RESULTS

Data for known carriers, for possible carriers, and for a group of normal subjects are given in Tables I and II. Unfortunately it was not possible to obtain meaningful values for aldolase on all of the serum specimens because some were haemolysed. Phosphocreatine kinase estimations, on the other hand, are unaffected in these circumstances.

TABLE I.—*Known Carriers*

Subject	P.C.K. (μ M/ml./hr.)	Aldolase D.H.A. Units
R.W.	22.0	56.4
L.S.	13.1	16.8
D.G.	9.5	4.7
S.V.	7.4	9.4
A.D.	6.3	66.8
J.P.	3.3	11.0
E.N.	1.9	—

Units and normal values:

Aldolase: Dihydroxy acetone (D.H.A.) units. Normal adult range ($\bar{x} \pm 2$ S.D.) for 73 subjects = 12.8 ± 8.8 (Hughes, 1962).

Phosphocreatine kinase (P.C.K.): μ moles creatine per ml. serum per hour at 37° C. Normal adult range ($\bar{x} \pm 2$ S.D.) = 1.6 ± 1.0 (40 female subjects).

Values in this table (and also in Table II) which are in *italics* lie above the normal range as defined above.

TABLE II.—*Possible Carriers*

Mothers with One Affected Son			Females with Affected Sibs			Maternal Aunts of Patients		
Sub- ject	P.C.K. μ M/ ml./hr.	Aldo- lase D.H.A. Units	Sub- ject	P.C.K. μ M/ ml./hr.	Aldo- lase D.H.A. Units	Sub- ject	P.C.K. μ M/ ml./hr.	Aldo- lase D.H.A. Units
F.D.	22.2	11.6	E.C.	66.5	—	N.M.	3.9	11.0
B.B.	13.5	26.1	B.P.	5.1	15.0	R.K.	2.6	11.0
V.S.	3.8	26.6	Z.N.	4.4	—	E.H.	2.0	9.7
C.B.	2.1	—	M.P.	3.2	11.6	P.C.	1.5	16.7
T.C.	1.8	1.2	G.N.	2.4	—	J.G.	1.1	10.6
P.R.	1.4	5.3	Y.W.	2.3	87.5*			
M.S.	1.0	12.4	B.C.	2.1	—			
			T.S.	1.8	16.0			

* This value is probably normal because the subject was an infant aged 2 years, and serum aldolase levels in infants are sometimes considerably higher than in adults (Friedman and Lapan, 1958). All other subjects were aged 10 years or older.

DISCUSSION

These results confirm recent reports that a high proportion of carriers of the Duchenne type of muscular dystrophy show serum enzyme abnormalities. In our subjects the proportion of known carriers with raised phosphocreatine activity (7 out of 8) is similar to that reported by Aebi *et al.* (1962)—that is, 8 out of 9. Compared with this enzyme, the proportion with raised aldolase levels (2 out of 7) was smaller.

A considerable number of cases arise by fresh mutation in the mothers (Walton, 1960), who would not themselves exhibit systemic changes. Consequently deviations from normal would be expected less frequently in mothers with only one affected son and no relevant family history. The results shown in Table II are consistent with this expectation. The proportion of female sibs of patients who had serum phosphocreatine kinase activity above the normal range, and were therefore probably carriers, was 50%. In view of the small numbers this result is partly fortuitous even though it is the proportion to be expected from the mode of inheritance, assuming that all the mothers were in fact carriers. In the remaining subgroup of maternal aunts all were normal with one exception (N. M.).

These data lend further support to the hope that serum enzyme studies can be used successfully for the detection of carriers of this form of muscular dystrophy.

However, studies on a larger group are still necessary to assess accurately the reliability of the method.

SUMMARY

Serum phosphocreatine kinase and aldolase have been estimated in a number of female relatives of patients with the Duchenne form of muscular dystrophy. Seven out of eight known carriers had serum phosphocreatine kinase levels above the normal range; three also had elevated serum aldolase. A smaller proportion of possible carriers also showed serum enzyme abnormalities.

I thank Dr. J. N. Walton and Dr. J. van den Bosch for making available the family histories of the subjects in the investigation, and Professor J. N. Cumings for helpful advice and criticism. Dr. van den Bosch was also kind enough to provide a number of serum specimens.

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Medical Memoranda

Seminoma in Identical Twins

Testicular neoplasms are uncommon, comprising about 1.5 to 2% of all malignant tumours in the male (Dean, 1935; Gilbert and Hamilton, 1940). About two-fifths (Dixon and Moore, 1952) to two-thirds (Gordon-Taylor and Wyndham, 1947) of all testicular new growths are seminomas. A familial incidence of rare tumours is of interest, but only a few accounts of a familial occurrence of testicular neoplasms are on record. Raven (1934) reported the cases of two brothers, the younger of whom died at the age of 19 from a metastasizing testicular growth. The elder brother underwent a left orchidectomy for seminoma testis at the age of 38. Lownes and Leberman (1939) also described the cases of two brothers, the younger of whom died, aged 32, of a seminoma with pulmonary metastases. The older brother had a right orchidectomy at the age of 53 for a malignant growth regarded by the authors as a teratoma. Willis (1960) mentions a similar occurrence in brothers aged 15 and 31 years respectively, whose grandfather had died at the age of 30 from "bilateral ulcerated cancer" of the testes.

