

Intervals between arteriography, start of treatment with captopril, and diagnosis of occlusion of renal arteries in three patients with renovascular hypertension. Plasma creatinine concentrations were measured at beginning and end of indicated periods

Case No	Age (years)	Sex	Stenosis (%)	Arteriography to start of treatment with captopril		Start of treatment with captopril to occlusion of renal artery	
				Intervals (weeks)	Plasma creatinine ($\mu\text{mol/l}$)	Intervals (weeks)	Plasma creatinine ($\mu\text{mol/l}$)
1	62	M	75-90	1	89-89	13	89-120
2	64	M	75-90	12	201-208	13	208-270
3	51	M	> 90	16	122-127	21	127-148

Conversion: SI to traditional units—Creatinine: $1 \mu\text{mol/l} \approx 0.01 \text{ mg/100 ml}$.

with captopril, chlorthalidone, and atenolol (13 weeks) decreased blood pressure to 155/88 mm Hg and increased plasma creatinine concentration to $120 \mu\text{mol/l}$ (1.41 mg/100 ml). Arteriography, performed immediately before percutaneous transluminal angioplasty, showed the left renal artery completely occluded.

Case 2—A 64 year old man with recent hypertension (210/126 mm Hg) seemed to have 75-90% stenosis of the left renal artery. Treatment with captopril and chlorthalidone (13 weeks) decreased blood pressure to 178/96 mm Hg. Renal function was slightly impaired before treatment began (creatinine clearance 77 ml/min). Plasma creatinine concentration rose from 200 to $270 \mu\text{mol/l}$ (2.26 to 3.05 mg/100 ml) during treatment. Arteriography before percutaneous transluminal angioplasty showed the left renal artery completely occluded after treatment.

Case 3—A 51 year old man after two years of hypertension (170/110 mm Hg) seemed to have severe stenosis (>90%) of the right renal artery and impaired renal function (plasma creatinine concentration $129 \mu\text{mol/l}$ (1.46 mg/100 ml), creatinine clearance 86 ml/min). Treatment (16 weeks) with metoprolol, hydrochlorothiazide, and amiloride did not affect blood pressure or renal function. Treatment with captopril and chlorthalidone (21 weeks) decreased blood pressure (118/84 mm Hg) and increased plasma creatinine concentration to $148 \mu\text{mol/l}$ (1.67 mg/100 ml). We decided to perform percutaneous transluminal angioplasty. Arteriography before this, however, showed a completely occluded right renal artery.

Comment

It is difficult to prove that treatment with captopril contributed to the occlusion of the severely stenotic renal arteries in these patients, especially as two used β blockers, which are also said to provoke renal artery thrombosis.¹ We do, however, consider that captopril was a precipitating factor because: firstly, intervals were short between arteriography, start of treatment, and diagnosis of renal artery occlusion; secondly, plasma creatinine concentrations increased during treatment but not before it began; and, thirdly, blood pressure responded well to captopril, making it less probable that rapid progress of the atherosclerotic lesion because of persisting high blood pressure would have lead to occlusion of the renal arteries.

Reports on renal artery thrombosis during inhibition of angiotensin converting enzyme are scanty,^{2,3} but inhibitors of angiotensin converting enzyme are thought to provoke renal artery occlusion.³ Hollenberg, however, found no renal artery thrombosis in 269 patients treated with captopril.⁴

These are the only such cases known to the manufacturers of captopril (personal communication, Dr A J Spijker, Squibb Benelux),^{2,3} and the Netherlands Centre for Monitoring Adverse Drug Reactions has not received any report on renal artery thrombosis and inhibition of angiotensin converting enzyme (personal communication, B H C Stricker).

The blood pressure lowering effect of long term treatment with captopril is said to guide prediction of curability of renovascular hypertension. The experiences in our patients made us and others³ reluctant to prescribe inhibitors of angiotensin converting enzyme for more than one month, especially when severe renal artery stenosis is present.

1 Shaw AB, Gopalka SK. Renal artery thrombosis caused by antihypertensive treatment. *Br Med J* 1982;285:1617.

2 Williams PS, Hendy MS, Ackrill P. Captopril-induced acute renal artery thrombosis and persistent anuria in a patient with documented pre-existing renal artery stenosis and renal failure. *Postgrad Med J* 1984;60:561-3.

3 Tillman DM, Malatino LS, Cumming AMM, et al. Enalapril in hypertension with renal artery stenosis: long-term follow up and effects on renal function. *Journal of Hypertension* 1984;2(suppl 2):93-100.

4 Hollenberg NK. Medical therapy of renovascular hypertension: efficacy and safety of captopril in 269 patients. *Cardiovascular Reviews and Report* 1983;4:852.

