PostScript

LETTERS TO THE EDITOR

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Squawks in pneumonia

Squawks are short inspiratory wheezes that have been described in hypersensitivity pneumonitis and other fibrotic disorders. Little attention has been paid to the fact that they also occur in patients with pneumonia. In the course of studying the correlation of automated lung sound analysis with disease states in patients at a community teaching hospital, we noticed that squawks appeared to be more common in patients with pneumonia than we expected. We therefore examined the occurrence of squawks more systematically in 500 subjects who had been examined with a multichannel lung sound analyser (Stethographics Model STG-1602), as previously described.[1]

Seventy eight of the subjects in this population had a clinical diagnosis of pneumonia. All participants had been asked to breathe more deeply than normal with their mouths open. Two 20 second samples were taken. The Institutional Review Board of the Faulkner Hospital approved the study. Two experienced observers, blinded to the clinical diagnosis, used playback and waveform displays to identify squawks. They were defined according to the criteria initially described by Earis *et al* and adopted by CORSA.[2 3] All channels from each subject were replayed and the waveforms of the data in the time domain were simultaneously examined. Only those sounds which fit both the auditory and waveform characteristics were considered to be squawks for the purposes of this study.

Squawks were present in 12 of the 78 patients with pneumonia and in none of 224 patients considered to have no significant lung disease. They were found in four of 18 patients with interstitial pulmonary fibrosis, two of 41 patients with bronchial asthma, one of 79 patients with COPD, and in none of 56 patients with congestive heart failure. We also noted squawks in a patient with radiation pneumonitis and in one of the two patients in our database with hypersensitivity pneumonitis.

In nine of the 12 patients with pneumonia the squawks were in the same location as the radiological opacifications. In one patient the squawk was in a different location and in another the chest radiograph did not show evidence consistent with pneumonia until 4 days after the squawk was detected. In the remaining patient a squawk was heard over the left posterior mid chest. The portable chest radiograph was interpreted as technically suboptimal due to the patient's body habitus. The clinicians caring for this patient made a diagnosis of pneumonia and treated him accordingly.

Interestingly, one patient who clearly had congestive heart failure on a number of occasions had a squawk when we examined him. On re-examining his record he also had a diagnosis of pneumonia on that day. Similarly, there were two patients with squawks (one with COPD and one with asthma) who, on the day that the squawks were noted, had acute febrile illnesses consistent with pneumonia. In one patient the presence of a squawk led to reinterpretation of the chest radiograph as showing an area of opacification consistent with pneumonia.

The squawks in this study all had a distinctive sound that is readily distinguished from crackles, rhonchi, rubs, and most wheezing noises. Occasionally wheezes can be short and have a similar sound, but this occurs rarely as an isolated finding in inspiration. The squawks in this study all had sinusoidal waveforms as illustrated in the time-amplitude plot shown in fig 1. The mean (SD) duration of these sounds was 64 (49) ms (range 16–228) and the mean (SD) frequency was 425 (110) Hz (range 200–667). These findings are similar to those of Earis *et al*.[3]

When a squawk is accurately identified, the question arises—what does it mean? In a patient who is not acutely ill, investigations to rule out hypersensitivity pneumonitis and the other fibrotic conditions mentioned above should be considered. In a patient with a clinical picture consistent with pneumonia, the presence of a squawk offers some objective evidence to support the diagnosis. It would seem reasonable to suggest that the patient should be followed up to be sure that the squawk disappears when the acute illness resolves to exclude the possibility that the acute illness was the mode of presentation of a chronic pulmonary disorder. Squawks can be helpful in providing evidence for pneumonia in lung areas where radiological visualisation may be suboptimal, such as below the dome of the diaphragm or in the retrocardiac region.

