He had drunk about 250 ml of concentrated hydrochloric acid (32% w/v, 8N, pH 0.9) used for cleaning toilet bowls. He had acid burns on his lips and oral mucosa and mild tenderness in the epigastrium but no other abnormal physical signs.

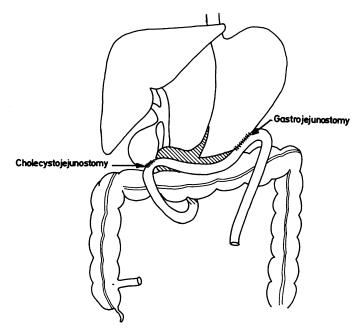
He was given oral antacids and was fed intravenously. Barium meal examination five days later showed that the oesophagus and proximal part of the stomach were normal, but the distal part and the prepyloric antrum were narrowed and irregular; the duodenum was normal. Endoscopy showed a normal oesophagus and mild inflammation of the proximal part of the stomach. The mucosa of the distal part and antrum showed severe necrosis and ulceration. Narrowing of the pyloric canal prevented inspection of the duodenum.

A psychiatrist diagnosed reactive depression and arranged for a social worker to help ease the family's financial problem. On discharge the patient was well and eating normally.

He was readmitted seven weeks later having vomited undigested food and passed pale stools and dark urine for four days. He looked ill, dehydrated, and jaundiced and had a dilated stomach with a succussion splash. Biochemical investigations confirmed obstructive jaundice, and barium meal examination and gastroscopy showed complete pyloric stenosis.

Ultrasound scanning of the abdomen showed a dilated extrahepatic biliary system down to the ampulla of Vater but no other abnormalities; the pancreas was normal.

The dehydration and clotting disorders were corrected, and intravenous feeding was begun. Because of deepening jaundice and general deterioration laparotomy was done. The prepyloric antrum and duodenum down to the duodenojejunal flexure were considerably thickened and contracted, and the gall bladder and common bile duct were dilated. Palpation of the head



Gastrojejunostomy and cholecystojejunostomy created to bypass obstructed stomach and common bile duct. Hatched area shows extent of damage caused by ingested acid to stomach and duodenum.

of the pancreas was normal. A duodenotomy showed that the wall of the duodenum was thickened, and its lumen, which was almost obliterated, contained no bile. The mucosa was inflamed and ulcerated, and the duodenal papillas were unidentifiable. A full thickness biopsy specimen of the duodenum was taken and the duodenotomy closed. A loop of jejunum was sutured to the gall bladder and stomach to create a cholecystojejunostomy and a gastrojejunostomy, bypassing the head of the pancreas and the duodenum (figure). Histological examination of the duodenal biopsy specimen showed ulceration of the mucosa, round cell infiltration, and extensive fibrosis of the duodenal wall.

He recovered completely, the jaundice cleared, and he gained weight with a normal diet. Computed tomograms of his abdomen three and six months later showed no evidence of tumour in the biliary tree or pancreas.

### Comment

Clinically important corrosive injury to the upper gastrointestinal tract after ingestion of mineral acid occurs most commonly in the prepyloric antrum and sometimes in the oesophagus.1 Damage has also been reported in the proximal duodenum and the ileum in cases of severe ingestion.25

The cause of obstructive jaundice in this patient was thought to be either a coexisting lesion in the biliary tree or pancreas, such as

stones or a tumour, or the result of duodenal injury caused by ingestion of acid. A lesion was unlikely because of the lack of evidence at laparotomy and the negative results of computed tomography. Thus it appears that if severe fibrosis in the duodenal wall spreads into the papillary area complete obstruction of the common bile duct may occur, leading to obstructive jaundice.

- Di-Costanzo J, Cano N, Martin J, Noirclerc M. Surgical approach to corrosive injuries of the stomach. Br J Surg 1981;88:879-81.
   Herrington JL. Stenosis of the gastric antrum and proximal duodenum resulting from the ingestion of a corrosive agent. Am J Surg 1964;107:580.
   Maull KI, Scher LA, Greenfield LJ. Surgical implications of acid ingestion. Surg Gynecol Obstet 1979;148:895-8.

(Accepted 15 November 1984)

Department of Surgery, Ealing Hospital, Middlesex UB1 3HW DAVID P SELLU, CHM, FRCS, registrar

# Loss of form in young athletes due to viral infection

Many athletes experience sudden and unexplained deterioration in performance in training or competition. This is often attributed to overtraining or psychological factors, but a medical illness may cause a temporary loss of form in a previously fit athlete. In the past year I have seen 12 athletes complaining of loss of form with no features suggesting an underlying medical cause. No medical abnormality was found in eight of them, though four were undergoing the stress of academic examinations. The four others showed evidence of a recent viral infection and are reported on here.

