in the site usually occupied by a distended urinary bladder, and on being moved manually from its position it slipped upwards to lie above the umbilicus and tended to remain there. Its extreme mobility was presumably attributable to a mobility of the retroperitoneal structures as a whole, since the cyst did not possess a long pedicle but was attached to the pancreas by a short and broad base.

In the reported cases the investigations carried out have included "straight" *x*-ray film of the abdomen, *x*-ray film of chest, barium meal, barium enema, and intravenous pyelogram. The necessity for these procedures, involving considerable exposure of the infant to radiation, is doubtful, since vomiting in infancy which persists in spite of conservative measures raises the question of surgical exploration, which is put beyond doubt by the finding of an abdominal tumour. There is unlikely to be any difficulty in distinguishing the tumour from the "tumour" of hypertrophic pyloric stenosis.

There is no general agreement on the form surgical treatment should take, though it is commonly stated that excision of the cyst is the ideal treatment but that it is not always feasible owing to close adhesion to surrounding structures. Where excision is regarded as impracticable, the choice of treatment lies between marsupialization and anastomosis of the cyst to the stomach, duodenum, or jejunum. In the present case it was felt that complex excision should not be attempted and that the wall of the cyst was so thin as to render an anastomosis with any part of the gastro-intestinal tract unsafe. Marsupialization, a less extensive procedure than either excision or anastomosis, was therefore carried out with satisfactory results.

I am indebted to Dr. K. Cuddihy, Kilkenny, for the examination of the cyst wall and fluid.

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Idiopathic Myoglobinuria

Idiopathic myoglobinuria is a rare disease in which myoglobin is liberated from muscle tissue and excreted by the kidney. We encountered an example of this condition in a previously healthy young man, and have investigated methods for the detection of the urine pigments.

CASE REPORT

The patient, a youth of 19, had always led a sedentary existence; on the afternoon of September 27, 1959, he played football for the first time for two years. During the game he sustained a kick on the left thigh which, although it did not prevent his finishing the game, left a small bruise. Later that evening he noticed pain and stiffness in the muscles of the lower back and thighs, and weakness of both legs. The pain prevented sleep that night, and by the next morning the weakness of the legs and back had increased to an alarming extent, so that he was not able to sit up unaided in bed. He then noticed for the first time that his urine was dark red in colour.

He was admitted to hospital 18 hours after the onset of symptoms. His general condition was satisfactory, temperature, pulse, and blood-pressure being normal. Because of profound weakness of the extensor muscles of the back and thighs he was unable to lift himself unaided from the supine to the sitting position, or to raise either leg off the bed. Although the muscles of both thighs were tender on palpation, there was no swelling of the affected muscle groups. Knee reflexes, although diminished, were equal. There was no abnormality of other muscle groups, and examination of other systems revealed nothing abnormal.

Investigations.—The urine passed on admission—that is, 24 hours after the game of football—contained no albumin, sugar, red blood cells, or casts, but was port wine in colour, and contained myoglobin and metmyoglobin. The blood count, serum bilirubin, liver-function tests, and electrolytes were normal, and no excess urinary porphyrins were detected. The plasma creatine and creatinine were normal, but the excretion in the urine showed characteristic changes:

Urine creatinine	1.8 g./litre	24 hours after onset of symptoms
„ creatine	0.2 g./litre	
Urine creatinine	0.7 g./litre	36 hours after onset of symptoms
„ creatine	0.2 g./litre	

Muscle biopsy from an affected muscle group in the thigh, performed five days after admission to hospital, showed normal voluntary muscle, with no evidence of breakdown of fibres or loss of striation (Dr. K. A. D. Turk).

After admission there was a steady improvement with return of power to the affected muscles; within 24 hours there was no detectable weakness or muscle tenderness, and the knee reflexes had returned to normal. Urine passed on the morning after admission still contained myoglobin although only in about one-twentieth of the concentration in the first specimen. At no time during the illness did he develop oliguria or urea retention.

