asymptomatic.⁴ All raw pork handled by the patient was of local origin. *Y enterocolitica* has not been reported in British pigs. The only neurological complication previously recorded was a case of Guillain-Barré syndrome.⁵ Neuralgic amyotrophy was recognised during the last war. It is of unknown aetiology but may follow dysentery or Reiter's syndrome. Severe pain in the shoulder girdle is followed by rapid wasting. Sensory loss is minimal and recovery usually good. Extensor plantars may occur.

When enteritis and abdominal pain is followed by lower-limb polyarthritis, salmonellae, shigellae, and yersinia should be sought in the acute phase by culture and serology. Underestimation of the true prevalence is likely.

¹ Ahvonen, P, Sievers, K, and Aho, K, *Acta Rheumatologica Scandinavica*, 1969, **15**, 232.

² Ahvonen, P, *Annals of Clinical Research*, 1972, **4**, 39.

³ Aho, K, et al, *Arthritis and Rheumatism*, 1974, **17**, 521.

⁴ Mollaret, H G, *Pathologie et Biologie*, 1971, **19**, 189.

⁵ Parsonage, M J, and Aldren Turner, J W, *Lancet*, 1948, **1**, 973.

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Yersinia arthritis

Reactive arthritis associated with *Yersinia enterocolitica* infection is common in parts of Europe, particularly Scandinavia, but has never been documented in the UK. We describe such a case.

Case report

A 54-year-old London office clerk was well until May 1977, when she began to complain of pain in the calves, heels, and ankles, and subsequently of swelling of the ankles and middle toe of the left foot, together with slight general malaise and transient low back pain. She had had no gastrointestinal or urogenital symptoms, nor had she recently been abroad or been in contact with any infectious diseases. Her family history was negative.

She was first seen at this hospital in August 1977. Her third left toe was swollen and tender, and the dorsum of the foot was warm and reddened. Physical examination, including eyes and skin, showed no other abnormality. She slowly improved, though with episodes of pain and swelling in other toes and right sternoclavicular joint. She was given phenylbutazone (which caused buccal ulceration) and aspirin but not antibiotics. By June 1978 she had completely recovered.

Results of investigations were: erythrocyte sedimentation rate 85 mm in first hour (later falling to normal); haemoglobin concentration 13.2 g/dl; white cell count $8.6 \times 10^9/l$ ($8600/mm^3$); rheumatoid and antinuclear factors not detected; serum urate concentration 0.3 mmol/l (5.5 mg/100 ml); radiographs of feet and sacroiliac joints normal; tissue typing, HLA-B27 positive. The table gives the bacteriological findings.

Agglutination titres in serum to various serotypes of *Yersinia enterocolitica* and to *Brucella abortus*

	September 1977*	October 1977	December 1977	March 1978
<i>Yersinia enterocolitica</i> 0:9	+ 1/640	+ 1/640	+ 1/40	<1/20
<i>Yersinia enterocolitica</i> OH:9 ..	+ 1/1280			
<i>Yersinia enterocolitica</i> 0:3	<1/20			
<i>Brucella abortus</i> ..	+ 1/1280			<1/20

*Stools negative for yersinia and other pathogens.

Cross-absorption tests for *Y enterocolitica* and *Brucella abortus* are complicated by almost complete cross-reactivity between these organisms. Use of an enzyme-linked immunosorbent assay (ELISA) to detect and quantify antibodies against brucella and yersinia shows antigenic differences between *Br abortus* and *Y enterocolitica* 0:9 not detected in tube-agglutination studies.¹ ELISA testing of serum collected in September 1977 gave IgM and IgG titres of 260 and 1400 to *Y enterocolitica* 0:9; 290 and 920 to *Br abortus*; and 150 and 1250 to *Y enterocolitica* 0:3 respectively. The higher IgG titre to *Y enterocolitica* 0:9 than *Br abortus* and the cross-reactivity

between *Y enterocolitica* serotypes 0:9 and 0:3 strongly suggested infection with *Y enterocolitica* 0:9.

