the expiratory valve to malfunction and so obstruct expiration (see Figs. 1 a & b). After the correction of this problem, EEP normalised and the patient was able to be weaned rapidly from her infusions and made an uneventful recovery.

We present this case to emphasise a useful clinical adage: "all that wheezes may not be asthma". In this case the severe expiratory obstruction was assumed to be due to the patient's underlying disease process, resulting in the unnecessary use of bronchodilator therapy, sedation and mechanical ventilation. Anaesthetic practice teaches the trainee always first to "check the tube" for mechanical causes of high inflation pressures before assuming it is due to bronchoconstriction, a principle that applies equally to patients in the ICU. Initially our patient undeniably had severe status asthmaticus and because the clinical findings and past history supported this diagnosis, other possible causes for the markedly elevated airway pressures in this patient were not considered at first. The initial recording of an EEP of 30 cm H_2O, which was extraordinarily high, was assumed to be due to the ongoing severe bronchospasm and air trapping, an erroneous assumption in retrospect, which contributed to the delay in diagnosis of the faulty respiratory valve. A false sense of security was also engendered by the drop in the EEP to 10 cm H_2O with a reduction of the ventilator rate. With attempted increases in the rate, the EEP once again climbed to very high levels which prompted a subsequent rate reduction, however a full and thorough search for extrinsic causes that could have accounted for this phenomenon should have been carried out at this stage, but was not. On day three of the patient's course there was an obvious disparity between the findings on clinical examination and the pressures being recorded by the ventilator, and this led to discovery of the incorrectly seated expiratory valve.

We commend to readers always to check and, if necessary, recheck their equipment at an early stage, especially in patients with poorly compliant lungs whose clinical course may not be straightforward and, above all, treat the patient not "the numbers". Elevated airway pressures do not always mean the patient has underlying bronchospasm. Marked elevation of the EEP such as was noted in this case always requires further prompt assessment and investigation in any intensive care patient.

We emphasise that this problem arose due to a technical error in the assembly of the expiratory channel and was not a ventilator fault per se. It is stated clearly in the Servo 300 operating manual that on assembly of the expiratory channel the expiratory valve tube must not be bent or twisted [1]. Discussion with the Siemens Servo 300 distributor in New Zealand and review of the published literature showed that a malfunction such as this has not been previously reported with this particular ventilator.

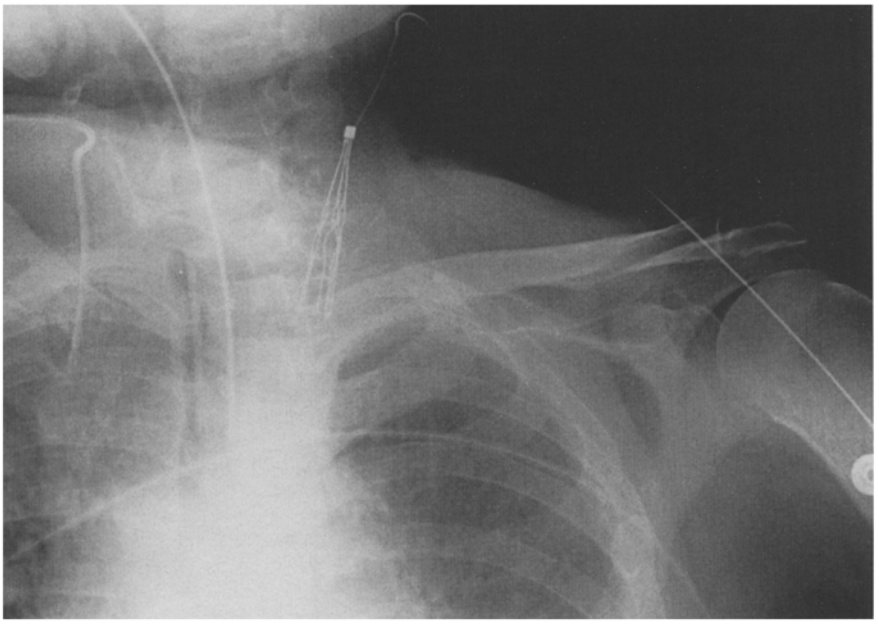

Fig. 1 Chest X-ray film (magnification) showing the unwound guidewire entangled in the Greenfield filter in the left internal jugular vein

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J. R. Gowardman (✉) · J. Torrance
Intensive Care Unit, Wellington Hospital, Riddiford Street, Private Bag 7902, Wellington South, New Zealand
FAX: +64 (4) 3855962
e-mail: wshsjg@mash.wnhealth.co.nz

A. Argiris
J. Rademaker
M. Mahmud

Dislodgment of an inferior vena cava filter to the internal jugular vein

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Sir: Complications associated with inferior vena cava (IVC) filters include insertion-associated complications, such as misplacement, air embolism, hematoma, and pneumothorax, insertion-site deep venous thrombosis, penetration or obstruction of the IVC, and filter migration [1]. There have been case reports of migration of the IVC filter into the right atrium or pulmonary artery, which may lead to life threatening complications [1]. We recently encountered an intriguing complication when an IVC filter was dislodged to the internal jugular vein due to guidewire manipulation.

