Planning Association advice for a "missed pill" should be followed,²⁷ and wearers of soft contact lenses warned that these may be permanently stained. Itching, rashes, and gastrointestinal reactions may also occur. Contacts must be advised of the risk of late secondary disease occurring despite prophylaxis.

We advocate the use of rifampicin at a dose of 20 mg/kg a day (up to a maximum of 600 mg daily) for four days for three groups of contacts of patients with invasive *Haemophilus influenzae* type b disease. With the exclusion of pregnant or breastfeeding women, anyone with severely impaired hepatic function, and children aged under 3 months, prophylaxis should be offered to (a) all household members in households where there is an index case of *Haemophilus influenzae* type b disease, irrespective of age, and another child aged under 3 years; (b) all classroom contacts—both teachers and children—where two or more cases of the disease have occurred within 120 days—that is, where spread of the organism may have occurred within the class; and (c) all index cases of the disease before discharge from hospital. Prophylaxis should not be offered routinely to household contacts when there are no other children aged under 3 years, to non-household contacts of single cases, or to ward contacts in hospital outbreaks.

When prophylaxis is given all defined contacts must be identified and treatment started as early as possible. These recommendations are consistent with and amplify those of the British Paediatric Association.²⁸ A more detailed review of *H influenzae* type b chemoprophylaxis may be found in the *Communicable Disease Report*.²⁹

KEITH A V CARTWRIGHT

Consultant Microbiologist,
Public Health Laboratory,
Gloucester Royal Hospital,
Gloucester GL1 3NN

NORMAN T BEGG

Consultant Epidemiologist,
PHLS Communicable Disease Surveillance Centre,
London NW9 5DF

DAVID HULL

Chairman, British Paediatric Association
Standing Committee on Immunisation,
University Hospital, Nottingham NG7 2UH

- 1 Cochi SL, Fleming MD, Hightower AW, *et al*. Primary invasive *Haemophilus influenzae* type b disease: a population-based assessment of risk factors. *J Pediatr* 1986;108:887-96.
- 2 Tudor-Williams G, Frankland J, Isaacs D, *et al*. *Haemophilus influenzae* type b disease in the Oxford region. *Arch Dis Child* 1989;64:517-9.
- 3 Howard AJ, Dunkin KT, Millar GW. Nasopharyngeal carriage and antibiotic resistance of *Haemophilus influenzae* in healthy children. *Epidemiol Infect* 1988;100:193-203.
- 4 Michaels RM, Norden CW. Pharyngeal colonization with *Haemophilus influenzae* type b: a longitudinal study of families with a child with a meningitis or epiglottitis due to *H. influenzae* type b. *J Infect Dis* 1977;136:222-8.
- 5 Li KI, Dashevsky B, Wald ER. *Haemophilus influenzae* type b colonization in household contacts of infected and colonized children enrolled in day care. *Pediatrics* 1986;78:15-20.
- 6 Glode MP, Daum RS, Goldmann DA, *et al*. *Haemophilus influenzae* type b meningitis: a contagious disease of children. *BMJ* 1980;280:899-901.
- 7 Filice GA, Andrews JS Jr, Hudgins MP, *et al*. Spread of *Haemophilus influenzae*. *Am J Dis Child* 1978;132:757-9.
- 8 Ward JO, Fraser DW, Baraff LJ, *et al*. *Haemophilus influenzae* meningitis: a national study of secondary spread in household contacts. *N Engl J Med* 1979;301:122-6.
- 9 Campbell LR, Zedd AJ, Michaels RH. Household spread of infection due to *Haemophilus influenzae* type b. *Pediatrics* 1980;66:115-7.
- 10 Granoff DM, Basden M. *Haemophilus influenzae* infections in Fresno County, California: a prospective study of the effects of age, race, and contact with a case on incidence of disease. *J Infect Dis* 1980;141:40-6.
- 11 Band JD, Fraser DW, Ajello G. Prevention of *Haemophilus influenzae* type b disease. *JAMA* 1984;251:2381-6.
- 12 Immunization Practices Advisory Committee (ACIP). Update: prevention of *Haemophilus influenzae* type b disease. *MMWR* 1985;25:170-80.
- 13 Fleming DW, Leibenhaut MH, Albanes D, *et al*. Secondary *Haemophilus influenzae* type b in day-care facilities: risk factors and prevention. *JAMA* 1985;254:509-14.
- 14 Osterholm MT, Pierson LM, White KE, *et al*. The risk of subsequent transmission of *Haemophilus influenzae* type b disease among children in day care: results of a two-year statewide prospective surveillance and contact survey. *N Engl J Med* 1987;316:1-5.
- 15 Murphy TV, Clements JF, Breedlove JA, *et al*. Risk of subsequent disease among day-care contacts of patients with systemic *Haemophilus influenzae* type b disease. *N Engl J Med* 1987;316:5-10.
- 16 Makintube S, Istre GR, Ward JL. Transmission of invasive *Haemophilus influenzae* type b disease in day care settings. *J Pediatr* 1987;111:180-6.
- 17 Broome CV, Mortimer EA, Katz SL, *et al*. Use of chemoprophylaxis to prevent the spread of *Haemophilus influenzae* b in day-care facilities. *N Engl J Med* 1987;316:1226-8.
- 18 Cox F, Trinchler R, Rissing JP, *et al*. Rifampicin prophylaxis for contacts of *Haemophilus influenzae* type b disease. *JAMA* 1985;254:1043-5.
- 19 Boies EG, Granoff DM, Squires JE, *et al*. Development of *Haemophilus influenzae* type b meningitis in a household contact treated with rifampicin. *Pediatrics* 1982;70:141-2.
- 20 Murphy TV, McCracken GH Jr, Moore BS, *et al*. *Haemophilus influenzae* type b disease after rifampicin prophylaxis in a day care center: possible reasons for its failure. *Pediatr Infect Dis J* 1983;2:193-8.
- 21 Wilde J, Adler SP. Molecular epidemiology of *Haemophilus influenzae* type b: failure of rifampicin prophylaxis in a day care center. *Pediatr Infect Dis J* 1986;5:505-8.
- 22 Robbins JB, Schneerson R, Pittman M. *Haemophilus influenzae* type b infections. In: Germanier R, ed. *Bacterial vaccines*. London: Academic Press, 1984:310-2.
- 23 Edmonson MB, Granoff DM, Barenkamp SJ, *et al*. Outer membrane protein subtypes and investigation of recurrent *Haemophilus influenzae* type b disease. *J Pediatr* 1982;100:202-8.
- 24 Schaad UB, Nelson JD, McCracken GH Jr. Recrudescence and relapse in bacterial meningitis of childhood. *Pediatrics* 1981;67:188-95.
- 25 Liston TE. Pathogenesis and prevention of recurrent infection after *Haemophilus influenzae* bacteremia. *Clin Pediatr* 1984;23:215-9.
- 26 Cates KL, Krause PJ, Murphy TV, *et al*. Second episodes of *Haemophilus influenzae* type b disease following rifampicin prophylaxis of the index patients. *Pediatr Infect Dis J* 1987;6:512-5.
- 27 Anonymous. Contraceptives. *British National Formulary* 1990;20:266.
- 28 Nicoll A, Rudd P, eds. *Manual of infections and immunizations in Children*. Oxford: Oxford University Press, 1989:71.
- 29 Cartwright KAV, Begg NT, Hull D. Chemoprophylaxis for secondary *Haemophilus influenzae* type b disease. *Communicable Disease Report* 1991;1:R2-6.

