War Medicine Series

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CRUSHING INJURY

BY

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In the heavy air raids on London in the autumn of 1940 we attended some patients who had been pinned for several hours underneath the debris of their own bombed houses. Shock was present, which responded adequately to transfusion: the limb which had been compressed was found to be swollen and sometimes pulseless. To our surprise these patients developed renal failure with casts in the scanty urine and died in uraemia about the seventh day.

This appeared to us at that time a new entity: it was described as such under the title of "Crush Injuries with Renal Impairment" (Beall *et al.*, 1941; Bywaters and Beall, 1941; Mayon-White and Solandt, 1941). Since then, however, we have discovered descriptions of it by German authors from the war of 1914-18. During that war Frankenthal (1916) and others directed attention to the local muscle damage, and it was not until later that the renal lesion was adequately emphasized (Minami,* 1923). The condition had actually

occurred in 1909 in people buried beneath the debris of the Messina earthquake, but as the relief expedition arrived on the scene a fortnight after the eruption, only the survivors with gangrene of soft parts were seen (von Colmers, 1909).

Apart from German books on military surgery, we have found no other references to it and no further records of cases. This is curious, since, even apart from the almost continuous warfare since 1918 somewhere in the world; such accidents do occur in the mining industry and have recently been described (McClelland, 1941). Since the first few cases in air-raid casualties were published the condition has been generally recognized here. Hospitals in the Emergency Hospitals Scheme of the Ministry of Health have been asked to notify cases to the Medical Research Council, which now has records of over 70 cases. It is on this group, through the kindness of

the M.R.C. and its observers throughout the country, that the following observations are based. About 5% of air-raid casualties in an urban area may be of this type.

Clinical Description

The patient has usually been pinned by the limbs beneath heavy beams or pieces of masonry for a period of several hours. If seen soon after release from this compression he generally appears comparatively well, apart from other coincidental wounds, fractures, etc. Soon, however, he enters the first phase of shock, due to loss of plasma through the damaged capillary walls into the extravascular tissue spaces of the affected limb, which becomes swollen (see Chart). As a consequence, the haemoglobin and corpuscular content of the blood rises relative to the plasma, and vasoconstriction occurs to compensate for the decrease in circulating blood volume. During this first phase the skin is pale, cold, and moist, but the blood pressure is maintained at a nearly normal level. Later, however, vasoconstriction may become unable further to compensate for this leakage of plasma into the damaged area; the blood pressure then falls rapidly. This is the second phase of shock. If fluids such as serum, citrated plasma (plain or recalcified), and blood are given intravenously at this stage in adequate amounts the blood pressure can be restored to normal levels.

This is the phase of recovery from shock. There may, however, be failure to respond to intravenous protein-containing fluids if the blood pressure has been allowed to remain at too low a level—below 80 mm. Hg—for too long a time.

Thereafter the general condition of the patient causes no anxiety until the first or second urine passed is noted to be bloody and to contain a pigment in solution similar in many ways to haemoglobin and giving a positive reaction to the benzidine peroxidase test. The urine also contains albumin, creatine, pigmented granular casts, and sometimes pigmented granules somewhat resembling erythrocytes; it is highly acid (pH 4.6 to 6.0).

The daily output of urine may become very small despite an adequate fluid intake, but the pigment disappears as a rule after the second day.

Meanwhile, however, the damaged limb has swollen, is hard, tense, and cannot be indented. This swelling is usually progressive for the first four or five days, except possibly in cases associated with blood-vessel damage. The skin shows small petechial haemorrhages, erythematous wheals, and large blisters, related to the areas of direct contact pressure. These blisters are often thought to be the result of burns. They are not. They may occur in crops, related in time to the return of circulation to the damaged part. There is anaesthesia of the limb, often of a patchy distribution due to damage of the terminal nerve fibres, and sometimes of a nerve-trunk distribution.* Paralysis, which may be of any severity, is usually present; in many cases arterial pulsation in the peripheral part of the limb is absent, the limb is cold and pale, and

CLINICAL COURSE OF A CRUSHING INJURY CASE

Buried 6 hours. Note from above down:

- (1) Return of circulation to legs as measured by oscillometer readings.
- (2) Increase in leg volume measured by spiral bandage.
- (3) Haemoconcentration and later haemodilution shown by haemoglobin level.
- (4) Blood-pressure fall and restoration after transfusion.
- (5) Decreased urine output with rising blood urea. Urine contained pigment granules and casts and failed to concentrate urea above 670 mg./100 c.cm.

