

no antibodies to brucella when tested 9 months later is evidence against a common source of infection. Brucellosis in domestic animals has not been recorded in Sweden since the 1950s, and human brucellosis acquired in this country has not been reported since then. The possibility of coital transmission of brucella has long been suggested, but only recently has brucellosis been isolated from human spermatozoa.³

We thus conclude that the woman was probably infected by her boyfriend, possibly by sexual intercourse during genital manifestation of the disease.

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Capnocytophaga canimorsus infection after dog-bite

SIR,—Dr Gallen and Dr Ispahani (Feb 2, p 308) report a case of fulminant *Capnocytophaga canimorsus* septicaemia. We have seen another case of severe *C canimorsus* disease, in which immediate suspicion of the infection made prompt and adequate treatment possible.

A 74-year-old man with cardiac failure (New York Heart Association class III-IV) was bitten by a dog. Signs of local infection then developed, and he was started on roxithromycin 150 mg twice daily (allergy to penicillins was suspected). The local reaction subsided, but treatment was discontinued after three doses because of diarrhoea. Fever, lethargy, and increasing confusion developed over the next two days. On admission several days after the bite the patient was unresponsive to verbal stimuli, but reacted with agitation to painful stimuli. Temperature was 37.3°C, pulse 108/min, and blood pressure 110/70 mm Hg. Lumbar puncture showed 240 leucocytes/ml (80% granulocytes) and few gram-negative rods, whose morphology and gliding motility raised the possibility of *C canimorsus*. Treatment with rapidly increasing intravenous doses of penicillin (the drug of choice¹) was instituted. The diagnosis was confirmed by positive cultures from CSF. A lumbar puncture 20 h after admission showed more than 2000 leucocytes/ml and was strongly positive for endotoxin by the limulus test; blood culture and this test on serum were negative. The patient was kept on a ventilator for 5 days and slowly improved. Penicillin 5 MU three times daily was given for 15 days, but heart failure and urinary tract infections delayed discharge until 67 days after admission.

This case confirms that *C canimorsus* infections typically follow dog-bites, and that patients with no obvious immunocompetence can be infected. In our patient early antibiotic treatment was discontinued, and this may have contributed to the dissemination of the disease. We fully agree with Gallen and Ispahani that early antibiotic treatment and debridement are important.

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Detection of *Chlamydia pneumoniae*

SIR,—*Chlamydia pneumoniae* is a common cause of bronchitis and pneumonia,¹ but diagnosis is difficult because of non-specific clinical presentation, poorly defined serodiagnostic criteria, and lack of availability of both culture and serodiagnostic tests. Preliminary evidence suggests that enzyme immunoassay (EIA) and direct fluorescent antibody (DFA) techniques for identification of *C pneumoniae* may be useful.²

To assess the minimum number of inclusions detectable by these techniques, we inoculated serial dilutions of *C pneumoniae* elementary bodies onto McCoy cell monolayers (in triplicate) and incubated them for three days. One monolayer at each dilution was then stained with chlamydial fluorescein-conjugated lipopolysaccharide antibody and inclusions were counted. A second monolayer was swabbed and tested with a commercially available EIA ('STD-EZE', Abbott). The third monolayer was swabbed, rolled onto a glass slide, and tested by a commercially available DFA test ('Microtrak', Syva, USA) with lipopolysaccharide rather than major outer-membrane protein fluorescein-conjugated antibody. *C pneumoniae* inclusion counts with EIA and DFA for serial dilutions are given below:

Log ₁₀ dilution	Inclusion count	EIA results (cut-off 0.234)	DFA results
-2	> 675	> 3 (reactive)	Many (reticular bodies)
-3	70	0.779 (reactive)	Few
-4	15	0.254 (reactive)	Rare
-5	0	0.012 (NR)	Negative
-6	0	0.082 (NR)	Negative
-7	0	0.084 (NR)	Negative

NR = non-reactive.

There was good specificity for both EIA and DFA tests compared with inclusion counting, and both techniques could detect about twelve inclusions per specimen. These preliminary results suggest that chlamydial EIA and DFA tests should be studied further.

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Effect of massive dose vitamin A on morbidity and mortality in Indian children

SIR,—We wish to draw K. Vijayaraghavan and colleagues' (Dec 1, p 1342) attention to one or two points. In their double-blind trial, after the administration of placebo, the prevalence of night blindness and Bitot spots decreased significantly from 6% to 2.9%. The reason for this reduction was not explained.

On the basis of Vijayaraghavan and colleagues' table III (morbidity cases) and the percentages of patients who received zero, one, or two doses of vitamin A or placebo given in their results section, we have calculated the morbidity rate according to dose (table). Morbidity at zero dose for the placebo group is greater than that for the treatment group ($p = < 0.005$). The baseline morbidity in the placebo group was probably high. After one dose of vitamin A morbidity increased in the treatment group to 87% but did not change in the placebo group. Why should morbidity increase drastically after one dose of vitamin A? The treatment group might not have been homogeneous. We also find perplexing the fact that morbidity in the placebo group increased by nearly 26% from one to two doses.

Your editorial in the same issue (p 1350) states that the field workers who were engaged in the distribution of vitamin A or placebo provided information about management of illness with special reference to diarrhoea, respiratory infection, and measles.

MORBIDITY STATUS ACCORDING TO DOSE OF VITAMIN A OR PLACEBO*

	Vitamin A	Placebo
No of doses		
0	397/615 (65%)	638/889 (72%)
1	2274/2615 (87%)	1857/2587 (72%)
2	4405/4461 (99%)	4511/4608 (98%)
Total	7076/7691	7006/8084

*Cases/recipients (aged 1-5 yr).