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REPLY

We thank you for your informative comments on the diving reflex. You noted no episodes of the diving reflex during cold ocular irrigation of the anterior chamber of the eye and we would like to respond to your comments.

The generation of any reflex is at best problematic and its occurrence is multifactorial. Although the diving reflex was not noted during cold saline anterior chamber ocular irrigation, it has never been described during anterior eye chamber irrigation. The circumstances described in your letter are different from our paper. The diving reflex classically has been described during cold ocular irrigation in the distribution of the fifth division of trigeminal nerve supplying the cornea, eyelid and the skin sensory areas surrounding the eye.¹ One also must question if clinical circumstances might inhibit the oculocardiac reflex. Topical ocular 0.75% bupivacaine was used, yet it is stated that the afferent impulses from the anterior chamber are unblocked. It is possible the bupivacaine has diffused over the time course of the surgery and partially or totally blocked the sensory input necessary to elicit the reflex. The innervation of the anterior chamber may also be different from that of the surrounding skin of the eye.² This skin sensory input triggered by cold solution may be needed to prompt the reflex. It is also common to use vagolytic drugs of the belladonna alkaloid family for mydriatics. Their use is not noted and would also block the reflex.

We believe the diving reflex does occur under general anaesthesia. However, we feel it is not a common occurrence but that when it occurs it is problematic. The diving reflex is well known in other branches of clinical medicine, primarily cardiology where the diving reflex is used to terminate supraventricular tachycardias in both children and adults.³⁻⁵ It is also known to cause bradycardia in awake humans through the parasympathetic nervous systems.^{6,7} The goal of our paper was to remind clinicians that the diving reflex may occur and why it may happen.

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Interleukin-6 and tumour necrosis factor during cardiopulmonary bypass

To the Editor:

Surgery stimulates the production of a variety of endogenous mediators that affect the response of the host to tissue injury. It has been reported that immunological function may be depressed more after cardiac surgery than after other surgical procedures.¹ Therefore, to assess further the effect of cardiopulmonary bypass on immunological function, we measured interleukin-6 and tumour necrosis factor in 14 patients (ages 6.1 ± 2.5 yr) scheduled for coronary artery bypass grafting. Plasma interleukin-6 and tumour necrosis factor concentrations were determined using the Human Interleukin 6 Elisa Kit and a highly sensitive sandwich enzyme immunoassay.^{2,3} Plasma interleukin-6 and tumour necrosis factor concentrations increased during cardiopulmonary bypass (42.1 ± 2.9 pg · ml⁻¹ to 71.1 ± 6.0 pg · ml⁻¹, $P < 0.05$ and 4.2 ± 0.4 pg · ml⁻¹ to 7.1 ± 0.5 pg · ml⁻¹, $P < 0.05$, respectively) compared to the presurgery baseline (repeated measures ANOVA). The maximum concentration of interleukin-6 occurred before skin closure (116.6 ± 8.3 pg · ml⁻¹, $P < 0.01$ compared with control). Tumour necrosis factor concentration was maximum on the first postoperative day (9.9 ± 0.8 pg · ml⁻¹, $P < 0.01$). Surgical injury induces both a local inflammatory reaction and stress responses involving the immune and haematopoietic systems. These responses are associated with increased production of a variety of endogenous mediators and collectively are called the acute phase response. Cytokine may be instrumental in orchestrating this acute phase response. The increase in IL-6 after tissue injury precedes changes in interleukin-1 or other acute phase proteins and induces a broad spectrum of alterations in other immunologic and endocrinologic factors. Cardiopulmonary bypass has been associated with an array of postoperative clinical problems including coagulopathy,

increased capillary permeability, fever and end-organ injury that could be related to disruption of immunology function. We demonstrated increases of IL-6 and NF concentrations during cardiopulmonary bypass. Although many factors could account for the increases the changes may reflex surgical and/or cardiopulmonary bypass-induced depression of immunity in these patients. But, in cardiac surgery, it should be kept in mind that many factors including contact activation by blood interfacing with non-endothelial extracorporeal circuits, hyperthermia during rewarming, and foreign antigen administration can also produce cytokin and inflammatory responses.