Necropsy showed blanching and necrosis of muscle in both legs and a typical swollen kidney.

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the onset of gangrene is to be feared. It is important to record the state of the circulation, which can be followed from time to time with the oscillometer. Sometimes Art and sometimes Nature will restore the circulation; often then the pulsation is greater on the damaged than on the sound side. Whether gangrene sets in or not, the severely damaged patient will continue in a peculiar state of mental apathy, alternating with phases of anxious and apprehensive awareness of his condition, until the end of the first week. During this time vomiting is a prominent symptom, and the blood pressure gradually rises to a level much in excess of the normal. The blood urea, potassium, and phosphate concentrations rise progressively, and the CO_2 -combining power of the plasma falls. The urine flow is greatly diminished and, despite the high blood urea, there is failure of the tubules to concentrate urea and other solids, and also failure to reabsorb chloride, even though the blood chloride concentration is low: tubular dysfunction is therefore indicated. Pain in the loins (possibly evidence of stretching of the renal capsule) occurs not infrequently about the fourth or fifth day. Some patients have had severe abdominal pain and rigidity, and have been opened without anything definite being found. This symptom may be related to the kidneys.

The end of the first week is the critical period: in those patients who recover a sudden diuresis is seen, and thereafter the urinary output will rise gradually to very high levels. At the same time the blood urea level falls; the urea-clearance test (of van Slyke) may show improvement of tubular function, although this may not reach the normal level until several months after the injury. During recovery granular and epithelial casts may be noted from time to time in the urine. In the damaged areas skin and muscle may slough away; infection may occur. This is the "pressure necrosis" observed in base hospitals when the initial phases of the syndrome have passed unnoticed. Restitution fibrosis may occur if the damage is slight, a condition resembling Volkmann's ischaemic contracture.

In more severely damaged patients at this critical period some cardiac irregularity may be noticed, and electrocardiograms taken at this time show changes not unlike those seen in experimental potassium poisoning. The serum potassium level may be greatly increased, but it is difficult to say whether it is high enough to cause symptoms (as in the recent work on dogs by Hoff *et al.*, 1941). Too little is known about the balance between intra- and extra-cellular potassium at increased blood levels to be dogmatic on this point. However, we know that potassium is very soon and very easily lost from damaged muscle (Baetjer, 1935) and that it cannot be adequately excreted if there is severe renal impairment. Hoff *et al.* (1941) are of the opinion that, in man, death in uraemia from ordinary causes associated with a high terminal potassium is not a "potassium death," because the kidney can excrete this substance even in extreme oliguria. If, however, there is an increased flow of potassium into the blood stream from intracellular sources (and, in general, intracellular potassium is about 10 times as concentrated as extracellular potassium) it seems possible that this substance may be exerting toxic effects. Measures, such as insulin injection, directed towards the storage of circulating potassium might be found of value in cases in which there are indications of recovery of renal function.

Death occurs very suddenly: the patient may call out in great alarm, and be found pale and sweating, with dilated alae nasi. He may die within one or two minutes, or recover until a similar episode an hour or so later finally terminates his life.

Pathology

Changes are seen in the adrenals, muscle, vessels, and skin, and in the kidneys. There may be other traumatic lesions, such as fat embolism, contusion, or blast-lung. The muscles show one or more of the following types of change (Bywaters, Belsey, *et al.*, 1942):

1. Part of the muscle is blanched, unduly friable and necrotic, resembling fish flesh. The sharp demarcation line corresponds to the skin-pressure areas. This type is due to direct-pressure ischaemia.

2. The whole of the muscle is white and necrotic. This is usually seen in those muscles contained within tight fascial compartments, and it is thought that this necrosis may be due

to rise of pressure within the compartment, with subsequent necrosis. It is for this type that fascia-splitting operations have been proposed.

3. The muscle may appear grossly normal, without pallor, but patchy necrosis of isolated fibres may be seen microscopically. It is this type that is associated with arterial spasm, due perhaps to periarterial haemorrhage or direct arterial damage, and sometimes associated with rupture of a main limb vessel without any prolonged crush. This type is thought to be due not to direct-pressure ischaemia but to vasogenic ischaemia. These changes depend upon the survival time and upon the extent of circulatory return.