A 84-year-old man was admitted to the Intensive Care Unit because of massive upper gastrointestinal bleeding. His stay was complicated by thrombosis of the left superficial femoral vein, diagnosed by Doppler sonography. A stainless steel Greenfield filter was inserted percutane-

ously into the infrarenal IVC the same day. An attempt to place a left internal jugular vein catheter was made 5 days later. During withdrawal of the 60 cm-long guidewire, significant resistance was encountered. A chest X-ray film (Fig. 1) showed relocation of the IVC filter to the internal jugular vein. The guidewire remained entangled in the filter and was unwound at its end point. No damage to the filter was evident. An attempt to recover the dislodged filter and the guidewire via an incision over the left internal jugular vein was unsuccessful and finally the guidewire was sutured to the vein wall. The patient eventually died from unrelated complications.

Our case illustrates a very rare but preventable complication of central venous line insertion in patients with IVC filters. Previous reports have described dislodgment of IVC filters due to guidewire manipulation to the right atrium [2, 3] and brachiocephalic vein [4]. It appears that the J-tipped guidewire can "hook" the filter through its central hole and entangle in it. The fact that the incidence occurred early after IVC filter placement, before endothelization ensues (usually within 2–4 weeks) [4], probably facilitated the dislodgment of the filter. Measurement of the distance between the site of insertion and the IVC on the patient's skin prior to central venous catheter placement is recommended as a practical way to avoid this complication.

A. Argiris (✉)[1] · J. Rademaker · M. Mahmud
Department of Medicine, Beth Israel Medical Center, First Avenue at 16th Street, New York, NY 10003, U.S.A.
Present address:
[1] Department of Internal Medicine, Yale-New Haven Medical Center, New Haven, CT 06520, USA

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G. Rajan
J.W. Sleigh

Lymphocyte counts and the development of nosocomial sepsis

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Sir: A generalised depression in immune function has been noted following major surgery and trauma[1] and is associated with the development of infection and sepsis. An important indicator of this 'anergy' is the reduction in numbers of circulating lymphocytes. Gennari and others have found in postoperative patients that most of the lymphopenia is explained by a reduction in the CD3 subset (T-cells) [2, 3]. We conducted a study to determine if the total lymphocyte count was related to the subsequent development of nosocomial infection in patients from a general intensive care unit (ICU).

A review of the data of 76 adult patients (55 male) requiring 5 or more days of intensive care treatment during 1996 was carried out. Demographic information, the total white cell count, the lymphocyte count, antibiotic use and positive bacteriological cultures were noted daily. The standard definition for the diagnosis of sepsis was used [4]. The admitting diagnosis was post-trauma in 21 of the patients, postoperative in 26, and medical in 29. The mean age (and lymphocyte count) of the patients for each group was 39.5 years (1.2 cells/nl) for post-trauma, 65.1 years (1.2 cells/nl) for postoperative, and 48.3 years (1.1 cells/nl) for the medical group. In each diagnostic group a similar proportion (34–38 %) of patients developed the criteria for sepsis.

We arbitrarily divided the periods when the patients were free of sepsis into 3-day blocks. If the patient developed sepsis on the last day of the block, or in the subsequent 1–2 days after the block; that particular block was designated as being associated with the development of a septic episode. If no sepsis developed, that block was considered free of sepsis. We then compared the mean lymphocyte counts for septic-associated blocks, with those when no sepsis developed. As shown in Table 1, a severe lymphopenia of 3 days duration was associated with a greatly increased chance of developing an infection ($p < 0.01$, Chi-squared test). If the lymphocyte count was above 1.5 cells/nl, the probability of developing a septic episode was only 20 %. The significance of this effect persisted even when additional other possible confounding factors (age, APACHE II score, diagnosis, antibiotic treatment and length of stay) were included in a multivariate analysis (multiple logistic regression).

Table 1 Comparison of probability of developing sepsis compared with lymphocyte count. (The numbers in brackets are the number of sepsis-associated 3-day blocks divided by the total number of blocks.)

Lymphocyte count (cells/nl)	Percentage of patients becoming septic
0–0.8	53 % (51/97)
Greater than 0.8	29 % (51/172)

We concluded that a decreased lymphocyte count of at least 3 days duration is associated with a greatly increased risk of the development of nosocomial sepsis in intensive care patients. This raises questions about the possibility of using prophylactic antibiotic treatment, or even drug therapy to stimulate T-cells, in the prevention of nosocomial sepsis.

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G. Rajan · J.W. Sleigh (✉)
Intensive Care Unit, Waikato Hospital, Hamilton, New Zealand
FAX: + 64 (7) 839 8761
e-mail: sleighj@hwl.co.nz