

the diagnoses of only 76 out of the 140 patients with a positive Haemocult test result, however, these being a colorectal neoplasm, diverticulitis, Crohn's disease, and angiodysplasia. What were the diagnoses in the other 64 patients? What were the diagnoses and their incidences in the original 802 patients (for comparison)? What proportion of the latter had no detectable disease? Incidentally, what were the original presenting symptoms which defined the patients being studied?

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<sup>1</sup> Llewelyn DEH. Mathematical analysis of the diagnostic relevance of clinical findings. *Clin Sci* 1979;57:477.

<sup>2</sup> Llewelyn DEH. Applying the principle of logical elimination to probabilistic diagnosis. *Med Inform* 1981;6:1-25.

<sup>3</sup> Eddy DM, Clanton CH. The art of diagnosis. *N Engl J Med* 1982;306:1263.

\* \* \* We sent a copy of this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—Dr D E H Llewelyn has misinterpreted the aim of our study, which was designed to compare Haemocult occult blood testing with the present routine methods of large bowel investigation, using proctosigmoidoscopy and double contrast barium enema. Our conclusion states that a positive test is a useful indicator for the need to proceed to urgent colorectal investigation since only 12 cases had a false positive result.

We agree with Dr Llewelyn's observations regarding the predictive value for neoplastic disease: of the 602 patients with a negative Haemocult test 28 (4.6%) had neoplastic disease, of which two were colonic cancers, six rectal cancers, and the remainder adenomatous polyps. Six patients who had had a normal barium enema and sigmoidoscopy with a negative Haemocult test were subsequently found to have a neoplasm at colonoscopy (one colon cancer, five polyps). We would like to re-emphasise that while a negative Haemocult test does not exclude colorectal neoplasia a positive result alerts the doctor to the need for early, full colorectal investigation, including colonoscopy.

Although we accept that the false positive rate for neoplasia was 14.6%, the overall false positive rate was 5.6%; the difference being due to appreciable disease such as ulcerative colitis. The 76 patients with positive Haemocult tests in whom the diagnosis was stated referred only to those patients with both diverticular disease and an additional lesion and those with neoplastic lesions. The diagnoses of all the 140 patients with positive tests are shown in

the table, together with the diagnoses of the negative group for comparison. Because of the study design the data from the 60 patients who did not comply with testing were excluded. The original presenting symptoms were all documented independently by the doctors, who decided which patients had suspected colorectal disease requiring barium enema examination. The presenting symptoms and other details are currently being analysed with a view to further publication.

This study was carried out on patients with symptoms referred to hospital. We consider that the Haemocult test correlated closely enough with the final diagnosis to make a positive result of clinical value. We are currently assessing the test in general practice to see if it remains a helpful indicator of colorectal disease and can therefore be used to help select patients for early referral.

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#### Acute respiratory distress in diabetic ketoacidosis

SIR,—I was heartened to see that other doctors recognise the dangers of giving crystalloid in diabetic ketoacidosis (5 March, p 760) but amazed at the suggestion that the cause of the pulmonary oedema was low colloid osmotic pressure or albumin. Both patients had normal albumin values when they were observed to be hypoxic and cyanosed with changes on the chest x ray film (43 g/l on day three for case 1; 48 g/l on day one for case 2). Even if the albumin had been low, as it did indeed become, it must be considered in conjunction with the left atrial pressure when predicting whether pulmonary oedema would occur.

It is a basic physiological principle that the majority of a crystalloid solution will move into the interstitial space in normal patients and even more in hypotensive patients.<sup>1</sup> This is related to the relative volume of the intravascular and interstitial spaces not the colloid osmotic pressure. Eventually, enough crystalloid will be given to dilute the albumin in the intravascular space, but long before this the interstitial space has been grossly over-

expanded because crystalloid is a fluid which is mainly distributed to this space; when the interstitial space is overexpanded in the lung it causes pulmonary oedema. This is initially why pulmonary oedema was seen before the albumin or colloid osmotic pressure was low. I suggest that these problems can be overcome by using correct replacement fluid to expand the appropriate body spaces. Where there is hypovolaemia, as there was in these two cases, colloid should be used to resuscitate the patient. This is a medical emergency. The sodium in the colloid replaces sodium losses, and 5% dextrose should be used to replace water losses. There is no indication to use physiological saline to resuscitate patients with diabetic ketoacidosis.