In summary, short inspiratory wheeze-like sounds are found in pneumonia. Other conditions that can cause them are chronic restrictive disorders but these are relatively uncommon compared with pneumonia. When there is no evidence of these restrictive disorders and an acute syndrome consistent

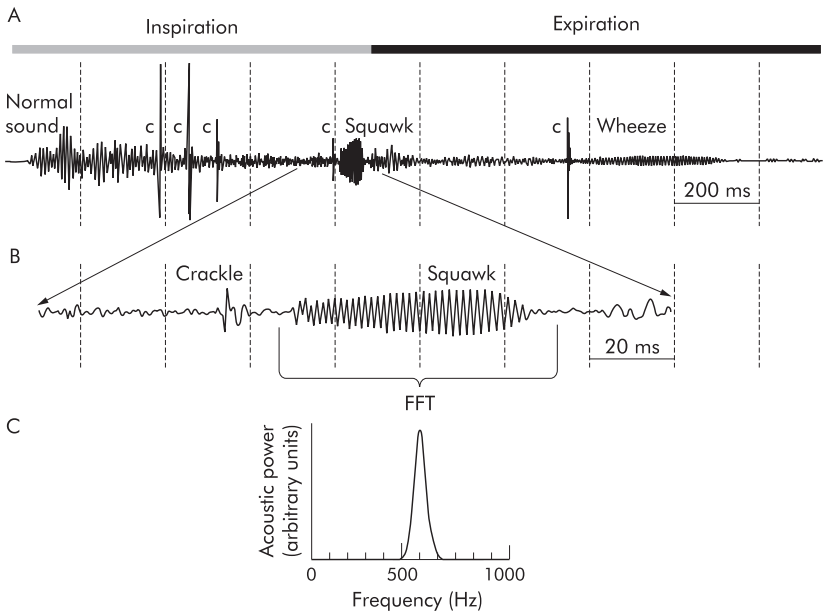

Figure 1 Time-amplitude plot of a sound recorded at the lung bases posteriorly (channel 14) in a patient with pneumonia. Waveforms are presented in both the unexpanded (A) and expanded (B) modes. (A) The unexpanded waveform shows one full breath. The solid bars above the unexpanded wave mark the respiratory cycle (the light bar indicates inspiration and the dark bar indicates expiration). The normal inspiratory sound can be seen as having almost random waveform fluctuations. Fine crackles (c) look like spikes on an unexpanded waveform. A squawk is present at the end of inspiration. One fine crackle and a wheeze are present during expiration. (B) The expanded squawk waveform exhibits monophonic sinusoidal fluctuations lasting approximately 60 ms. (C) The squawk sound in the frequency domain shows a single peak at 600 Hz. In this patient similar squawks appeared in three consecutive breaths during 20 s of deeper than normal breathing. They occurred approximately in the same location on the chest and at the same time in the respiratory cycle. These observations were typical of the squawks detected in our study. FFT = fast fourier transform.

with respiratory infection is present, squawks can provide relatively specific—although not very sensitive—evidence of pneumonia.

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BTS guidelines for the management of pleural infection

We have read the BTS guidelines for the management of pleural infection[1] and are concerned about the proposed antibiotic choices for the initial treatment of culture negative or pending pleural infection. Section 2.8 of the text and table 2 detail the antibiotic choices but, in our opinion, leave considerable gaps in antibacterial cover against the likely pathogens. In particular:

- Amoxycillin (text) is not reliably active against *Staphylococcus aureus*.
- Clindamycin has no activity against Gram negative aerobic organisms (especially not *Haemophilus influenzae* as mentioned in the text).
- Benzyl penicillin (table) rarely now has activity against *Staphylococcus aureus* and we suggest that relying on ciprofloxacin is unwise. In addition, this combination will not cover many anaerobic bacteria.
- We do not consider chloramphenicol is an appropriate agent to use in this category of patients in view of the serious side effect profile.
- Third generation cephalosporins such as ceftazidime and cefotaxime have unreliable activity against many anaerobic bacteria.
- Pneumococci are considerably less susceptible to ceftazidime than to other cephalosporins and penicillins[2]; the policy (table), however, suggests its use as a single agent.
- Piperacillin (text) is no longer available in the UK except in combination with a β-lactamase inhibitor.

