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Correspondence

Pulmonary artery catheterization and mortality in critically ill patients

Editor—We read with interest the paper by Murdoch and colleagues.[1] We would like to draw attention to problems with the study design which render the results and conclusions unreliable, but which were not mentioned in the accompanying editorial.[2]

When studying the effects of any medical intervention, the outcomes for patients who receive the intervention are compared to a control group. Variables measured on patients in both groups prior to the start of treatment (and hence which are unaffected by treatment) are termed covariates. For example these may be age, sex, diagnosis, or severity of illness. The main reason for randomly assigning treatment in a randomized controlled trial is to balance covariates between the treated and control groups.[3] In an observational study such as that performed by Murdoch and colleagues, where treatments are not assigned at random, covariates may not be balanced between the intervention and control groups and this needs to be adjusted for during the analysis stage. For example, if an intervention is performed more frequently in patients who have a high probability of death than in those who have a low probability of death, an unadjusted comparison of deaths in those receiving and those not receiving the intervention will be misguided, as the higher death rate is likely to be incorrectly attributed to the intervention. This is clearly demonstrated in early retrospective studies of pulmonary artery catheterization (PAC) in acute myocardial infarction where catheters were predominantly inserted in those patients with congestive heart failure or cardiogenic shock, with the inevitable result that the patients with pulmonary artery catheters had a higher mortality.[4] The propensity score is an attempt to adjust for an imbalance between covariates in the intervention and control groups. It is calculated as the probability that a subject will receive the intervention, given the known set of covariates for that subject. Estimating this probability allows the creation of a 'quasi-randomized experiment'.[5] In other words, if two subjects are found who have the same propensity score, and one of them received the intervention but the other did not, then they can be considered to have been randomly assigned to each group (i.e. they had an equal likelihood of getting the intervention or being a control), and the analysis can proceed. In contrast to randomization which balances both known and unknown covariates, propensity scoring only balances known intervention that have been used during its calculation. If other (unused) covariates exist which influence the decision to provide an intervention, the groups may not be balanced for these. If these covariates are independently related to outcome, results obtained after adjustment using this propensity score will be incorrect. It is therefore vital to include in the calculation of the propensity score all covariates that are related to both the provision of the intervention and also to outcome.

From the above it can be seen that there are three problems with the analysis used by Murdoch and colleagues. First, little attempt was made to investigate if there were other covariates related both to the decision to use PAC and also to the outcome. In the original study of PAC using propensity scores, Connors and colleagues[6] investigated which covariates had most influence on clinicians' decisions to use PACs by asking seven different clinicians to list the factors involved in the decision, and checking this list with a further 13 independent clinicians. All these covariates were used in the construction of their propensity score. In addition, Connors carefully checked for the magnitude that missing covariates would have to have to alter the result by a defined amount using two statistical techniques. The study by Murdoch used an entirely different method. The covariates entered into the logistic regression analysis to determine the propensity score were apparently selected solely on the basis that they were available in the database and some covariates that self-evidently should have been included (e.g. diagnostic group) were not. No checks were made for the effects of missing covariates. It is entirely possible that the Leeds database simply does not contain enough information to produce an accurate propensity score based on all the pertinent covariates.

Second, Murdoch used variables from both before and after PAC to construct the propensity score. The value of some variables recorded following PAC will almost certainly be dependent on the catheter being present. If vasoactive drug use prior to PAC use influenced the decision to provide PAC then these covariates should be used in the propensity score. However, if the decision to use these drugs was made on the basis of readings from a pulmonary artery catheter, they become a surrogate marker of the presence of a pulmonary artery catheter. When surrogate markers are included in regression equations to predict the use of the catheter the equations are, of course, highly but spuriously predictive. Thus the calculation of propensity score to predict an intervention must not contain variables that depend on the intervention. Rosenbaum and Rubin, in one of the core papers on propensity scores, clearly state that measurements should be made prior to treatment assignment.[7]

In statistical parlance, the reason measurements must be made prior to treatment assignment is as follows: the central question is to estimate the average effect on the outcome variable Y of treatment 1 *versus* treatment 2 for the relevant population. Considering the relevant population as a random sample of the entire population under scrutiny, the estimate of this effect is the average value of the appropriate difference between the conditional expectation of the outcome variable Y on the set of predictors in the two treatment groups. Therefore the predictors used to make assignment decisions must be recorded before the intervention takes place, then the expectation of outcome in each group is estimated (conditional on the predictors in each treatment group). Finally, the average difference between the estimated conditional expectations over the estimated distribution of the predictors is computed. These three steps must be followed carefully to estimate causal effects of an intervention.

Third, a more general point is that non-randomized studies rely on natural variations in patient treatment, in other words, that similar patients are sometimes given an intervention but at other times not. If all patients with the same condition are treated in the same way comparison between a treated and control group cannot be made because the two groups do not co-exist. If PAC was 'protocol driven' in Leeds, as suggested in the manuscript, variations between clinicians would be minimal, and it would not be possible to define the two groups. Lack of natural variation is suggested by the fact that norepinephrine, epinephrine and dobutamine were virtually never used in the control group. This might explain why Murdoch chose not to perform the case-by-case matching analysis undertaken by Connors; with a very skewed distribution of covariates between the control and treatment group it may be very difficult to match patients. A judgement on how much treatment variation existed in Murdoch's data could be made by comparing the number of patients who received a PAC and the number who did not receive a PAC in

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each quintile or other subdivision of the propensity score, though these figures were not provided. Murdoch and colleagues do state that there was no significant difference between the covariates in each quintile except the lowest, though this may have been due to the small number of subjects in each group leading to wide confidence intervals.

We suggest that the majority of conclusions in the paper, and the detailed analysis of the possible reasons for the difference between Murdoch's results and those of Connors in the accompanying editorial, are based on results that are fundamentally flawed and sadly add little to the ongoing debate about pulmonary artery catheters. In particular, the paper provides no supportive evidence for the statement that a randomized controlled trial of PAC would have insufficient power to demonstrate an effect on mortality even with very large numbers of patients. We believe that a randomized trial of PAC is imperative to resolve some of the current uncertainties surrounding their use.