This type of episode had never occurred previously in the patient or other members of his family. The youth had never been very energetic or taken strenuous exercise. He subsequently admitted to having had similar pain in the back and thighs, and slight weakness of the legs, lasting 24 hours, on two previous occasions, both associated with an infection of the upper respiratory tract. He had not noticed any alteration in the colour of the urine during these episodes.

IDENTIFICATION OF MYOGLOBIN

The identification of the urine pigment is usually made spectroscopically, although Whisnant, Owings, Cantrell, and Cooper (1959) used the ultracentrifuge and paper electrophoresis. The latter test takes several hours and it is advisable to have an authentic sample of human myoglobin for comparison. Both haemoglobin (Hb) and myoglobin (Mb) are rapidly oxidized in urine to their met-derivatives, the spectral characteristics of which are not very different. Direct examination of the urine may therefore show, as in this case, both MbO_2 and metMb, which may not be easy to identify. Berenbaum, Birch, and Moreland (1955) showed that the absorption maxima of HbCO and MbCO were sufficiently separated to permit this method to be used to identify the pigment present in the urine of their case. The spectra of a number of derivatives were therefore compared to see which would provide the best distinction between Hb and Mb.

A Hb solution was used as a control, and absorption spectra were measured with Unicam SP 500 spectrophotometer. The absorption maxima found, and those previously reported, are given in the Table.

The acid met-derivatives (urine, buffered to *p*H 6, plus 2 drops dil. $K_3Fe(CN)_6$) did not give sharp maxima in the visible region of the spectrum, possibly because of "background" absorption by other urine pigments. These spectra would be of little use as a diagnostic test, although the Soret bands (406 and 410 mμ) were clearer. The absorption spectra of the met-cyanide derivatives

(prepared by adding 1 drop NaCN to the acid met-solutions) were virtually identical, except for the Soret region, where maxima again showed a definite shift in wavelength. The reduced compounds (sodium dithionite in the absence of air) also showed significant shifts in the Soret bands. The oxy-derivatives were prepared by reduction with sodium dithionite, followed by oxidation by shaking with air for several minutes. These were not very stable, but the spectral differences (Fig. 1) are easily seen. The carboxy-compounds were prepared by bubbling coal-gas through the solution for several minutes before and after adding a little sodium dithionite, and then covering the solution with a layer of liquid paraffin. These derivatives were relatively stable, with clear differences in spectra (Figs. 2 and 3).

Absorption Maxima of Hb and Mb Derivatives. The Hb Maxima are Taken from Lemberg and Legge (1949) and Those for Mb from Smith and Gibson (1959). The Values Found in this Case are Shown in Parentheses

Compound	Absorption Maxima		Shift of Mb band (mμ)
	Hb (mμ)	Mb (mμ)	
Acid met- ..	630 (630) 500 (496) 405–407 (406)	630 (640) 500 (494) 407–408 (410)	0 (+10) 0 (−2) 0 to +3 (+4)
Met-cyanide ..	540 (540) 412–416 (420)	540 (540) 422 (422)	0 (0) +6 to +10 (+2)
Reduced- ..	555 (556) 430 (430)	552–555 (556) 432–435 (433)	0 ,, −3 (0) +2 ,, +5 (+6)
Oxy-	576–578 (576) 540–542 (542) 412–415 (413)	582 (580) 544 (548) 418–420 (420)	+4 ,, +6 (+4) +2 ,, +4 (+6) +3 ,, + (+7)
Carboxy- ..	568–572 (568) 538–540 (538) 418 (419)	580 (578) 540–542 (542) 422–424 (423)	+8 ,, +12 (+10) 0 ,, +4 (+4) +4 ,, +6 (+4)

The absorption maxima of the Mb derivatives were nearly all at higher wavelengths than those of Hb. The absorption minima of the oxy-compounds (HbO_2, 560 mμ; MbO_2, 568 mμ) and carboxy-compounds (HbCO, 554 mμ; MbCO, 560 mμ) were also displaced in the same direction. If the spectrophotometer is used, with either the oxy- or carboxy-derivatives, these spectral differences provide a reliable means for the identification of the pigment, and by using the Soret bands the method may be rendered more sensitive.

