Simple Foot Restraint

To the Editor:—Some surgical procedures require the operating table to be tilted to one side. Under these circumstances, despite the use of a knee-strap, the patient's dependent lower leg may slide off the table, particularly if the patient is obese. The same can also occur without the table being tilted, if a wedge or inflatable device is placed under the patient's hip to produce a pelvic tilt as is done routinely during cesarean section.

In situations where we cannot be assured that both legs will securely remain in their intended position throughout surgery, we maintain proper leg position in the following manner: the end of a 11 cm wide Kerlix bandage (Kendall Company, Boston, MA) of appropriate length is doubled up to a length of about 60 cm, and a knot is tied to form a loop which is wide enough to easily slide around the foot to a position just above the external malleolus. For added safety, some padding (e.g., polyurethane foam pad) can be placed between the gauze and skin, particularly during operations of longer duration. The other end of the gauze is attached to the rail on the contralateral side of the operating table (fig. 1).

The use of tape instead of bandage material is simpler and cheaper, but causes some medial traction of the ankle. A 5 cm wide tape is applied to the dependent ankle and, beginning at the medial aspect of the heel, it is carried around the posterior and lateral aspects of the heel. Before continuing, a piece of gauze of appropriate size is placed over the anterior surface of the foot to protect the hair. The tape is then brought around the front of the foot and across the width of the table underneath the other foot to the contralateral side of the table, where it is attached to the side and undersurface of the table. In obese patients undergoing cesarean section, the foot restraint can be used in combination with the side brace described by Palmer et al. 1

I have used this technique with good success and no complications for many years.

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Measurement of the Longitudinal Distribution of Pulmonary Vascular Resistance from Pulmonary Artery Occlusion Pressure Profiles

To the Editor:—Collee et al. 2 have applied pulmonary artery occlusion pressure profile analysis to determine the longitudinal distribution of pulmonary vascular resistance in adult respiratory failure. The pressure profile after pulmonary artery occlusion is a biexponential decay. 2 Collee et al. analyzed this decay by analogy to an electrical circuit containing two resistors (arterial and venous) and two capacitors (arterial and capillary). Pulmonary capillary hydrostatic pressure (p_c) was estimated by extrapolation of the slow component of the biexpo-
Fig. 1. Two capacitor model of the pulmonary circulation. Arterial and venous resistance are represented by $R_a$ and $R_v$. Blood flow into the lung segment is denoted $I$. Pulmonary artery pressure ($p_a$) and pulmonary capillary pressure ($p_c$) are represented as circuit nodes. Arterial capacitance and capillary capacitance are denoted $C_a$ and $C_e$, respectively. Left atrial pressure is represented by LAP, and inflation of an exclusive balloon is represented by the opening of the switch which results in the decay of $p_c$ toward LAP.

ential decay. Unfortunately, this analysis is inconsistent with an exact solution which may be obtained by applying basic circuit theory to the circuit model shown in figure 1. The slow component of the biexponential decay does not simply represent the decay of pulmonary capillary pressure following pulmonary artery occlusion, because flow continues into the pulmonary capillary bed from the pulmonary arterial bed distal to the balloon. This flow occurs because the model includes an arterial capacitance containing pulmonary arterial blood volume which must discharge following occlusion. The biexponential decay following pulmonary artery occlusion is, therefore, not simply the sum of an exponential describing pressure decay over the arterial bed plus an exponential describing decay over the venous bed. Instead, the final biexponential decay is dependent upon the mathematical interactions among model components. It is incorrect to state, “The slower exponential represents the pulmonary capillary bed pressure discharging through the pulmonary venous resistance.” Rather, from the moment of occlusion, the arterial and capillary capacitors simultaneously discharge, resulting in a biexponential decay.