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Editor—The letter by Young and colleagues raises three questions regarding the statistical methods we used in our paper[1] examining the effect of pulmonary artery catheter use on mortality in intensive care patients. We welcome the opportunity to respond and will take their points in turn.

The first point is in regard to choice of variables used in calculating our propensity score. It is true that we did not seek expert opinion in determining factors influencing the clinicians' decision making process with regard to insertion of a PA line. The validity of this technique—asking 'expert' opinion to identify variables which determine practice is questionable—variables are often suggested which in real life are not used. Our database was constructed prospectively to collect data which were determined locally as important in determining patient outcome and in describing haemodynamic manipulation including pulmonary artery catheterization. Within this, our choice of variables to create a propensity score is strongly supported by the area under the curve of the ROC plot. This suggested a more predictive score than that obtained by Connors and colleagues.[2] We agree that there may be covariates missing from our analysis, but in view of the high AUC the effect of such missing covariates is unlikely to be much greater than those in the Connors study. Since the publication of the Connors paper, better approaches to the assessment of the sensitivity of regression results to unmeasured confounders have been developed. Lin and colleagues have shown that the exposure effect and the effects of measured and unmeasured confounders can be formulated through a regression model, allowing inferences about the true exposure effect by making simple adjustments to the point interval estimates.[3] Application of this approach to the Connors study led Lin to recalculate the Connors point estimate for the odds ratio for mortality with a PA catheter as close to unity, and possibly as low as 0.8, a finding very close to the results we have published in our study. Moreover, Connors went on to estimate the magnitude of the effect of a potential confounder on risk of death or chance of allocation to PA catheterization as six-fold before a relative risk of death of 1.0 could be misrepresented as 1.21. However, Lin has pointed out—and Connors confirmed—that the Connors group inadvertently described the odds ratio of death as the probability of death. This led them to overestimate substantially the size of an unmeasured covariate needed to misrepresent a beneficial or zero effect of PA catheterization as an adverse effect, bringing the likely effects into line with the Lin calculation above. This potential effect of small unmeasured covariates may similarly apply to our study.

Diagnostic group was available in our database, but was not included in the propensity score model. This was because diagnosis was unimportant in determining catheter use in comparison with physiological derangement. A single diagnosis can encompass a wide degree of patient illness, while simple diagnostic categories fail to represent the real range of pathophysiological disturbance. By contrast, analysis of Project Impact data has shown that in American intensive care units organizational characteristics are highly significant variables regarding PA catheter use.[4] The presence of full time ICU staff is associated with a two-thirds decrease in catheter use OR 0.36 (95% CI 0.28–0.45); this variable is not included in the Connors study.

The second point raised relates to the use of inotropes to construct the propensity score. We accept that the PA catheter is likely to have influenced the choice of inotrope, and so individual inotropes should not have been included in the model. We are grateful to Young for pointing out this error. We had originally intended analysing only whether or not any inotrope had been used (as a single binary variable) as on our unit inotrope resuscitation is commenced prior to PA catheter insertion and regard as a primary indication for a PA catheter. Recalculating the propensity score with inotropes as a single binary variable results in inotrope use being excluded from the propensity score, but otherwise has little effect on the model, area under the ROC plot, propensity score, or PA catheter predictions of mortality.

The third point raised relates to lack of natural variation between the two groups. Young correctly points out that our 'protocol driven' approach results in a skewed distribution of our propensity score between those receiving and not receiving a PA catheter. Distribution of the number of patients in each quintile is as follows:

quintile 1	PA=44	no PA=751,
quintile 2	PA=155	no PA=627,
quintile 3	PA=321	no PA=464,
quintile 4	PA=588	no PA=205,
quintile 5	PA=726	no PA=61

The proportion of patients in each quintile has similar covariate distribution. This we believe is due to the predictive nature of the propensity score in correctly identifying PA catheter insertion and minimal random treatment—a less skewed distribution would only be achieved if there were a greater random effect in the decision to insert a PA catheter and would undermine a consensus view on decisions for their insertion. This skewedness is not extreme, and is in fact very similar to that in an example by Rosenbaum and Rubin[5] (treatment of coronary artery stenosis shows higher propensity score in the surgical population compared to the medical population) which they deemed 'acceptable'.

Finally, we did not state in our paper that a randomized study should not take place; merely that it would be difficult to carry out and unlikely in our opinion to 'answer' the debate.

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Lymphatic drainage of the thoracic paravertebral space

Editor—Karmakar and colleagues[1] are the latest to report contralateral spread following a thoracic paravertebral injection to relieve the pain of fractured ribs. Injection of bupivacaine 20 ml into a thoracic paravertebral catheter produced an ipsilateral block extending over seven thoracic segments. Iopamidol, a radio-opaque contrast, was then injected into the catheter to confirm placement. Contrast was noted in the ipsilateral paravertebral space and demonstrably spread across the mid-line at T6, at approximately the level of the tip of the catheter. No corresponding contralateral sensory blockade was found nor was there any haemodynamic change, suggesting that spread was not via the epidural space.

Large molecule, water-soluble, radio-opaque contrast media, like Iopamidol, are scavenged by the lymphatic system. The paravertebral space forms a watershed for lymphatic drainage. The bulk is to local nodes and then to tributaries of the thoracic duct. The latter are inconsistent and form a plexiform network lying anterior and lateral to the vertebral bodies.[2] Laterally, extrapleural material is scavenged by the lymphatics of the intercostal space. Some lymphatics from the subpleura join bronchial lymphatics. This may be particularly relevant as spread tends to be from the left hemithorax into the right hemithorax. Classical studies of the movement of bronchogenic tumour cells have shown spread from the left lung across the mid-line through lymphatic bridges which are relatively constant, one of which, notably, is at the T6 level.[3]

Though there is usually little delay in the process of lymphatic drainage of contrast, it is not difficult to envisage a situation of the usual drainage systems and paths of least resistance being disturbed by a primary process such as trauma,[1 4] or a pathological process such as infection and tumour.