COMMENT

The diagnosis before admission to hospital was haematuria secondary to renal injury. When this was disproved by examination of the urine deposit other possibilities considered were March haemoglobinuria, porphyria, and myoglobinuria. The differentiation between these pigments is impossible by the naked eye, but may be made spectroscopically, as described above.

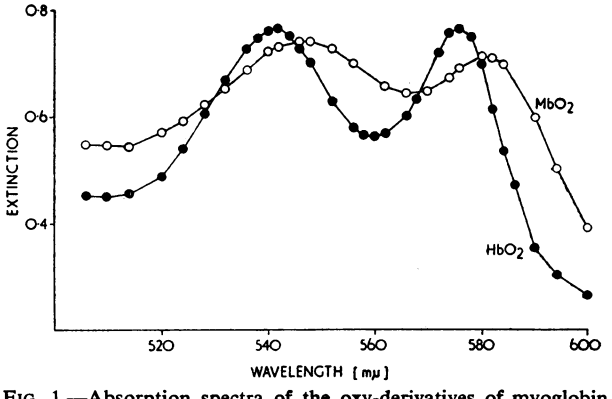

FIG. 1.—Absorption spectra of the oxy-derivatives of myoglobin (MbO_2, —○—○—) and haemoglobin (HbO_2, —●—●—).

Myoglobin, a haem-pigment present in red muscle, is excreted in the urine after breakdown of muscle tissue. This may occur secondary to injury to voluntary muscle, as in the crush syndrome, or to primary muscular disease, such as dermatomyositis or muscular dystrophy (Acheson and McAlpine, 1953), or consequent to the ingestion of some toxic agent, as in Haff disease.

In primary idiopathic myoglobinuria there is no known primary muscular disease. Muscle pain, cramps, or paralysis may occur temporarily, but these are probably due to the breakdown of muscle cells, liberating myoglobin. There are fewer than 20 cases of this type in the literature (Elek and Anderson, 1953; Berenbaum *et al.*, 1955; Whisnant *et al.*, 1959). We consider our case to be of this type. The symptoms did not develop until several hours after the game of football, and the amount of primary muscular trauma was minimal, and could in no way be compared with the secondary myoglobinuria of the crush syndrome. The relatively trivial course of the illness suggested that there was only a minor degree of muscle breakdown, and this may explain the absence of any histological change, such as that described by Berenbaum *et al.* (1955).

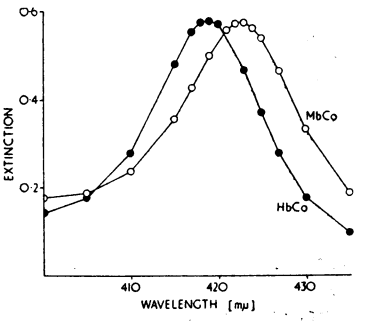

FIG. 2.—Absorption spectra of the Soret bands of the carboxy-derivatives of myoglobin (MbCO, —○—○—) and haemoglobin (HbCO, —●—●—).

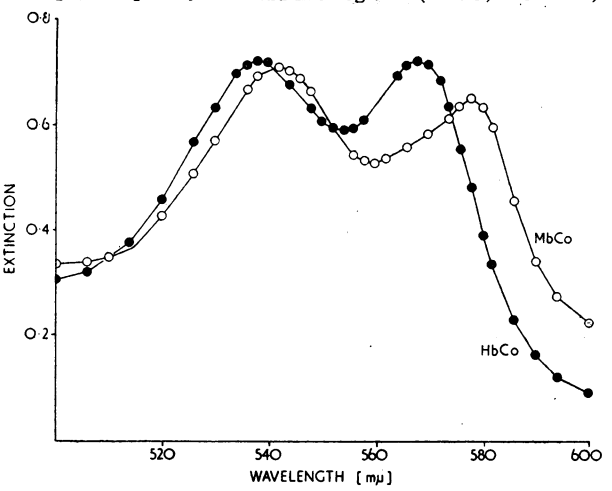

FIG. 3.—Absorption spectra of the carboxy-derivatives of myoglobin (MbCO, —○—○—) and haemoglobin (HbCO, —●—●—).

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