So far as we have been able to ascertain there have been only two previous reports of testicular neoplasms in identical twins. In the first of these, by Champlin (1930), one of the twins died at the age of 24 from cerebral and abdominal metastases of a testicular

tumour three months after orchidectomy and two years after first noticing a testicular enlargement. The second twin had a right orchidectomy for a seminoma at the age of 31, and was alive and well four and a half years later. Domrich (1939-40) reported testicular embryonal carcinomas occurring in identical twins, one of whom died at the age of 35, three months after orchidectomy. We observed two brothers, identical twins, who bore a remarkable likeness to each other in that they were both cryptorchids and developed seminomas in the fifth decade of their lives.

CASE REPORTS

Twin I, a childless married man then aged 40, was seen in March, 1954, complaining of lower abdominal pain, and occasional shooting pain down to the perineum. He was found to have emphysema, bilaterally undescended testes, and a palpable tender mass 10 cm. above the symphysis pubis. A right abdominal testicular mass was removed in June, 1954, when masses of involved lymph nodes were noted to be present along the abdominal aorta. He received a post-operative course of radiotherapy, and was fit and well seven years after laparotomy.

Operation Specimen.—The excised testis measured 15.5 by 10 by 7 cm. and weighed 693 g. It was encapsulated, but at one aspect the tunica albuginea was superimposed by a flat, button-shaped tumour mass, measuring 2 cm. in diameter and 8 mm. in height. Microscopical examination showed the testicular mass to be a typical seminoma, while the substance of a para-aortic lymph node had been replaced by identical tumour tissue.

Twin II was admitted to hospital in January, 1959, at the age of 44 on account of a large lower abdominal mass. He was unmarried and a cripple owing to post-encephalitic Parkinsonism since the age of 10, but was of unimpaired intelligence. Bilateral cryptorchidism was noted. At laparotomy the mass was found to be a tumorous abdominal left-sided testis. It was removed, but there was considerable involvement of the para-aortic lymph nodes, and the pelvis was almost completely filled with growth. Pulmonary metastases were absent but he died of cachexia and bronchopneumonia two months after laparotomy.

Operation Specimen.—The excised testis measured 15 by 13 by 11 cm. and weighed 1,240 g. It was only partly surrounded by an intact capsule. Microscopical examination showed again the typical features of a seminoma.

COMMENT

The increased likelihood of malignant change in undescended testes is well known, and is reported to be 48 times greater than in scrotal testes; or, expressed differently, 1 in 20 abdominal testes might be expected to become neoplastic, and 1 in 80 inguinal testes (Dixon and Moore, 1952). Both twin brothers had undescended testes and malignant change occurred in both within a few years of each other in the fifth decade. In one twin the tumour arose in the left, in the other in the right testis. It is also known that bilateral cryptorchidism is present in a third brother. Combined surgery and radiotherapy resulted in cure in twin I, but in twin II the disease was too far advanced for successful treatment.

We are grateful to Professor R. A. Willis for confirming the histological diagnoses and reading the manuscript.

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Steatorrhoea due to Dissecting Aneurysm of the Superior Mesenteric Artery

Failure of the blood supply to the small intestine as a result of thrombosis or embolism of the superior mesenteric artery has been described as an uncommon cause of steatorrhoea in four cases (Hawkins, 1957; Joske *et al.*, 1958; Shaw and Maynard, 1958). The following additional case presented difficulty in diagnosis.

CASE HISTORY

The patient, a retired fisherman aged 51, was admitted to another hospital in March, 1960. For four months he had suffered from frequent attacks of left-sided lower abdominal pain, unrelated to meals and of increasing severity and frequency. The pain had been associated with anorexia, vomiting, and constipation. By the time of admission he had lost 3 st. (19 kg.) in weight.

The chief findings were as follows: mild generalized rheumatoid arthritis; slight left lower abdominal tenderness; rectum full of faeces; blood-pressure, 190/120 mm. Hg; proteinuria, 1-7 g./l. (Esbach); blood urea, 45 mg./100 ml.; serum concentration of Na⁺, K⁺, Cl⁻, and HCO₃⁻, normal; haemoglobin, 14.1 g./100 ml. White blood cells, 12,200/c.mm. (neutrophil polymorphs 87%). Plain x-ray of abdomen, normal. Sigmoidoscopy, normal. Barium enema, a few diverticula of the pelvic colon only.

The patient's condition seemed to improve after evacuation of the rectum, and he was discharged.

Dissecting aneurysm of superior mesenteric artery. (Weigert Elastica. $\times 8.5$.)