produced by a V-Z infection.^{1 2} V-Z titres were cited for only one of Dr Wilson's cases and were in keeping with recent V-Z infection.

I would agree with Dr B E Juel-Jensen (22 October 1977, p 1086) that V-Z is a much more likely cause of Bell's palsy than herpes simplex.

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¹ Ross, C A C, Sharpe, J H S, and Ferry, P, *Lancet*, 1965, 2, 708.

² Schmidt, N J, Lennette, E H, and Magoffin, R L, *Journal of General Virology*, 1969, 4, 321.

Antibiotic resistance in *Streptococcus pneumoniae* and *Haemophilus influenzae*

SIR,—We would like to reply to Dr M Rahman's letter (19 August, p 569) concerning the results of the survey of antibiotic resistance in respiratory pathogens (24 June, p 1657). We agree with him that it is important to know the incidence of antibiotic resistance in a particular locality, but the limitations of the data should be recognised if the information is not derived from a study of large numbers of strains. This is illustrated by Dr Rahman's results if they are subjected to statistical analysis. In the table we have added 95% confidence limits¹ to the incidence of resistance in strains obtained from Nottingham to the overall results obtained in the study and to the incidences of resistance reported from Sutton-in-Ashfield. Contrary to the conclusions of Dr Rahman the results from Sutton-in-Ashfield conform to the national averages and are similar to the results from Nottingham, and the incidences of resistance in Sutton-in-Ashfield did not change materially between 1977 and 1978.

We have also included in the table the results reported by Dr A P Gillett and his colleagues from Birmingham (22 July, p 278). These authors record a significantly higher incidence of ampicillin resistance in *Haemophilus influenzae* than the national average derived in our survey. As all of their isolates were obtained from children (the majority from the respiratory tract) it was suggested by them that the difference in the results may have been explained by a relatively higher incidence of strains of type b. We studied 206 isolates derived from children under 10 years of age. One hundred and eleven of these strains were isolated from respiratory tract sources and only seven (6.3%) of them were capsulated (consisting of four (3.6%) of type b, one of type e, and two of type f), a serotype distribution similar to that reported by Alexander.² It is rather unlikely that the prevalence of typable strains in Dr Gillett's study would have diverged sufficiently from these results to have explained the

discrepancy in the incidence of antibiotic resistance.

It is possible that the incidence of ampicillin resistance may have increased in the United Kingdom since our survey was undertaken. However, it may also be that the incidence of β -lactamase producers reported by Dr Gillett and his colleagues was an overestimate. Firstly, they have compared the zones of inhibition exhibited by haemophili around a 2- μ g ampicillin disc with those obtained with the Oxford staphylococcus. Using the methods described in our survey report it was found that the diameters of inhibition exhibited by ampicillin-sensitive, non- β -lactamase-producing strains of *H influenzae* averaged 26 mm (range 19-33 mm), and few strains produced zones of inhibition as large as did the Oxford staphylococcus (average 31 mm diameter). Secondly, the phenol red/penicillin technique³ for the detection of β -lactamase production may occasionally be liable to misinterpretation, depending on the form of the penicillin substrate used. Escamilla describes the use of buffered penicillin G (benzylpenicillin) for this test, a result being recorded as positive if the indicator registers an intense yellow colour and negative if the colour remains unchanged or turns a red or pale pink colour. If unbuffered penicillin is used further intermediate colours varying from orange to yellow may be occasionally obtained with strains that have been shown to be non- β -lactamase-producers by other methods.

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¹ *Documenta Geigy Scientific Tables*, 7th edn, p 85. Macclesfield, Geigy Pharmaceuticals.

² Alexander, H E, *American Journal of Diseases of Children*, 1943, 66, 160.

³ Escamilla, J, *Antimicrobial Agents and Chemotherapy*, 1976, 9, 196.

Antibiotics in endocarditis

SIR,—It is with some diffidence that I would gently take issue with Dr Celia Oakley on a few points in her otherwise useful article on endocarditis in your excellent series on the use of antibiotics (12 August, p 489).