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Bleeding from the $S\bar{v}O_2$ monitoring port of PA catheter during cardiac surgery

To the Editor:

Although pulmonary artery (PA) catheter entrapment or failure is rare,¹ we report bleeding from the fibreoptic $S\bar{v}O_2$ port of a PA catheter due to surgical suture following catheter displacement which disabled the continuous monitoring of $S\bar{v}O_2$ during cardiopulmonary bypass.

After an 8 Fr balloon-tipped, flow-directed fibreoptic pulmonary artery catheter (OPTICATH™, model P7110-EP8-H, Abbott Laboratories) was connected to a saturation/cardiac output computer (Oximatrix™ 3 System, Abbott Laboratories), and *in vitro* calibration, the PA catheter was placed in a patient scheduled for

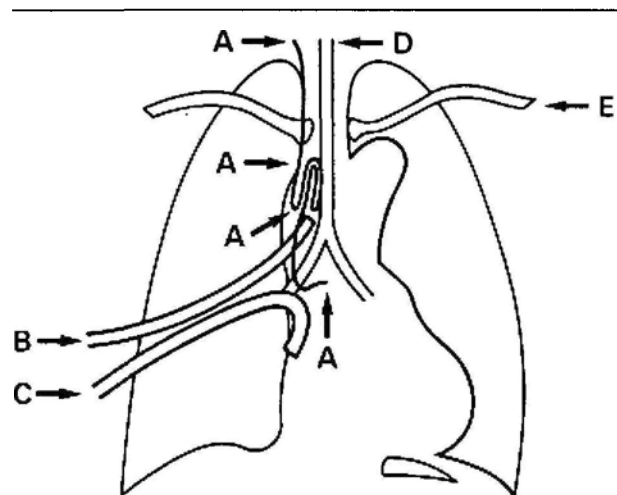


FIGURE 1 The slack PA catheter in the SVC. A, PA catheter; B, SVC cannula; C, inferior vena cava cannula; D, trachea; E, clavicle.

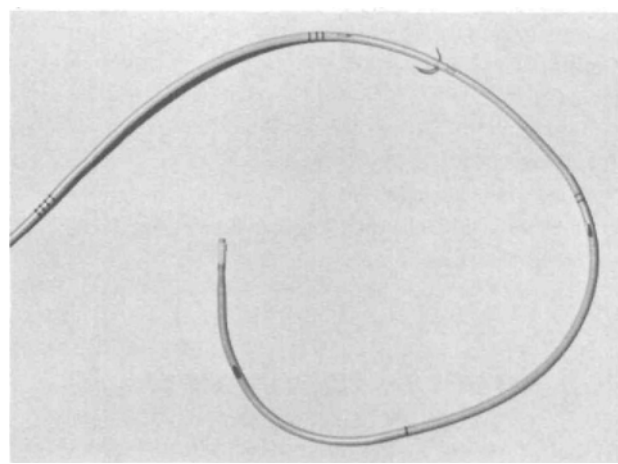


FIGURE 2 Distal portion of PA catheter after removal. A needle hole was situated at 26 cm from the catheter tip and communication with the extra-lumen of the catheter at 19 cm.

aortic valve replacement. Measurement of PA wedge pressure (PAWP) and continuous monitoring of $S\bar{v}O_2$ were possible. For cardiopulmonary bypass, the surgeon inserted a 34 Fr superior vena cava (SVC) cannula, which was fixed to the right atrial (RA) wall using a double-armed surgical suture. Following cannulation, the configuration of the PA tracings changed to a right ventricular waveform and we could not obtain PAWP. We verified the slack of the PA catheter in the SVC under fluoroscopy (Figure 1). As soon as extracorporeal circulation was instituted, the saturation/cardiac output computer system displayed a light intensity signal alert. Fifteen minutes later, we noticed blood leaking around the connection at the optical module from the fibreoptic