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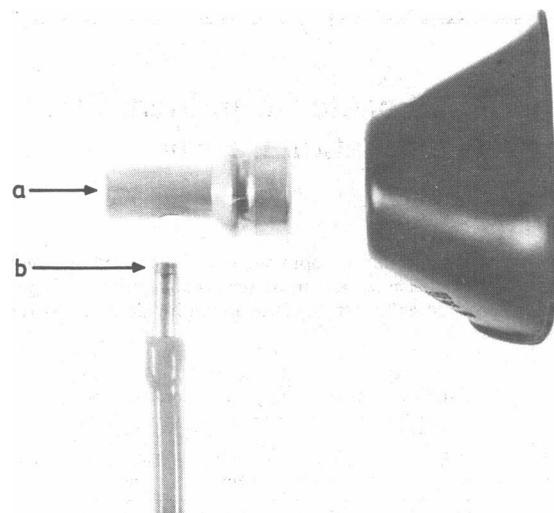
Cross infection with *Streptococcus pneumoniae* through a Resuscitaire

Transmission of *Streptococcus pneumoniae* between members of the same family and between patients in the same ward has been reported.¹⁻³ We report here the transmission of *S pneumoniae* type 6 between two infants resuscitated consecutively with the same piece of equipment (Resuscitaire) 48 hours apart.

Case reports

A 31 year old woman was admitted in labour with ruptured membranes. Caesarean section was performed after a prolonged and difficult labour and fetal distress. The baby was asphyxiated at birth with an Apgar score of 3 at one minute and 9 at five minutes, thus requiring immediate intubation and resuscitation. Despite ventilation and a high dosage regimen (50 mg/kg every eight hours) of benzyl penicillin and gentamicin (2.5 mg/kg every 12 hours) the baby died after eight hours.

A gastric aspirate taken at birth yielded *S pneumoniae* type 6. At necropsy severe bronchopneumonia was found and *S pneumoniae* type 6 was isolated from the bronchi and lung tissue. Twenty four hours after delivery the mother was



Resuscitaire and its components, including T piece connector (a) and thread of T piece (b).

clinically unwell. A high vaginal swab and midstream specimen of urine yielded *S pneumoniae* type 6. She then developed a wound infection 48 hours post-operatively from which *S pneumoniae* type 6 was also isolated. She was treated with parenteral benzylpenicillin 2 MU every six hours for six days, made a satisfactory recovery, and was discharged. Follow up high vaginal swab specimens yielded negative results.

Forty eight hours after the first infant had been resuscitated a second baby born by caesarean section required intubation and ventilation for fetal distress. After 12 hours this infant developed signs of infection, and a gastric aspirate taken at birth grew *S pneumoniae* type 6. High dose penicillin and gentamicin were started immediately after birth, and the baby gradually improved and made an uneventful recovery. The second mother was screened for pneumococci, but no pathogens were isolated.

Extensive investigations were carried out, including bacteriological screening of all attending staff, equipment in the labour ward, and the environment. It was found that these two babies had been ventilated consecutively on the same Resuscitaire. In the interval between these two births three infants had been born

who required only oxygen for a brief period, and the same facemask had been used. None of them had developed any signs of infection. On the initial screening *S pneumoniae* type 6 was isolated from the T piece connector of the face mask (figure). Repeat examination of the same facemask and connector seven days later yielded *S pneumoniae* type 6 from the thread of the T piece, which was contaminated with secretions encrusted in it.

Comment

It was standard practice in the labour ward to wash the facemask in one piece with soapy water, then rinse and dry it. The T piece connector was never dismantled to be cleaned. On this particular day the ward was exceptionally busy, and it is possible that there was a lapse in the cleaning methods. It became clear from the first bacteriological sampling of the face mask that *S pneumoniae* could survive routine washing and drying, as presumably this was done after use on each baby, but the length of time that the pneumococcus could survive (seven days) in the thread of the T piece connector was surprising. The problem of cleaning the masks was overcome by ordering three autoclavable circuits for each Resuscitaire, which were dismantled after use, washed, and sent for sterilisation.

This is an unusual route for transmission, and to our knowledge cross infection with *S pneumoniae* through resuscitation equipment has not previously been documented. This report is also a salutary reminder that scrupulous attention is required when dealing with any equipment that comes into intimate contact with patients, particularly infants.

1 Fenton A, Spencer RC, Savill S, Grover A. Pneumococcal bacteraemia in mother and son. *Br Med J* 1981;287:259-60.

2 Shaw PJ, Robinson DL, Watson JG. Pneumococcal infection in a mother and infant. *Lancet* 1984;ii:47.

3 Davies AJ, Hawkey PM, Simpson RA. Pneumococcal cross infection in hospital. *Br Med J* 1984;288:1195.

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Prevention of bacterial endocarditis: does nasal intubation warrant prophylaxis?

