3 Buchanan CR, Law CM, Milner RDG. Growth hormone in short, slowly growing children and those with Turner's syndrome. *Arch Dis Child* 1987;**62**:912-6.
4 Preece MA. Experience of treatment with pituitary derived growth hormone with special reference to immunological aspects. In: Milner RDG, Flodh H, eds. *Immunological aspects of human growth hormone*. Oxford: Medical Education Services, 1986:9-16.
5 Powell-Jackson J, Weller RO, Kennedy P, *et al*. Creutzfeldt-Jakob disease after administration of human growth hormone. *Lancet* 1985;ii:244-6.
6 Weller RO, Steart PV, Powell-Jackson JD. Pathology of Creutzfeldt-Jakob disease associated with pituitary-derived human growth hormone administration. *Neuropathol Appl Neurobiol* 1986;**12**:117-29.
7 Preece MA. Creutzfeldt-Jakob disease: implications for growth hormone deficient children. *Neuropathol Appl Neurobiol* 1986;**12**:509-15.
8 Brown PB, Gajdusek C, Gibbs CJ, Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;**313**:728-30.
9 Watanabe S, Tsunematsu Y, Fujimoto J, Komiyama A. Leukaemia in patients treated with growth hormone. *Lancet* 1988;i:1159.
10 Delemarre-Van de Waal HA, Odink JH, de Grauw TJ, de Waal FC. Leukaemia in patients treated with growth hormone. *Lancet* 1988;i:1159.
11 Fisher DA, Job J-C, Preece MA, Underwood LE. Leukaemia in patients treated with growth hormone. *Lancet* 1988;i:1159-60.
12 Frisch H, Thun-Hoenstein L, Balzar E. Leukaemia and growth hormone. *Lancet* 1988;i:1335.
13 Redman GP, Shu S, Norris D. Leukaemia and growth hormone. *Lancet* 1988;i:1335.
14 Stahnke N, Zeisel HJ. Growth hormone therapy and leukaemia. *Eur J Pediatr* 1989;**148**:591-6.
15 Milner RDG, Barnes ND, Buckler JMH, *et al*. United Kingdom multicentre clinical trial of somatrem. *Arch Dis Child* 1987;**62**:776-9.
16 Tanner JM, Whitehouse RH, Hughes PCR, Vince FP. Effect of human growth hormone treatment for 1 to 7 years on growth of 100 children with growth hormone deficiency, low birth weight, inherited smallness, Turner's syndrome and other complaints. *Arch Dis Child* 1971;**46**:745-82.
17 Standen GR, Hughes IA, Geddes D, Jones BM, Wardrop CAJ. Myelodysplastic syndrome with trisomy 8 in an adolescent with Fanconi anaemia and selective IgA deficiency. *Am J Hematol* 1989;**31**:280-3.
18 Brown PB. The decline and fall of Creutzfeldt-Jakob disease associated with human growth hormone therapy. *Neurology* 1988;**38**:1135-7.
19 Clayton PE, Shalet SM, Gattamaneni HR, Price DA. Does growth hormone cause relapse of brain tumours. *Lancet* 1987;i:711-3.
20 Clayton PE, Price DA, Shalet SM, Gattamaneni HR. Craniopharyngioma recurrence and growth hormone therapy. *Lancet* 1988;i:642.
21 Whitehead HM, Hadden DR, Carson DJ. The Northern Ireland experience of growth hormone therapy for short stature. *Ulster Med J* 1989;**58**:153-60.
22 Office of Population Censuses and Surveys. *Mortality statistics. Review of the registrar general on deaths by cause, sex and age in England and Wales*. London: HMSO, 1988. (Series DH2 1986.)
23 Goujard J, Entat M, Maillard F, *et al*. Human pituitary growth hormone (hGH) and Creutzfeldt-Jakob disease: results of an epidemiological survey in France, 1986. *Int J Epidemiol* 1988;**17**:423-7.
24 Farwell J, Flannery JT. Second primaries in children with central nervous system tumours. *J Neurooncol* 1984;**2**:371-5.
25 Kingston JE, Hawkins MM, Draper GJ, Marsden HB, Wilson LMK. Patterns of multiple primary tumours in patients treated for cancer during childhood. *Br J Cancer* 1987;**56**:331-8.
26 Arslanian SA, Becker DJ, Lee PA, Drash LA, Foley TP. Growth hormone and tumor recurrence. Findings in children with brain neoplasms and hypopituitarism. *Am J Dis Child* 1985;**139**:347-50.
27 Rodens KP, Kaplan SL, Grumbach MM, Teller WM. Does growth hormone therapy increase the frequency of tumor recurrence in children with brain tumors? *Acta Endocrinol (Copenh)* 1987;**283**(suppl):188-9.
28 Sherwood MC, Stanhope R, Preece MA, Grant DB. Diabetes insipidus and occult intracranial tumours. *Arch Dis Child* 1986;**61**:1222-5.
29 Stanhope R, Preece MA, Brook CGD, Grant DB. Is diabetes insipidus during childhood ever idiopathic? *Br J Hosp Med* 1989;**41**:490-1.
30 Cochius JI, Mack K, Burns RJ, Alderman CP, Blumbergs PC. Creutzfeldt-Jakob disease in a recipient of human pituitary-derived gonadotrophin. *Aust N Z J Med* 1990;**20**:592-3.
31 New MI, Brown P, Temeck JW, *et al*. Preclinical Creutzfeldt-Jakob disease discovered at autopsy in a human growth hormone recipient. *Neurology* 1988;**38**:1133-4.
32 Committee on Health Care Issues, American Neurological Association. Precautions in handling tissues, fluids, and other contaminated materials from patients with documented or suspected Creutzfeldt-Jakob disease. *Ann Neurol* 1986;**19**:75-7.