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<sup>1</sup> Hillman KM. Resuscitation in diabetic ketoacidosis. *Crit Care Med* 1983;11:53-4.

#### Is weighing babies in clinics worth while?

SIR,—While I agree entirely with the plea by Professor D P Davies and Dr T Williams (12 March, p 860) for accurate weighing of babies and recording the weight on centile charts I would like to emphasise the value of measuring, in addition, the length of babies and recording this likewise on the centile chart. The two readings together give a more accurate profile of the infants' well being.

I have found that correlating the length and weight of babies is particularly useful: (a) to reassure mothers who are anxious lest their babies are overweight when it can be shown that their infants' weight is in proportion to its length; and (b) to convince mothers who have contented but starved babies that their baby is too thin relative to its length. Once convinced the mother will be much more likely to act on advice regarding feeding.

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#### The Heimlich manoeuvre

SIR,—Dr E Saphier (12 March, p 897) says that he has never met anyone who has met anyone who has performed a Heimlich manoeuvre. I performed this manoeuvre for the first and only time some eight months ago. I was called to a neighbour one Sunday lunch time, the story being that an old lady of 82 had collapsed while eating her Sunday lunch. I was there in less than a minute to find a rather obese old lady deeply cyanosed and not breathing and lying on the floor. I assumed that there was a bolus of food impacted in her larynx so I put a finger in her mouth, removed her false teeth, but could not feel a bolus at that level; I did not attempt to feel further back in the throat. The Heimlich manoeuvre may be quite easy to perform in someone in an upright position, but it was extremely difficult to get the old lady into a sitting position, and I had to kneel on the floor behind her to get my hands in the correct position.

I carried out the manoeuvre 12 times and at that stage thought that it was not going to work when a large food bolus some 8 cm by 2 cm appeared in her mouth which I then

Final diagnosis and Haemocult results in 742 symptomatic patients with suspected colorectal disease

Diagnosis	Haemocult result			
	Positive		Negative	
	No of patients	%	No of patients	%
Colon cancer	22	(15.7)	4	(0.7)
Rectal cancer	6	(4.3)	5	(0.8)
Adenomas	14	(10.0)	20	(3.3)
Haemorrhoids only	15	(10.7)	50	(8.3)
Diverticular disease only	19	(13.6)	173	(28.7)
Haemorrhoids and diverticular disease	7	(5.0)	22	(3.7)
Ulcerative colitis	25	(17.9)	20	(3.3)
Crohn's disease	8	(5.7)	14	(2.3)
Other colitis	2	(1.4)	0	(0)
Proctitis	0	(0)	4	(0.7)
Angiodysplasia	1	(0.7)	3	(0.5)
Irritable bowel syndrome	0	(0)	109	(18.1)
Upper gastrointestinal disease	8	(5.7)	12	(2)
Appendicitis ("chronic")	0	(0)	11	(1.8)
Non-gastrointestinal disease	0	(0)	29	(4.8)
Miscellaneous (leukaemia, etc)	1	(0.7)	10	(1.7)
Anaemia	0	(0)	4	(0.7)
Normal	12	(8.6)	112	(18.6)
Total	140		602	

extracted. She immediately started to breathe and within two or three minutes was pink again. She made a total recovery and half an hour later was asking for the rest of her Sunday lunch. She appears to have suffered no cerebral damage from this and has no recollection of the whole episode. Thus if attempting this manoeuvre do not give up if it is unsuccessful the first few times.

