this complication may be kept to a minimum by overhydration before treatment or by occluding the ureters during instillation with Fogarty balloon embolectomy catheters.[11] Steroids, epsilonaminocaproic acid, silver nitrate,[12] and phenol[13] have also been used, but all have either proved too toxic or been ineffective. Infusion of vasopressin does stop bleeding (as it does with oesophageal varices) but only for as long as the infusion is continued.[14] Helmstein balloon compression will halt the bleeding in 80% of cases,[15] but—like chemical instillation—it causes fibrosis and a reduction in bladder capacity.[16] This can be a serious problem when the bladder is already thickened and contracted as a result of the primary disease.

Arterial embolisation of the bladder may control the bleeding, especially that due to severe pelvic trauma, but it does not always work in practice.[1] While ligation or embolisation of large vessels seems to be safe more peripheral embolisation has been associated with a real risk of necrosis of the bladder.[17] One practical point worth mention is that simple transurethral diathermy sometimes actually worsens the bleeding; the best chance of success with diathermy is to use the minimum current necessary to produce coagulation.[18] If this fails, alum seems to be a safe treatment to use before resorting to more toxic agents such as formalin, which require regional or general anaesthesia for their instillation.

Some patients will not respond even to these measures, and supravesical urinary diversion may be necessary—for, surprisingly, diversion alone may stop the bleeding. The same may be achieved in gravely ill patients by nephrostomy or cutaneous ureterostomy.[19] A few patients, however, will continue to bleed uncontrollably from the bladder. Emergency cystectomy and diversion must then be considered despite its attendant morbidity and mortality. The prognosis for patients who stop bleeding depends on the nature of the underlying disease.[19]

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Adolescent soma and psyche

Everyone knows that adolescents may be difficult and are best kept at arm's length until their hormones settle down. Those with symptoms, whether physical or emotional, often fall between two stools—too old for paediatricians or child psychiatrists but not yet fully adult and therefore not of prime interest to general physicians and psychiatrists. The patterns of presentation of disorders in adolescents may, therefore, be omitted from both undergraduate and postgraduate teaching.

Some centres in the United States are now specialising in adolescent medicine, and one in Nashville has recently published a study of the link between recent life events and certain somatic and behavioural disorders.[1] Using an adolescent life events questionnaire,[2] the authors found that those patients with recurrent abdominal or chest pain for which no organic cause could be identified reported more stress than patients being seen for routine check ups, acute minor illness, stable chronic illness, or pain with a clinically diagnosed organic cause.[1]

The life events questionnaire was administered independently of the clinician and the scores were not disclosed until diagnostic evaluation was completed. The score for negative life changes in the preceding year was highest for patients referred for behavioural problems (22·5) and recurrent non-organic pain (13·2). Other conditions gave mean scores of 3·5-4·1. The events present most often included death and illness in the family, increased arguments between parents, failures at school, and financial changes.

Clearly a strong case can be made for including an inquiry about adverse life events in any evaluation of adolescent symptoms. Such an inquiry may help patients and parents to recognise that doctors rate psychological factors as important; it may also convince parents who have been reluctant to consider stress as a possible factor in their teenagers' symptoms. And it may point the way to possible intervention strategies.

Organic disease, too, occurs after stressful events more frequently than chance allows (reviewed by Connolly[3] and Graham[4]). Much of the research has been poorly designed, however; one criticism is that a depressed patient may view events as causal when they may have been produced by the psychiatric disorder or have a common root. For example, a husband may desert a depressed wife, who then blames the desertion for causing her depression.

In practical terms, what should alert a general practitioner or physician to the possibility of predominant psychological factors in adolescents presenting with somatic disorders? Three psychiatric conditions commonly present in this way and may be more effectively treated—and sometimes cured—if recognised early. Sadly, they are often missed by physicians until the disorder has become chronic.

Firstly, the masquerade syndrome, described by Waller and Eisenberg, is a variant of school refusal, or phobia, in which separation anxiety (in both parent and child) leads to abdominal pain, vomiting, and headaches but only on school days.[5] It disappears if permission is given to stay home. It is more frequent in children with life threatening disease; and it responds well to immediate return to school.[6] Patient and parent often do not alert the physician to the unnecessary school absence, and it must be inquired about if the patient is not to be educationally crippled.