(Accepted 10 January 1991)

Dengue haemorrhagic fever: a risk of returning home

M G Jacobs, M G Brook, W R C Weir, B A Bannister

Coppetts Wood Unit, Royal Free Hospital Department of Infectious and Tropical Diseases, London N10
M G Jacobs, MB, *senior house officer*
M G Brook, MD, *senior registrar*
W R C Weir, MRCP, *consultant physician*
B A Bannister, FRCP, *consultant physician*

Correspondence and requests for reprints to: Dr Brook.

BMJ 1991;**302**:828-9

The number of imported cases of infection with dengue virus each year is rising as a consequence of increased travel and an increase in the worldwide incidence of dengue.[1] Almost all travellers who become infected with the virus experience a benign, if uncomfortable, febrile illness. We report on two adult residents of the United Kingdom who visited their country of origin and developed a severe, life threatening complication of this infection known as dengue haemorrhagic fever.

Case reports

Case 1—A 73 year old Malaysian woman had been living in the United Kingdom for many years. She spent two months visiting Malaysia and on her return complained of unremitting fever, anorexia, and nausea. By the fourth day she had developed nose bleeds, haemoptysis, haematuria, and a generalised petechial rash. Tests showed severe thrombocytopenia (platelet count 3×10^9/l) with a low white cell count (total white cells $1{\cdot}6\times10^9$/l; neutrophils $0{\cdot}48\times10^9$/l) and haemoglobin concentration (86 g/l). Virus related haemophagocytic syndrome was diagnosed on bone marrow aspiration.[2] Management consisted of treatment with broad spectrum antibiotics appropriate for neutropenia and platelet transfusions. She made an uneventful recovery. Infection with dengue virus was diagnosed by an eightfold rise in antibody titres.

Case 2—A 41 year old Pakistani man returned to the United Kingdom, where he had been living for many years, after a two week visit to Pakistan. He developed a confluent petechial rash after six days of fever, nausea, diarrhoea, and headache. Tests showed thrombocytopenia (platelet count 18×10^9/l) and a severe coagulopathy (partial thromboplastin time 66 s; thrombin time >120 s). He was treated with blood products and made an uneventful recovery. Infection with dengue virus was diagnosed by an eightfold rise in antibody titres.

Comment

Dengue viruses, comprising four distinct serotypes of flavivirus, are transmitted from infected to susceptible humans by *Aedes* mosquitoes. This vector is ubiquitous in the tropics, where dengue is a major public health problem. The first infection with dengue virus, usually in early childhood, may pass unrecognised or cause a self limiting febrile illness. This is followed by lifelong homotypic immunity, but after a few months of cross protection the subject is susceptible to infection with other serotypes.[3] Considerable evidence suggests that haemorrhagic complications are associated with subsequent infection with a second serotype. This explains an early observation that foreign visitors who have not been exposed to dengue virus previously develop uncomplicated dengue during outbreaks of dengue haemorrhagic fever among indigenous people.[4] It is believed that "immune enhancement" of viral replication underlies the development of severe, haemorrhagic disease: non-neutralising, cross reacting antibodies opsonise virus of a second serotype and enhance uptake into mononuclear phagocytes, in which the virus replicates.[5]

Large epidemics of dengue haemorrhagic fever among children are frequently reported in the tropics, particularly in South East Asia and the Caribbean, with an associated mortality of 2-10%. This syndrome is rare in adults and has not, we believe, been reported before in travellers returning to the United Kingdom. We presume that our patients acquired dengue haemorrhagic fever as a result of infection with a second serotype of dengue virus after a delay of many years since their first exposure to the virus. This potential problem should be remembered whenever people return to the tropics, and they should be

encouraged to be assiduous in their measures to prevent mosquito bites while abroad, especially if there is an epidemic of dengue at their destination.

1 World Health Organisation. Dengue haemorrhagic fever: diagnosis, treatment and control. Geneva: WHO, 1986.
2 Risdall RJ, McKenna RW, Nesbit ME, *et al.* Virus-associated hemophagocytic syndrome. *Cancer* 1979;**44**:993-1002.
3 Sabin AB. Research on dengue during world war II. *Am J Trop Med Hyg* 1952;**1**:30-50.
4 Halstead SB, Udomsakdi S, Singharaj P, Nisalak A. Dengue and chikungunya virus infection in man in Thailand, 1962-1964. III. Clinical, epidemiologic, and virologic observations on disease in non-indigenous white persons. *Am J Trop Med Hyg* 1969;**18**:984-96.
5 Halstead SB. Pathogenesis of dengue: challenges to molecular biology. *Science* 1988;**239**:476-81.

