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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 \( P_a(t) = e^{-2\beta} + e^{-\alpha} + LAP \). 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 to 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\(^2\) 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 \( C_c \cdot R_v \), 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.

GEORGE COLLEE, M.B., CH.B.
Department of Anesthesia
Massachusetts General Hospital
Boston, Massachusetts 02114

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