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REPLY

The specific issue addressed in the Refresher Course was the potential value of various monitors in detecting hypoxaemia during clinical anaesthesia. It is clear that the transcutaneous oxygen tension ($PtcO_2$) does not reflect absolute PaO_2, being anywhere from 30 to 130 per cent of PaO_2 values. Notwithstanding this marked variability, it has been suggested that $PtcO_2$ might be useful in detecting hypoxaemia by indicating PaO_2 trends. In a limited study of anaesthetized adults, we observed that the $PtcO_2$ did not consistently follow PaO_2 changes. From a larger study of patients and a review of the literature, Dr. Tremper has apparently reached the opposite conclusion.

Dr. Tremper's assessment appears to have been based upon significant correlation coefficients between $PtcO_2$ and PaO_2 values at normal and high PaO_2 levels. Ours was based upon the lack of reliability and the very long response times of $PtcO_2$ in following induced reductions of PaO_2. The different conclusions appear related in the first place to differences in data analysis, and specifically to the use of the correlation coefficient in assessing consistency of trend detection. For example, in Figure 2 of Dr. Tremper's original report[1] it appears that PaO_2 values measured every 15 minutes during the course of an anaesthetic increased or decreased ten different times. Since the $PtcO_2$ correlated significantly with these PaO_2 values, it was concluded that the $PtcO_2$ "trended with the PaO_2 values." However, the data presented in the Figure show clearly that with five of the ten changes in PaO_2, the $PtcO_2$ moved in the opposite direction. These inappropriate shifts of $PtcO_2$ cannot all be accounted for by changes in cardiovascular variables. Thus, a direct analysis of Dr. Tremper's data as well as our own, reveals that the $PtcO_2$ does not consistently follow PaO_2 trends. Furthermore, this analysis points out the danger of inferring consistency of $PtcO_2$ performance on the basis of correlation coefficients alone.

Before one accepts the $PtcO_2$ as an indicator of peripheral tissue oxygenation, it is important to remember that it is not a physiological oxygen tension but one which is induced artificially at the surface of the skin, and that it is critically dependent upon and influenced by the factors used to induce it. These include (1) local heating of the skin which vasodilates the circulation and shifts the oxyhaemoglobin dissociation curve to the right (thereby artificially increasing blood PO_2) and (2) the addition of a considerable resistance to oxygen diffusion at the surface of the skin (thereby artificially increasing PO_2 values in the skin). Any $PtcO_2$ value represents a complicated and variable interaction between the effects of these inducing factors and the effects of the principal physiological influences on $PtcO_2$, i.e., the PaO_2, the cutaneous oxygen consumption and the rate of local cutaneous perfusion which is in itself affected by PaO_2. Hence, the varying and unpredictable relationships between $PtcO_2$ and PaO_2. It seems to me that if $PtcO_2$ is to become a meaningful indicator of tissue oxygenation and/or a reliable index of change of PaO_2 or perfusion, some method will have to be found to control or independently assess the influence of the added variables.

For the moment, modern ear oximeters are far more direct, accurate, rapidly responsive and reliable detectors of hypoxaemia in anaesthetized adults.

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Recurrence of bronchial asthma after adrenalectomy for phaeochromocytoma

To the Editor:

Although it is well known that plasma epinephrine plays an important role in the relaxation of bronchial smooth muscles,[1–3] there has been only one case report of asthmatic attacks recurring after removal of a phaeochromocytoma in an asthmatic.[4] We wish to report such an episode which occurred

in an asthmatic patient after adrenalectomy for a phaeochromocytoma producing predominantly epinephrine.

A 62-year-old, 60 kg, 162 cm, male with a history of bronchial asthma was scheduled for removal of a phaeochromocytoma. He had suffered from frequent asthmatic attacks since the age of three years and had in the past been treated with nebulized or subcutaneous sympathomimetics. At age thirty, he had an episode of status asthmaticus, requiring mechanical ventilation. Since that episode the severe attacks had not recurred and he had required no anti-asthma medications in the last 25 years.

At age fifty-four the first paroxysmal hypertensive attack with encephalopathy occurred. Since then, paroxysmal hypertension, with systolic blood pressure exceeding 200 mmHg, occurred two or three times a year. He had otherwise been asymptomatic and normotensive. The diagnosis of phaeochromocytoma was made by the history of paroxysmal hypertension, determination of plasma and urinary catecholamine levels, a diabetic pattern glucose tolerance test, and computer tomographs of the adrenal regions.

Preoperative plasma epinephrine and norepinephrine concentrations in a normotensive period were 820 $pg \cdot ml^{-1}$ and 816 $pg \cdot ml^{-1}$, respectively (normal range: epinephrine $<120\ pg \cdot ml^{-1}$, norepinephrine 40–350 $pg\ ml^{-1}$). One week volume expansion therapy with prazosin (1–3 $mg \cdot day^{-1}$) and whole blood transfusion (1200 ml) was carried out. No bronchospasm was present before surgery.