The kidneys (Bywaters and Dible, 1942) are enlarged and swollen (except in patients dying early); they may show a white zone in the cortex, which is wet and glistening. Microscopically, the collecting tubules and the distal convoluted tubules contain a brown pigment material even in patients dying within one or two days of the injury: the proximal convoluted tubules show cloudy swelling and desquamation. In cases surviving for 3 to 4 days further changes are seen, consisting of areas of necrosis in the boundary zone and in the neighbourhood of distal convoluted tubules in the cortex. These are usually pronounced in the typical case ending fatally at the end of the first week from renal failure: there is oedema and cellular proliferation surrounding the large hyaline casts which are protruded into the interstitial tissue in these regions. Some polymorph infiltration may be seen within the collecting and distal convoluted tubules. The glomerular capsule may show cuboidal epithelium. This appearance is not entirely specific, since it occurs in certain other conditions. For instance, such casts, composed of haem pigment, occur after intravascular haemolysis if the plasma haemoglobin exceeds the renal threshold of 135 mg. per 100 c.cm. (Gilligan *et al.*, 1941). One of the most common causes of haemoglobinuria nowadays is mismatched transfusion. Not only is the morphologic lesion similar, with pigmented casts and, in certain cases, areas of focal necrosis, but the clinical course is also similar, with anuria and death in uraemia.

Pathogenesis

At first it seemed we might be dealing with some such haemolytic reaction, since our first cases had had serum and blood transfusion, but there were three reasons why this appeared improbable. First, the blood used was Group O; secondly, there was no clinical evidence such as chill, rigor, headaches, limb pains, vomiting, or jaundice following transfusion, nor was there any increased colour in the plasma, such as one would expect if any serious haemolysis had occurred. Thirdly—and this seemed the most cogent argument—no such reactions had occurred in any of our shocked and transfused casualties other than buried cases. Later we were to learn that this syndrome developed without any therapy other than "rest and quiet." Extravascular haemolysis, as in a haematoma, has been invoked to explain certain cases (Longland and Murray, 1941). In general, however, following extensive bruising of soft parts, the uptake of freed haem pigment is so slow that there is no rise above the renal threshold, and seldom even jaundice (except locally) unless there is associated liver dysfunction.

Discarding this theory, we were struck by the pallor of the damaged muscle at necropsy. Normal muscle owes its colour to a pigment called "myohaemoglobin," which differs from haemoglobin in several ways, since, although the iron-containing porphyrin group is the same, the globin portion is smaller. For instance, the spectral bands are 30 A.U. nearer the red, and the carbon monoxide span is 30 and not 60 A.U. There are three conditions in which myohaemoglobinuria occurs: perhaps the best known is paralytic equine myohaemoglobinuria. Five cases have been recorded in man, and we have seen a case recently, probably falling into this group: muscle necrosis is associated with myohaemoglobinuria, renal damage, and an increased blood potassium after exercise. In this disease, in the Haff disease, and in epizootic myohaemoglobinuria the muscles are blanched and are the seat of degenerative changes. The next step was therefore to identify this pigment in the urine (Bywaters, Delory, *et al.*, 1941). We were able to do this, using the Hartridge reversion spectroscope and comparing the pigment with myohaemoglobin extracted from perfused human muscle. Dr. Rimington confirmed our

findings, and in addition demonstrated another important characteristic. As the molecule is one-quarter the size of that of haemoglobin, it will filter through smaller pores of semi-permeable membranes, and it will also sediment more slowly in the ultracentrifuge. Both of these things occurred, and they are of some clinical importance because they explain the curious fact that no pigment is seen in the blood stream: as the renal threshold is so much lower than that for haemoglobin, there is prompt excretion and no such accumulation in the blood stream as occurs with the latter pigment.

We regarded this identification of myohaemoglobin (Bywaters, Delory, *et al.*) as important not because it might be the cause of the renal failure (although that is possible) but because, like the creatinuria it was associated with, it definitely incriminated muscle damage. Cases had been cited (often, however, poorly investigated) in which there was no evidence of tissue damage other than lacerations. Since then we have found that crush lesions of trunk muscles often go unnoticed except for this indirect evidence of myohaemoglobinuria. Cases do occur, however, in which muscle necrosis follows not direct compression ischaemia but ischaemia due to arterial spasm or rupture of main arteries, and these cases may also develop renal failure (Bywaters, Belsey, *et al.*, 1942).

