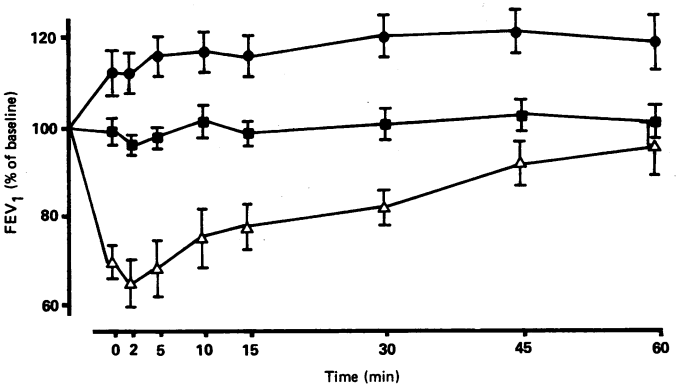
by more than 20% of the baseline value participated in the remainder of the study. On two occasions subjects inhaled single blind 4 ml solutions of isotonic saline and of nebulised isotonic ipratropium bromide free of benzalkonium chloride or edetic acid and had their FEV_1 measured over 60 minutes. On two further occasions they inhaled double blind 4 ml solutions of increasing concentrations of nebulised benzalkonium chloride (0·125 to 5·0 g/l) or edetic acid (0·25 to 10·0 g/l) until the FEV_1 had fallen by >20%. All solutions were nebulised by compressed air using an Inspiron nebuliser at a flow rate of 8 l/min. The aerosol was inhaled through a mouthpiece during tidal breathing from a starting volume of 4 ml in the nebuliser to dryness.

The airway response was expressed as percentage change in FEV_1 from the baseline value, and the differences between the challenge solutions were evaluated by analysis of variance and application of the Newman-Keul's test. The PC_{20} FEV_1 values (cumulative concentration of benzalkonium chloride or edetic acid causing a 20% fall in FEV_1) were obtained from each concentration-response curve and geometric mean values calculated for the group.

There was no significant difference between the baseline FEV_1 measurements on the different study days. After inhaling 4 ml Atrovent six patients developed bronchoconstriction with a mean (SEM) fall in FEV_1 of 35·3 (5·4)% two minutes after inhalation (figure). When these six subjects inhaled 4 ml preservative free ipratropium bromide solution all showed bronchodilatation, the mean FEV_1 increasing to 112·3 (4·9)% of baseline at two minutes (figure). Inhalation of 4 ml saline produced no significant change in airway calibre. The change in FEV_1 after inhaling Atrovent was significantly different from that after inhaling preservative free ipratropium bromide at all time points after inhalation ($p<0·05$) and from the response after saline for the first 30 minutes ($p<0·05$).



Changes in airway calibre after inhalation of nebulised Atrovent (△), preservative free ipratropium bromide (●) and saline (■) in the six asthmatic subjects in whom the FEV_1 fell >20% after inhalation of 4 ml solution of nebulised Atrovent. Each point represents the mean FEV_1 expressed as per cent of baseline, and each bar the SEM.

Inhalation of the preservatives administered separately produced dose related bronchoconstriction, which persisted for longer than 60 minutes. The cumulative geometric mean (range) PC_{20} FEV_1 was 0·30 g/l (0·13-2·0) for benzalkonium chloride and 2·40 g/l (1·2-12·8) for edetic acid.

Comment

This study shows that Atrovent nebuliser solution, the current isotonic formulation of ipratropium bromide, may cause bronchoconstriction in asthmatic subjects. This bronchoconstriction is due to the benzalkonium chloride and edetic acid in the solution, since both agents were potent bronchoconstrictor agonists when inhaled alone.

Benzalkonium chloride, a mixture of quaternary benzyldimethyl alkylammonium chlorides, may cause bronchoconstriction by releasing spasmogenic mediators from mast cells within the bronchial wall, since it has been shown to release histamine and 5-hydroxytryptamine from rat serosal mast cells.[2,3] Although the mechanism by which edetic acid causes bronchoconstriction is uncertain, it probably relates to its action as a chelator of calcium ions.[4,5]

All six subjects who developed pronounced bronchoconstriction with the isotonic Atrovent solution showed bronchodilatation after inhaling preservative free ipratropium bromide. These results suggest that an ipratropium bromide nebuliser solution free of preservatives is likely to be a more effective bronchodilator than the currently available solution. We recommend that nebuliser solutions should be made available without benzalkonium chloride, edetic acid, or other agents which may provoke bronchoconstriction in asthmatic patients.

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1 Mann JS, Howarth PH, Holgate ST. Bronchoconstriction induced by ipratropium bromide in asthma: relation to hypotonicity. *Br Med J* 1984;289:469.
2 Read GW, Kiefer EF. Benzalkonium chloride: selective inhibitor of histamine release induced by compound 48/80 and other polyamines. *J Pharmacol Exp Ther* 1979;211:711-5.
3 Coleman JW, Holgate ST, Church MK, Godfrey RC. Immunoglobulin E decapeptide-induced 5-hydroxytryptamine release from rat peritoneal mast cells. *Biochem J* 1981;198:615-9.
4 Downes H, Hirshman CA. Importance of calcium in citric acid-induced airway constriction. *J Appl Physiol* 1983;55:1496-1500.
5 Downes H, Hirshman CA. Calcium chelators increase airway responsiveness. *J Appl Physiol* 1985;59:92-5.

