THE FICKLE FINGERPRINT OF FATE
Sleep deprivation is amongst the many reasons why your editors are glad they are not intensivists, but diagnostic impossibility is another one. The ventilated patient who is deteriorating, who may or may not have a new pulmonary infiltrate, which may or may not be due to pulmonary oedema, atelectasis, haemorrhage, infection or other reasons presents a huge diagnostic challenge—is it ventilator associated pneumonia, and how to confirm it, given any sampling procedure has to traverse a likely heavily infected endotracheal tube and trachea? The penalty of not treating may be death of the patient, and the penalty of giving out potent broad spectrum antibiotics is resistant organisms, the front page of the Daily Mail, and now being reported to the GMC. Fowler and colleagues (see page 320, Hot topic) report that a high lower airway pathogen load has a fingerprint in breath samples analysed for volatiles by thermal desorption/gas chromatography/time-of-flight mass spectrometry (or a highly expensive and sophisticated toy to you). Still preliminary, and if the measurements could be refined so antibiotic sensitivity profiles could be determined (not that we are asking for much) they could be even more powerful. We await with great interest the intervention trial that shows these measurements can be used to improve clinical outcome—please send the results to Thorax!

IF IN DOUBT, CUT IT OUT
A familiar refrain from the surgical corner of lung cancer multidisciplinary meetings up and down the country, struggling to deal with an epidemic of solitary pulmonary nodules (SPN). Could BTPNA keep the surgeons away (BTPNA=biopsy by Bronchoscopic Trans-Parenchymal Nodule Access)? Felix Herth and colleagues (see page 326, Editors’ choice) report a first in man trial of this technique. The procedure involves identifying an optimum airway wall point of entry and establishing an avascular transpulmonary path through which biopsies of peripheral SPNs can be carried out. It was performed immediately pre-operatively in 12 patients undergoing a lobectomy for suspected lung cancer. Biopsy was successful in 10, revealed consistent histological findings in all, and caused no obvious harm to the resected lobe. Most promising preliminary findings, but we need to know much more about safety and the ability of BTPNA to positively identify benign disease before we can expect the surgeons’ chorus to chant ‘if BTPNA OK, then we must be away’.

MOVING OR SHAKING?
A perennial editorial bleat is the need to look carefully at the components of airway disease, and not rush to huddle them in an umbrella. This has been particularly true for those perennial leaky favourites ‘asthma’ and ‘COPD’, but it is also important to think clearly about chronic suppurative lung diseases. We know that a great treatment in one such (rhDNase in CF) may make matters worse in another (primary ciliary dyskinesia, PCD). Is the same true for physiological measurements? Lung clearance index (LCI) is a hot physiological topic, increasingly being used as an endpoint in randomised controlled trials in CF, but according to two previous groups, of no use in PCD. Not so, say Mieke Boon and colleagues; in milder PCD, LCI is very valuable (see page 339). They discuss reasons for the discrepancies, also summarised in an editorial (see page 305). This needs to be resolved; there are no randomised trials of treatment in PCD, and good end-points are going to be needed, as momentum across Europe gathers for collaborative work in the field. Whatever the explanation, one clear message: check the equipment and protocols that you propose to use for your study is actually useful in your proposed population, and do not rely uncritically on what has been published before.

AN AIR-RAISING PROBLEM?
This radiograph is from a sick, haemodynamically unstable one year old, who died despite skilled medical help. What’s the diagnosis? See Images in Thorax (see page 395).

Andrew Bush, Ian Pavord, Editors-in-Chief

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THE AIRWAY
FROM JAMES BOND TO THE AIRWAY
QV A 149 is a fixed dose combination of the long-acting β2-agonist (LABA) indacaterol and the long-acting muscarinic antagonist (LAMA) glycopyrronium. It is one of a number of fixed dose LABA/LAMA combinations for once daily treatment of patients with COPD coming to a pharmacy near you. Does it have any advantages over the existing and popular formoterol and tiotropium taken separately, the former twice daily? Roland Buhl and colleagues (see page 311) think it might. They report that significantly more patients report a clinically important change in transitional dyspnoea index (TDI) with QVA149. Whether this is a function of the drugs, dose, duration of action or the fact that once daily leads to better adherence than twice is unclear. The potentially large beneficial effect of once daily combination treatment on treatment adherence could not be assessed in this trial as it involved a double dummy placebo and was carried out in a highly adherent clinical trial population. The authors’ assertion that any benefits are likely to be much greater in real world settings seem to us to be fair, but will it be enough to convince sceptical payers? And philosophically, should the taxpayer cough up for a more expensive treatment for patients who cannot be bothered to take a cheaper alternative?