Was this pigment responsible for the renal failure? In the similar case of haemoglobinuria with renal failure following intravascular haemolysis the simplest theory is that the pigment blocks the tubules. Baker and Dodds (1925) demonstrated that in acid urine with high salt content the pigment changes to the less soluble acid haematin and is precipitated: with large and repeated doses under these conditions renal failure could be induced. Hence alkalinization of the urine (Bushby *et al.*, 1940) became the practice with all patients in whom a haemolytic reaction was to be feared.

Certainly, in these crush cases, oliguria, due to shock, and an acid urine are seen, and myohaemoglobin behaves similarly to haemoglobin under these conditions. Animal experiment has shown that in the rabbit, whose muscles contain no myohaemoglobin, muscle necrosis reproduces all the signs and symptoms of crushing injury except myohaemoglobinuria and renal failure. When myohaemoglobin in a quantity comparable to that excreted in human crush cases was injected into the rabbit acidified with ammonium chloride, we were able to produce death from renal failure. It seemed possible that partial blockage brought about a rise of intrarenal pressure, and this in turn led to irreversible renal failure.

The proof of this theory will lie in the prevention of renal failure in man by administration of alkali. As yet the validity of the theory is undecided, and the exact mechanism by which muscle necrosis produces renal failure has not been fully explained.

Associated Features

Crushing injury is often associated with other lesions which complicate both the clinical picture and the treatment. Of these the most common is haemorrhage, which may be internal or external. Haemodilution occurs after a variable number of hours, and if haemoconcentration due to plasma loss occurs at the same time the net change in haemoglobin may be *nil*. Fat embolism (Robb-Smith, 1941), often associated with fractures, may occur. The lung symptoms are manifest a few hours up to a day after the injury, and consist of pain in the chest, dyspnoea, pallor or cyanosis, and congestion. Systemic embolization occurs later, on the second or third day, with fat globules in the urine, petechiae, and a surly stupor or delirium. Thus uraemia has seldom an opportunity to develop unless the fat embolism is of a minor grade producing a delayed pneumonic process. Blast or concussion injury of lung is less common (Zuckerman, 1941): it may be present as slight dyspnoea with frothy blood-stained sputum and a ruptured drum or, later, lobar pneumonia. Another condition sometimes met with is carbon monoxide poisoning.

Differential Diagnosis

Oliguria may be due to oligaeic shock alone, to dehydration from vomiting, or inadequate intake, to blockage by acetyl-sulphapyridine crystals, or to a mismatched transfusion: examination of the urine and blood, investigation of the blood pressure, and an intake chart are necessary to differentiate these conditions. Cortical necrosis has occurred after traumatic

injury to the liver with necrosis. This is very rare. There is often a temporary retention of urine, disclosed as such by the passage of a catheter.

Haematuria from direct injury and haemoglobinuria from exposure to cold are differentiated by examination of the urine and the use of the spectroscope.

Limb-swelling may be due to a haematoma: the skin wheals are absent and there is no doughy feel to the muscle. Skin wheals may be mistaken for burns.

Prognosis

The prognosis depends on many factors—first, on the amount of muscle necrosis. If compression is for a shorter period than one hour the muscle survives unless arterial spasm prolongs the ischaemia. If longer than 4 hours, necrosis ensues. For periods between, tissue survival may depend on temperature. Curiously, periods of burial longer than 12 hours seem less serious than shorter periods, perhaps because only the least-hurt patients survive, perhaps because of a natural self-amputation and failure of the circulation to return to the part. The amount of necrosis in our animal experiments determined the height and duration of the haemoconcentration. Once the patient has recovered from shock, renal damage decides the outcome. Arm crushes have a better prognosis than those of the leg, perhaps owing to the smaller amount of muscle. The presence of other injuries adversely affects the result; the rapidity with which treatment can be started probably also does. About one-third of 70 cases have recovered; these were mostly minor cases without severe shock or uraemia—a class of case that is being increasingly recognized. However, a case has been seen (Bywaters, Belsey, *et al.*, 1942) in which the blood urea rose to 470 mg., and yet, without any treatment at all, on the critical seventh day a diuresis occurred and was followed by recovery. Claims of successful treatment have therefore to be very fully substantiated.