### Case reports

Case 1-A 15 year old middle distance runner complained of loss of stamina and inability to manage his normal training schedule. His competition performance had also deteriorated. The problem had been preceded by a mild infection of the upper respiratory tract and a sore throat not serious enough for him to have consulted a doctor. Examination showed several small supraclavicular lymph nodes. Atypical mononuclear cells were visible in a blood film, and a screening test for infectious mononucleosis (Monospot) gave positive results. Training was temporarily reduced, and he had regained his form after four months.

Case 2-An 18 year old cross country runner complained of two months of malaise, tiredness, and difficulty in training. She had not had any upper respiratory tract symptoms. Examination gave normal results. Estimation of viral titres showed a considerable increase in Coxsackie B2 (1/512), indicating recent infection. After a short recovery period she regained her form over three months.

Case 3-An 18 year old cross country runner presented with loss of stamina, being unable to maintain his former training schedule. He had had no recent symptoms of the upper respiratory tract. Physical examination gave negative results, but he had a raised aspartate transaminase activity of 57 IU/I (normal range 12-42), which suggested mild hepatitis. A Monospot test gave positive results, indicating recent infectious mononucleosis. Six months later he was still complaining of tiredness and aching legs and had not been able to repeat previous performances.

Case 4-A 20 year old international sprinter had had an infection of the upper respiratory tract two months previously. She had subsequently felt weak and dizzy during training and had been unable to maintain her former training capacity. Examination gave negative results. Measurement of viral titres showed a pronounced increase in Coxsackie B3 (1/256), indicating recent infection. Her loss of form persisted throughout the track season.

## Comment

Two of these highly trained athletes had had no prodromal symptoms, and two had had minor symptoms of the upper respiratory tract. All had evidence of recent viral infections as shown by increased titres of antibodies against Coxsackie B or Epstein-Barr virus, and one had morphologically abnormal white blood cells suggesting recent viral infection.1

Viral infections are blamed for many minor illnesses. They may be subclinical and may give rise to symptoms beyond the acute infective phase. This post-viral syndrome produces various physical abnormalities, including excessive intracellular acidosis of skeletal muscles and persisting abnormal function of T cells.23 Pether

described two cases of bacterial meningitis in sportsmen who took part in sporting activities within a week of symptoms suggesting influenza; one of the patients died.4 Sutton et al described a 42 year old patient who died from heart failure after swimming. Coxsackie virus B4 was isolated from damaged myocardial tissue.5

Inquiry about recent minor illness should be standard practice in athletes with unexplained loss of form. It may also be worth considering a viral cause, as infections that are subclinical in the normal population may greatly affect maximum performance in athletes. Athletes with such infections might be tempted to increase their training load when a temporary reduction would be more appropriate.

Sprunt TP, Evans FA. Mononuclear leukocytosis in reaction to acute infection. Johns Hopkins Hospital Bulletin 1920;31:410-7.
 Arnold DL, Boe PJ, Radda GK, Styles P, Taylor DJ. Excessive intracellular acidosis of skeletal muscle in a patient with post viral exhaustion/fatigue syndrome. Lancet 1984j::1367-9.
 Hamblin TJ, Hussain J, Akbar AN, Tang YC, Smith JL, Jones DB. Immunological reason for chronic ill health after infectious mononucleosis. Br Med J 1983;287:85-8.
 Pether IVS. Bacterial meningitic after influence.

 4 Pether JVS. Bacterial meningitis after influenza. Lancet 1982;i:804.
 5 Sutton GC, Harding HB, Trueheart RP, Clark HP. Coxsackie B4 myocarditis in an adult: successful isolation of virus from ventricular myocardium. Aerospace 1067:39:66. Med 1967;38:66-9

(Accepted 7 November 1984)

#### Department of Respiratory Medicine, Western Infirmary, Glasgow G11 6NT

A ROBERTS, BSC, MRCP, medical registrar and medical adviser to the Scottish Amateur Athletic Association

## Tamoxifen as primary treatment of breast cancer in elderly or frail patients: a practical management

Tamoxifen is the most widely used agent in hormonal treatment of advanced breast cancer. It may have a role as primary treatment for elderly or frail patients who are unfit for surgery or primary radiotherapy.

### Patients, methods, and results

In 1977 we started using tamoxifen (10 mg thrice daily) instead of surgery as primary treatment for elderly or frail patients with histologically confirmed breast cancer. We report on the first 100 patients (mean age 76.3 years; 11

weeks (range 6-135). Median duration of tamoxifen treatment was 23 months in patients with a complete response (range 5-48), 18 months in patients with a partial response (range 6-55), and 15 months in the group showing no change. Two patients with a complete response and 10 with a partial response subsequently relapsed, giving a median duration of response of 19 months (mean 24 months, range 9-55); four received breast irradiation, two were

given aminoglutethimide, and six did not receive any further treatment. In 10 patients the disease progressed during treatment.