Mathematically, the experimentally observed biexponential decay may be expressed as $p_a(t) = e^{at} + h e^{bt} + \text{LAP}$. Collee et al. estimated $p_c$ from extrapolation of the slow component which corresponds to the formula $p_c = h + \text{LAP}$. In contrast, the correct solution obtained from circuit theory is $p_c = \frac{g + h}{g\beta + h\gamma} + \text{LAP}$. When the magnitudes of the time constants ($\beta, \gamma$) of the biexponential decay differ substantially, the two estimates of $p_c$ will be similar; however, when the magnitudes of the time constants are comparable, the value of $p_c$ predicted from circuit theory may differ substantially from the value obtained by extrapolation of the slow component. For example, in an intact dog, Holloway et al.² observed time constants of 1.2 s and 2.4 s, with mean pulmonary artery pressure of 12.8 mmHg and mean pulmonary venous pressure of 2.5 mmHg. Applying circuit theory, pulmonary capillary pressure is computed as 3.8 mmHg, which is 4.5 mmHg less than the value of 8.3 mmHg obtained by extrapolating the slow exponential. The ratio of the arterial component of pulmonary vascular resistance to the total pulmonary vascular resistance is 87% when computed from circuit theory, rather than 44% as obtained by extrapolation of the slow exponential. This example illustrates the general phenomenon that the method of extrapolating the slow component will tend to overestimate capillary pressure and underestimate the arterial component of pulmonary vascular resistance. Time constants of similar magnitude are frequently observed experimentally, and may be particularly common in states such as hypoxia, which preferentially increase precapillary resistance. In unilateral hypoxia, we have observed that hypoxia changes the longitudinal distribution of pulmonary vascular resistance and results in time constants which are similar in magnitude.⁴

Regional alveolar hypoxia is a common finding in respiratory failure, and undoubtedly was present in the patient population studied by Collee et al. They were unable to demonstrate a relationship between the severity of lung injury and the estimated longitudinal distribution of pulmonary vascular resistance. In contrast, a relationship between the severity of lung injury and total pulmonary vascular resistance has been shown.⁵ The failure of Collee et al. to demonstrate a relationship to the longitudinal distribution of pulmonary vascular resistance may be due to systematic error from the simple estimation technique, and different results might have been found had the analysis from circuit theory been used. We suggest that the method of extrapolation of the slow exponential decay component should be abandoned in favor of an exact analysis based upon circuit theory which properly accounts for the interactions of model elements.

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In Reply.—The electrical analogue model we used is intended as a clear simplification of the pulmonary circulation which usefully serves to illustrate why one can expect a bi-exponential decay of pressure in the pulmonary artery distal to an occluding Swan–Ganz catheter balloon. As they derive, the resulting trace is of the form \( P_a(t) = e^{-\alpha t} + e^{-\beta t} + P_{LAP} \). The question is whether or not the late part of this curve is significantly influenced by the rapid (\( \beta \)) exponential. As we discuss in the original text, the estimation of pulmonary capillary pressure by extrapolation of the late part of the bi-exponential curve back to the time of pulmonary artery occlusion is valid only if the time constants of the two exponential components of the trace are significantly different. To check this hypothesis, we built the electrical circuit used as the analogue model. We found accurate separation of the two exponentials was possible when the ratio of the rapid:slow exponential time constants was \( \leq 0.3 \). At ratios of \( > 0.3 \), a single exponential fit of the trace was possible.

The time constant of the rapid exponential component is difficult to quantify due to pressure trace artefacts produced by catheter movement early in wedging. As can be seen from our published trace (fig. 1 in our article), the time constant of the rapid phase is considerably shorter than 1 s, falling from 38 cm H\(_2\)O (= PAOP + 20 cm H\(_2\)O) to 28 cm H\(_2\)O (PAOP + 10 cm H\(_2\)O) in approximately 0.15 s, suggesting a time constant of approximately 0.2 s in this example. Holloway et al.'s work in dogs measured half times of 0.9 s and 2.8 s for the rapid and slow components of the biexponential pressure decay.

The relative values of pulmonary arterial capacitance (\( C_a \)) and pulmonary capillary capacitance (\( C_c \)) are not known in human acute respiratory failure, and the distribution of pulmonary vascular resistance between arterial (\( R_a \)) and venous (\( R_v \)) beds may be significantly different from that found in healthy lungs. The time constants of the rapid and slow components of the wedging pressure decay are predicted by the products \( C_a \cdot R_a \) and \( C_c \cdot R_c \), respectively. Drs. Siegel and Pearl correctly point out that these time constants are unknown, and that our data may, therefore, contain unpredictable inaccuracies. Because we were always able to easily identify separate fast and slow phases of the decay of pulmonary artery pressure following balloon inflation, we believe the inaccuracy is small. The technique certainly provides a closer estimate of pulmonary capillary filtration pressure than either mean pulmonary artery pressure or pulmonary artery occlusion pressure. The mathematical analysis suggested would provide an extremely accurate solution of a very inaccurate model of the pulmonary vascular tree. Moreover, the rapid exponential is difficult (if not impossible) to quantify in the clinical setting, making measurement of the variables in the formula a source of considerable error. We suggest that, while the technique we used undoubtedly contains unknown inaccuracies, it remains a useful approximation to the measurement of pulmonary capillary pressure. It would seem wrong to abandon it without further investigation of its merits.

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