Summarizing, extent of injury, degree of haemoconcentration, oliguria, and blood urea rise constitute the four chief determining factors of prognosis.

Finally, one patient whom I saw through the kindness of Dr. Grant, whose patient he was, had drunk four pints of beer just before the establishment was demolished. They dug him out 11 hours later with both legs broken. In spite of a fairly high blood potassium level he recovered, but showed little haemoconcentration or uraemia. The possible protective action of this beverage might be worth investigating.

Treatment

The natural tendency under emergency conditions is to treat shock first, then the local limb condition, and lastly the renal failure on or about the third or fourth day, when signs of renal damage are obvious. Since it is possible that the renal damage occurs soon after the re-establishment of an adequate blood supply to the damaged limb, these three aspects should be considered in the reverse order.

1. Ample alkaline fluid (3 litres daily), by mouth preferably, or by rectum, intramuscularly or intravenously, must be given rapidly at the earliest possible moment, to produce an alkaline diuresis. If possible, this should be done while the patient is still trapped under the wreckage. We favour for ingestion an alkaline fluid flavoured with citrus juice or peppermint and glucose, containing sodium citrate and sodium bicarbonate, 3 g. of each to the ounce. Coffee or tea is also useful, and morphine should be given if required. For intravenous injection, isotonic sodium citrate (3.8%) or sodium lactate (M/6) should be used. Enough alkali should be given to make the urine alkaline: this will mean probably 30 to 40 g. daily or more by mouth, or a lesser quantity by intravenous injection. If tetany is induced, calcium gluconate should be given intramuscularly. Potassium salts must not be used. Sodium sulphate (2%) may also be injected intravenously in combination with the above measures to maintain urine output.

2. With the kidneys thus protected, it is safe to restore the circulation by combating shock, if this is present. Plasma or serum should be given, or, if there has been much haemorrhage, blood. A large quantity may have to be administered: so long as the arterial blood pressure remains low no harm need be feared. Saline is of no value. Overheating the patient is dangerous.

3. Local treatment to excise wounds and set fractures may now be undertaken. The limb may have to be amputated: if this possibility is considered on the first day it should be done soon, to prevent the absorption of autolytic products. A tourniquet may be useful. Amputation later may not save life, and should be done only if the limb is non-viable. Active measures to restore circulation in the damaged limb (only permissible once it is assured that there is an adequate alkaline diuresis) include elevation of the leg, incision of fascial sheaths, stripping of a damaged artery, or the use of alternate positive and negative pressure. Vasodilatation is best assured by giving alcohol and warming the body. The limb should be kept cool rather than hot, perhaps with bags of ice. This delays autolysis and decreases local metabolic needs in ischaemic tissue (Allen, 1938). Whether limitation of swelling by plaster-of-Paris (Trueta), tight binding, or inflatable rubber bags (Patey and Robertson, 1942) will do more than limit plasma loss into the leg (Duncan and Blalock, 1942) is uncertain. There is as yet no evidence that it will prevent renal damage.

It must be remembered that we are treating not only an injured limb but a human being who has passed through an exhausting and terrifying ordeal. Pain must be relieved and steps taken to see that the patient is comfortable in body and mind.

If by these means a fatal outcome, either from shock on the first day or from renal failure on the seventh, can be avoided, we have left a patient with severe muscle damage. These muscles may remain paralysed or may shorten, giving rise to Volkmann's contracture. They should be adequately splinted and exercised. If sloughing and gangrene occur, infection should be controlled.

In conclusion, this was until recently a little-known type of injury: we still know very little about its exact mechanism and how to treat it. Further observations on the effects of controlled and adequately followed treatment are needed.

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CANCER RESEARCH IN YORKSHIRE

The Yorkshire Council of the British Empire Cancer Campaign, according to its sixteenth annual report, has carried through its last year's work without any further curtailment of research, and the programme has been about half that of a normal year. Apart from research, the Council continues to support the services of the radium officer and physicist in the radium department of Leeds General Infirmary, where a new deep-therapy apparatus has lately been installed, towards the cost of which the Council has granted £1,000.