Secondly, disorders of appetite are often present in adolescence but they may be missed in the early stages—

when treatment is easiest and most effective. Anorexia nervosa commonly follows teasing because of plumpness. Parents may collude with the girl in condoning the request for slimming pills or diet from the physician. Large amounts of weight may be lost unobtrusively when garments are concealing. All adolescents presenting with abnormalities of appetite or weight should be weighed and measured; and the doctor needs to review their progress regularly, charting the weight on standard charts, and referring for specialist treatment if the weight continues to fall or rise abnormally.[7]

Finally, manic-depressive disorder commonly starts in adolescence and is the cause of most sudden and unexpected suicides in young people. It may present with hypochondriasis. Any young person who presents with recurrent symptoms for which no organic cause can be found (pain, headache, menstrual symptoms, fatigue, and so on) should be specifically examined for depression and a family history obtained. Such action may be life saving.

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The trypanosomiases

Trypanosomiases kill thousands of people in Africa and South America every year, a fact recognised by their inclusion in the Special Programme for Research in Tropical Diseases sponsored by the World Health Organisation and the World Bank.[1] Earlier this year the *British Medical Bulletin* published a comprehensive review of the human diseases caused by these protozoa. In South America and east Africa animals provide a reservoir of the trypanosome species which infect man, and closely related species remain a serious impediment to the rearing of cattle and other livestock throughout tropical Africa. Brazil is most severely affected by South American trypanosomiasis, with 120 000 new cases of Chagas' disease each year, but most other countries of south and central America have regions affected by severe, if often very local, disease.[1]

African trypanosomiasis is almost invariably fatal if untreated. The annual incidence is now at least 20 000 and has more than doubled since 1979 as a result of epidemics in Cameroon, Sudan, and Uganda. The end stage of west African trypanosomiasis, the diffuse meningoencephalitis known as sleeping sickness, has been recognised for centuries, though its cause was not identified until 1901. Soon afterwards Bruce proved that the disease was transmitted by tsetse flies. In 1908 the first recorded case of a more rapidly progressive form of trypanosomiasis occurred in a European who was travelling in south east Africa. The west African variant is now termed *Trypanosoma brucei gambiense* and the east African *T brucei rhodesiense*. The two strains cannot infallibly be distinguished either morphologically or by newer techniques such as analysis of isoenzymes and deoxyribonucleic acid. The human diseases show considerable overlap, though the east African form generally runs a more acute course. Duggan and Hutchison reviewed 109 cases of African trypanosomiasis in Europeans and found that all these patients had an initial disease characterised by feverish episodes of diminishing severity and frequency. This was followed by a quiescent phase, which varied from a few weeks in "Rhodesian" disease to as long as three years in one "Gambian" case, culminating in late disease with widespread manifestations.[2] Ecologically the two African forms are distinct diseases,[3,4] though the traditional view that the eastern is a zoonosis and the western a purely human infection has been blurred by the discovery in both wild and domestic animals in west Africa of trypanosomes with enzyme markers (zymodemes) identical with those of species infective for man.

The febrile episodes of the first phase of disease coincide with peaks of parasitaemia, during which the trypanosomes multiply by binary fission. Most are of the same antigen type and are killed when the host responds by producing IgM. A few trypanosomes, however, have other antigen types which continue to multiply during remissions until one overgrows the others to cause a recrudescence. At least 60 different antigen types have been detected from a single cloned strain of *T brucei rhodesiense*.[5]

Antigenic variation does not occur in American trypanosomiasis, but the pathogenesis of late disease—which fortunately appears to develop in only a few of those infected—seems no longer to require the presence of the parasite. Chemotherapy is then too late to prevent progression, so that this disease can be treated only in its early stage, which may closely resemble glandular fever but usually goes unrecognised.[6]

Eradication of the trypanosomiases from either continent is impossible without wholesale destruction of wildlife. The development of vaccines faces immense obstacles: antigenic variation in African trypanosomes and the risk of accelerating autoimmunisation in the American disease. In its late stages Chagas' disease is untreatable, and the drugs used in the treatment of advanced African trypanosomiasis often produce serious adverse effects. The outlook is not, however, entirely hopeless—it is just that medical strategies will always play a relatively small part in the control of these diseases. The recent upsurge of African disease is due to the disruption of control programmes by civil unrest and anarchy. Chagas' disease could be eliminated if the rural population at risk in South America could be rehoused in modern dwellings, in which the reduviid bug vectors cannot live. These are social and political diseases.

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