Table 1 *Comparison of 326 white blood cell differential counts by Hemalog-D (HD) and standard microscopy (SM) (± 1 SD in brackets) with total white cell count between 5 and 20 × 10⁹/l*

Hemalog-D count result	*Count number*	*Count*	*Neutrophil*	*Lymphocyte*	*Monocyte*	*Eosinophil*
Positive remainder < 5	76	SM	70·5 (11·2)	22·5 (11·9)	4·80 (2·40)	1·80 (2·50)
		HD	69·1 (12·2)	21·3 (10·8)	3·1* (1·9)	1·9 (2·3)
Positive remainder > 5	91	SM	66·1 (13·1)	26·2 (12·7)	7·8 (2·4)	2·3 (2·8)
		HD	66·3 (10·9)	20·8† (10·2)	1·7* (1·4)	2·3 (2·4)
Negative remainder < 5	66	SM	62·7 (9·3)	30·1 (10·1)	6·1 (2·1)	2·9 (1·7)
		HD	63·4 (8·5)	28·2 (9·1)	6·4 (1·8)	2·8 (1·4)
Negative remainder > 5 SM monocyte count < 10	101	SM	54·2 (11·9)	35·2 (9·7)	6·1 (1·9)	3·6 (3·9)
		HD	56·8 (10·9)	32·1‡ (9·6)	11·9* (1·9)	3·6 (3·7)
Negative remainder > 5 SM monocyte count > 10	61	SM	56·3 (13·1)	29·4 (12·1)	14·1 (7·2)	2·8 (1·8)
		HD	59·6 (11·1)	28·8 (11·1)	14·9 (5·1)	2·7 (1·4)

*P < 0·01 †P < 0·05 ‡P < 0·025 All others P > 0·1.

Table 2 *Suitable correction of Hemalog-D (HD) monocyte count by addition, or subtraction, of remainder to obtain monocyte counts similar to standard microscopy (SM) monocyte count*

Hemalog-D count results	*Count number*	*Monocytes (SM)*	*Monocytes (HD)*	*+ Remainder*	
Positive remainder > 5	91	7·8 (2·4)	1·7 (1·4)	7·6 (1·3)	Monocytes (HC) + remainder 8·3
Negative remainder > 5 SM monocyte < 10	101	6·1 (1·9)	11·9 (1·9)	6·1 (1·6)	Monocytes (HD) − remainder 5·8

visual monocyte counts greater than 10, good correlation was obtained (Table 1).

A positive remainder in the Hemalog-D can be due to monocytes with low esterase activity not identified as monocytes in the esterase channel, mistaken identification of monocytes with high peroxidase activity as other cells, or the mistaken identification of neutrophils with high esterase activity as monocytes. A negative remainder in the Hemalog-D can be due to excess cells being mistakenly identified by the machine as monocytes in the esterase channel, or mistaken identification of monocytes as eosinophils or neutrophils in the peroxidase channel.

Accordingly, from our results, we think that the machine monocyte count could be adjusted to approach the visual count when the remainder was greater than ± 5 (with white blood cell counts between 5 and 20 × 10⁹/l and no other alarm signals) by adding the machine remainder to the machine monocyte count, and the machine monocyte count could similarly be adjusted when the remainder was greater than −5 and less than −10 (with visual monocyte count less than 10), if the machine remainder was subtracted from the Hemalog-D monocyte count.

Thus simple addition or subtraction of remainders of less than ± 10 expanded to a range of differential counts which could be accepted from the Hemalog-D without subsequent visual microscopy of blood films.

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J AZNAR
A VAYÁ
JJ OSUNA
M MONFORTE
Department of Clinical Pathology, Ciudad Sanitaria "La Fé", Valencia, Spain

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

Drs Smibert and Graevenitz (May issue, page 509) question the reliability of tests for growth at 42°C for distinguishing *Campylobacter fetus* subsp. *intestinalis* from *C. fetus* subsp. *jejuni*. During the past two years or so we have tested some 1000 campylobacter isolates from both man and animals, and we agree that the ability to grow at 25°C is the more reliable characteristic for this purpose; most campylobacters of the *jejuni* group fail to grow at 30°C, let alone 25°C. However, nalidixic acid resistance is not confined to subspecies *intestinalis* and *fetus* (*venerealis*); some *jejuni* strains are also resistant (no

inhibition with 30 μg disc) and they appear to form a distinct subgroup. We have tested 46 such organisms: they do not grow at 25°C, but most grow freely at 45°C, are relatively salt-tolerant, and produce coccoid forms in cultures much earlier than with most *jejuni* strains. We have isolated them commonly from locally caught seagulls, but also occasionally from other animals and man. Thus the nalidixic acid sensitivity test is a useful adjunct to the 25°C test, but it is not infallible.

We, too, have found that while all *jejuni* group organisms grow freely at 42°C, a few isolates of what are ostensibly subsp. *intestinalis* (25°C positive and with typical morphology) do manage to grow at this temperature. Such strains, which were mostly isolated from bovine faeces, have generally produced more H_2S than orthodox *intestinalis* strains, notably in iron-containing medium. Of course, one has to be careful in performing tests near the upper limit of temperature tolerance; the cut-off point is sharp, and unless the temperature is accurately controlled results will be variable. We hope to publish a full account of our studies shortly.