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Inability of trained nurses to perform basic life support

Effective basic life support within the first three to four minutes after a cardiac arrest increases the chance of survival. In hospital a large proportion of cardiac arrests occur on the wards, where the nurses have to perform basic life support before the arrival of the crash team in three to five minutes. Though it is essential that nurses can perform basic life support competently, there has been no report of the skills in basic life support of trained nurses in the United Kingdom. The first aim of this study was to assess the effectiveness of these skills in trained nurses and the second to examine the relation between self assessed skills and actual skills.

Subjects, methods, and results

Fifty three nurses (15 sister/charge nurses, 28 staff nurses, and 10 state enrolled nurses) were studied. They had been qualified for an average of 4·8 years (range 1-16 years) and had trained at 43 hospitals throughout the United Kingdom. All the nurses were attending an orientation course for newly appointed staff at the hospital. Nurses were tested without prior warning for two minutes on a manikin (recording Resusci-Anne (Laerdal)). Each nurse's performance was rated on a scale of 0-5 with a checklist derived from the Resuscitation Council (UK) guidelines 1984. Their experience with basic life support was determined by a questionnaire covering their grade, years since qualification, hours spent in resuscitation training, whether a manikin had been used in training, time since they had last attended a basic life support revision course, and number of cardiac arrests attended. They also estimated the percentage of all resuscitation attempts at the Royal Free Hospital in 1984 that had resulted in the patient being discharged alive. Nurses rated their confidence in being able to resuscitate a patient and how many points out of 10 they would gain on a test of resuscitation skills and knowledge where the average score was 5.

Nurses' basic life support skills and self assessed ability in relation to seniority

	All nurses (n=53)	Sisters/ charge nurses (n=15)	Staff nurses (n=28)	State enrolled nurses (n=10)
Total basic life support skills score*				
0	30	9	18	3
1	0	0	0	0
2	8	2	3	3
3	8	3	3	2
4	7	1	4	2
5	0	0	0	0
Self assessed ability† (mean (SD))	4·4 (1·9)	5·5 (1·8)	3·9 (1·6)	4·3 (2·1)

* Basic life support skills score=sum of 1=assessment, open airways, check breathing and pulse (AABC); 2=effective ventilation and compression (irrespective of ratio), 1=effective ventilation and compression (ratio between 4:1 and 9:1), 1=effective ventilation and compression (5:1 or 15:2) (maximum score=5).
† Self assessed ability assessed with a 9 point rating scale: 0=not at all confident in being able to resuscitate a patient, 8=extremely confident.

None of the 53 nurses performed basic life support adequately (score of 5)[1]; 30 were assessed as completely ineffective (score of 0) (table). None of the six measures of experience was related to basic life support skills. The nurses overestimated the success of resuscitation as an intervention, with the estimated mean percentage survival rate for hospital arrests over one year being 41·1% (SD 23·4%) (actual survival 10%). Nurses scoring highest on basic life support skills, however, estimated the procedure as significantly more successful

(mean 58·8 (23·1)) than those with lower scores (mean 37·8, (22·5); Mann-Whitney U test $Z=2·22$, $p<0·05$). Self assessed ability was not related to objectively assessed basic life support skills.

Sisters/charge nurses were significantly more confident than staff nurses about performing basic life support; (Kruskal-Wallis one way analysis of variance: χ^2 5·91, $p<0·05$; Mann-Whitney U test: $Z=2·34$); in fact, however, they were no more competent. Nurses who had attended more arrests were more confident about performing basic life support (Kruskal-Wallis one way analysis of variance: χ^2 15·55, $p<0·005$), although again they were no more competent. There was no relation between the post held and the number of arrests attended.

Comment

Our results show that the basic life support skills of nurses trained in the United Kingdom are as poor as those reported for nurses in the United States[2,3] and for preregistration and postregistration doctors.[4,5] Self assessed ability was unrelated to objectively assessed performance. Experience of attending cardiac arrests and seniority were associated with increased confidence but were not matched by an increase in skills. As nurses cannot accurately assess their own skills at basic life support, this study highlights the fact that compulsory retraining programmes are necessary, as those with poor skills will not necessarily seek further training.

1 Resuscitation Council. *Resuscitation for the citizen*. London: Resuscitation Council, 1984.
2 Gass EA, Curry L. Physicians' and nurses' retention of knowledge and skills after training in CPR. *Can Med Assoc J* 1983;128:550-1.
3 Kaye W, Mancini ME. Retention of cardiopulmonary resuscitation skills by physicians, registered nurses and the general public. *Crit Care Med* 1986;14:621-3.
4 Casey WF. Cardiopulmonary resuscitation: a survey of standards among junior hospital doctors. *J R Soc Med* 1984;77:921-4.
5 Skinner DV. Cardiopulmonary skills of preregistration house officers. *Br Med J* 1985;290:1549-50.

