In nine patients fractures were apparent for the first time at three weeks: three were of Lister's tubercle, one of the distal radius, one of the lunate, and four of the scaphoid. One of these patients (studied retrospectively) was excluded as only views of the wrist had been taken initially. Therefore, three patients (2% of patients presenting with clinical scaphoid injuries) were ultimately proved to have scaphoid fractures. All three fractures were observed only in those patients who still had tenderness at three weeks. No fractures were observed in patients without tenderness at three weeks.

*Outcome of treatment of 159 patients for clinical carpal scaphoid injury*

| | No | Average duration of treatment (days) | |
|---|---|---|---|
| | | Time in plaster | Entire treatment |
| No tenderness at 3 weeks | 120 | 22 | 32 |
| Tenderness at 3 weeks | 39 | 46 | 92 |

## Comment

Clinical scaphoid fracture is a definite entity. The 2% incidence we found by radiography at three weeks is low and correlates with the findings of others.[3] These fractures healed without complications but required three months' immobilisation. Patients with tenderness at three weeks and no fractures evident on radiography pose a problem. Serious consideration should be given before the joint is immobilised in plaster in these cases: a delay in treatment does not preclude union even of proved fractures.[4]

Treatment demands clinical and radiographic examination to exclude a fracture or injuries such as scapholunate dissociation. Radiographs may be supplemented with a view in 30° angulation to the elbow.[5] When no fracture is observed the wrist should be immobilised with an easily fitted splint. Patients should be reassessed clinically and radiologically at three weeks, and those with a visible fracture should start conventional treatment. Patients with normal radiographs who still complain of tenderness should be reviewed again, but attendances beyond six weeks should be discouraged. This regimen will reduce the use of plaster casts and outpatient attendances. Reusable splints may be cost effective, though plaster casts are sometimes necessary for soft tissue injuries. Initial tenderness over the scaphoid is not necessarily specific to fractures of the scaphoid; the rate of fractures missed initially (3%) is compatible with the rate missed on examination by junior doctors.

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# Body building and myoglobinuria: report of three cases

Myoglobinuria is a syndrome characterised by the passage of dark urine due to massive muscle necrosis. It is related either to disorders of muscle metabolism or to excessive exercise of presumably normal muscle.[1,2] Several cases of exertional rhabdomyolysis have been reported, such as in status epilepticus, prolonged myoclonus, dystonia, military training, marathon running,[1,2] and weight lifting.[3] We report three cases of myoglobinuria that occurred after a first session of body building.

## Case reports

*Case 1*—A 20 year old medical student, a keen skier and tennis player, had never experienced cramps or myalgias after exercise, including a four hour tennis match. After his first one hour session of body building, aimed chiefly at strengthening pectoral and abdominal muscles, he complained of severe muscle pain and weakness and of passing dark urine. Several days later he still had pain and his father, a doctor, suggested routine laboratory tests and measurement of serum creatine kinase activity. The routine tests gave normal results but serum creatine kinase activity was 10 times higher than normal. Four months later he was referred to us. Neurological examination showed an athletic young man with normal strength and reflexes. Serum creatine kinase activity and results of electromyography were normal. The forearm ischaemic exercise test[4] induced a normal increase in venous lactate. An open biopsy sample of quadriceps muscle showed normal morphology and ultrastructure. Staining with periodic acid Schiff, Sudan black B, and oil red O and histochemical staining for lactate dehydrogenase, succinic dehydrogenase, reduced nicotinamide adenine dinucleotide tetrazolium reductase, cytochrome c oxidase, phosphorylase, phosphofructokinase, and adenylate deaminase gave normal results. Biochemical investigations on muscle homogenate (table) showed no enzyme deficiencies.

*Case 2*—A 23 year old student regularly played competitive football. After his first session of body building in the same club as case 1 he experienced muscle pain and weakness and passed dark urine. One week later the pain persisted and he attended his family doctor (father in case 1). Routine laboratory tests showed nothing abnormal, but serum creatine kinase activity two weeks after the episode of dark urine was five times higher than normal. The patient was admitted one month later and neurological findings, serum creatine kinase activity, and results of electromyography and the forearm ischaemic exercise test were all normal. Morphology, ultrastructure, and results of histochemical and biochemical studies of an open biopsy sample of quadriceps muscle were also normal.

*Case 3*—A 34 year old doctor was a skier and horse rider. After his first session of body building at a different club from that in cases 1 and 2 he complained of muscle tenderness and weakness and voided dark urine. Three days later the serum creatine kinase activity was 180 times the normal value but routine laboratory tests gave normal results. At 10 days the muscle pains had disappeared and creatine kinase activity had returned to normal. On admission two months later neurological findings, result of the forearm ischaemic exercise test, creatine kinase activity, results of electromyography, and a quadriceps muscle biopsy specimen (table) were all normal.