We suggest that the antibiotic choices in the BTS guidelines for the management of pleural infection should be changed to the following:

- For community acquired pleural infection, either cefuroxime + metronidazole *or* co-amoxiclav *or* (for the penicillin/cephalosporin allergic individuals) clindamycin + ciprofloxacin, all administered intravenously. Oral treatment choices would be co-amoxiclav *or* clindamycin + ciprofloxacin.
- For hospital acquired infection, clinicians should seek guidance from the local microbiologists but the following choices would be reasonable in the interim: piperacillin + tazobactam *or* cefotaxime/ceftriaxone + metronidazole *or* meropenem.

Relating to the initial diagnosis of pleural infections, we were also concerned that mycobacteria were mentioned only once in the article. Pleural fluid has a poor yield for diagnosis of tuberculosis and more emphasis should have been placed on the routine use of pleural biopsy for histology and culture of tuberculosis which has much higher diagnostic rates. The algorithm should include the investigation of pleural tuberculosis.[3]

In conclusion, we would commend the inclusion of a medical microbiologist in discussions leading to guidelines dealing with the diagnosis and treatment of infections.

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Authors' reply

We would like to thank Dr Roberts and colleagues for their interest in the BTS guidelines on pleural infection[1] and for their thoughtful letter. In our view, guidelines (particularly the first set in an area) exist partly to stimulate a debate which subsequently better informs care.

Since the advisory regimens in the guidelines were first drafted (and they are ''advisory'' and to be used in line with local microbiological advice), the microbiology of pleural infection has been greatly clarified (not least by the joint BTS/MRC trial in pleural infection). This same work is also identifying high risk patient groups, clarifying drain type choice, and accurately defining intrapleural treatment. We have no doubt that these new data, as well as some of the points raised by Dr Roberts and colleagues, will strengthen the next revision of the BTS guidelines. The recent data show that only 10% of community acquired infections are staphylococcal, while 50% of hospital acquired infections are due to staphylococcal disease and 66% of these are MRSA infections. Thus, a regimen with limited staphylococcal cover may be appropriate in community infection (although thorough anaerobic cover is needed here), but isolated meropenem in hospital acquired infection (suggested by Roberts and colleagues) would be ineffective for 25% of patients in this setting. Here we might currently favour vancomycin + meropenem (or similar). The BTS/MRC trial suggests that about 50% of patients with hospital acquired pleural infection are currently receiving ineffective empirical antibiotics—emphasising the importance of clarifying this issue. The suggestion of a combination of clindamycin + ciprofloxacin in community acquired infection seems a particularly elegant improvement on the regimen of clindamycin alone advocated in some US centres and mirrored in our original suggestions.

We share the view that, when the pleural infection guidelines are next updated, a microbiologist should be on the drafting panel and not only included during peer review. The drafting panel for these guidelines was a compromise between size and inclusivity in all the therapeutic areas, since it had to cover all the pleural syndromes. This, for example, led to the absence of an oncologist for the malignant effusion guideline and a physician with particular skills in cystic fibrosis for the pneumothorax guideline (again peer review was the chosen method for including these specialists). On the plus side, this led to an efficient guideline generation process. We have previously encouraged the BTS to constitute separate groups for each of the guidelines as they come up for future review for just this reason.

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1 **Davies CWH**, Gleeson FV, Davies RJO, *et al*. BTS guidelines for the management of pleural infection. *Thorax* 2003;**58**(Suppl II):ii18–28.

Bronchodilator reversibility testing in COPD

In their paper on bronchodilator reversibility testing in COPD, Calverley and colleagues[1] come to the intuitively sensible conclusion that, in severe COPD, bronchodilator responsiveness is a continuous variable. However, this conclusion is based on an analysis in which the change in forced expiratory volume in 1 second (FEV_1) effected by inhalation of a bronchodilator aerosol is related to the baseline (that is, initial) level. As a result, the reported bronchodilator responses are subject to the error that can result from regression to the mean.[2] The error can be minimised by relating the change to the mean level instead of the initial level, and it would be reassuring to see the data expressed in this form.

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