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COMMENT

Thank you for the opportunity to reply to Dr. Ghani's letter. The test Dr. Ghani describes points out again how important it is to check breathing circuits for proper function before each use.

I routinely visually inspect each circuit to assure that the blue inner tube does extend from the patient end connector to protrude slightly outside the circuit at the fresh gas connecting nipple. I then personally connect the circuit to the gas machine and perform the Pethick test and finally pressurize the circuit to 30 cm of water pressure for a few seconds to assure there are no significant leaks. This routine testing takes about 20 seconds. Perhaps now I will include the Ghani test and the Foex-Crampton Smith manoeuvre. Both tests are easily performed and give further assurance the inner tube of a Bain Breathing Circuit® is intact. The Foex-Crampton Smith manoeuvre also nicely checks the integrity of the hard circuitry of the gas machine from the connection at the fresh gas nipple of the circuit back to the rotameters. I would encourage all readers to look up the original publication on the Foex-Crampton Smith manoeuver, as it makes delightful reading!

The inner tube of the Bain Breathing Circuit is recessed at the patient end thus necessitating the use of a plunger from a 2 or 3 ml syringe to carry out the Ghani test or the Foex-Crampton Smith manoeuvre. The inner tube is recessed to prevent obstruction to exhalation by the elbow connector or the endotracheal tube connector. The minimal dead space is of no consequence even to the premature baby.

Finally I would like to emphasize that everyone must routinely carry out safety checks of any anaesthesia circuit before each use to assure its proper function.

James A. Bain MD FRCP(C)
Department of Anaesthesia
Victoria Hospital
London, Ontario

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Oxygen monitoring of bleomycin-treated patients

To the Editor:

Oxorn, *et al.*,[1] discussed an important aspect in the anaesthetic management of the surgical patient previously treated with bleomycin. Although our experience with these patients undergoing thoracotomy[2] suggests that they do tolerate higher FiO_2's than previously recognized,[3] like Oxorn *et al.*, it is still our practice to administer the lowest concentration of oxygen compatable with safe levels of arterial oxygenation.

Oxorn *et al.* described their experience with an indwelling in vivo arterial oxygen tension monitoring system. They detailed the potential hazards of intra-arterial cannulation as well as the very real shortcomings of other methods of oxygen monitoring. Their article failed to mention an accurate, non-invasive oxygen monitor that is currently available. We now use a pulse-oximeter (Nellcor, Hayward, CA) for continuous monitoring of oxygenation on our high risk patients. Unlike the less dependable ear oximeters, the Nellcor device uses a sensor that is easily wrapped around any pulsating artery. For convenience we use a finger. The monitor digitally displays arterial hemoglobin-oxygen saturation (SaO_2), and is extremely accurate over a wide range of hemodynamic conditions.[4] For bleomycin-treated patients, we use the lowest "safe" amount of supplemental oxygen as determined by reducing the FiO_2 until an SaO_2 of 90–95 per cent is reached. Non-invasive finger pulse oximetry is an ideal means of continuously monitoring patients at risk for hypoxemia or for oxygen toxicity such as the bleomycin-treated patient described by Oxorn.

Jay B. Brodsky MD
Mark S. Shulman MD
Department of Anesthesia
Stanford University School of Medicine
Stanford, California

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Hypertension associated with cardiopulmonary bypass

To the Editor:

In the recent article by Townsend *et al*[1] the authors conclude that the renin-angiotensin system is not the primary mediator of cardiopulmonary bypass associated hypertension, at least during fentanyl anaesthesia. In that study, as outlined in the method, all patients were anaesthetised with a single bolus of fentanyl and received halothane or enflurane if hypertension occured prior to cardiopulmonary bypass. After institutions of cardiopulmonary bypass no further anaesthestic was given. Recent studies[2,3] have indicated that fentanyl levels on cardiopulmonary bypass may fall to subtherapeutic levels very quickly. One study has demonstrated that this may be a result of binding of fentanyl to certain membrane oxygenators and siliconized tubing.[3] In the absence of any other anaesthetic agent, I question therefore whether CPB associated hypertension is hypertension associated with lack of anaesthesia.

I would suggest that any study which involved the use of fentanyl as a primary anaesthetic agent during cardiopulmonary bypass is subject to the possibility of patients having inadequate anaesthesia and massive sympathetic output may occur on that basis. Perhaps studies including fentanyl as a major component of anaesthesia during bypass should include drug levels as part of that study to demonstrate whether anaesthesia is adequate during the period of study.

Gerald V. Goresky MD FRCP(C)
Department of Anaesthesia
Alberta Children's Hospital
Calgary, Alberta

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REPLY

We are grateful for the opportunity to reply to Dr. Goresky's letter. The abstracts he quotes provide convincing evidence of the sequestration of fentanyl in the lungs and in the membrane oxygenator during cardiopulmonary bypass (CPB) and explain the substantial decrease of plasma fentanyl concentration that has been observed during CPB. We use bubble rather than membrane oxygenators during CPB and, in a previous study,[1] did not see any dramatic decrease in plasma fentanyl concentration with institution of CPB.

We agree that the most likely cause of the hypertension in our patients was an adrenergic response which, perhaps, could have been diminished by administration of additional anaesthesia. However, the purpose of our study was to determine whether the renin-angiotensin system also had a causative role in CPB-associated hypertension. Our results suggest it does not.

We dispute whether it is possible to determine the adequacy of anaesthesia by measuring plasma fentanyl concentrations. When fentanyl is administered as a continuous infusion there is a tendency for higher plasma levels to be associated with a reduction in the incidence of hypertension during CPB.[1] However, the relationship is inconsistent and unpredictable and we suspect,[2] like others,[3] that it is not possible to block the response to noxious stimuli completely, at least at fentanyl doses used in clinical practice.

G.E. Townsend MD FRCP(C)
J.E. Wynands MD FRCP(C)
D.G. Whalley MB FRCP(C)
D.R. Bevan MB MRCP FFARCS
Department of Anaesthesia
Royal Victoria Hospital
Montreal, Quebec

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