intima and perhaps to the inclusion of the cholesterol moiety of the chylomicrons in the intimal tissues.

An altogether different possibility is suggested by the studies of Fullerton *et al.* (1953), Waldron and Duncan (1954), Poole (1955), and O'Brien (1955) concerning the relationship between lipaemic plasma and accelerated clotting-times. The first two groups of workers have claimed to find significant acceleration of the clotting-time after a fatty meal, especially in the presence of pronounced visible lipaemia. Poole has shown that rapid centrifugation, with the removal of fat particles from the plasma, increases recalcified plasma clotting-times by the order of two and a half times in the absence of platelets. Subsequent addition of the fat particles restored the clotting-time to its former value. If this work is confirmed then the present finding of increased visible lipaemia in men with atherosclerosis will appear to reveal an obvious clue to the possible aetiology of arterial thrombosis, at least in a proportion of cases.

Oliver and Boyd (1953) preferred to explain the relative resistance in atherosclerotics to the clearing action of heparin by diminished production of a heparin-like substance from the mast cells of a diseased arterial system.

Summary

Following the administration of a meal of 60 g. of fat the extent and duration of the lipaemia has been measured by optical and chemical methods in 35 men with cardiac infarction, 33 healthy men matched for age, and 14 healthy pre-menopausal women.

The coronary and male control series have been compared. Results of the optical density readings showed significantly greater degrees of visible lipaemia at five to seven hours after the meal in the coronary series. A wide range was encountered in both series, leading to a considerable overlap.

The fasting serum also showed greater optical density in the coronary series.

Results of the chemical estimations showed total lipids in the fasting state to be significantly higher in the coronary series. The difference in the mean values was 133 mg. per 100 ml. of serum. Total lipids after the meal tended to rise and fall with changes in the visible lipaemia.

In the female controls the lipaemia was generally less than in the men. These differences did not reach statistical significance.

It is concluded that in some men with coronary artery disease the clearing of visible lipaemia after a fatty meal is delayed and that the relative failure of the clearing mechanism may continue to exert its influence after many hours.

Possible ways in which this phenomenon may be related aetiologicaly to atherosclerosis are briefly discussed.

I am indebted to Professor C. Bruce Perry for his interest in this investigation. The physicians and surgeons of the United Bristol Hospitals have allowed me to investigate their patients. Dr. Fraser Roberts, of the London School of Hygiene, generously gave statistical advice, and Miss Duncan, of the Department of Public Health in Bristol, carried out all the statistical calculations. Miss Margaret Haynes performed the chemical estimations.

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Medical Memoranda

Hypertension Due to Hydronephrosis: Relief after Nephrectomy

The following case is reported because of the problem which it poses in the aetiology of hypertension due to renal disease.

CASE REPORT

In September, 1953, I was asked to see a European man, then aged 31, because of the discovery of a non-functioning left kidney on excretion pyelography. This had been found by his own doctor during an investigation for hypertension, which had been discovered when the patient had attended about a month previously complaining of waking at night "with his heart thumping" and shortness of breath. At such times he suffered nausea but did not vomit. These symptoms had arisen two weeks before he had consulted his physician, and had never occurred previously. When aged 13 he had been confined to bed for nine months with rheumatic fever. He had later been treated with Lugol's iodine for "thyroid disease," but had since played games without ill effect. While on active service in 1943 he had had an attack of left renal colic lasting a few hours, but no investigations were undertaken because of the exigencies of the situation.

On examination the relevant findings were an apparently healthy mesomorph showing no evidence of disease except a blood pressure of 170/110. He smoked 40 cigarettes a day, and weighed 187 lb. (84.8 kg.). An E.C.G. tracing revealed moderate left ventricular stress, and the optic fundi showed grade 1 hypertensive retinopathy. Urinalysis disclosed nothing abnormal.

In view of the rapid onset and nature of the symptoms, I thought it advisable to exclude the possibility of phaeochromocytoma: an amylobarbitone sedation test favoured primary hypertension, a benzodioxane test was negative, and presacral perirenal pneumography did not show any abnormality of the adrenal glands. Ascending pyelography showed a large pelvic hydronephrosis of the left kidney, and the appearances suggested an "aberrant" artery as the likely cause.

Operation.—On October 22 I removed the left kidney through an incision in the bed of the twelfth rib; freeing the upper pole did not affect the blood pressure. The kidney was largely a hydronephrotic sac, with just a thin rim of renal tissue. The primary cause appeared to be a large aberrant artery arising from the distal aorta and passing to the lower pole of the kidney; the true renal vessels were a little smaller than normal.

Histology.—"The kidney is the seat of a large internal hydronephrosis, with marked thinning of the surrounding parenchymal kidney tissue. Sections through the thinned kidney tissue show a marked degree of chronic interstitial nephritis, fibrosis and hyaline degeneration of the majority of the glomeruli, narrowing and obliteration of many of the tubules and dilatation of others, which contain pink-staining hyaline material. The arterioles show the changes associated with hypertension—namely, thickening of the walls due to elastosis, and intimal thickening and consequent narrowing of the lumina. The characteristic arteriolonecrotic lesions of malignant hypertension are not found here."

On the day following operation his blood pressure had dropped to 115/75, and remained at that level until his discharge from hospital, except for a transient rise to 140/90 on his first day out of bed. He made an uninterrupted recovery, and a follow-up note from his physician on November 4, 1953, stated that the patient's blood pressure was 120/75, and that the E.C.G. tracing showed a return towards normal.

For further information about the patient's progress, I am indebted to Dr. Morley Smith. When last seen (April, 1956) his blood pressure was 135/80 and weight 159 lb. (72.1 kg.), and this in spite of divorce, reaffirmation, a broken engagement, considerable alcoholic excess, and a sexual life bordering on satyriasis. So I think it reasonable to claim a cure.