(Accepted 8 January 1991)

Influence of undergraduate teaching on medical students' attitudes to rectal examination

T W Hennigan, P J Franks, D B Hocken, T G Allen-Mersh

Department of Surgery, Charing Cross and Westminster Medical School, London W6 8RF
T W Hennigan, FRCS, *lecturer*
P J Franks, PHD, *research fellow*
D B Hocken, FRCS, *lecturer*
T G Allen-Mersh, FRCS, *consultant*

Correspondence to:
Mr Hennigan.

BMJ 1991;**302**:829

The confidence of general practitioners in their ability to diagnose a condition based on rectal examination and a belief that they have been thoroughly taught rectal examination at medical school appreciably influence general practitioners' frequency of rectal examination.[1] We investigated medical students' experience of rectal examination during training and assessed whether teaching at medical school influences attitudes to rectal examination.

Subjects, methods, and results

We sent a questionnaire to 119 final year medical students in one medical school. General surgical subspecialty interest (breast, gastrointestinal, vascular, urological, or general) of the four firms that each student had been attached to and whether attachments were in teaching or non-teaching hospitals were recorded. Students were asked about the number of rectal examinations they had performed for specific anorectal conditions and in total; formal teaching of rectal examination, seniority of teacher, when they were taught, and whether they were taught on anaesthetised patients; reasons for omitting routine rectal examination; and confidence in their diagnosis of specific anorectal conditions based on rectal examination. The end points were categorised and analysed using Kendall's τ C test.[2] Overall score for confidence in diagnosis was determined by summing the values (yes=1, no=0) for the five conditions (range 0 to 5). Confidence score was dichotomised around the median (0 to 3, 4 to 5).

We received replies from 114 medical students (96% response rate). The median category for total number of rectal examinations performed was 11 to 30; 23 had done fewer than 10 examinations and 19 had never felt a rectal cancer. The table shows the main results.

Only 32 students routinely performed a rectal examination when examining patients. Factors that deterred students from rectal examination were being told not to do so by medical staff (35 students), embarrassment (14), refusal of patients (10), and lack of chaperon (three). Students who had done more than the median number of rectal examinations were significantly more confident about diagnosing rectal cancer (τ C=0·174, p=0·013), benign prostatic hyperplasia (τ C=0·150, p=0·006), prostate cancer (τ C=0·142, p=0·028), and anal fistula (τ C=0·157), p=0·030) than were those who had done fewer than the median. Confidence was significantly greater about diagnosing benign prostatic hyperplasia (τ C=0·108, p=0·026), prostatic carcinoma (τ C=0·185, p=0·004), rectal carcinoma (τ C=0·135, p=0·032), and anal fistula (τ C=0·143, p=0·031) among students who had done more than two non-teaching hospital attachments than it was among those who had done fewer.

Factors influencing students' confidence of diagnosis based on rectal examination

	Confidence score		τ C	p Value
	0-3	4-5		
Consultant teaching:				
No	62	19	0·156	0·022
Yes	19	14		
Formal teaching:				
No	26	5	0·139	0·033
Yes	55	28		
Teaching on anaesthetised patients:				
No	29	5	0·170	0·015
Yes	52	28		
No of attachments to gastrointestinal or urological firms:				
0-2	19	1	0·168	0·005
3-4	62	32		
No of attachments to teaching hospital firms:				
0-2	56	26	−0·079	0·005
3-4	25	7		

Comment

Formal teaching (especially by a consultant) encouraged the view among medical and nursing staff that students are expected to do rectal examinations. Only 33 students had been formally taught rectal examination by a consultant. More confident students did more rectal examinations. Easily deterred students would become more confident if clinicians encouraged rectal examination. The outpatient clinic is ideal for one to one teaching and minimises patient and student embarrassment. Attachment to a firm with a subspecialty interest that regularly used rectal examination in diagnosis and treatment increased confidence.

Possible reasons for attachment to a non-teaching hospital increasing confidence include the presence of fewer students and the heavier general surgical emergency workload, which provides a greater variety of anorectal conditions. Students in non-teaching hospitals did not receive more formal tuition than those in teaching hospitals.

Though the factors affecting confidence identified might seem obvious, there is scope for improvement. A fifth of students had done fewer than 10 rectal examinations and 54% had been deterred from routine rectal examination. Frequency of rectal examination after graduation is influenced by attitudes acquired during training.[1] Consultant teaching and emphasis on the importance of rectal examination by students will increase confidence and produce doctors who are more willing to perform rectal examination.

We thank the medical students for completing the questionnaires.

1 Hennigan TW, Franks PJ, Hocken DB, Allen-Mersh TG. Rectal examination in general practice. *BMJ* 1990;**301**:478-80.
2 SPSS Incorporated. *Statistical package for the social sciences—X: user's guide.* 2nd ed. New York: McGraw Hill, 1986.

(Accepted 21 January 1991)