

miss a diagnosis of sarcoidosis or tuberculosis and for the procedure to still be classified as a true negative. Fourthly, it should be emphasised that CUSUM analysis is suited to ongoing audit of the EBUS service beyond the learning curve and that issues with training and competence may be identified before 100 cases are reached. A final point is an inaccuracy in the definition of Q in the description of the CUSUM methodology. The paper states $Q = \ln((1-p_1)/(1-p_0))$, where it should be $Q = \ln((1-p_0)/(1-p_1))$. The value of s obtained is however correct, so does not represent an error in calculation by the authors.

In our institution, a tertiary teaching centre, EBUS-TBNA has been performed by two physicians (NN and SJ) since February 2008. In order to maximise our learning process, a gastroenterologist (SP) with expertise in endoscopic ultrasound (EUS) attended the first 25 of our procedures. The CUSUM chart for our initial 120 cases (reached in November 2008) is shown in figure 1. Only patients with abnormal nodes were included in the analysis and nodes <1 cm in the short axis were excluded. Real-time evaluation of aspirates was available by an on-site cytologist in 23 (19%) cases. The chart demonstrates a short learning curve with a rise in the curve and a learning period over the first 20 patients. After this, the curve reaches a steady state below the alert line, indicating that the target sensitivity was being met and performance remained acceptable for the duration of the series. In contrast to the data from Kemp *et al*, isolated mediastinal lymphadenopathy was the indication for EBUS-TBNA in 53 (44%) of the patients in our initial cohort. The sensitivity of EBUS-TBNA for our first 120 patients undergoing EBUS-TBNA was 90% with a diagnostic accuracy of 93% and negative predictive value of 83% when the disease prevalence was 68%. No false positives were observed and therefore the specificity and positive predictive values were 100%.

EBUS-TBNA is an important procedure for the diagnosis of mediastinal lymphadenopathy and its use will continue to spread. Where available, inviting gastroenterologists

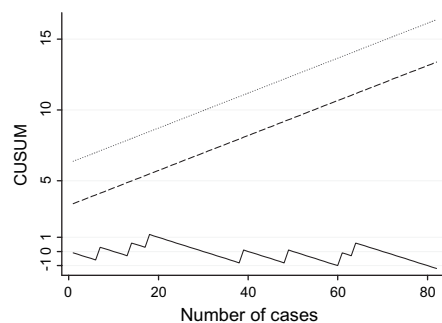


Figure 1 CUSUM chart of the learning curve of endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) at our centre using an acceptable false-negative rate of 10% and an unacceptable rate of 15%.

and pathologists into the bronchoscopy suite may help to shorten the learning curve.

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Smokers commonly misperceive that nicotine is a major carcinogen: National survey data

In vitro testing has shown that nicotine may play a role in making cancers more aggressive,¹ but the currently available evidence does not suggest that nicotine in itself induces cancer.² Despite this, many smokers believe that nicotine does cause cancer. For example, in a USA-based study it was found that 65% of smokers believed nicotine causes lung cancer and 71% believed it caused oral cancer.³ Furthermore, some smokers regard nicotine replacement therapy (NRT) as also being carcinogenic.⁴ These findings are concerning since misperceptions about nicotine may result in underutilisation of NRT. Therefore, we aimed to assess these views in New Zealand (NZ) smokers, with the context being a country in which NRT is provided in a heavily subsidised form and widely distributed via the national quitline service.

Data were collected through the NZ arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) which derives its sample of smokers from the NZ Health Survey (a representative national sample). From this sample we surveyed adult smokers in two survey waves (n=1376 and n=923) 1 year apart (with wave 2 in 2008/early 2009). Here we focus on those who completed both surveys (to facilitate comparisons over time). Further details of the methods, including response rates, attrition and weighting processes, are available in online reports (at: <http://www.wnmeds.ac.nz/itcproject.html>).

When asked if ‘the nicotine in cigarettes is the chemical that causes most of the cancer?’, most smokers in wave 1 (52.6%) said that it was true, 36.7% said it was false (the correct answer) and 10.7% could not say. The proportion answering ‘true’ was fairly similar in wave 2 at 52.1%. In a multivariate model (that adjusted for demographics, socioeconomic position, mental health and smoking-related beliefs and behaviours), certain groups of smokers were significantly more likely to believe that nicotine was carcinogenic. These included older smokers (≥ 50 vs <35 years); Māori smokers (vs European/other, adjusted OR (aOR)=1.77, 95% CI 1.22 to 2.58); and Asian smokers (vs European/other, aOR=3.25, 95% CI 1.35 to 7.83). One of two forms of financial stress was significantly associated with this misperception (aOR=1.57, 95% CI 1.03 to 2.41 for not spending on household essentials) but the individual and small area deprivation measures were not. Of 13 other variables considered (covering mental health, smoking beliefs and behaviours), only having a higher AUDIT score (reflecting an increased risk of hazardous alcohol use), was significantly associated with this misperception.

