; 
33:1549–56
6. De Blasi RA, Palmisani S, Alampi D, Mercieri M, Romano R, Collini S, Pinto 
G. Microvascular dysfunction and skeletal muscle oxygenation assessed by phase-
modulation near-infrared spectroscopy in patients with septic shock. Intensive 
Care Med 2005; 31:1661–8
7. Calandra T, Cohen J. International Sepsis Forum Definition of Infection in 
the ICU Consensus Conference: The international sepsis forum consensus con-
33:1538–48
8. Spöhr F, Hettrich P, Bauer H, Haas U, Martin E, Büttiger BW: Comparison of 
two methods for enhanced continuous circulatory monitoring in patients with 
Score (APS II) based on a European/North American multicenter study. JAMA 
1993; 270:2957–63
Care 2000; 4:217–25
11. Michard F. Changes in arterial pressure during mechanical ventilation. 
Anesthesiology 2005; 103:419–28
12. Feissel M, Michard F, Faller JP, Teboul JL. The respiratory variation in 
inferior vena cava diameter as a guide to fluid therapy. Intensive Care Med 2004; 
30:1854–7
$\text{BP}$ induced by passive leg raising predict response to fluid loading in critically 
ill patients. Chest 2002; 121:1245–52
14. Bourgoin A, Leone M, Delmas A, Garnier F, Albanèse J, Martin C. Increas-
ing mean arterial pressure in patients with septic shock: Effects on oxygen 
15. Cui W, Kumar C, Chance B. Experimental study of migration depth for the 
photons measured at sample surface. Proc SPIE 1991; 1451:180–91
16. den Uil CA, Klijn E, Lagrand WK, Brugs JF, Ince C, Spronk PE, Simoons ML. 
The microcirculation in health and critical disease. Prog Cardiovasc Dis 2008; 
51:161–70
17. Cohn SM, Nathens AB, Moore FA, Rhee P, Puyana JC, Moore EE, Belizan 
GM: $\text{StO}_2$ in Trauma Patients Trial Investigators: Tissue oxygen saturation predicts 
the development of organ dysfunction during traumatic shock resuscitation. 
J Trauma 2007; 62:44–54
Stewart CJ, Hemphill C, Manley GT. Continuous muscle tissue oxygenation in 
critically injured patients: A prospective observational study. J Trauma 2006; 
61:780–8
ANESTHESIOLOGY REFLECTIONS

The Infant Lungmotor

During and after World War I, Chicago’s Life Saving Devices Company promoted its “Infant Lungmotor” for resuscitating young victims of drowning, smoke inhalation, and birth asphyxia. During such crises, an assistant would apply the device’s facemask to the apneic infant as the lead resuscitator would hand-pump air through two lengths of metal tubing connecting the distal end of the Lungmotor to the double-nippled mask. Air or oxygen volume delivered per “Lungmotor breath” could be reduced by inserting the attached piston-limiting pin into slots designated for “SMALL”- or “MEDIUM”-sized infants. By 1928, Yale physiologist Yandell Henderson would record in JAMA that these Lungmotors were “inevitably applied neither gently nor moderately.” (Copyright © the American Society of Anesthesiologists, Inc. This image appears in color in the Anesthesiology Reflections online collection available at www.anesthesiology.org.)

George S. Bause, M.D., M.P.H., Honorary Curator, ASA’s Wood Library-Museum of Anesthesiology, Park Ridge, Illinois, and Clinical Associate Professor, Case Western Reserve University, Cleveland, Ohio. UJYC@aol.com.