The report embodies an account by Prof. E. D. Passey of some continuing work on experimental carcinogenesis in the department of experimental pathology and cancer research in Leeds University. The induction of pulmonary cancer in mice by intranasal administration of carcinogenic hydrocarbons has produced an unexpected result in the appearance of breast tumours in several experimental animals, suggesting that the genetic constitution of an animal is not a determinative factor in the occurrence of mammary cancer when some of the highly potent carcinogenic compounds are concerned. Another piece of work in this department has been an inquiry into the part played by heredity in the production of mammary cancer. The inquiry so far, which has included the investigation of the family history of 600 women in Leeds with cancer of the breast

with a view to finding the incidence of cancer in their relatives, lends little support to the suggestion that heredity plays an important part in breast cancer in this country. Breast cancer does not appear to stand out as a family disease. Only in four of the 600 families was breast cancer reported as occurring in more than two relatives of the basal patient.

In the department of pathology and the field laboratory of the University of Sheffield Prof. H. N. Green reports attempts to correlate the results of investigations on bacterial growth and on the abnormal tissue growth of cancer. One aspect of this work, which is still proceeding, is the testing of chemical carcinogens, in view of their growth-inhibiting action on the whole animal, for a possible inhibitory action on bacterial growth. It is pointed out that if the mode of action of a carcinogen can be studied on bacteria the rate of progress in such studies may be speeded up enormously. Moreover, it is conceivable that there is no fundamental difference between the bacterial cell which has developed resistance to a cancer-producing agent and the tissue cell which in developing resistance displays, in relation to other types of tissue cell, the properties recognized as cancerous. A report from the same department describes two experiments designed to discover whether the epithelial proliferation induced by oestrogens in the breast and uterus and in the accessory sex glands would render them more susceptible to the action of a carcinogen which has no specific effect on these tissues. It appears that, provided the amount of carcinogen is sufficiently high to produce tumours with certainty in all the animals treated, or if the tissue is not susceptible to the carcinogen used, the effect of possible auxiliary or complementary factors in carcinogenesis is completely hidden.

VENEREAL DISEASE: APPLICATION TO SCOTLAND OF REGULATION 33B

The Department of Health announces that in the application to Scotland of the Defence Regulation dealing with venereal disease "special practitioner" is defined as a registered medical practitioner who provides treatment under a local authority's venereal disease scheme approved by the Secretary of State for Scotland; or a specialist in venereal disease in H.M. Forces or Dominions Forces; or any other practitioner designated for the purpose by the Secretary of State for Scotland.

The Secretary of State has made arrangements similar to those made by the Minister of Health (*British Medical Journal*, Nov. 21, 1942, p. 616), and copies of the general directions, forms, and notes may be obtained from the Secretary, Department of Health for Scotland, St. Andrew's House, Edinburgh, 1, or from the medical officer of health of any county or large burgh.

EFFECTS OF RADIATION ON X-RAY WORKERS

The Minister of Health has issued a circular (No. 2718) bringing to the notice of local authorities and voluntary hospitals a service which the National Physical Laboratory is prepared to render for testing the amount of radiation received by workers in x-ray departments. Although the service is intended primarily for hospitals, it is open to any person who uses x-ray apparatus or to any institution which employs workers who use x-ray apparatus.

The initial test (which should normally suffice) consists of each worker carrying a dental film in the breast or waistcoat pocket for a week. These films (one film per person) can be obtained on request from the Director, National Physical Laboratory, Teddington, Middlesex, together with a record card and instructions for use. On receipt of the exposed film and the record card, and a remittance of two shillings per film, the Laboratory will furnish a report. If the results are satisfactory, the test should be repeated, and fresh application made to the Laboratory, at three-monthly intervals. If the initial test shows unsatisfactory results, the Laboratory will be prepared on request to undertake the more elaborate investigations required. The fee for any such investigation will be of the order of £5, together with any travelling or subsistence expenses incurred.

C. L. Prince and S. A. Vest (*J. Urol.*, 1941, **46**, 1129), who record the fifteenth genuine case of leiomyosarcoma originating in the prostate in a man aged 52, state that leiomyosarcomas form a definite entity and differ pathologically and clinically from other sarcomas of the prostate. They are of a somewhat lower degree of malignancy in the adult than other types of sarcoma of the prostate. The average duration of life in leiomyosarcoma of the prostate is 3.2 years in patients over 20 and 2½ months in patients under 20. The authors' case is the first in which a pre-operative diagnosis was made and complete removal of the prostate carried out. The patient was completely well and free from metastases one year after the operation.