A requiem for vagotomy

Despite the last ditch efforts of surgeons

In the early years of this century Latarjet, a surgeon-anatomist from Lyons, proposed vagotomy for relieving the abdominal pains of tabetic crises.¹ In the 1960s we laughed at his naivety but acknowledged his anatomical contribution by giving his name to the nerve that we strive to preserve when performing proximal gastric vagotomy. In the 1970s this operation was hailed as the most physiologically sound one for duodenal ulcer. Perhaps Latarjet is now laughing at us as peptic ulcer surgeons join the ranks of the unemployed.

The incidence of serious complications of duodenal ulcer and the need for operation began falling in Britain and the United States in the early 1970s, well before the widespread introduction of cimetidine. The long term safety of cimetidine and the emergence of more and more powerful ways of limiting or even abolishing the secretion of gastric acid has given gastroenterologists and general practitioners the confidence to manage medically almost all the exacerbations of peptic ulcer disease. What a change has happened in the past 20 years.

In 1901 Moynihan, in a lecture describing the impact of gastrojejunostomy on the management of duodenal ulcer disease, said, "Gentlemen, we need look no further."² (In those days there were no women abdominal surgeons in Britain.) In 1938 Ogilvie wrote, "The only safe course is to advise gastrectomy in all ulcers that are not rapidly healed by rest and diet. We can do this without hesitation because we know that the patients will live happily ever afterwards."³ The careful documentation of the high incidence of symptomatic and metabolic consequences of operations, particularly resection, has made their optimism seem almost ludicrous.

Surgical prophets now, as then, are inviting the indigestible prospect of eating their own words. Mindful of those risks I predict that vagotomy for ulcer will soon go the way of vagotomy for tabes; made obsolete by the conquest of spiral organisms. Perhaps we have not yet entirely conquered peptic ulcer disease by eradicating *Helicobacter pylori*, but we do have effective means of controlling acid secretion. Nowadays, the

term "intractable to medical treatment" is confined to those who are so unreliable or impoverished that they cannot take medication to control the exacerbations. The residual role of the surgeon is in treating the complications of bleeding, perforation, or stenosis, and even this role is diminishing. Stenoses are now amenable to balloon dilatation, bleeding can be controlled by endoscopically delivered topical treatment, and perforations that do not seal spontaneously usually need only sealing, not additional curative operations.