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### Salmonella gastroenteritis associated with erythema nodosum

SIR,—In their article implicating salmonella gastroenteritis as an aetiological agent in erythema nodosum (5 March, p 765) Dr W M Morrison and others point out that: "Concurrent streptococcal infection cannot be totally excluded in these three patients."

We have recently treated a 22 year old woman who presented with typical lesions of erythema nodosum and gave a two week history of passing up to four loose, watery bowel motions (without blood or mucus) a day. In addition, she complained of colicky left iliac fossa discomfort, anorexia, and vomiting. There was no past or family history of bowel disease. She was not taking any drugs. Sigmoidoscopy showed a congested rectal mucosa which bled easily on contact. *Salmonella typhimurium* was cultured from three consecutive stool samples, which failed to grow yersinia, shigella, or campylobacter. Sedimentation rate (Westergren) was 95 mm in the first hour. A full blood count was normal. Throat swab and blood culture were sterile. Mantoux test (1:100 old tuberculin units) was negative. Antistreptolysin titre was 12 units. A chest x ray examination was normal. Kveim antigen test was negative. During two weeks' bed rest the diarrhoea and the cutaneous lesions resolved. A further series of four consecutive stool samples was sterile, and the sedimentation rate fell to normal. When seen for review two months later the patient was without symptoms and sigmoidoscopy was normal.

In this case we were confident that a recent streptococcal infection had been excluded. We would, therefore, agree with the authors that salmonella gastroenteritis should be considered as a causative agent in erythema nodosum.

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### Endoscopy after gastric surgery

SIR,—We were interested to read the study by Mr P A Farrands and others (5 March, p 755)—their discovery of two asymptomatic gastric cancers is worrying. While we did not find any patients with invasive cancer in our study of 65 patients who had had partial gastrectomies, 14 had moderate dysplasia and 28 mild dysplasia.<sup>1</sup> Of the 14 with moderate dysplasia 12 agreed to biannual follow up and we hope to publish the five year results shortly.<sup>2</sup> One patient has had a total gastrectomy after developing severe dysplasia, but despite a careful search no invasive carcinoma was found. The remaining patients have not shown progression of dysplasia, and we have therefore

recommended annual follow up for moderate dysplasia.

In view of these less worrying findings we were concerned about the use of the term "carcinoma in situ" in the paper of Mr Farrands and others since severe dysplasia, by definition, implies changes which fall short of the full criteria of carcinoma in situ. Morson *et al*<sup>3</sup> proposed that the term be dropped in favour of "intra-mucosal carcinoma" where there was invasion of the lamina propria and emphasised, as we do, that the degree of risk associated with epithelial dysplasia is not yet established. We agree with Morson *et al* that severe dysplasia is not on its own an indication for surgical intervention and have suggested that this decision should rest on the results of more than one set of multiple biopsies.<sup>2</sup> Mr Farrands and others suggest endoscopic screening from five years after gastric surgery, but this is surely a counsel of perfection and in practice is unworkable. While we would agree that for cases with mild dysplasia a five yearly follow up is reasonable patient compliance is likely to be a problem, as we are finding in our first five year review of the mild dysplasia cases reported in our original paper.

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<sup>1</sup> Savage A, Jones SM. Histological appearances of the gastric mucosa 15-27 years after partial gastrectomy. *J Clin Path* 1979;**32**:179-86.

<sup>2</sup> Mortensen NJMcC, Savage A, Jones SM. Early results of a prospective study of gastric stump dysplasia. *Br J Surg* 1981;**68**:815.

<sup>3</sup> Morson BC, Sobin LH, Grundmann E, *et al*. Precancerous conditions and epithelial dysplasia in the stomach. *J Clin Path* 1980;**33**:711-21.

### Meningoencephalitis associated with Chlamydia trachomatis infection

SIR,—The report by Dr J M Goldman and others of a case of meningoencephalitis and serological evidence of *Chlamydia trachomatis* infection (12 February, p 517) requires some comment. The patient also had urinary pus cells, which may have been due to *C trachomatis* urethritis. The authors suggested that a causal link between this *C trachomatis* infection and meningoencephalitis should be seriously considered.

