CORRESPONDENCE

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In Reply:—Pauca incorrectly claims we "concluded that thermoregulation and anesthesia produced the post-CPB [aortofemoral blood pressure gradient]." Our study was an investigation of factors that could contribute to this phenomenon. Rewarming during cardiopulmonary bypass often causes sweating.[1] Because forearm blood flow increases during sweating[2,3] and increased upper extremity flow will produce greater resistive losses along the brachial and radial arteries, we tested the hypothesis that thermoregulatory sweating is associated with a femoral-to-radial artery blood pressure difference (gradient). Our hypothesis was confirmed by the presence of increased upper extremity blood flow and a consistent 5-mmHg difference between radial and femoral artery mean pressures in volunteers during thermoregulatory sweating.[4] We therefore postulated that thermoregulatory factors contribute to observed aortoradial artery blood pressure differences in the postcardiopulmonary (CPB) bypass period. A conclusion about the role of thermoregulation in the production of the post-CPB aortoradial artery blood pressure difference can be made only after completion of a carefully planned and controlled study that adequately examines thermoregulatory responses in patients undergoing cardiopulmonary bypass.

Pauca argues that data obtained in young, healthy volunteers are not helpful in understanding physiologic responses in middle-aged and elderly patients undergoing cardiopulmonary bypass. We disagree. We studied young healthy volunteers to minimize complexity and to isolate the effects of thermoregulatory factors and anesthesia on blood pressure differences. Older surgical patients will have varying degrees of vascular disease and will have less compliant arteries. However, these conditions would likely increase the central-to-radial artery blood pressure differences during sweating because increased flow (due to distal vasodilation) through a fixed proximal resistance will augment the pressure gradient. The reported forearm blood flow measurements were performed exactly as described.[4] The hand was deliberately not excluded by a wrist tourniquet because we were interested in total forearm blood flow—including that traversing hand arteriovenous shunts. Nonetheless, we also measured forearm flow during hand compression, which comparably excludes distal flow (table 1). Hand compression during vasoconstriction (with minimal arteriovenous shunt flow) had no effect on forearm blood flow. As expected, hand compression decreased forearm blood flow when shunt flow was large. These interesting but irrelevant data do not, of course, alter the conclusions of our study.

Table 1. Forearm Blood Flow with and without Hand Compression

	Without (ml/100 ml)	With (ml/100 ml)	*P*
Vasoconstriction	7 ± 6	7 ± 5	0.21
Vasodilation	11 ± 5	8 ± 6	0.11
Mild sweating	15 ± 6	12 ± 7	0.07
Intense sweating	17 ± 9	13 ± 11	0.41
Cool-down	10 ± 9	8 ± 7	0.06
Propofol/nitrous oxide	14 ± 6	9 ± 6	0.02

Pauca incompletely describes the effect of heating on forearm blood flow. Cutaneous blood flow in the forearm will increase greatly if sweating is present.[2,3] The increase in flow during sweating results from active vasodilation in nonacral skin.[2,3] Pauca is thus incorrect when stating that the large increase in forearm blood flow during sweating was due to "failure to exclude the hand circulation." Induction of anesthesia increased forearm blood flow because propofol[5] and nitrous oxide[6] reduce the vasoconstriction threshold, thus increasing arteriovenous shunt flow.[7] It is therefore not surprising that hand compression during propofol/nitrous oxide decreased forearm blood flow.

Finally, Pauca concludes on theoretical grounds that our fingertip flow measurements were performed incorrectly and inaccurate. His first concern is that we positioned the venous occlusion cuff over the proximal rather than middle phalanx. However, the review that Pauca cites to support his position specifies that many investigators position the cuff proximally because there is less inflation artifact (an increase in fingertip volume resulting from blood pushed distally during cuff inflation).[8]

Pauca's other concern about our plethysmographic measurements is that we based flow on the linear phase of the volume *versus* time curves rather than uniformly using the first few seconds after venous cuff inflation. Proper interpretation of the volume *versus* time curves requires identification of the linear region and use of this portion to determine the slope. Always using the slope of the first few heart beats is obviously incorrect; the finger will not have begun to fill at low flows, whereas the finger volume will be saturated at high flows. Pauca illustrates this error in his figure: The slope indicated by his dashed line obviously reflects a period during which finger volume is saturated and no longer increasing linearly. A slope calculated from this dashed line would grossly underestimate flow, whereas the solid line (as in our original publication) provides the correct value.

