Is investigation of patients with haemoptysis and normal chest radiograph justified?

We read with interest the article by Thirumaran et al.1 They described their experience of 270 consecutive patients referred with haemoptysis and a normal chest radiograph. In their study they found the incidence of respiratory malignancy within this group was 9.6% (26 individuals) and of these 22 were primary lung malignancies. This is slightly higher than the 3–6% incidence previously reported in the literature.2–4

We have delivered a nurse-led clinic for patients referred via the 2 week wait system with haemoptysis and a normal chest x-ray. Patients were stratified initially into high risk or low risk groups according to age and smoking history. An algorithm was then devised to guide further investigation (available as Figure 1 online).

A total of 348 patients were seen in this clinic (215 male, 133 female) between 2003 and 2008. Leicester has a large ethnic minority population and 41 (11.8%) of the 348 patients were of South Asian origin.

Thirty-four patients referred, on detailed history taking, did not have haemoptysis at all. The presenting problems reported by these patients included bleeding gums, ill-fitting dentures and epistaxis.

The investigation modality of choice was CT scanning of the thorax and upper abdomen. A total of 165 patients were referred for CT scan at their initial consultation with the nurse. Fibreoptic bronchoscopy (FOB) was only utilised to confirm tissue diagnosis in patients diagnosed with a thoracic malignancy.

Twenty-three patients (6.6%) were diagnosed with a lung malignancy in our clinic. The other common diagnostic causes of haemoptysis are shown in Table 1.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td>23</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>52</td>
</tr>
<tr>
<td>Infection</td>
<td>107</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>70</td>
</tr>
<tr>
<td>ENT</td>
<td>24</td>
</tr>
<tr>
<td>Cardiac</td>
<td>6</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>5</td>
</tr>
<tr>
<td>Not haemoptysis</td>
<td>34</td>
</tr>
<tr>
<td>Anticoagulation therapy</td>
<td>8</td>
</tr>
<tr>
<td>Vascularis</td>
<td>4</td>
</tr>
<tr>
<td>DNA follow-up</td>
<td>13</td>
</tr>
<tr>
<td>Died</td>
<td>2</td>
</tr>
</tbody>
</table>

A possible thoracic malignancy and freeing up resources in the urgent 2 week wait clinic.

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► An additional figure is published online only. To view this file please visit the journal online (http://thorax.bmj.com).

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The learning curve for EBUS-TBNA

We read with interest the paper by Kemp and colleagues5 which utilises cumulative sum (CUSUM) to analyse the learning curves associated with endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA). The retrospective study from five centres demonstrated that variable learning periods are required to attain proficiency in the procedure, and a pooled sensitivity of 67.4% was observed.

The authors are to be commended on using CUSUM to calculate the learning curves for EBUS-TBNA; however, several points deserve comment. First the study only includes patients undergoing EBUS-TBNA for the diagnosis or staging of lung cancer. In clinical practice, the procedure is also commonly employed for the diagnosis of isolated mediastinal lymphadenopathy, and these procedures should be incorporated in the learning process. Secondly, the authors included non-malignant nodes in the CUSUM analysis. Therefore, it may be possible to inadequately sample a benign node and for the result to be assigned as a true negative. This highlights the importance of reporting the disease prevalence for each cohort. Thirdly, utilising the criteria employed in this paper, there is potential to

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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 <1 cm in the short axis were excluded. Real-time evaluation of aspirates was available by an on-site cytol- ogist 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 83 (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 and pathologists into the bronchoscopy suite may help to shorten the learning curve.

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**REFERENCE**


**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, 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=1576 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); Maori 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 7.85). 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, menthol 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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