We agree that spread of local anaesthetic is likely to have occurred through the lax tissues of the prevertebral fascia as demonstrated in various cadaver studies,[5] but feel that it should not be presumed that contrast media *in vivo* will mimic the spread of local anaesthetic and that a radiological artefact produced by the scavenging of the contrast by the lymphatic system cannot be discounted.

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Acknowledgement

We are grateful to Dr Thomas F Molnar, Head of Department Thoracic Surgery, University Medical School of Pécs, Hungary for information and reference about the spread of malignant cells.

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Editor—Thank you for the opportunity to reply to Nel, Shanahan and Conacher's letter in which they raise the question that the contralateral spread of contrast seen after the paravertebral injection in our patient[1] could have been an artefact produced by scavenging of contrast by the lymphatic system. The correspondents have raised an interesting question, which requires discussion; to our knowledge it has never been addressed in relation to radiocontrast studies in regional anaesthesia.

We agree that the thoracic paravertebral space forms a watershed for lymphatic drainage, and lymphatic vessels on either side of the vertebra anastomose with each other and those from the contralateral side to form a lymphatic plexus along the lateral and anterior surface of the vertebrae.[2] In addition, the subcarinal lymph nodes act as a lymphatic bridge between the two hemithorax providing a pathway for contralateral spread of malignant cells from the left lung.[3]

Any foreign material, contrast in our case, injected interstitially may be taken up by the lymphatic capillaries

after which it is transported with the lymph to the regional lymph nodes and phagocytized. The exact mechanism involved in uptake by the lymphatic capillaries is not known but passage through the endothelial junctions and/or pinocytosis through the cells are thought to be involved,[4] and depend on the size and the number of particles injected.[4] Small diameter particles (less than a few nanometers) are mostly exchanged through the blood capillaries; larger particles (diameter, a few tens of nanometers) are absorbed by the lymphatic vessels; and particles which are hundreds of nanometers in diameter are trapped in the interstitial space for a long time.[4] Iopamidol, as acknowledged by Nel, Shanahan and Conacher, is a large molecule and so may not be so readily or rapidly taken up by the lymphatic vessels. Moreover, due to the low pressure in the lymph vessels,[5] there is usually some delay before the absorbed contrast appears in a regional lymph node where it is filtered and scavenged by the macrophages.

In our report, the anteroposterior chest x-ray demonstrating contralateral spread was taken immediately after the contrast injection,[1] which takes about 1–2 min. Moreover there are no suggestions from the pattern of contrast spread that it occurred via the lymphatics, as the opacified lymphatic channels would appear as thin continuous lines of contrast.[6] Even if trauma related changes resulted in accelerated uptake by the lymphatics as suggested by the correspondents, within the time frame that the chest x-ray was taken this would be negligible and unlikely to produce the demonstrated radiological images. A CT scan would have been ideal to demonstrate the physical spread to the contralateral side.[7]

Based on the above facts, and the wealth of evidence demonstrating contralateral spread after thoracic paravertebral injection in cadavers[8] and *in vivo*,[1 9] with[9] or without[1] contralateral anaesthesia, we still believe that physical spread via the subserous layer of connective tissue contributed to the contralateral spread of contrast in our patient.[1]

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Anaesthetic management of lobectomy for lung abscess or bronchopleural fistula

Editor—We read with interest Pfitzner and colleagues' account of the anaesthetic management of a patient requiring lobectomy for a cavitating lung abscess complicated by haemoptysis.[1] We have previously described our ventilatory management of a 36-yr-old patient with a bronchopleural fistula complicating necrotizing group A β-haemolytic streptococcal pneumonia[2] and we feel the technique we described provides an alternative approach to such patients.

Whilst mechanically ventilated on the intensive care unit, our patient developed a large air leak (500 ml per 800 ml tidal volume), and adequate ventilation became impossible. Her underlying pneumonia was complicated by generalized acute lung injury in the non-pneumonic areas of her lungs resulting in profound hypoxaemia. Under fibrescopic guidance, we placed a single 2 ml size 7F Fogarty embolectomy catheter alongside the single lumen tracheal tube sequentially into the right intermediate, lower and then middle lobe bronchi. After placement in the right middle lobe bronchus, the balloon was inflated and there was almost complete cessation of the air leak. Effective ventilation of all other lung segments was continued. Using this technique we confirmed the presence of a bronchopleural fistula affecting solely the right middle lobe.

During her subsequent middle lobectomy she was ventilated using a left-sided double lumen tracheal tube with a size 7F Fogarty catheter placed via the tracheal lumen of the tracheal tube into the right middle lobe bronchus. Attempted single left lung ventilation, to improve surgical access, resulted in rapid arterial desaturation to Sa_{O_2} 65% which was attributed to the on-going acute lung injury in the non-pneumonic areas of lung. Ventilation of the left lung and the right upper and lower lobes with selective blockade of the middle lobe using the Fogarty catheter as a bronchial blocker maintained saturations of 94–96%. Right middle lobectomy was performed and the patient eventually made a full recovery.

We published this case report[2] because we felt that this technique of bronchial blockade, previously described by other authors,[3 4] has a useful role in the ventilatory management of patients who require isolation of particular lobes for a variety of reasons. Our patient had severe necrotizing middle lobe pneumonia complicated by bronchopleural fistula and could not be adequately oxygenated by one-lung ventilation due to coexistent generalized pulmonary dysfunction. We feel that the technique of selective bronchial blocking is worth considering in this difficult group of patients.

