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Reference

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How specific is the rapid urease test for diagnosing *Campylobacter pylori?*

We read with interest, the letter to the editor by Vaira *et al.*[1] We describe our experience with the rapid urease test, which differs from that reported by Vaira *et al*[1] and from those reported earlier.[2-5]

Three pieces of antral biopsy specimen were taken from 53 patients with dyspepsia. Two pieces were transported to the microbiology laboratory, one piece for Gram staining and another for culture in the Campylobacter medium; a third piece was immersed in CLO-gel, as described by Marshall *et al.*[5] Results were read after 20 minutes, three hours, and 24 hours. Twelve (20·69%) cases grew *Campylobacter pylori* (table).

The sensitivity of the rapid urease test has been shown to vary from 59–97%,[1-4] but our study shows a sensitivity of 100%. Unlike studies showing 100% specificity[1 3 5] this study showed a specificity of 95·7% at 20 minutes, 93·5% at three hours, and 80·4% at 24 hours. Unlike other studies there were no false negative results with the test,[2] but there was a false positive rate of 4·4% at 20 minutes, 6·5% at three hours, and 19·6% at 24 hours.

Vaira *et al* concluded that a positive *C pylori* (CP) test before 20 minutes of incubation is strong evidence of *C pylori* infection. To substantiate the authors' observation, it would be important to know the sensitivity, specificity, false positive and false negative rates at 20 minutes, three hours, and 24 hours of the CP test compared with those of the 2% RUT and CLO-tests. Results of the CLO-test were read at 20 minutes, 90 minutes, and 24 hours; results of the 2% RUT were read at three hours, four hours, and six hours; results of the CP test were read at 15 minutes, 20 minutes, and two hours. We feel that it would have been better if the results were read at the same time intervals with these three tests.

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1 Vaira D, Holton J, Cairns S, *et al.* Urease tests for Campylobacter pylori: care in interpretation. *J Clin Pathol* 1988;**41**:812–3.
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Drs Vaira, Holton, and Salmon comment:

In reply to Bhasin *et al*, our conclusion that "a positive Campylobacter pylori test (CP-test) before 20 minutes' incubation is strong evidence of *C pylori* infection" is based on the results of our rapid 6% urea test (CP-test) and does not refer to other urease tests.

The specificity and sensitivity of the tests at different times are given in the test of our original letter.

All the three tests were compared—the 2% urea test; 6% urea test (CP-test), and CLO-test—were done at five, 10, and 20 minutes. The results at one, three, and 24 hours are also given in our letter.

Table *Comparative efficacy of various tests for Campylobacter pylori*

			Rapid urease test at:		
	Culture	*Gram staining*	*20 minutes*	*3 hours*	*24 hours*
Positive	12	10	14	15	21
Negative	46	48	44	43	37
Sensitivity		83·33%	100%	100%	100%
Specificity	100%	100%	95·65%	93·48%	80·43%
False positive		0	4·35%	6·52%	19·57%
False negative		16·67%	0	0	0

Demonstration of aluminium on bone using different staining techniques and spectrophotometry

We were interested to see the paper of Ellis *et al.*[1] To our knowledge, it is the first time that anyone has attempted to validate the technique we originally described in 1985[2] for showing the presence of aluminium within bone, and we note with some pleasure that they have been able to confirm our results.

We have now examined more than 1800 biopsy specimens using the solochrome azurine technique and have had an opportunity on many occasions to compare the stain distribution with that perceived by energy and wavelength dispersive electron probe analysis, secondary ion mass spectrometry, and laser microprobe mass analysis, and believe we can resolve two of the anomalies described by Ellis *et al.*

The "false" positivity that they describe is not false but real. Atomic absorption spectrophotometry (AAS) by its nature gives a measure of aluminium expressed as a proportion of total dried weight of bone. Localised deposits of aluminium, as frequently occur after treatment of aluminium related renal osteodystrophy (AIROD), would be "diluted" out by AAS analysis giving a low mean aluminium concentration, whereas in truth, the local concentration may be relatively high and well within the concentration range detected by solochrome azurine. Thus by comparison with AAS, solochrome azurine may appear to be reacting with bone containing only low aluminium concentrations. The obverse may also apply. Aluminium may be deposited diffusely within bone but with local concentrations too low to be detected by solochrome azurine. We have found this to be particularly true in patients with a moderately decreased glomerular filtration rate in regions like ours where the ionic aluminium content of tap water is relatively high. This group of patients gradually accumulate aluminium in bone but the concentration in the extracellular fluid is

never high enough to inhibit bone cell function significantly.

The "unrecognised substance that inhibits the aluminium technique" is much less mysterious than it at first appears. The aluminium technique is undertaken at acid pH. Even in the short time that the stain solution is applied to the tissue, the acid conditions initiate decalcification with the result that the local concentrations of calcium and phosphate at bone surfaces (including the cut surface of the trabeculae and cortices) are high. It has been known since 1954[3] that high phosphate ion concentrations inhibit the aluminium staining reaction and this is almost certainly the cause of the observed failure of staining.

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Fungus ball of the urinary tract

I read with interest the account by Morton *et al* of urinary bladder fungus ball in a man of 71.[1] As they comment, there is scant mention of this condition, few cases having been reported, but thrush of the urinary bladder was reviewed by Winner and Hurley[2] and, more recently, candida pyelonephritis has been reviewed by Odds,[3] who observed that diabetes is the most common single underlying condition. Hurley and Winner published an illustrated account of the pathogenesis of experimentally induced renal candidosis with a brief review of cases in man affecting the upper renal tract.[4] Hydronephrosis with formation of fungus ball in the pelvis of the ureter is a feature of this rare disease. The fungus ball described in the bladder by Morton *et al* may well have had its origin in fungal pyelonephritis.

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2 Winner HI, Hurley R. *Candida albicans*. London: Churchill, 1964:158.
3 Odds FC. *Candida and candidosis*. 2nd Edition. London: Baillière Tindall, 1988:170–4.
4 Hurley R, Winner HI. Renal moniliasis in the mouse. *J Pathol Bacteriol* 1963;**86**:75–82.

Some new titles

The receipt of books is acknowledged, and this listing must be regarded as sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits.

An Endoscopic Approach to Bilio-Pancreatic Disease. L Familiari. (Pp 196; £33.) Piccin Nuova Librerta, S.p.A., Padua, Italy; distributed by Gazelle Book Services, Lancaster. 1988. ISBN 88-299-0404-X.

Directory of Ongoing Research in Cancer Epidemiology. Ed MP Coleman, J Wahrendorf. IARC Scientific Publications No. 93. (Pp 662; £26.) Oxford University Press. 1988. ISBN 92 832 1193 6.

From Hippocrates to Virchow: Reflections on Human Disease. JM Byers. (Pp 160; $32.) Raven Press. 1988. ISBN 0-89189-257-5.

The Biochemical Pathology of Astrocytes. Neurology and Neurobiology. Vol 39. Ed MD Norenberg, L Hertz, A Schousboe. (Pp 662; $118.) Alan R Liss. 1988. ISBN 0-8451-2741-1.

Quantitative Bioassay. Analytical Chemistry by Open Learning. D Hawcroft, T Hector, F Rowell. (Pp 300; soft cover £11·50.) John Wiley. 1987. ISBN 0-471-91401-0.