MB SKIRROW
J BENJAMIN
Worcester Royal Infirmary, Castle Street Branch, Worcester WR1 3AS

'Ultra-fast' alkaline phosphatase isoenzyme

Koett and colleagues, in your December 1979 issue, requested investigators to report their experience (if any) of 'ultra-fast' alkaline phosphatase isoenzyme in serum with substrates other than the alpha naphthol ASMX phosphate they used. Over the past 10 years I have used cellulose acetate electrophoresis (on Sepraphore III) and indoxyl phosphate substrate to examine alkaline phosphatase isoenzyme patterns in more than a thousand serum specimens with raised total alkaline phosphatase activity. I have never demonstrated 'ultra-fast' alkaline phosphatase of albumin mobility in any of these samples.

SIDNEY B ROSALKI
The Royal Free Hospital, Pond Street, Hampstead, London NW3 2QG

Staphylococcal enterocolitis and inflammatory bowel disease

We were interested by the articles of Willoughby *et al.* and Price *et al.* (Volume 32, page 986 and page 990 respectively). We are also studying the role of rectal biopsy in the diagnosis of acute colitis and have encountered two patients with infective colitis who are of particular interest.

Patient 1

A 55-year-old housewife was well and on no medication until February 1976 when she had a sudden onset of severe watery diarrhoea associated with flu-like symptoms. At the time she attributed her symptoms to the ingestion of a pork pie 2-3 hours before the onset of her illness. Her GP prescribed cotrimoxazole and kaolin to no effect, and she was admitted three weeks later to an infectious diseases hospital for investigation. On admission she was unwell and pyrexic. The abdomen was distended but there was no tenderness. Investigations revealed Hb 11·8 g/dl, WBC 12·0 × 10^9/l (82% neutrophils), ESR 50. Stool cultures showed *Staphylococcus aureus* +++ (ie, massive overgrowth of staphylococcus with relative absence of the normal Gram-negative enteric flora) (GL Gibson—personal communication). The organism was resistant to penicillin and ampicillin but sensitive to tetracycline, cotrimoxazole, cloxacillin, lincomycin, fucidin, and erythromycin. No other pathogens were detected on routine culture although *Clostridium difficile* and pathogenic *Escherichia coli* were not sought. Ten days after admission she was transferred to this hospital where investigations showed the serum albumin was low at 30·3 g/l. Sigmoidoscopy was normal and rectal biopsy revealed a mild acute proctitis compatible with an infective aetiology.[1,2] Barium enema revealed severe ulceration of the whole colon but with rectal sparing. A small bowel meal was normal. She was treated with steroids and intravenous hyperalimentation (IVH) and improved rapidly and was discharged after 18 days. She has since had no further bowel symptoms and sigmoidoscopy, rectal biopsy, and barium enema nearly three years after her acute illness were normal.

Patient 2

A 25-year-old male factory worker, who was on no medication, presented in May 1978 with a three-week history of watery diarrhoea of sudden onset, fever, and weight loss. His GP had prescribed sulphasalazine, 3 g/day, to no effect and he was admitted. On examination he was toxic with a temperature of 39°C. Sigmoidoscopy revealed no mucosal abnormality. Stool taken at sigmoidoscopy and plated immediately on to blood agar showed a massive overgrowth of staphylococci with almost complete absence of normal enteric flora. This finding was subsequently confirmed in stool samples sent to the routine laboratory, and the organism was identified as *Staph. aureus* resistant to penicillin but sensitive to tetracycline, erythromycin, fucidin, clindamycin, gentamicin, and cloxacillin. A search for other routine faecal pathogens, including *Campylobacter* species, was negative. *Cl. difficile* and its toxin were not sought. In addition, no pathogenic *E. coli* were found in a faecal sample obtained five days after admission, by which time the flora had apparently returned to normal. Other investigations showed Hb 13·2 g/l, WBC 7·6 × 10^9/l (78% neutrophils with a toxic shift and 2% myelocytes). The ESR was 46 mm/h. Plasma albumin was low at 33·5 g/l. Barium enema showed gross colonic ulceration with rectal sparing similar to that of patient 1. A small bowel meal was normal. Rectal biopsy (Fig. 1a and 1b) showed characteristic changes of infective colitis.[1,2] He was treated with steroids and IVH and showed dramatic improvement; he was discharged 18 days after admission. Six months after discharge from hospital he suffered a further attack of diarrhoea necessitating treatment with steroids. Sigmoidoscopy during this second attack showed minimal erythema only but a barium enema showed continuing ulceration in the descending and transverse colon. Faecal bacteriology was not performed on this occasion.

These patients are of interest for three reasons. Firstly, the bacteriological findings strongly suggest the diagnosis of staphylococcal enterocolitis. A massive overgrowth of staphylococci in the faeces to the exclusion of the normal enteric flora is uncommon and probably diagnostic of staphylococcal enterocolitis.[3] These cases appear to be highly unusual since we have been unable to find similar reports in the literature. They do suggest, however, that staphylococcal enterocolitis may occur in previously healthy individuals who are not on antimicrobials. This suggestion is supported both by the observation that staphylococci have been recovered from