Oestrogen receptor state was determined in 37 patients, 35 of whom had concentrations >20 fmol/mg cytosolic protein—that is, were rich in receptor. The proportion of these patients who responded (74%) was similar to the proportion overall. The median oestrogen receptor concentration was 300 fmol/mg cytosolic protein. Patients with locally advanced (T4) disease responded less well. Side effects to tamoxifen occurred in 33 patients: dry mouth (13 patients), fatigue (10), transient nausea (10), vomiting (four), vaginal dysaesthesia (two), and vaginal discharge (two). One patient stopped treatment because of persistent nausea. Fourteen deaths occurred: six due to vascular disease in responders, and three due to vascular disease and five to disseminated carcinoma in non-responders.

#### Comment

Elderly patients with breast cancer can create problems in management when primary surgery or radiotherapy is considered to be inappropriate. Results of using tamoxifen as primary treatment have been encouraging in terms of response rates and possibly survival.23 The response rate in this study (68%) compares well with that in previous reports. The similar response in patients rich in oestrogen receptor and those whose receptor state was unknown suggests that such tumours in the elderly should be regarded as rich in oestrogen receptors.

We emphasise the slow time to response (median 15.5 weeks). The actuarial survival of our patients at five years was 52% compared with 42% (not significant) in a historical control group of elderly women treated by surgery or irradiation. Only five of the 14 deaths were due to carcinoma, and these were among non-responders. Tamoxifen was well tolerated by the patients: only one third experienced side effects, which were generally transient.

We believe that tamoxifen is an excellent and appropriate primary treatment for elderly women with breast cancer.

We thank Dr R A Hawkins for performing the oestrogen receptor assays, which are supported by a grant from the Cancer Research Campaign.

- 1 Hayward JL, Carbone PP, Heuson JC, Kumaoka S, Segaloff A, Rubens RD. Assessment of response to therapy in advanced breast cancer: a project of the programme on clinical oncology of the International Union Against Cancer, Geneva, Switzerland. Cancer 1977;39:1289-94.
- Preece PE, Wood RAB, Mackie CR, Cuschiera A. Tamoxifen as initial sole treatment of localised breast cancer in elderly women: a pilot study. Br Med J 1982; 224:869-70.
   Bradbeer JW, Kyngdon J. Primary treatment of breast cancer in elderly women with tamoxifen. Clin Oncol 1983;9:31-4.

Influence of oestrogen receptor state and size of tumour on response to treatment with tamoxifen

Response	No of patients	Oestrogen receptor state of patient			Size of tumour			
		Unknown	Rich*	Poor	<2 cm (T₁)	>2-5 cm (T <sub>2</sub> )	>5 cm (T <sub>3</sub> )	Locally advanced (T <sub>4</sub> )
Complete Partial No change Progressive disease	39 29 22 10	23 19 15 6	16 10 7 2	2	1	19 11 4 3	9 3 1 1	10 15 16 6
Total	100	63	35	2	2	37	14	47
No (%) responding	68 (68)	42 (67)	26 (74)	0	1 (50)	30 (81)	12 (86	i) 25 (53)

<sup>\*≥20</sup> fmol/mg cytosol protein.

aged 60-69, 26 aged 70-74, 35 aged 75-80, and 28 aged over 80) who received tamoxifen for an indefinite period or until their disease progressed (range five to 55 months). Over the same period of underwent surgery or radiotherapy; in 1980, however, we began giving primary endocrine treatment to most women over 70. Thirty eight of the patients studied had other major systemic disorders (for example, vascular disease, arthropathy, and dementia). After biopsy staging was confined to full blood count, tests of liver function, and chest and pelvic radiology. Two dimensional measurements of the tumours were assessed using criteria of the International Union Against Cancer<sup>1</sup> as indicating partial response, complete response, no change, or progressive disease.

The table shows the response and how this was influenced by the oestrogen receptor state and size of the tumour. Tumour was localised to the breast and axilla in 89 patients. Of 68 patients with objective regression of tumour, 39 had complete resolution. Median time to achieve best response was 15.5 (Accepted 16 November 1984)

### University Department of Clinical Oncology, Western General Hospital, Edinburgh EH4 2XU

SIMON G ALLAN, MB, MRCP, lecturer in medical oncology ALAN RODGER, MB, FRCS, consultant radiotherapist JOHN F SMYTH, MD, FRCP, professor of medical oncology ROBERT C F LEONARD, MD, MRCP, senior lecturer in medical oncology

### University Department of Clinical Surgery, Royal Infirmary, Edinburgh

UDI CHETTY, MB, FRCSED, senior lecturer in clinical surgery A PATRICK M FORREST, MD, FRCR, professor of surgery

Correspondence to: Dr S G Allan.