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Editor—We are grateful for the opportunity to comment on McCormick and Wilson's letter, which describes the management of a very different patient from ours.[1] Their patient with group A β-haemolytic streptococcal pneumonia, ARDS and a bronchopleural fistula with a large air leak, was already being mechanically ventilated at the time that right middle lobectomy was performed.[2] To achieve adequate ventilation and oxygenation pre- and intra-operatively, two-lung ventilation with selective blockade of the right middle lobe bronchus with a Fogarty catheter was required, and they are to be congratulated on achieving a successful outcome in this very sick patient.

In patients with a cavitating lung abscess and otherwise reasonably healthy lungs, a safer management plan in our view involves: first, prompt lung separation to prevent contamination of the good lung; and second, refraining from ventilating the lung with the abscess until the lobectomy is complete and the airway cleared by bronchial suction.[1] If ambient pressure oxygenation[3,4] is applied to an operated lung from which nitrogen has been excluded, there will be little risk of marked arterial desaturation in the vast majority of cases. However, this management plan would be inappropriate for a patient with ARDS, since single-lung ventilation would almost certainly be associated with an appreciable degree of shunting through both the ventilated and the non-ventilated lungs.

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Effects of isoflurane, sevoflurane and propofol on jugular venous oxygen saturation

Editor—Nandate and colleagues recently reported the results of their study of the effects of isoflurane, sevoflurane and propofol on jugular venous oxygen saturation.[1] We write to express our concerns about the technique used for induction and maintenance of anaesthesia in the propofol group.

Our concerns are based upon a report by Vuylsteke and colleagues (from the same institution) of a high incidence of amnesic awareness associated with this technique.[2] In the earlier study, anaesthesia was induced with midazolam $50\,\mu g\,kg^{-1}$ and fentanyl $12\,\mu g\,kg^{-1}$, and maintained with a propofol infusion. Using the isolated forearm test for 45 min after induction, it was found that four of the five subjects responded to command, leading the authors to conclude that: 'The use of amnesic drug will avoid a conscious recall but patients who experienced amnesic awareness may suffer from post-traumatic disorder. A midazolam–fentanyl induction does not prevent amnesic awareness and should be abandoned.'[2] As far as we are aware, this statement has not been retracted. In the propofol group of the current study midazolam $100\,\mu g\,kg^{-1}$ and fentanyl $15\,\mu g\,kg^{-1}$ were used, but there is no evidence that these doses prevent awareness. It is unclear why this technique has not been abandoned.

In both studies a morphine premedication was used and anaesthesia was maintained with a propofol $3\,mg\,kg^{-1}\,h^{-1}$ infusion. We used the TIVA Trainer, a computerized pharmacokinetic simulator (© F Engbers, The Netherlands) to estimate the drug concentrations this regimen will produce 45 min after induction of anaesthesia, when skin incision and sternotomy are likely to occur. In a 70 kg male, the estimated blood concentrations will be propofol $1.2\,\mu g\,ml^{-1}$, fentanyl $3.5\,ng\,ml^{-1}$ and midazolam $43\,ng\,ml^{-1}$. These concentrations may be sufficient to prevent memory of events at this stage, as it has been shown that in healthy volunteers the Cp50 for loss of memory for words is $0.62\,\mu g\,ml^{-1}$ for propofol and $56\,ng\,ml^{-1}$ for midazolam.[3]

However, it is less certain that this combination is adequate to prevent response to command or awareness. Kazama and colleagues studied the interaction between fentanyl and propofol during abdominal surgery.[4] Using their graphs, it is evident that a combination of propofol $1.2\,\mu g\,ml^{-1}$ and fentanyl $3.5\,ng\,ml^{-1}$ is *below* the Cp50 for somatic and haemodynamic responses to skin incision, peritoneal incision, and abdominal wall retraction.

Russell has said that a benzodiazepine/opiate technique can at best be said to provide 'general amnesia' and not general anaesthesia.[5] For the first 45 min of the current study, while the blood propofol concentration was slowly increasing from zero, the authors relied on the (decreasing) midazolam and fentanyl levels to maintain unconsciousness. Lack of recall does not necessarily imply adequate anaesthesia. Had they chosen to look for it, the authors may well have found that many of their subjects responded to command during this period.

Is anaesthesia adequate if the patient responds to command, but later shows no sign of recall? Some anaesthetists may feel it is, while others are unsure.[6] The comments of Vuylsteke[2] and Russell[5] seem to indicate that they feel that response to command may indicate inadequate anaesthesia. Until this issue is resolved we feel that if a study involves an anaesthetic technique that relies on a benzodiazepine/opiate combination to provide unconsciousness, the investigators should seek and report clinical signs of awareness. Tests for post-operative recall and adverse psychological sequelae should also be performed.

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Editor—We certainly share the concerns raised by Absalom and Miles and have indeed not retracted our findings published in 1996. We would like, however, to point out that: our small study conducted in 1996 and published as an abstract has never reached the status of peer-reviewed publication due to the small sample size; as pointed out by Absalom and Miles, the anaesthetic technique used in the present study was different with a dose of midazolam twice as high as previously described and a slightly higher dose of fentanyl; and, the time elapsed between induction (and initiation of the propofol infusion) and skin incision was longer in the present study because of the insertion of a retrograde jugular bulb catheter.

Anaesthetic regimen used in cardiac anaesthesia have to compromise between anaesthesia and haemodynamic stability. The aim of our study was to compare three different anaesthetic regimen that are currently used. In the absence of monitoring that would allow us to detect 'amnesic awareness', we felt that we were not diverging from what is still considered standard and safe practice. None of the patients, who were all reviewed post-operatively, spontaneously reported any recall of their surgery.

B. F. Matta
A. Vuylsteke
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Adult epiglottitis: an under-recognized, life threatening condition

Editor—I read the case reports from Ames and co-workers with interest, and some surprise. Was case 1 really epiglottitis? The description of the case does not comment on the appearance of the epiglottis, other than the mass. The symptoms recorded are suggestive of laryngeal obstruction, and would appear to be confirmed by the clinical findings of a mass rather than a diffusely swollen, hyperaemic epiglottis, typical of the epiglottitis described in the other two cases.

