Medical Memoranda

Primary Dislocation of the Left Shoulder-joint in the Boxing-ring

In the course of a light-heavyweight bout between R.A.F. College, Cranwell, and Cambridge University a cadet was seen suddenly to hold his left elbow with his right glove and to be completely disabled. The round ended almost immediately, and he subsided into the chair in his corner in great pain.

It is often difficult to see some decisive incident in the ring (hence multiple judges as well as a referee), and in this instance I was "stymied" by the referee and saw nothing. As I climbed up to investigate, a colleague in the front row helpfully hazarded the opinion that the man had dislocated his shoulder. On examination this "spot" diagnosis was seen to be perfectly correct, with the classical flattening of the deltoid region and the bulge of the head of the humerus anteromedially and subcoracoid. It was not the moment or the place for bothering the patient about the detailed mechanism whereby this extraordinary happening had come about. I was puzzled because, in boxing, the *left* upper limb is mainly concerned with the safe piston-like movements of leading and guarding, in which no considerable abduction is involved. It would have been much more explicable in the case of the *right* shoulder-joint, where wide abduction occurs in swinging a right hook to the jaw. Moreover, this was not a recurrence. The man had never dislocated his shoulder before, and so lacked the notorious tendency to recurrence. I wrote to the patient and to the S.M.O. at Cranwell R.A.F. College, asking if they could throw any further light on the dislocation, and I also questioned his opponent, our own light-heavyweight.

The cadet replied: "It was very difficult, as I didn't actually feel it happen at all. The only sensation was of not being able to raise my left arm, and then I thought it must be cramp. I didn't realize the shoulder was dislocated until I returned to my corner at the end of the round." He continued: "I have asked independently all the members of the team who were watching, and . . . it appears that . . . I attempted a left hook, and while my arm was fully extended (my punch having missed) he hit my arm with a right-hand punch."

The opponent confirmed this missed hook, and stated that he himself had a very sore right ulna from the violent impact of his own upthrusting *forearm* blow in warding off the said hook.

COMMENT

It would appear that the patient's left shoulder-joint was relaxed, with flaccid muscular control, at the moment when his arm was widely abducted and the blow missed. Simultaneously the Cambridge man violently forced the arm much further upwards, bruising his own forearm, and levering the head of the humerus downwards out of its shallow socket and through the capsule of the joint.

It is hardly necessary to recall that the stability of the shoulder-joint (with its shallow glenoid cavity providing little or nothing to hold the head of the humerus) depends on the tension of the tendons passing under, over, or through the joint and the elastic compressing pull of the muscles acting upon it. The dislocation happened at the moment of misfire, when all these structures were relaxed.

I have never seen this accident in the boxing-ring before, during thirty years at the Cambridge ringside, nor have I heard of it elsewhere. It would seem that primary dislocation of the shoulder-joint in the boxing-ring is an extremely rare accident, comparable in rarity with dislocation of the hip-joint at "rigger" (Woods, 1949).

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Agranulocytosis During Treatment with an Amoebicide

A case of agranulocytosis developing in the course of treatment of amoebiasis with "camoform" is described. It appears to be the first time in which such a haematological change has been noted during camoform medication.

CASE REPORT

In November, 1955, in Calcutta, an Englishwoman aged 27 complained of abdominal discomfort and dyspepsia. She had never been ill before. She had one child aged 15 months. On clinical examination no abnormality except moderate obesity was found. A stool examination revealed mucus and a few precystic and cystic forms of *Entamoeba histolytica*, no cellular exudate, and no ova. A blood count showed: erythrocytes, 4,510,000; Hb, 12.6 g./100 ml.; leucocytes, 5,200 (polymorphs 70%, lymphocytes 27%, monocytes 0%, eosinophils 3%). Her urine contained no abnormality. Camoform, one tablet (250 mg.) four times a day, was prescribed. On the fifth day of treatment her temperature was 101° F. (38.3° C.), and she had mild photophobia and mild pains in the limbs and neck. On the following day the fever had abated to 100° F. (37.3° C.), and she had very little photophobia or pain. She did not complain of sore throat and no pharyngeal abnormality was found on examination. On that day her leucocytes numbered 2,300 (polymorphs 71%, lymphocytes 24%, monocytes 3%, eosinophils 2%).

Camoform treatment was stopped after a total of 16 tablets (4 g.) had been taken. On the sixth day the leucocytes had further decreased to 2,000 (polymorphs 56%, lymphocytes 40%, monocytes 2%, eosinophils 2%). On the seventh day the lowest readings were obtained: leucocytes, 1,200 (polymorphs 56%, lymphocytes 40%, monocytes 1%, eosinophils 3%).

The patient was admitted to hospital on the sixth day and treatment instituted. Penicillin was given eight-hourly in doses of 400,000 units intramuscularly (crystalline penicillin G procaine 300,000 units, with buffered crystalline penicillin G potassium 100,000 units) for five days. "Pent-nucleotide," 10 ml. daily, was given intramuscularly for the first three days. Cortisone acetate, 150 mg. daily in divided doses, was also given for four days. Afterwards the dose of cortisone acetate was gradually reduced. On the third day of this treatment the leucocytes rose to 1,800 (polymorphs 68%, lymphocytes 30%, monocytes 1%, eosinophils 1%), and there was a consistent improvement onwards. Five days later a blood count showed: erythrocytes, 4,250,000; Hb, 12 g./100 ml.; leucocytes, 5,100 (polymorphs 54%, lymphocytes 34%, monocytes 2%, eosinophils 10%). Four months later the patient was still well.

COMMENT

In a series of 20 cases Barrios (1954) reported no haematological toxic signs, though three of his patients developed anorexia, transient albuminuria, or headache. Abdominal pain, headache, nausea, dizziness or vertigo, sweating, or palpitation were observed in East Africa by De Mello and De Mello (1955), but they, too, do not report any blood dyscrasia. In Honduras, Hoekenga and Batterton (1954) gave camoform, 1.5 g. daily for seven days, to a series of 18 patients with amoebiasis, with no ill effect.

We have used camoform in a large number of cases of amoebiasis, but this case of agranulocytosis is the only one in which a serious toxic reaction followed the use of the drug.

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