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Fibreoptic bronchoscopy and the use of antibiotic prophylaxis

McShane and Hone recently suggested that nasal intubation for anaesthesia causes bacteraemia and should be covered by antibiotics in patients at risk of endocarditis.[1] Fibreoptic bronchoscopy, however, which is usually carried out by introducing the bronchoscope through the nose, does not apparently lead to bacteraemia.[2,3] This discrepancy may be the result of differences in the time at which the various authors looked for bacteraemia; in the case of fibreoptic bronchoscopy the available evidence relates to the period after bronchoscopy rather than during bronchoscopy. We have looked for bacteraemia immediately after the start of fibreoptic bronchoscopy and during the procedure, a period which corresponds more closely to that chosen by McShane and Hone.

Patients, methods, and results

We studied 73 patients with respiratory disease undergoing bronchoscopy. Seven of the patients had recently taken antibiotics (ampicillin or amoxycillin in four cases, erythromycin in three), but the remainder had not taken antibiotics for at least one week. The bronchoscope (Olympus BF10) was passed through the nose after the nasopharynx had been sprayed with lignocaine (4%). Blood samples were taken immediately before and one to 10 minutes after the start of bronchoscopy. This period was chosen to cover bacteraemia which might occur on introducing the bronchoscope and also any subsequent bacteraemia caused by manipulating the instrument during the procedure. After preparation of the venepuncture site with 70% isopropyl alcohol 20 ml of blood was taken and divided between aerobic and anaerobic blood culture bottles (London Analytical and Biological Media Ltd). The bottles were incubated for 72 hours before being subcultured. A second and final subculture was performed at nine days. Transbronchial biopsy samples were not taken during any of the bronchoscopies.

One to two minutes after the start of bronchoscopy one of 22 patients had a positive culture. With increasing intervals, up to 10 minutes, only one further positive result was obtained from 51 patients. Four blood cultures taken before bronchoscopy were also positive (table). Of 292 blood culture bottles inoculated, eight gave positive results (2·7%); all isolates were organisms commonly found on the skin (coagulase negative staphylococcus, *Corynebacterium* sp) or in the environment (*Bacillus* sp). There was no significant difference between the number of positive cultures obtained before and after the start of bronchoscopy ($p>0·5$, χ^2 analysis with Yates's correction for small numbers).

Details of isolates obtained from five patients with positive blood cultures*

Case No	Time of blood culture in relation to the start of bronchoscopy: Before	After
1	*Bacillus* sp (1 bottle)	No growth
2	Coagulase negative Staphylococcus (both bottles)	Coagulase negative Staphylococcus (1 bottle)
3	*Corynebacterium* sp (1 bottle: 2nd subculture)	No growth
4	Coagulase negative Staphylococcus (1 bottle)	No growth
5	No growth	Coagulase negative Staphylococcus (both bottles)

*All isolates were obtained from the first subculture unless otherwise indicated.

Comment

The nasal passages are usually colonised with coagulase negative staphylococci and *Corynebacterium* spp, organisms which are also common contaminants of blood cultures. Other organisms likely to be found in the nasal flora include *Staphylococcus aureus, Neisseria* spp, and *Haemophilus* spp, none of which are common causes of bacterial endocarditis. In the study of McShane and Hone half of the organisms isolated were *Staph epidermidis* (a coagulase negative staphylococcus), but they did not consider the possibility that any of these were contaminants.[1] Of the other isolates, most were organisms which rarely cause endocarditis: *H influenzae, Corynebacterium hofmannii,* and *Streptococcus pyogenes*. McShane and Hone isolated an α haemolytic streptococcus from only two patients; in both cases this was *Str sanguis*, a common cause of bacterial endocarditis. Their failure to take control blood cultures before nasal intubation means that these isolates may have represented pre-existing bacteraemia in patients with poor oral hygiene.

We did not find any difference between the number of positive cultures obtained before bronchoscopy and the number obtained during the procedure. The number and nature of isolates were entirely consistent with our usual experience of contaminated blood cultures. Whether or not the risk of bacteraemia is likely to be appreciably increased by taking transbronchial biopsy samples remains to be determined.

Our results support the view that antibiotic prophylaxis is not warranted in patients at risk of endocarditis when undergoing fibreoptic bronchoscopy. Furthermore, we believe that there is insufficient evidence to justify the use of such prophylaxis in patients undergoing nasal intubation.

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1 McShane AJ, Hone R. Prevention of bacterial endocarditis: does nasal intubation warrant prophylaxis? *Br Med J* 1986;292:26-7.
2 Kane RC, Cohen MH, Fossiecki BE Jr, Tvardzik AV. Absence of bacteremia after fiberoptic bronchoscopy. *Am Rev Respir Dis* 1975;111:102-4.
3 Pereira W, Kovnat DM, Kahn MA, Iacovino JR, Spivack ML, Snider GL. Fever and pneumonia after flexible fiberoptic bronchoscopy. *Am Rev Respir Dis* 1975;112:59-64.

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