Comment

"Reactive arthritis," which signifies arthritis associated with infection elsewhere in the body, may occur after intestinal infections with salmonellae and shigellae in contrast to cases of sexually acquired reactive arthritis,² some of which are associated with *Chlamydia trachomatis* in urethral smears, and a minority of which show the classical triad of Reiter's syndrome. Most patients with reactive arthritis possess HLA-B27. *Y enterocolitica* infection is a well-recognised cause of reactive arthritis.³ A self-limiting polyarthritis occurs about three weeks after infection; gastroenteritis may be so mild as to escape notice. By the time the arthritis appears stool culture is usually negative and a diagnosis is made from agglutination titres. Affected joints include knees, ankles, fingers, toes, and sternoclavicular joints.

In our patient serology showed a high agglutination titre to *Y enterocolitica* 0:9, later becoming negative. Cross-reactivity with *Y enterocolitica* 0:3 was found on ELISA testing but not in ordinary agglutination. Carlsson et al,¹ using hyperimmune rabbit antiserum under similar conditions, found little or no cross-reactivity between *Br abortus* and *Y enterocolitica* 0:3. Furthermore, in our patient *Br abortus* titres became negative in the absence of chemotherapy. We concede that the "reactive" nature of the arthritis is presumptive, since we did not obtain synovial fluid.

Between 1970 and 1976 only two cases of *Y enterocolitica* infection were reported in Britain, both patients being infected abroad.^{4,5} Since then increasing numbers of cultures of *Y enterocolitica* and sera with high antibody titres to *Yersinia* have been received at the Leicester Public Health Laboratory, including sera from five patients who developed arthritis after gastrointestinal episodes. If this increase reflects a true rise in the incidence of yersinia infection in Britain more attention will have to be paid to *Y enterocolitica* infection as a cause of polyarthritis of apparently unknown origin.

We are indebted to Dr A A Lindberg, of the National Bacteriological Laboratory, Stockholm, for the ELISA testing of serum collected in September 1977.

¹ Carlsson, H E, Hurvell, B, and Lindberg, A A, *Acta Pathologica et Microbiologica Scandinavica*, sect C, 1976, **84**, 168.

² Keat, A C, et al, *British Medical Journal*, 1978, **1**, 605.

³ Aho, K, et al, in *Infection and Immunology in the Rheumatic Diseases*, ed D C Dumonde, ch 44, p 341. London, Blackwell Scientific, 1976.

⁴ Chessum, B, et al, *British Medical Journal*, 1971, **3**, 466.

⁵ Baldock, N E, and Catterall, M D, *British Journal of Dermatology*, 1975, **93**, 719.

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Is intrinsic factor really affected by cimetidine?

The fact that intrinsic factor (IF) is secreted from the parietal cell in man¹ has led to speculation that blockade of histamine H_2 receptors by cimetidine might result in impairment of absorption of vitamin B₁₂. There have been conflicting reports concerning the acute effect of cimetidine on stimulated IF output. Previous paired studies in healthy men have shown that while the concomitant administration of cimetidine 100 mg/h decreased the peak and total IF output during the infusion of pentagastrin 6 µg/kg/h this reduction was not statistically significant. No change was seen in peak IF concentration or the time course of the secretory response,² but studies in two groups of patients with duodenal ulcer, one of whom received cimetidine

Mean intrinsic factor concentration (U/ml) and mean intrinsic factor output (U/15 min) in seven healthy men on three separate study days.

while the other acted as a control, suggest that cimetidine reduces both the output and concentration of IF.³ Similar results followed cimetidine 300 mg by mouth and histalog stimulation.⁴ We tried to evaluate further the effect of cimetidine on the IF response to pentagastrin stimulation and to investigate the effect of a period of treatment with cimetidine on this response.

Methods and results

Studies were carried out in seven healthy men. After an overnight fast a nasogastric tube was passed. Gastric secretion was aspirated continuously, 15-minute samples collected, the volume recorded, and IF concentration assayed by radioimmunoassay.⁵ Each subject was studied on three separate occasions one week apart. On study day 1 gastric juice was collected for 30 minutes before an intravenous infusion of cimetidine 100 mg/h. After 45 minutes pentagastrin 6 µg/kg/h was infused simultaneously and continued for a further 90 minutes. On the second study day pentagastrin alone was infused for 90 minutes, followed immediately by cimetidine 200 mg intravenously. Cimetidine 2 g/day in divided doses was then taken for the next week, the first dose two hours after the intravenous injection and the last dose two hours before the start of aspiration on study day 3, when pentagastrin was again infused for 90 minutes. IF output and concentration were compared by means of an analysis of variance.