Although there is no uniform opinion about the need for antibiotic prophylaxis in patients susceptible to bacterial endocarditis, it is generally accepted that procedures known to cause a high incidence of bacteraemia

incidence of bacteraemia of 12% after such intubation in children,³ but no similar information exists for adults. This study attempted, firstly, to determine the incidence of bacteraemia in adults after nasal intubation; secondly, to examine the influence of the type of endotracheal tube used on the incidence; and, thirdly, to determine whether the volume of blood sampled influenced the yield of positive cultures, as has recently been reported.⁴

Patients, methods, and results

After gaining approval from the anaesthetic research committee we studied 71 patients (45 women, 26 men) presenting for elective surgery requiring nasotracheal intubation. All were free of cardiac disease and did not have evidence of sepsis or a current or recent history of treatment with antibiotics. Swabs from the anterior nares were taken from 48 patients before induction. Anaesthesia was induced by thiopental sodium 4-6 mg/kg, and nasotracheal intubation with a Magill tube was facilitated by the use of succinylcholine chloride 1 mg/kg and a Magill's forceps. One to three minutes after intubation and minimal inflation of the cuff to a "no leak" level 6 ml of blood was sampled from an antecubital vein after preparation of the skin with 70% isopropyl alcohol. The "Bactec system" of blood culture (Johnston Laboratories, Towson, Maryland, USA) was used, with 3 ml of blood being put into both aerobic and anaerobic bottles. In 49 patients 36 ml of blood was sampled, so that 30 ml could be inoculated into 120 ml of nutrient broth containing 0.05% Liquoid. All bottles were screened regularly for a week and subcultured, and identification was performed when appropriate. Statistical analysis was by χ^2 test.

The mean age of the patients was 31. Twelve (17%) positive blood cultures were obtained (table). Use of a cuffed endotracheal tube in 53 patients was associated with positive blood cultures in 11 (21% incidence), while only one positive culture was obtained after use of a non-cuffed tube in 18 patients ($p=0.06$). The 30 ml samples cultured in 49 cases detected four organisms undetected by the 3 ml samples but failed to detect organisms in two cases in which the 3 ml bottles yielded positive cultures. Nasal swab in 48 patients detected normal nasal flora.

Comment

Our finding of a 17% incidence of bacteraemia after nasal intubation shows that this procedure should be covered by antibiotic prophylaxis in susceptible patients. The incidence after use of cuffed tubes (21%) is more important as this is the type of endotracheal tube most commonly used.

Staphylococci are the most commonly isolated organisms in infective endocarditis occurring after cardiac surgery.¹ Though the skin of the patient or surgical team is thought to be the source,² nasal intubation might also be a source, as it is commonly used for postoperative ventilation of these patients. Our results, showing staphylococci to be part of the normal flora of the nose and to have caused six cases of bacteraemia, support this view.

Although the additional 30 ml samples of blood increased the detection of organisms compared with standard techniques, this difference was not significant. Thus the greatest benefit is seen when 30 ml sampling is used in conjunction with standard techniques.

We thank our anaesthetic, surgical, nursing, and laboratory colleagues for their help and cooperation.

Details of 12 patients with positive blood cultures

Case no	Age (years)	Sex	Tube size (mm)	Results of blood samples		Results of nasal swabs
				2×3 ml	30 ml	
1	56	M	8.0	<i>Haemophilus influenzae</i>	Not done	Not done
2	18	F	6.5	<i>Corynebacterium hofmannii</i>	Not done	Not done
3	35	F	7.0	<i>Streptococcus sanguis</i>	Not done	<i>Staph epidermidis</i>
4	26	M	8.0	<i>Staphylococcus epidermidis</i>	<i>Staph epidermidis</i>	<i>Staph epidermidis</i> , <i>Staph aureus</i>
5	14	F	6.5	<i>Staph epidermidis</i>	<i>Staph epidermidis</i>	<i>Staph epidermidis</i> , <i>diphtheroids</i>
6	18	M	8.0	<i>Staph epidermidis</i>	<i>Staph epidermidis</i>	<i>Staph epidermidis</i> , <i>Staph aureus</i>
7	42	M	7.0*	<i>H influenzae</i>	<i>H influenzae</i> ; group F β -haemolytic streptococci at 8 days	<i>Staph epidermidis</i>
8	19	F	7.0	<i>Staph epidermidis</i>	No growth	<i>Staph epidermidis</i>
9	18	M	8.0	<i>Str sanguis</i> (anaerobic only)	No growth	<i>Staph epidermidis</i>
10	24	M	8.0	No growth	<i>Staph epidermidis</i>	Not done
11	21	F	7.0	No growth	<i>Staph epidermidis</i>	<i>Staph epidermidis</i>
12	20	F	7.0	No growth	<i>Str pyogenes</i>	<i>Staph epidermidis</i>

*Cuffless tube; all others were cuffed tubes.

should be covered with antibiotics.^{1,2} In otherwise clean surgery, however, little information exists on whether the mode of anaesthesia used ever causes appreciable bacteraemia, thereby warranting prophylaxis. Nasal intubation is widely used in both surgery and intensive care. Berry *et al* showed an

1 Shulman ST, Amren DP, Bisno AL, *et al*. Prevention of bacterial endocarditis. A statement of health professionals by the committee on rheumatic fever and infective endocarditis of the council on cardiovascular disease in the young. *Circulation* 1984;70:1123-7.

2 Working Party of the British Society for Antimicrobial Chemotherapy. The antibiotic prophylaxis of infective endocarditis. *Lancet* 1982;ii:1323-6.