The authors state that there are no reports linking epiglottitis with the smoking of heroin. The presentation of heroin users with systemic candidiasis is well described, however. The most common presentations are with endophthalmitis, or cutaneous or osteoarticular manifestations,[1–3] and infection has been attributed to contamination of the lemon juice, used as a dilutent for the heroin, with *Candida albicans*. Perhaps in this case it was the isolation of Candida that was more relevant than the epiglottitis?

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1 Shankland GS, Richardson MD, Dutton GN. Source of infection in candida endophthalmitis in drug addicts. *BMJ* 1986; **292**: 1106–7
2 Collignon PJ, Sorrell TC. Disseminated candidiasis: evidence of a distinctive syndrome in heroin abusers. *BMJ* 1983; **287: 861–2**
3 Newton-John HF, Wise K, Looke DF. Role of lemon in disseminated candidiasis of heroin abusers. *Med J Aust* 1984; **140**: 780–1

Editor—May I congratulate Ames and colleagues on their recent reminder that adult epiglottitis is under-recognized and potentially lethal.[1] A few years ago, Stuart and Hodgetts made similar points in the *British Medical Journal*.[2] Like all important messages, it bears repetition to each new generation of doctors, since epiglottitis can progress from being no more than a bad sore throat to total airway obstruction within a few hours.

I fully agree with Ames that an airway needs to be established as soon as the diagnosis is made, even if airway obstruction appears minimal. I agree also that the performance of a tracheostomy under local analgesia is unwise, since patients with epiglottitis find it easier to breathe sitting up rather than lying down. The key question is, how does one intubate these patients safely? Breathing the patient down with an inhalational technique can lead to acute loss of the airway, as happened in case 3 of Dr Ames's paper and also to one of my patients.[3] The solution, I believe, is to perform a cricothyroidotomy under local analgesia and insuffate oxygen into the lungs via a mini-tracheostomy tube or equivalent *prior* to inducing general anaesthesia with intravenous agents and succinylcholine. In the event of a difficult intubation, the patient can be kept well oxygenated by this route until a tracheostomy is performed.

With regard to the issue of tracheostomies, I would query whether they are necessary once the patient has been safely intubated. Adult epiglottitis normally resolves within 24–48 h once antibiotics have been started. It seems unnecessary to subject a young patient to the long-term complications of tracheostomy, such as tracheal stenosis, when the alteration is a day or two on the intensive care unit attached to a ventilator.

W. Konarzewski
Colchester

1 Ames WA, Ward VMM, Tranter RMD, Street M. Adult epiglottitis: an under-recognized, life-threatening condition. *Br J Anaesth* 2000; **85**: 795–7
2 Stuart MJ, Hodgetts TJ. Adult epiglottitis; prompt diagnosis saves lives. *BMJ* 1994; **308**: 329–30
3 Konarzewski WH. Adult epiglottitis: heightened awareness saves lives. *BMJ* 1994; **308**: 719

Editor—I thank Sherrard and Konarzewski for their interest in our case reports.[1] Despite Dr Sherrard's comments regarding case 1, I firmly believe that the diagnosis of 'epiglottitis' was correct. The symptoms, signs and radiological investigations are compatible with the diagnosis. Furthermore, while the swollen epiglottis completely obscured the larynx, fibreoptic examination demonstrated diffuse swelling of the aryepiglottic structures which is characteristic of the adult form of the disease (Figure 4 in the article).

I appreciate Dr Sherrard's comments regarding lemon juice as a carrier medium for Candida. Lemon juice is used to dissolve street heroin before its intravenous use.[2] Although the patient was not directly questioned as to how he prepared this drug, it is unlikely that he first dissolved it in lemon juice prior to smoking it. As detailed in our discussion, the causal organism or precipitant of epiglottitis in adults is often difficult to identify. While we were unable to determine definitively the aetiology of epiglottitis in this case, the most likely causes were considered to be fungal or thermal epiglottitis. The direct effects of heroin inhalation as a causative factor is unlikely as diacetylmorphine has been found to be fungicidal.[3]

I am grateful to Dr Konarzewski for emphasizing the key issues in our article. I agree that *an* alternative and safe course of action would include an awake cricothyroidotomy prior to inducing anaesthesia and securing the airway. The technique is well described and associated with few complications.[4] However, although not typically the case in these patients, tracheal intubation may be difficult and a tracheostomy would then become necessary. In case 1, although the patient was intubated, he underwent a tracheostomy because the diagnosis was initially uncertain and the epiglottitic mass required further evaluation. The trachea was decannulated 9 days after the original presentation. In retrospect, case 2 may well have been managed without proceeding to a tracheostomy. At the time, however, with the difficulty in establishing an airway, it was considered the safest course of action to provide and maintain a stable airway. Lastly, in case 3, attempts at tracheal intubation had already failed and a tracheostomy became necessary. I agree however that, in general, a tracheostomy and its concomitant complications are best avoided, but realize this may not always be possible.

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1 Ames WA, Ward VMM, Tranter RMD, Street M. Adult epiglottitis: an under-recognized, life-threatening condition. *Br J Anaesth* 2000; **85**: 795–7
2 Newton-John HF, Wise K, Looke DF. Role of lemon in disseminated candidiasis of heroin abusers. *Med J Aust* 1984; **140**: 780–1
3 Shankland GS, Richardson MD, Dutton GN. Source of infection in candida endophthalmitis in drug addicts. *BMJ* 1986; **292**: 1106–7
4 Ames WA, Venn P. Complications of the transtracheal catheter. *Br J Anaesth* 1998; **81**: 825–9

Post-operative nausea and vomiting—time for balanced antiemesis

Editor—Further to the editorial by Heffernan and Rowbotham,[1] I note there was no mention of low dose midazolam infusion as an option for patients with refractory post-operative nausea and vomiting (PONV). I agree totally with the concept that PONV probably requires a 'balanced approach' for effective treatment of a multifactorial problem.