ONE HUNDRED YEARS AGO Dr W B Hammond (*The Voice*, No 3) gives his method of self-treatment of stammering. He considers it a functional disorder of that part of the brain which presides over the faculty of speech. Having himself been a sufferer, he is able to speak as one having authority. He describes his method in the following words.

"If the attention of the stammerer can be diverted from himself and his articulation, he will often speak to others as calmly and as perfectly as he does to himself when alone. Now, there are various ways of accomplishing this object, but the one that I found most effectual was the performance of some slight muscular action synchronously with the articulation of the difficult syllables. The words that troubled me most were those that began with the *explosive* consonants—those that require the sudden opening of the lips for their enunciation—*b, p,* and *t*. I could no more have repeated the alliterative lines, 'Peter Piper picked a peck of pickled peppers,' etc., to other persons without stammering, than I could have walked to the moon, though perfectly able to say the whole piece through without a flaw when speaking alone. With each troublesome word, especially with one beginning a sentence, I made some slight motion with the hand or foot, or even with a single finger, and I found that this plan enabled me to get the word out

No significant difference was seen between any study days in either mean peak IF concentration (10.64, 10.18, and 11.99 U/ml on study days 1, 2, and 3 respectively) or in the area under the IF concentration curve (figure). Peak IF output was reduced by 42.7% on study day 1 ($P < 0.05$) and 41.6% on study day 3 ($P < 0.01$). Total IF output during the infusion of pentagastrin was reduced by 37.5% on study day 1 ($P < 0.01$) and by 30.3% on study day 3. The difference between study days 2 and 3 was not statistically significant. No significant difference was seen between study days 1 and 3 with respect to either IF output or concentration.

Comment

The reduction in total and peak intrinsic factor output produced by cimetidine during pentagastrin infusion was entirely accounted for by the concomitant reduction in the volume of gastric secretion. The discrepancy between our findings and those of Fielding *et al*³ may arise because they compared IF output between two separate groups. There is considerable variation in IF output between individuals and between the sexes. Binder and Donaldson⁴ used a different secretagogue (histalog) and gave it by single subcutaneous injection rather than continuous intravenous infusion, and this may explain the difference between their findings and ours.

The IF response to pentagastrin was unaffected by seven days' regular administration of cimetidine at high dose, after initial release of preformed stores. While synthesis of IF was not directly measured in these studies the depletion of IF stores in the parietal cell by pentagastrin immediately before giving cimetidine and the subsequent response to repeat stimulation one week later, while pharmacologically active quantities of cimetidine were still circulating, suggest that cimetidine has no effect on the synthesis of IF.

¹ Jacob, E, and Glass, G B J, *Clinical and Experimental Immunology*, 1971, 8, 517.

² Burland, W L, *et al*, in *Proceedings of the 2nd International Symposium on Histamine H₂ Receptor Antagonists*, ed W L Burland and M A Simkins. Amsterdam and Oxford, Excerpta Medica, 1977.

³ Fielding, L P, *et al*, in *Proceedings of the 2nd International Symposium on Histamine H₂ Receptor Antagonists*, ed W L Burland and M A Simkins. Amsterdam and Oxford, Excerpta Medica, 1977.

⁴ Binder, H J, and Donaldson, R M, *Gastroenterology*, 1978, 74 (2), 371.

⁵ Hansen, H J, *Biochemistry*, 1966, 16, 287.

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without stammering. With the enunciation of 'Peter,' for instance, I would tap the side of my body with the hand just as I opened my lips, and the word was articulated without the least halting. In the procedure, the attention is diverted from the effort to speak to the performance of the muscular action mentioned, and hence the speech becomes more automatic than it is with stammerers generally. And this is the only system of curing stammering. It consists in efforts to render the speech automatic. No orator thinks of his articulation when he is making a speech; no one in ordinary conversation thinks whether or not he will be able to pronounce a certain word, or to acquit himself well in the management of his tongue and lips. His mind is concerned with his thoughts, with what he is going to say—not with the manner in which he will articulate, and the more thoroughly we can succeed in bringing stammerers into the same way of procedure, the more successful will we be in our efforts to cure them."

He followed this method about two years before the cure was accomplished, and has succeeded in curing several of his young friends by recommending it to them. Sometimes it has failed, as all other plans sometimes fail; in some cases, probably from want of perseverance in carrying out the plan. (*British Medical Journal*, 1879.)