In summary, our strain-gauge measurements of forearm blood flow were appropriate for our study questions, and our fingertip flow measurements were performed correctly. Our methods and results thus support our conclusions that thermoregulatory and anesthetic-induced alterations in upper extremity vascular tone and blood flow influence the difference between femoral and radial artery blood pressures.

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End-tidal Carbon Dioxide Monitoring May Help Diagnosis of H-Type Tracheoesophageal Fistula

To the Editor:—End-tidal carbon dioxide (ET_{CO^2}) monitoring can be used to detect air embolism, circuit disconnection, endotracheal tube kinking, and rebreathing.[1] Recently, capnography assisted in the diagnosis of a tracheoesophageal fistula.

A 1-month-old, 3.2-kg infant was admitted with a diagnosis of gastroesophageal reflux. Increased temperature, respiratory rate, and leukocyte count and choking and brief cyanotic spells after feeding suggested aspiration pneumonia. Medical history included uncorrected cleft lip and palate and patent ductus arteriosus, patent foramen ovale, bilateral SVC, and right atrial and ventricular enlargement.

The infant was brought to the operating room for a Nissen fundoplication. Rapid sequence induction and intubation were done without positive-pressure ventilation by mask. Ventilation was begun *via* a 3.0 oral endotracheal tube, secured with the tip 9 cm at the alveolar ridge.

A central venous catheter was inserted. A chest x-ray showed the tip of the endotracheal tube to be midway between clavicles and carina. The incision was made, and after entering the abdomen, the surgeon noted air in the stomach. An oral-gastric suction catheter (position confirmed by surgeon) briefly emptied the stomach, but the stomach would refill. Ventilation peak pressure was reduced to 15 cmH_2O out of concern that air leaked around the endotracheal tube, accumulated in the pharynx, and then moved down the esophagus into the stomach.

It was then noted that, when the oral-gastric tube was suctioned, the previously square ET_{CO^2} capnogram waveform changed to a reduced, rounded form (fig. 1). A presumption of tracheoesophageal fistula was made. This was confirmed by bronchoscopy and esophagoscopy, which showed an H-type connection about halfway between the anterior esophagus and posterior trachea.

We assumed that suctioning the oral-gastric tube drew air from the lungs, *via* the tracheoesophageal fistula, into the esophagus and stomach. The result was a reduction of expired carbon dioxide, shown by the changed ET_{CO^2} capnogram. Gastric dilitation may have been caused by inspiratory pressure and/or partial intubation of the fistula itself.[2]

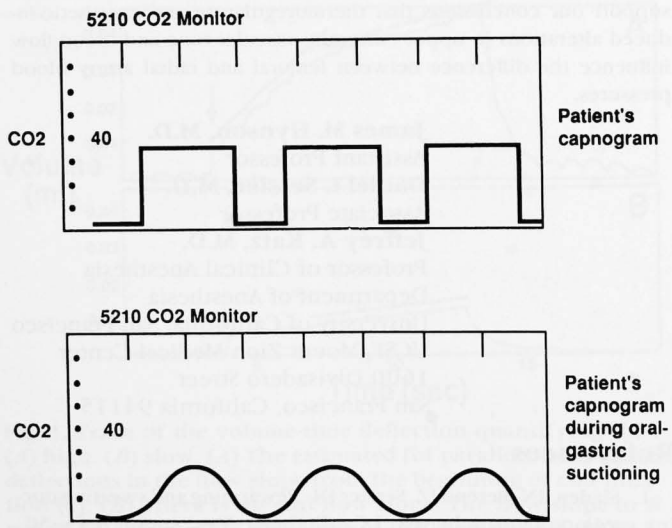

Fig. 1.