I refer readers to a recent study examining the effect of midazolam on persistent nausea and vomiting.[2] Low dose infusion of midazolam is thought to decrease dopamine input at the chemoreceptor trigger zone (CRTZ) in addition to decreasing anxiety. It may also decrease adenosine re-uptake. This leads to an adenosine-mediated reduction in synthesis, release and postsynaptic action of dopamine at the CRTZ. In addition to altering adenosine-mediated effects, midazolam probably reduces dopaminergic neuronal activity by binding to the gabba-aminobutyric acid–benzodiazepine complex. The study, although small in numbers, was terminated early due to statistically significant differences between the groups.

On a purely anecdotal level, I have used low dose midazolam infusions for several years and found it to be extremely efficacious, using a regimen of a 1 mg loading dose followed by a 1 mg h^{-1} infusion for an average sized adult. Over-sedation does not appear to be a problem. I feel low dose midazolam infusions are a useful treatment option in the management of refractory PONV.

C. Weidmann
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1 Heffernan AM, Rowbotham DJ. Post-operative nausea and vomiting—time for a balanced antiemesis. *Br J Anaesth* 2000; **85**: 675–6
2 DiFlorio T, Goucke CR. The effect of midazolam on persistent nausea and vomiting. *Anaesth Intensive Care* 1999; **27**: 38–40

Editor—We read with interest Heffernan and Rowbotham's editorial[1] and wish to make some comments. In the literature, there are numerous randomized double-blind controlled studies comparing one antiemetic to another or placebo for prophylaxis of post-operative nausea and vomiting (PONV). Even a meta-analysis of these studies failed to show that any antiemetic is superior in prevention of PONV.[2] A majority of the studies have enrolled patients anaesthetized with a nitrous oxide and a volatile technique.

There has been an increasing trend for inducing and maintaining anaesthesia using a combination of remifentanil and a target-controlled propofol infusion while ventilating the patients with oxygen and air. Omission of nitrous oxide and the use of total intravenous anaesthesia (TIVA) have been shown to be less emetogenic anaesthetic techniques.[3 4]

The neuropharmacology of PONV is now better understood. Multiple receptors including dopaminergic, muscarinic, cholinergic, opioid, histamine, serotonin and NK_1 mediate the emetic reflex.[5] A balanced antiemetic prophylaxis would need to block most or all of these receptors. This may not be a practical approach.

We believe that instead of balanced antiemetic pharmacological prophylaxis, an approach should be considered including the use of less emetogenic anaesthetic techniques and local or regional blocks for good post-operative pain relief. This may reduce the need for perioperative antiemetics.

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A. Mallick
Huddersfield
UK

1 Heffernan AM, Rowbotham DJ. Post-operative nausea and vomiting—time for balanced antiemesis. *Br J Anaesth* 2000; **85**: 675–7
2 Henzi I, Walder B, Tramer MR. Dexamethasone for the prevention of postoperative nausea and vomiting: a quantitative systematic review. *Anesth Analg* 2000; **90**: 186–94
3 Tramer M, Moore A, McQuay H. Propofol anaesthesia and post-operative nausea and vomiting: quantitative systematic review of randomized controlled studies. *Br J Anaesth* 1997; **78**: 247–55
4 Tramer M, Moore A, McQuay H. Omitting nitrous oxide in general anaesthesia: metanalysis of intraoperative and postoperative emesis in randomized controlled studies. *Br J Anaesth* 1996; **76**: 186–93
5 Kovac AL. Prevention and treatment of postoperative nausea and vomiting. *Drugs* 2000; **59**: 213–43

Editor—It is encouraging to see that our editorial is provoking good interest.[1]

Yes, we do acknowledge the fact that midazolam may be used in the treatment of persistent post-operative nausea and vomiting (PONV) by the mechanisms referred to in Weidmann's letter.[2 3] He and other authors refer to the fact that PONV is a multifactorial problem and that it is likely that combination therapy with more than one agent acting via different receptors will be required.[3–7] While the use of midazolam infusions in a small study does appear to significantly reduce the incidence of persistent PONV, it is not a method that can be resorted to lightly. The patients probably need high dependency care and this may not always be available for this patient group. However, in this study the authors have embraced the concept of balanced antiemesis; patients were also given metoclopramide, prochloperazine and droperidol, agents which act at different receptors.[2]

Nunez and Mallick postulate that using total intravenous anaesthesia (TIVA) and omitting nitrous oxide are less emetogenic techniques. We do agree with the authors that more local and regional techniques should be used in practice, which would decrease the incidence of PONV. However, a meta-analysis in 1997 confirms that TIVA studies are documented poorly and there is not enough evidence that it is an anaesthetic technique with a low emetogenic potency.[8] Since then, there has been no convincing evidence for an improvement in PONV with TIVA. One study shows an improvement in PONV but only in the first few hours post-operatively.[9] A second study shows a low incidence of PONV in both TIVA and inhalational anaesthesia groups.[10] Omitting nitrous oxide does not seem to be without its problems.[11] It appears only to decrease significantly post-operative vomiting if the risk of vomiting is high and does not affect nausea or complete control of emesis.