Premedication consisted of hydroxyzine 100 mg and atropine 0.5 mg IM. For epidural anaesthesia, 1.5 per cent lidocaine without epinephrine was given through a thoracic epidural catheter. After the establishment of epidural anaesthesia, general anaesthesia was induced with thiamylal 500 mg, IV, and the trachea was intubated after administration of succinylcholine 60 mg, IV. Anaesthesia was maintained with modified neuroleptanaesthesia (fentanyl 18 $\mu g \cdot kg^{-1}$, diazepam 10 mg and 66 per cent nitrous oxide in oxygen). Intraoperative hypertensive crises were treated successfully with intravenous administration of phentolamine and nitroglycerin. The plasma epinephrine and norepinephrine concentrations increased to 23,700 $pg \cdot ml^{-1}$ and 9,270 $pg \cdot ml^{-1}$, respectively during tumour manipulation. After the conclusion of the right adrenalectomy, the patient was awake and was extubated. No wheezing was noted during the operation.

On the first postoperative day, the patient complained of respiratory distress and expiratory wheezing was heard over the entire chest. The pHa was 7.45, $PaCO_2$ 42 mmHg, PaO_2 71 mmHg, BE 4.4 $mEq \cdot L^{-1}$ (FiO_2 0.21). He was given nebulized salbutamol ($15\ mg \cdot day^{-1}$) and orciprenaline ($60\ mg \cdot day^{-1}$) orally. The asthmatic attack improved only slightly. The plasma epinephrine and norepinephrine levels on the first postoperative day were markedly decreased, to $155\ pg \cdot ml^{-1}$ and $359\ pg \cdot ml^{-1}$, respectively. Although the patient had no symptoms on the second postoperative day, expiratory wheezing was still present on examination, and the arterial blood gas analysis revealed hypercapnia ($PaCO_2$ 46 mmHg). Expiratory wheezing was heard occasionally during the subsequent hospital days.

There are many nonspecific physical, chemical and pharmacologic stimuli such as histamine, cholinergic agonists, prostaglandin $F_2\alpha$, irritant gases, chemically inert dust, and cold air[3] which could initiate bronchoconstriction in asthmatics perioperatively. In our patient, it is also possible that effects of thiamylal, fentanyl, sympathectomy induced by thoracic epidural anaesthesia and the presence of a tracheal tube during light general anaesthesia might have caused bronchoconstriction during anaesthesia. However, we did not detect any bronchospasm the first postoperative day. Therefore, we suspect the sudden decrease in circulating epinephrine triggered the recurrence of bronchial asthama in this patient.

Epinephrine is a potent bronchial smooth muscle dilator. It has been shown in dogs that sympathetic relaxation of the airways is reduced more than 75 per cent after adrenalectomy, indicating that the physiologic sympathetic relaxation of airways occurs predominantly from circulating epinephrine.[1]

The patient's plasma epinephrine concentration decreased markedly after the removal of the phaeochromocytoma, to near the upper limit of the normal range on the first postoperative day. This rapid reduction might have contributed to the recurrence of the asthmatic attacks. Although the plasma epinephrine level was normal, beta-adrenergic dysfunction may have been present due to the preceding chronic excess plasma epinephrine. Receptor down-regulation or other causes of desensitization or tachyphylaxis to beta-stimulants[5]

likely developed, since the patient had been exposed to excess epinephrine for a long time. It has been shown that cyclic AMP increase due to epinephrine administration is impaired in asthmatics. Asthmatic patients have decreased beta-adrenergic receptor "availability" and thus beta-adrenergic dysfunction.[6,7] Furthermore, the withdrawal of the effect of prazosin which was given preoperatively, may trigger the recurrence of asthma, because alpha-blockade has been shown to increase cyclic AMP in asthmatic patients[8] and to inhibit the bronchoconstriction by alpha-agonists.[9]

In summary, since adrenal secretion of epinephrine plays an important role in the relaxation of bronchial smooth muscles, we should keep in mind that episodes of bronchial constriction can occur after adrenalectomy in asymptomatic asthmatic patients.

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Abnormal ECG pattern during battery operation of Mennen model 741 ECG monitor

To the Editor:

We wish to report a problem which occurred during our use of a recently acquired Mennen model 741 ECG Monitor (Mennen Medical Inc., Clarence, New York). During battery operation the monitor produced an abnormal wave form which incorrectly suggested a sudden change in the patient's status.

The monitor was being used during a general anaesthetic for hysterectomy in an ASA physical status 1 patient. The ECG indicated normal sinus rhythm for approximately one hour until it suddenly began to indicate an abnormal ECG pattern with widened QRS complexes and deep inverted ST segments, suggestive of ischaemia (Figure). The patient's clinical status appeared stable and satisfactory. Heart rate was 80 bpm and blood pressure 120/70 mmHg, both unchanged from previous values during the normal ECG pattern. A careful search for other factors which might lead to the abnormal ECG pattern proved negative.

After about 20 minutes it was realized that the electrical socket into which the monitor had been plugged did not have a power supply, and that therefore the monitor had been operating, from the outset, from its battery supply. The monitor was then plugged into an active electrical socket, which