*Biochemical studies of muscle (enzyme activities expressed as nmol/min/mg non-collagen proteins)*

| Enzyme | Case 1 | Case 2 | Case 3 | Control range (n=10) |
|---|---|---|---|---|
| Phosphorylase | 0·29 | 0·22 | 0·17 | 0·12 -0·30 |
| Phosphoglucomutase | 0·080 | 0·069 | 0·073 | 0·020-0·100 |
| Glucosephosphate isomerase | 0·97 | 0·77 | 0·95 | 0·37 -1·37 |
| Phosphofructokinase | 0·39 | 0·42 | 0·30 | 0·23 -0·47 |
| Aldolase | 0·67 | 0·70 | 0·62 | 0·61 -0·71 |
| Glyceraldehyde-3-P-dehydrogenase | 1·97 | 2·03 | 2·79 | 0·79 -4·03 |
| Phosphoglycerate kinase | 1·56 | 1·67 | 1·73 | 0·33 -3·17 |
| Phosphoglycerate mutase | 1·68 | 1·92 | 1·73 | 0·67 -3·03 |
| Enolase | 0·42 | 0·53 | 0·27 | 0·10 -1·34 |
| Pyruvate kinase | 1·43 | 1·22 | 1·57 | 0·55 -3·31 |
| Lactate dehydrogenase | 2·36 | 2·78 | 3·91 | 0·76 -5·60 |
| Carnitine palmitoyltransferase: | | | | |
| Forward | 0·628 | 0·260 | 0·170 | 0·147-0·587 |
| Isotope exchange | 0·690 | 0·340 | 0·299 | 0·132-0·772 |
| Reverse | 6·930 | 3·820 | 2·970 | 1·260-9·260 |

## Comment

These cases may be classified as myoglobinuria due to excessive exercise. The results of laboratory tests and investigations on muscle biopsy tissue ruled out myoglobinuria due to metabolic defects.[1,2] We cannot exclude unknown enzyme deficiencies as the cause of the myoglobinuria in these patients but the occurrence of a single episode in well trained subjects makes this unlikely. That the episode occurred under the same conditions in all three patients suggests a close relation between exercise and rhabdomyolysis.

Body building has not been mentioned among the different kinds of exercise causing myoglobinuria[1-3] but has been reported in association with increased serum creatine kinase activity in one patient.[5] In contrast with other types of physical activity, body building uses muscles usually less trained, such as the pectorals, trapezii, and abdominal muscles; hence it might cause myoglobinuria in people regularly engaged in sports. The occurrence of myoglobinuria in our patients after their first session of body building aimed at strengthening pectoral and abdominal muscles supports this hypothesis.

Given that our patients (a medical student, student, and doctor respectively) were particularly likely to attach importance to their symptoms we conclude that many other such cases may go unrecognised. We therefore recommend caution for anyone—including healthy sporting subjects—undertaking their first sessions of body building.

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# Blackwater fever caused by Plasmodium vivax infection in the acquired immune deficiency syndrome

Blackwater fever is usually associated with malaria caused by *Plasmodium falciparum* infection in susceptible subjects. It is not associated with other parasites in man.[1] We describe a case of blackwater fever caused by *P vivax* infection in a patient with the acquired immune deficiency syndrome (AIDS).

## Case report

A 37 year old African woman was admitted with fever, diarrhoea, and vomiting. Two years previously she had complained of intermittent attacks of fever of several months' duration, which had been followed by oral candidiasis and genital herpes. She had been treated with ketoconazole and ampicillin with some improvement, though she had continued to lose weight. She then developed cervical lymphadenopathy; biopsy showed non-specific lymphadenitis. A week before admission she had developed fever, diarrhoea, and vomiting. At first she was thought to have malaria, but she did not improve despite treatment with chloroquine. Typhoid fever was then suspected, but her symptoms did not respond to ampicillin.

On examination she looked ill and was mildly anaemic and dehydrated, but not jaundiced. Blood pressure was normal, and she was tender over the right flank. Haemoglobin concentration was 110 g/l, white cell count $8{\cdot}2\times10^9$/l, and erythrocyte sedimentation rate 45 mm in the first hour. Her glucose-6-phosphate dehydrogenase activity was not measured. A film of the peripheral blood did not show malaria parasites. She had antibodies to the human immunodeficiency virus (HIV) on enzyme linked immunosorbent assay (ELISA). Blood was not cultured for viruses or bacteria. No parasites or salmonellas were isolated from her stools. Her urine was sterile on culture, though it contained protein.