A. M. Heffernan
D. J. Rowbotham
Leicester

1 Heffernan AM, Rowbotham DJ. Post-operative nausea and vomiting—time for a balanced antiemesis. *Br J Anaesth* 2000; **85**: 675–6
2 DiFlorio T, Goucke CR. The effect of midazolam on persistent nausea and vomiting. *Anaesth Intensive Care* 1999; **27**: 38–40
3 DiFlorio T. The uses of midazolam for persistent postoperative nausea and vomiting. *Anaesth Intensive Care* 1992; **20**: 383–6
4 Matson A, Palazzo M. Postoperative nausea and vomiting. Adams AP, Cashman JN, eds. *Recent Advances Anaesth and Analg* 1995; 19: 107–26
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6 Pueyo FJ, Carroscosa F, Lopez L, Irabarren MJ, Garcia-Pedrajas F, Saez A. Combination of ondansetron and droperidol in the prophylaxis of postoperative nausea and vomiting. *Anesth Analg* 1996; **83**: 117–22
7 Ahmed AB, Hobbs GJ, Curran JP. Randomised, placebo-controlled trial of combination antiemetic prophylaxis for day-case gynaecological laparoscopic surgery. *Br J Anaesth* 2000; **85**: 678–82
8 Tramer M, Moore A, McQuay H. Propofol anaesthesia and post-operative nausea and vomiting: quantitative systematic review of randomized controlled studies. *Br J Anaesth* 1997; **78**: 247–55
9 Brooker CD, Sutherland J, Cousins MJ. Propofol maintenance to reduce postoperative emesis in thyroidectomy patients: a group sequential comparison with isoflurane/nitrous oxide. *Anaesth Intensive Care* 1998; **26**: 625–9
10 Fish WH, Hobbs AJ, Daniels MV. Comparison of sevoflurane and total intravenous anaesthesia for daycase urological surgery. *Anaesthesia* 1999; **54**: 1002–6
11 Tramer M, Moore A, McQuay H. Omitting nitrous oxide in general anaesthesia: metanalysis of intraoperative and postoperative emesis in randomized controlled studies. *Br J Anaesth* 1996; **76**: 186–93

Publications on paediatric anaesthesia

Editor—I read with interest the review by Brambrink and colleagues.[1] It confirms earlier observations on the low *per capita* output of German anaesthesiology.[2–5] This is true in comparison with the major English speaking and Scandinavian countries, and also with Switzerland and Austria. It was interesting to read that, at least in the field of paediatric anaesthesia, this is still the case if non-English language journals are included, considering a recent controversy on this point.[6–8]

Brambrink and colleagues made a mistake, however, concerning the names and database coverage of two German anaesthesia journals in Table 5. *Anästhesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie* (*AINS*) is indexed in Medline, but was **NOT** listed in the *Journal Citation Report* for the years analysed by Brambrink (1993–1998). It was only in 1999 that it was included for the first time under the abbreviation *Anasth Intensiv Notf* with an impact factor of 0.473. Consequently, *AINS* has no known impact factor for the years 1993–1998. Brambrink was probably misled by the abbreviation *Anasth Intensivmed* in the *Journal Citation Report* that stands for *Anästhesiologie & Intensivmedizin* (*A&I*), another German journal which is included in the Science Citation Index, but **NOT** in Medline. The mean impact factor of *A&I* for the years 1993–1998 is only 0.426, not 0.690 as given by Brambrink as it achieved an impact of 0.692 only in 1997 but was ranked lower for all other years.[9]

In the context of the methods used by Brambrink of counting only Medline-indexed publications and assigning an impact factor of zero to journals not listed in the journal citation reports 1993–1998, publication in *A&I* do not count at all and publications in *AINS* have no impact factor.

As 20 of the 22 paediatric articles in *AINS* were from Germany, this influences the country rankings in Tables 6 and 7 of Brambrink's paper. The already low total impact factor of Germany shrinks further from 88.38 to 74.58 and the mean impact factor from 0.875 to 0.738, making it even clearer that German paediatric anaesthesia in the mid-1990s was far behind its major competitors.

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1 Brambrink AM, Ehrler D, Dick WF. Publications on paediatric anaesthesia: a quantitative analysis of publication activity and international recognition. *Br J Anaesth* 2000; **85**: 556–62
2 Carnie J, Kumar B. An analysis of thirty years of contributions to the British anaesthetic literature (1953–1982). *Br J Anaesth* 1984; **56**: 1171–4
3 Schubert A, Glänzel W, Braun T. Scientometric datafiles. A comprehensive set of indicators on 2649 journals and 96 countries in all major science fields and subfields. *Scientometrics* 1989; **16**: 3–478
4 Pomaroli A, Hauffe H, Benzer A. Who publishes in the large anaesthesia journals? *Br J Anaesth* 1994; **72**: 723–5
5 Boldt J, Maleck W, Koetter KP. Which countries publish in important anesthesia and critical care journals? *Anesth Analg* 1999; **88**: 1175–80
6 van Aken H, Prien T. Forschungsqualität anästhesiologischer Universitäts-Abteilungen in Deutschland. *Anästhesiol Intensivmed Notfallmed Schmerzther* 1999; **34**: 793–4
7 Boldt J. Forschungsqualität anästhesiologischer Universitäts-Abteilungen in Deutschland. *Anästhesiol Intensivmed Notfallmed Schmerzther* 1999; **34**: 794–5
8 Maleck WH, Boldt J, Wickenhäuser R. Deutschsprachige Publikationen Deutscher Anaesthesiologischer Universitäts-Abteilungen. *Anästhesiol Intensivmed Notfallmed Schmerzther* 2000; **35**: 559–66
9 Boldt J, Haisch G, Maleck WH. Changes in the impact factor of anesthesia/critical care journals within the past 10 years. *Acta Anaesthesiol Scand* 2000; **44**: 842–9

Editor—We thank Dr Maleck for his interest in our work and are pleased to be given the opportunity to reply to his letter. Our analysis produced data on publication productivity and the international recognition of different countries/regions in the field of paediatric anaesthesia during the period from 1993 to 1998.[1] As Dr Maleck pointed out, we showed that the number of publications per German anaesthesiologist was smaller than for anaesthetists in other countries such as the European states or North America.

However, this does not necessarily imply that, as Dr Maleck stated, 'German paediatric anaesthesia in the mid-1990s was far behind its major competitors'.