COMMENT

The interesting features of this case are that in 1948 the patient's proposal for a considerable life insurance was accepted without question, which indicates that at that time his blood pressure was normal. The attack of renal colic in 1943 means that the kidney was functioning at that date, and, as it was the only obstructive episode which the patient remembers, it may have been the final effort of a failing kidney to overcome its "congenital" obstruction.

These points raise the interesting problem of why a "congenital" obstruction should take so long to make its presence felt, and why a non-functioning kidney should require such a lengthy period to become devascularized enough to cause hypertension. The short history of hypertensive symptoms suggests that there is a critical level of devascularization at which pressor effects become manifest. This effect must occur at the arteriolar or capillary level of the renal circulation, since the aberrant vessel was of medium calibre (about the diameter of a normal brachial artery) and the true renal vessels were but little smaller than normal.

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Pancoast Syndrome Due to Acute Osteomyelitis

Pancoast syndrome is a clinical and radiological entity which can be produced by a variety of pathological lesions at the root of the neck, in relation to the dome of the pleura and the neck of the first rib. Its characteristic features are: (1) pain around the shoulder and down the arm; (2) Horner's syndrome—that is, narrowing of the palpebral fissure, contracted pupil, enophthalmos, and loss of sweating on the same side of face; (3) dullness over the apex of the lung; (4) weakness of the arm and wasting of the small muscles of the hand; (5) x-ray changes: small homogeneous shadow at the extreme apex of the lung and destruction of the posterior portion of the first or second rib and corresponding transverse processes.

The underlying lesion is usually a malignant neoplasm, which by virtue of its site involves the first thoracic nerve and the sympathetic chain that lies in front of the neck of the first rib. In most cases the lesion is an extension of an apical bronchial carcinoma, though occasionally no connexion is found between the tumour and the lung, and the pathological types can be various (Aird, 1949).

In a few cases the syndrome is produced by non-neoplastic conditions—that is, an advanced tuberculous lesion of the apex (Sergeant, 1926; quoted by Morris and Harken, 1940) and hydatid cyst of the lung apex (Campi, 1955)—and aneurysm and trauma are mentioned as other possible causes (Pancoast, 1924, 1932).

The case reported below is an unusual one, the condition being an acute inflammation which gave rise to all the features of a Pancoast syndrome.

CASE REPORT

The patient, a female greengrocer's assistant aged 19, was admitted to the medical ward on August 19, 1955, with pain in her right shoulder and arm following a lifting strain a

week before. She had not responded to simple measures at home, and developed paraesthesia along the ulnar border of the forearm, pain in the right side of the chest, and pyrexia. There was a history of pneumonia at the age of 8 and occasional boils.

Examination on admission revealed a well-built but ill girl with a temperature of 102.4° F. (39.1° C.). Her head and right shoulder were held stiff, and the right arm showed hypotonicity and areflexia, with some impairment of sensation over the medial half of the forearm. There were no chest signs except for diminished air entry at the right base. The blood picture showed: Hb, 90%; white cells, 16,200 (polymorphs 89%); E.S.R., 30 mm./hour. Urinalysis: albumin + + +, no growth. Sputum: no tubercle bacilli. X-ray examination of the chest revealed some loss of translucency of the right apex, which subsequently increased in size.

On August 22 she was still toxic and her temperature was swinging between 101 and 104° F. (38.3 and 40° C.) in spite of chloramphenicol (250 mg. six-hourly). Further examination showed right-sided Horner's syndrome, swelling over the right supraclavicular fossa, dullness over the right apex, muscle spasm on the right side of the neck, tenderness over the right transverse process of the first dorsal vertebra, and slight wasting of the deltoid, spinati, biceps, and intrinsic muscles of the hand. On the sixth day her temperature dropped to normal after chloramphenicol had been replaced by chlortetracycline.

On August 29 she was still apyrexial, the swelling above the clavicle had diminished, and Horner's sign had become less pronounced. Blood culture yielded *Staphylococcus pyogenes*, sensitive to all available antibiotics, especially chlortetracycline and penicillin. Further x-ray films showed an encysted effusion in the right upper zone suggestive of abscess formation, and rarefaction and erosion of the right transverse process of the first dorsal vertebra and adjacent neck of the first rib suggestive of an acute osteomyelitis.

Subsequent progress remained satisfactory and both the abscess and the osteomyelitis gradually resolved on conservative measures alone—that is, a prolonged course of penicillin and immobilization of the neck in a Minerva plaster. She remained symptom-free, and at the end of October was transferred to a convalescent home. When last seen (March 16, 1956) she appeared to be in excellent health, there were no signs of any residual wasting or weakness in her arm, and radiologically the lesion above the right apex had completely resolved and the transverse process and rib fully recalcified.

COMMENT

This is a case of a previously healthy young woman who, after straining her shoulder, developed staphylococcal septicaemia, followed by acute osteomyelitis of the transverse process of the first dorsal vertebra and neighbouring part of the neck of the first rib with an abscess above the apex of the pleura and lung. Because of the anatomical situation of the lesion, she presented a symptom-complex identical with that encountered in apical neoplasms and known as Pancoast syndrome. I have not been able to trace any report of a similar case in the literature.

It may be of historical interest that the clinical part of this syndrome was first described by Edward Sellick Hare (1838), a house-surgeon at the Stafford County General Infirmary, and that in many countries outside Britain the entity is known as the Hare-Pancoast syndrome.

I am indebted to Dr. S. C. Dyke for permission to publish this case and to Mr. J. Leigh Collis for his helpful advice.

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