She was treated with intravenous fluid replacement, ampicillin 500 mg six hourly intravenously, and antiemetics. Three days later she was still feverish and had become delirious and confused. She seemed more anaemic and was jaundiced, though there was no evidence of a bleeding diathesis. She was tachypnoeic with bronchopneumonia, and a chest *x* ray film suggested infection with *Pneumocystis carinii*. Her urine was dark, and the output was reduced. No red cells were seen in the urine, but benzidine and guaicum tests showed free haemoglobin. Her haemoglobin concentration was 92 g/l, the white cell and platelet counts were normal, and the erythrocyte sedimentation rate was 40 mm in the first hour. A film of the peripheral blood showed that about 95% of the red cells had been invaded by *P vivax*. Analysis of the urine showed a protein concentration of 1 g/l but no red or white cell casts were seen.

Blackwater fever due to infection with *P vivax* was diagnosed, and she was treated with chloroquine, primaquine, and high doses of co-trimoxazole for suspected *Pn carinii* pneumonia. She died the same day, and permission for necropsy was refused.

## Comment

This may be the first report of blackwater fever due to *P vivax* infection, which is less common than *P falciparum* in east Africa. Over two years the patient had become progressively more immunocompromised by HIV infection and her symptoms had been mistakenly attributed to causes other than vivax malaria. Though the prevalence of subjects with antibodies to HIV is high in central Africa and parts of east Africa, there have been no reports that malaria is more severe or more common among patients with AIDS.[2] It is worth noting that she had severe parasitic invasion at the time of haemolysis, which is uncommon. Furthermore, only about 2% of red cells are usually invaded by parasites in patients with malaria caused by *P vivax*, increasing to over 5% if the patient is immunocompromised; in this case 95% were affected. *Pn carinii* pneumonia is an unusual complication of the disease. The diagnosis of blackwater fever itself might be questioned, particularly as glucose-6-phosphate dehydrogenase activity was not measured. This would be to miss the point that fatal infection with *P vivax* occurred in a patient with AIDS, and it is unfortunate that permission for necropsy was refused.

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# Adult epiglottitis due to Vibrio vulnificus

Adult epiglottitis is rare and characterised by dysphagia, upper airway obstruction, and toxaemia; it may progress rapidly to fatal asphyxiation.[1,2] We report on a man with β thalassaemia major who developed acute epiglottitis and septicaemia due to *Vibrio vulnificus*. We know of no previous report of this organism causing epiglottitis.

## Case report

A 22 year old man with β thalassaemia major receiving regular transfusions and desferrioxamine presented to the casualty department with a sore throat, which had started several hours earlier. He had had a splenectomy when aged 8 and had been prescribed phenoxymethylpenicillin V 250 mg twice daily prophylactically but had complied only occasionally. While in the casualty department he experienced difficulty in breathing and developed inspiratory stridor. He was unable to speak or swallow, and there was drooling of saliva. He had enlarged, discrete, mobile cervical glands, and one axillary gland was enlarged. The tonsils were enlarged and the throat inflamed and oedematous; his temperature was 39·8°C, pulse rate 100/min, and blood pressure 110/70 mm Hg. The chest was clear on clinical and radiographic examination. Haemoglobin concentration was 9·9 g/l, white cell count (corrected for nucleated red cells) $55\times10^9$/l (81% neutrophils), and platelet count $301\times10^9$/l. Acute epiglottitis was diagnosed provisionally and laryngoscopy performed under general anaesthesia.[3] A large, swollen, yet pale epiglottis was seen occluding the vocal cords. He was intubated with difficulty, and mechanical ventilation was started. He was started empirically on intravenous benzylpenicillin 1200 mg four hourly and erythromycin 500 mg six hourly.

The next day a Gram negative rod was isolated from a blood culture and throat swab. In view of this intravenous cefuroxime 1500 mg eight hourly was substituted for erythromycin. The white cell count rose to $74\times10^9$/l (90% neutrophils). Over the next 24 hours his condition improved but he remained feverish (37·5°C). Two days later laryngoscopy was repeated; the epiglottis was normal but the throat still inflamed. He was given a transfusion and chelation with desferrioxamine was continued. His condition stabilised well and he was extubated after 72 hours. He made satisfactory progress, continuing with intravenous antibiotics for a week before changing to oral phenoxymethylpenicillin 500 mg six hourly.

We tried unsuccessfully to identify the Gram negative rod by inoculating the organism into Analytical Profile Index 20E. It was found to be indole and oxidase positive and to have a darting motility and was therefore suspected to be a vibrio. The identification procedure was repeated with 1% sodium chloride as suspending medium, and salicin peptone water was inoculated. Disc sensitivity tests to vibriostatic agent 0129 showed inhibition by discs at 10 μg and 150 μg strengths. The organism was identified as *V vulnificus* based on its fermentation of lactose, mannitol, amygdalin, and salicin; positive results of tests for indole and oxidase; its motility; and its sensitivity to agent 0129.

Closer questioning of the patient elicited a bizarre story. Three days before admission he had bought a large pet fish, and the plastic carrier bag containing it had been accidentally set alight by a cigarette. He had instinctively dived into the bag to rescue the fish and had burnt his thumb, which he then put in his mouth; 72 hours later he developed epiglottitis.