First, publications in peer reviewed journals listed in Medline™ represent only one of the criteria which acknowledges the standing of 'paediatric anaesthesia' in a given country. Important additional factors such as, for example, reports in periodicals not listed in Medline™, chapters in books, contributions on the World Wide Web,[2] presentations at scientific meetings and—most importantly—the quality of day-to-day clinical practice, have to be taken into account in the appreciation of the international position of this anaesthetic subspecialty. Second, as discussed in our paper, differences in the publication activity of various countries may also be influenced by differences in the respective medical systems (structure of fund-raising, career development, financial support of scientific activity), or on other factors, such as language or training.[3] As mentioned in our paper,[1] a certain publication bias towards Anglo-American authors has been postulated to affect international publication patterns.[4 5]

We are grateful to Dr Maleck for the information that the German anaesthesia journal *Anästhesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie* (*AINS*) did not have an impact factor during the period from 1993–1998, although it was listed in Medline™ at that time. However, the 1999 Science Citation Index lists *AINS* with an impact factor of 0.473[6 7] (total citations=319, immediacy index=0.140, source items=136).[7] The Science Citation Index uses *A*NASTH *I*NTENSIV *N*OTF as the current abbreviation for *AINS*[7] (confirmed by the editorial office of the journal). We were misled by the abbreviation *A*NASTH *I*NTENSIVMED, used in the Science Citation Index[8] for another German anaesthesia journal (*Anästhesiologie & Intensivmedizin*) which, in contrast, is not listed in Medline™ and is therefore not included in our study sample. According to our method, publications in *AINS* should not be included to calculate a country's cumulative impact factor (cIF-country) as they do not have an impact factor. This results in a 15% lower cIF-country for German authors in paediatric anaesthesia (74.58), compared with the one we originally reported in Table 6 (88.38). Similarly, this reduces the mean impact factor (Table 7) of German publications on paediatric anaesthesia, which now stands at 0.738 instead of 0.875. For completeness, the one publication in *AINS* by US authors was subtracted leading to a small reduction in the cIF-country (from 1084.41 to 1083.41) and in the mIF (from 2.122 to 2.092) for US-American authors.

However, these calculations do not affect the ranking of the countries listed in both tables and, as Dr Maleck agrees, the reported results on this aspect of our study remain valid: German authors published less on paediatric anaesthesia during the period studied compared to their colleagues from the USA, UK, Japan, Canada, Scandinavia or France. Additionally, the relative international representation ('visibility'[9]) of the respective articles was lower when the impact factor was used for the calculation of the respective figures. However, as discussed in our paper, the value of the impact factor as a measure of quality remains a matter of controversy.[1 6 10]

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Mainz
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1 Brambrink A, Ehrler D, Dick WF. Publications on paediatric anaesthesia: a quantitative analysis of publication activity and international recognition. *Br J Anaesth* 2000; **85**: 556–62
2 Hernandez-Borges AA, Macias-Cervi P, Gaspar-Guardado MA, Torres-Alvarez de Arcaya ML, Ruiz-Rabaza A, Ormazabal-Ramos C. Assessing the relative quality of anesthesiology and critical care medicine Internet mailing lists. *Anesth Analg* 1999; **89**: 520–5
3 Boldt J, Maleck W, Koetter KP. Which countries publish in important anaesthesia and critical care journals? *Anesth Analg* 1999; **88**: 1175–80
4 Kolbitsch C, Balogh D, Hauffe H, Löckinger A, Benzer A. National publication output in medical research. *Anästhesiol Intensivmed Notfallmed Schmerzther* 1999; **34**: 214–7
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6 Hempelmann G, Krier C, Schulte am Esch J. AINS in Science Citation Index—Nutzen, Missbrauch und Grenzen des Impact Faktors (editorial). *Anästhesiol Intensivmed Notfallmed Schmerzther* 2000; **35**: 669–70
7 Journal Citation Reports. A bibliometric analysis of science journals in the ISI data base. Philadelphia; *Institute for Scientific Information*, 1999
8 Journal Citation Reports. A bibliometric analysis of science journals in the ISI data base. Philadelphia; *Institute for Scientific Information*, 1993–1998
9 Favaloro EJ. Medical research in New South Wales 1993–1996 assessed by Medline publication capture. *MJA* 1998; **169**: 617–22
10 Gisvold SE. Citation analysis and journal impact factors—is the tail wagging the dog? *Acta Anaesthesiol Scand* 1999; **43**: 971–3

Preventing epidural catheter obstruction

Editor—Most anaesthetists who combine general and epidural anaesthesia will be familiar with the problem of mechanical epidural catheter obstruction.

Rare manufacturing defects in the catheter[1 2] and early blood clot obstruction should be avoidable by careful inspection and by giving the 'test dose' prior to induction. More troublesome in our experience is late obstruction by intra-operative catheter kinking or stretching.[3] We have had several such cases during radical prostatectomy when the surgeon has insisted on steep Trendelenberg repositioning with exaggerated lumbar lordosis. Only then has the catheter obstructed, presumably kinked in a deepening skin fold. The problem is not rectifiable until post-operatively when the chance to optimize intra-operative regional analgesia has been missed.

Our solution to the problem uses a sterile, polyurethane nasogastric feeding tube ('Flowcare', Chatel Medical Devices, Chatel-St-Denis, Switzerland) cut to length. The tube is passed over the extracorporeal portion of the epidural catheter following removal of the epidural needle but prior to attaching the epidural filter. The guarded catheter is then taped to the back along the normal route with a thin layer of gauze padding separating it from the skin.

The stiff nasogastric tube provides a protective envelope for the fine epidural catheter and the gauze keeps the arrangement comfortable. Since using this simple technique, we have had no further problems with intra-operative mechanical catheter obstruction and we would commend the technique to others.

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Dr Lou Michels
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1 Chandhok D, Vijayakumar E. An unusual case of epidural catheter obstruction. *Anesthesiology* 1999; **91**: 895–6
2 Husemeyer RP. A defective epidural cannula. *Anaesthesia* 1980; **35**: 922
3 Khalouf FK, Kunkel FA, Freeman J. Stretching with obstruction of an epidural catheter. *Anesth Analg* 1987; **66**: 1202–3