Firstly, at a time when medical microbiologists and clinicians world wide appear to have agreed that the serum bactericidal level during therapy for endocarditis should have a titre of not less than 1:8 it is distressing to see in print a recommendation of 1:6.

Secondly, one wonders why the aminoglycoside antibiotics should be an exception when it comes to the intravenous route of administration. Toxicity in this group is not related to the brief peak found after intravenous use but rather to a prolonged high level—or, indeed, a

prolonged low level indicating accumulation of the drug. It is for this reason that both peak and trough levels of aminoglycoside antibiotics should be assayed daily, or at least on a regular basis. But neither of these identifies the unpredictable so-called "dampened" response of unknown aetiology.

Thirdly, her statement that "a combined fungicidal/antistaphylococcal skin cream provides further protection" is unsubstantiated in clinical fact and furthermore is likely to lead to a potentially dangerous complacency on the part of ward staff—if not of the clinician herself.

Fourthly, one doubts the wisdom of stopping the aminoglycoside component of combined therapy against viridans endocarditis once the blood culture reports are available. The decision whether or not to stop the aminoglycoside component is normally based on the clinical response, the relative minimum inhibitory concentrations, the serum bactericidal level (as mentioned above), and lastly the demonstration or otherwise of synergy.

Finally, why not use probenecid to boost penicillin concentrations in cases of endocarditis? There is no doubt about the renal blockade effect towards penicillin, and intermittent bolus intravenous doses of penicillin still raise the peak concentrations even further, but the whole curve is correspondingly higher than in the absence of probenecid.

Not all medical microbiologists, nor indeed clinicians, will necessarily agree with all the above comments, but these are the principles normally employed in treating such patients at St Vincents' Hospital, Melbourne.

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* * * We sent a copy of this letter to Dr Oakley, whose reply is printed below.—ED, *BMJ*.

SIR,—I would like to thank Professor Stratford for commenting on my article and I agree with his comments that not all medical microbiologists, nor clinicians, will necessarily agree with all his comments. It is nice to hear of the principles employed at St Vincent's Hospital in Melbourne although they differ a little from some of ours.

To take his first point, the recommendation of a serum bactericidal level during therapy of 1:6 rather than not less than 1:8 is not a very basic disagreement. When treating with penicillin, which is essentially a non-toxic drug, it is usual greatly to exceed the titre of 1:8 (even without probenecid), while when using an aminoglycoside one is inhibited by the toxicity of the drug from attaining more than the recommended peak and trough levels which he has also commented on.

With regard to the route of administration of aminoglycosides, I have used both the intravenous and the intramuscular routes but it has been my impression that toxicity in the elderly has been very much more frequent when giving bolus injections intravenously and I have attributed this possibly to the higher peak levels and returned to the intramuscular route in consequence.

The "potentially dangerous complacency" which the use of a fungicidal/antistaphylococcal skin cream may provoke is as unsubstantiated a clinical fact as the protection which it provides against secondary invaders! As I

Tetracycline resistance in Str pneumoniae and ampicillin resistance in H influenzae

Centre	<i>Str pneumoniae</i>		<i>H influenzae</i>	
	No of isolates	% resistant to tetracycline (95% confidence limits)	No of isolates	% resistant to ampicillin (95% confidence limits)
Birmingham	37	2.7 (0.07-14.6)	69	0 (0-5.21)
Nottingham	27	14.8 (4.19-33.73)	94	1.1 (0.03-5.79)
Total for all centres in survey	866	6.8 (5.13-8.5)	952	1.6 (0.93-2.74)
Sutton-in-Ashfield:				
1977	33	9 (1.92-24.33)	80	0 (0-4.5)
1978	50	6 (1.25-16.55)	116	2.5 (0.57-7.76)
Birmingham Children's Hospital, 1978			168	7.7 (4.13-12.75)