Those of us who worked so hard to perfect the operative control of acid secretion thought that in proximal gastric vagotomy, we had found the perfect operation and were about to say "Ladies and gentlemen, we need look no further" when we found that the dreaded disease was disappearing. I believe that even laparoscopic vagotomy will not prevent this nearly extinct operation from going the way of thoracoplasty and other once useful procedures. With so few occasions on which proximal gastric vagotomists can practise their technical art it is difficult either to keep in practice or to teach the next generation. In the early 1970s my surgical firm was performing 80-90 proximal gastric operations a year; now it is one or two a year. It seems a shame that the hard won skills are no longer needed, but I do not feel that this complex and theoretically attractive operation should still be part of the surgical training of the present generation of surgeons, at least

in the Western world that can afford the present generation of acid inhibiting drugs.

When Soll reviewed the options for managing refractory peptic ulcers in the *New England Journal of Medicine*⁴ he was taken to task by Morsch for failing to mention vagotomy and antrectomy or proximal gastric vagotomy.⁵ I support Soll's reply: although patients should be informed that surgery is an option, "the potential morbidity of operations, even if encountered by only a small number, makes elective surgery less advisable."

In many series the rate of ulcer recurrence after vagotomy is reported as between 15% and 40%.⁶ I think that gastric surgeons have to recognise that, despite valiant last ditch efforts to publish good reports of vagotomy, surgery is ever changing and vagotomy is on the way out.

J ALEXANDER-WILLIAMS

Professor of Gastrointestinal Surgery,
General Hospital, Birmingham B4 6NH

1 Latarjet A. Resection des nerfs de l'estomac. *Bulletin de l'Academie de Medicine Paris* 1922;97: 681-91.

2 Moynihan BGA. On duodenal ulcer and its surgical treatment. *Lancet* 1901;ii:1656.

3 Ogilvie H. Approach to gastric surgery: 2: ulcer of the stomach. *Lancet* 1938;ii:295.

4 Soll AH. Pathogenesis of peptic ulcer and implications for therapy. *N Engl J Med* 1990;322: 909-16.

5 Morsch HHC. Treatment of peptic ulcer. *N Engl J Med* 1990;322:998.

6 Hoffman J, Jensen HE, Christiansen J, Olesen A, Lord FB, Hauch O. Prospective controlled vagotomy trial for duodenal ulcer. Results after 11-15 years. *Ann Surg* 1989;209:40-5.

Vitamins, minerals, schoolchildren, and IQ

More questions than answers (still)

Once again research findings claiming to have important consequences for human health have reached the general public before the medical and scientific community has had time to evaluate them. This time it was through a programme in BBC television's *QED* series, entitled "Your Child's Diet on Trial—The Verdict," which claimed that schoolchildren's IQ could be improved by vitamin and mineral supplements. The results of only one of the three studies on which the programme was based were available at the time of the programme, published earlier that day in an obscure journal.¹

In this published study American schoolchildren were fed either placebo or increasing amounts of a multivitamin and mineral mixture. After treatment both groups performed better in a series of tests—probably explained by the learning effect. Any additional response in the children who received supplements was not linearly related to dose: in those children fed pills containing half or twice the recommended dietary allowances for the United States little response above placebo levels was found. It was only in the group receiving pills containing 100% of the recommended dietary allowances that there was anything of interest. Is it plausible, however, that intelligence would be so precisely sensitive to a given vitamin intake? As the authors point out, if more is not necessarily better then such a hypothesis begs further research. At the very least it would be important to regulate children's intake of supplementary vitamins and minerals.

Substantial variation existed around the mean IQ increments: the standard deviation of the change in non-verbal intelligence for the entire population studied was about three quarters that of the mean. The authors acknowledge that not everyone "benefited" from their pill but calculated that nearly

half did so when fed at the 100% recommended dietary allowances level. By the same reasoning, however it could be surmised from the variances that some children may have done worse, especially when fed 50% and 200% of the recommended allowances.

No physiological explanation exists of how vitamin and mineral supplementation could affect brain function in a well nourished subject. Although it would seem that additional information was collected, no before and after dietary or biochemical data has yet been provided on the nutritional state of the subjects. We clearly need to know if those children with the lowest habitual nutrient intakes improved the most.

So far, there have been three British studies published on this subject. The original one from Wales² claimed a significant response. Those carried out in London³ and Dundee⁴ have, however, failed to confirm these findings. Other studies are known to be under way. Any new findings should be published in such a way that they can be evaluated by fellow professionals before public claims are made.

ROGER G WHITEHEAD

Director,
MRC Dunn Nutrition Centre,
Cambridge CB4 1XJ

1 Schoenthaler SJ, Amos SP, Eysenck HJ, Peritz E, Yudkin J. Controlled trial of vitamin-mineral supplementation: effects on intelligence and performance. *Personality and Individual Development* 1991;12:351-62.

2 Benton D, Roberts G. Effect of vitamin and mineral supplementation on intelligence of a sample of schoolchildren. *Lancet* 1988;i:140-4.

3 Naismith DJ, Nelson M, Burley VJ, Gatenby SJ. Can children's intelligence be increased by vitamin and mineral supplements? *Lancet* 1988;ii:335.

4 Crombie IK, Todman J, McNeill G, Florey CDuV, Menzies I, Kennedy RA. Effect of vitamin and mineral supplementation on verbal and non-verbal reasoning of schoolchildren. *Lancet* 1990;335: 744-7.