The evidence for this association is provided solely by serology. In Myhre and Mardh's case<sup>1</sup> the titre of serum immunoglobulin G antibody shown by microimmunofluorescence against pooled D-K serotypes was very high indeed at 1/64 000 and subsequently declined. The patient also had a reactive arthritis which may explain these very high levels.<sup>2</sup> In the case described by Dr Goldman and others the antibody titres (again of IgG class against D-K serotypes by microimmunofluorescence) were more modest, but respectable, with a fourfold rise from 1/256 to 1/1024, suggesting a recent infection. Cerebrospinal fluid antibody titres were unremarkable.

Unfortunately, despite the statement that chlamydial IgG titres in genitourinary infection rarely exceed 1/64,<sup>3</sup> this is not our experience, and titres above this level do not necessarily imply systemic infection. In a series of 151 patients with uncomplicated

non-gonococcal urethritis 18% had IgG titres of 1/128 (to D-K serotypes by microimmunofluorescence), and about a third of these patients (5% of the total studied) had titres of 1/1024 (Munday P E, Thomas B J, unpublished data). Many also had immunoglobulin M antibody, but this was less likely if there had been a history of urethritis.

The geometric mean titre of chlamydial IgG antibody for all chlamydial urethritis is indeed quite low, being reduced considerably by the fact that some cases of *Chlamydia trachomatis* urethritis (proved on culture) are seronegative. If only seropositive (either culture positive or negative) cases of uncomplicated non-gonococcal urethritis are taken into account then the geometric mean titre would be raised considerably.

In view of these serological observations we would like to strike a note of caution and suggest that the serological evidence in the case of Dr Goldman and others is insufficient to support *C trachomatis* as an aetiological agent of encephalitis. Additional evidence is required. It is surprising that chlamydiae were not isolated from the urethra because cefotaxime is not active against them. If we assume that the urethritis was due to *C trachomatis*, however, it is very common in sexually active adults and it could occur easily by chance in a patient who also develops encephalitis.

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<sup>1</sup> Myhre E, Mardh PA. *Chlamydia trachomatis* infection in a patient with meningoencephalitis. *N Engl J Med* 1981;**304**:910-1.

<sup>2</sup> Keat AC, Thomas BJ, Taylor-Robinson D, Pegrum GD, Maini RN, Scott JT. Evidence of *C trachomatis* infection in sexually acquired arthritis. *Ann Rheum Dis* 1980;**39**:431-7.

<sup>3</sup> Treharne JD, Dines RJ, Darougar S. Serological responses to chlamydial ocular and genital infection in the United Kingdom and Middle East. In: Hobson D, Holmes KK, eds. *Non-gonococcal urethritis and related infections*. Washington DC: American Society for Microbiology, 1977:249-58.

### Dietary sodium restriction for mild hypertension in general practice

SIR,—Dr G A MacGregor and others (5 March, p 803) are mischievous to misquote the conclusion of our study (5 February, p 432) and then criticise it. We did not claim that: "Modest restriction of sodium intake does not lower blood pressure in patients with mild hypertension," only that there was no evidence of this effect in the patients in our study.

We are surprised by their criticism that our presentation of data was insufficient for detailed analysis for in this aspect of our paper we followed their example exactly.<sup>1</sup> They also warn against drawing broad conclusions from observations on 13 patients who restricted their sodium intake, but their own conclusions on the effect of sodium restriction are based on a study of 19 patients, representing a much wider range of pressures, and only 15 of these achieved a sodium intake of less than 120 mmol(mEq). These points aside we are pleased to reply to their comments and provide further information as requested.

We consider that small scale studies are most informative when a group of patients can be related clearly to a larger defined population. In our practice we have two types of hypertensive patient: those already on treatment because their blood pressures have been sustained above a