The finding that smokers in this national sample commonly have misconceptions about the carcinogenicity of nicotine is consistent with findings from the USA and the UK. This population of smokers also commonly have misperceptions around the relative harmfulness of ‘lights’, ‘roll-your-own’ tobacco, menthols and smokeless tobacco.⁵ How best to address all such misperceptions is complex, but at least for the nicotine and cancer issue evaluation work could be considered on: (1) inclusion of this information as part of warning labels on tobacco packets; and/or (2) mass media campaigns that highlight the relatively safety (and effectiveness) of NRT.

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Quality assurance in endobronchial ultrasound

In their study of endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA), Kemp and colleagues report variation in the learning curves for five operators, studied by using the cumulative sum (cusum) technique,¹ with which we have some experience.² The authors speculate on whether variations in lymph node size, prevalence of underlying diagnoses or rate of accrual of cases may explain these differences. We believe there may be other important influences. Successful EBUS-TBNA is a multi-disciplinary process: help is invaluable from colleagues in radiology for identification of suitable target nodes, in bronchoscopy nursing for adequate specimen preparation and in cellular pathology for confident diagnosis based on cytopathological specimens alone. In our experience, each of these aspects is subject to variation between centres. In addition, it is likely that access to prior positron emission tomography (PET) scanning, or different immunocytochemical stains, may have varied. In our view the results should be regarded as being those of the centres in question, and not those of the operators alone.

Kemp and others appear to have misinterpreted the cusum plots shown in their figure 1. The authors use the graphical representation of the cusum favoured by Kestin.³ In this representation, if the plot crosses two boundaries in succession from below, without crossing a boundary from above in between, unsatisfactory performance is confirmed for the procedure interval between the two upward crossings.⁴ Competence is confirmed by analogous downward crossing of two boundaries. Thus operator 4 demonstrates unacceptable performance between procedures 50 and 70 (these procedure numbers are approximate because the graphs reproduced are too small to permit their exact estimation), and to say that he has 'attained competence almost immediately' is not the whole story. Simi-

larly the cusum of operator 2 demonstrates unacceptable performance during the following procedure intervals: 32–43, 43–80 and 80–96. It never demonstrates satisfactory performance. Indeed, the only procedure intervals for which competence is confirmed in figure 1 or figure 2 are procedures 75–95 for operator 1 and 7–47 for operator 4. Therefore, only operator/centre 1 demonstrates competence by the end of the first 100 procedures. Indeed this is the only operator/centre with evidence of any learning—the others perform no better after 100 procedures than before. An alternative interpretation of the results, therefore, is that for some, and possibly most, operators or centres, no learning curve is expected in EBUS-TBNA at all, provided that standards substantially lower than those in the published literature are accepted.

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Authors' response

We agree with Drs Slade and Slade that success in endobronchial ultrasound-guided transbronchial needle aspiration relies on many factors other than the skill of the actual bronchoscopist and, as such, the term 'operator' may have been misleading. Nevertheless, the operator is going to have the greatest bearing on the results obtained. The article¹ was intended to highlight the need for more accurate methods of assessment of competency in any given task or procedure, using endobronchial ultrasound-guided transbronchial needle aspiration only as an example.

I am sure Drs Slade and Slade recognise that, as in medicine, there are valid alternative interpretations for data. In the referenced paper by Bolsin and Colson,² the discussion of Kestin's Cusum plots states that 'acceptable performance will be denoted on this format by a Cusum line which is roughly horizontal or down-sloping'—that is, a line crossing multiple decision intervals from above is not required to say that performance is acceptable. While a horizontal line does not indicate learning per se, this may not necessarily be an appropriate objective in more experienced practitioners/centres where the focus is on monitoring ongoing competence.

The interpretation of statistical methods is always open to differences, but there is little doubt that Cusum analysis allows the effective monitoring of practices and procedures and, when a change in outcomes is observed (whatever predetermined criteria are used), we as clinicians should reflect on our practice in order to determine which aspects of that practice require attention.

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Effect of statins on cancer in chronic obstructive pulmonary disease

We read with interest the article by van Gestel *et al*¹ reporting a protective effect of statins on cancer mortality in chronic obstructive pulmonary disease (COPD) patients and suggest here a plausible explanation.

Consistent with the literature, the study shows that COPD is associated with an elevated risk of lung cancer. Recently, we reported that COPD is pre-existing in 70% of lung cancer cases compared with 15% in unselected matched smokers.² We agree with van Gestel *et al*¹ that this link is likely to be secondary to a pro-inflammatory disposition resulting from both smoking and genetic susceptibility. In this regard serum interleukin (IL)-6, which is elevated by genetic and