determine β -lactamase production in 267 presumptive gonococcal isolates. Seven organisms had MICs to penicillin of > 2.0 mg/l, the remainder having MICs to penicillin of 0.03-1.0 mg/l. The Intralactam test identified the seven organisms with MICs of > 2.0 mg/l as β -lactamase producers, and none of the other 260 isolates gave a positive result with the test.

Due to Australia's proximity to south-east Asia, where β -lactamase producing *N. gonorrhoeae* strains are frequently encountered, we occasionally see such organisms in patients returning from these countries. There have also been some β -lactamase producing strains causing infections contracted locally. The standard treatment at the Venereal Diseases Clinic in Sydney is amoxycillin, 3 g orally, preceded by probenecid. This is highly effective except in the case of β -lactamase producing strains. For this reason we feel that rapid detection of β -lactamase production is desirable so that antibiotic treatment can be modified accordingly. In our experience, Intralactam strips provide a rapid, simple method for detecting β -lactamase production in *N. gonorrhoeae*.

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A human strain of *Campylobacter fetus* subsp. *intestinalis* grown at 42°C

A recent report of an infection with *Campylobacter fetus* subsp. *intestinalis* in a patient with multiple myeloma¹ leads us to mention several unusual features of the case. The strain was first isolated from a presumably traumatic joint. A blood culture was requested following that isolation. Tryptic Soy Broth and Thio-glycollate Medium (w/o Indicator) bottles (Pfizer Inc), inoculated with 5 ml blood each, remained clear (both contained

12 v/v % CO₂) but the organism grew in a one-day subculture to chocolate agar at 37°C in 8% CO₂. Most remarkable, however, was the fact that the *C. fetus* isolate grew at both 25 and 42°C. Since it proved to be resistant to nalidixic acid (no zone around the 30 µg disc) and grew in 1% glycine broth the identification of *C. fetus* subsp. *intestinalis* was established.²

Such strains have previously been reported from ovine sources³ but not from man. Conventional wisdom has it that the subspecies *intestinalis* and *fetus* (so far never isolated from man) grow at 25°C but not at 42°C while the subspecies *jejuni* grows at 42°C but not at 25°C. Our finding prompts us to re-emphasize that growth at 25°C is a more reliable characteristic in the differential diagnosis of *C. fetus* than growth at 42°C.² For the differentiation of clinical strains, we therefore recommend growth at 25°C and susceptibility to nalidixic acid rather than growth at both 25°C and 42°C.

We also want to emphasize the importance of 'blind' subcultures of blood culture bottles to suitable media, such as chocolate agar in a CO₂ atmosphere. It is rare to see turbidity due to *C. fetus* in blood culture media, presumably because of an inadequate growth atmosphere in commercial bottles.²

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Is there a place for the GammaCoat (¹²⁵I) Gentamicin Radioimmunoassay Kit in a routine clinical pathology laboratory?

Waterworth¹ has pointed out that a delay in obtaining results detracts from the value of a gentamicin assay service. In an attempt to minimise this delay a variety

of rapid methods have been devised. All of these have their protagonists, but none has yet achieved general acceptance. Included among these rapid methods is radioimmunoassay in a number of forms.²⁻⁵ Radioimmunoassay techniques are simple, sensitive, and specific.² Thus, when a kit, the GammaCoat (¹²⁵I) Gentamicin Radioimmunoassay Kit (Clinical Assays Cat. No. CA-540), recently became available in this country, we made a preliminary assessment of it.

Paired (pre- and post-dose) sera from patients receiving gentamicin therapy, either alone or in combination with other antibiotics, were assayed by our current overnight plate diffusion method and then, together with standards used to calibrate this method and controls of varying values made up in the laboratory, reassayed blind using the GammaCoat (RIA) kit. The principle of the GammaCoat technique and the recommended procedure for use of the kit are detailed in the instruction booklet. We followed the recommendations except that, for reasons of economy in the trial, only one tube per standard, control, or sample was used.

Altogether 109 samples were analysed by both methods. These comprised 15 standards, 12 controls, and 41 pairs of sera. The correlation between the results obtained by RIA and plate diffusion are shown in Figures 1 and 2. The linear correlation coefficient (r) for the standard/control results was +0.963 and for the patients' sera was +0.961. A slightly better correlation was obtained using a quadratic model (parabola) in which y (value by RIA method) = $-1.22 + 1.49x - 0.038x^2$ (where x = value by plate diffusion method) for the standard/control results and $y = -0.23 + 1.21x - 0.0257x^2$ for the patients' sera. Discrepancies seemed to be most marked at higher levels. Mahon *et al.*³ commented on discrepancies between the results by the two methods in their study and suggested that it might be due to the effects of multiple antibiotic therapy. This did not seem to be so in our case, other antibiotics present being inactive against our plate diffusion assay indicator organism, and a similar correlation being found for both patients' samples and standard/control samples. The RIA method may have been reading low at the higher levels, but we cannot explain why at present. An assessment of each pair of sera was made retrospectively by one of us (CDR), and it was found the variations