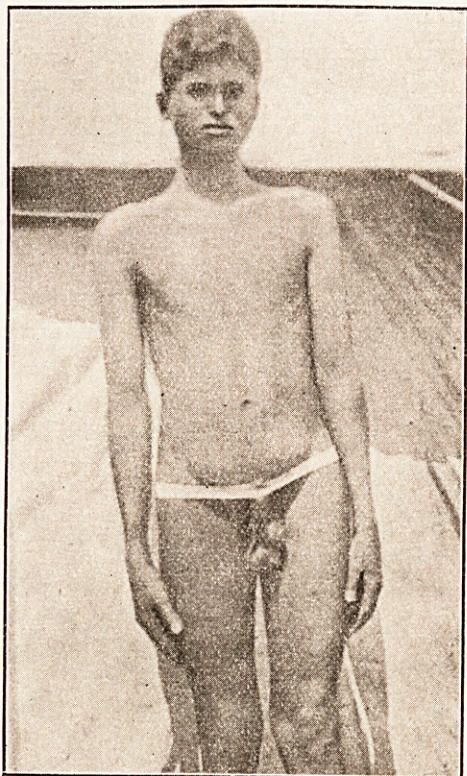


to have been no record of a similar case in medical literature up to date.

The proud owner of this Triple Testicle, an enrolled Follower, by name Ramasami, aged 19 years, a native of Trichinopoly District, in South India, arrived at the Followers' Central Depôt,



Kirkee, about August, 1918, and was medically examined when about to proceed in a draft for Overseas service about October or November, 1918.

The third testis was found above the left normal one, with its own adnexa-globus major and minor—and its own cord, distinctly traceable alongside the other normal left cord, up to the left internal abdominal ring, and was moored to the left wall of the left sac of the scrotum, above the normal left testis. The components of its cord were equally distinguishable. Even the "testicular feel" on pressure over this gland was the same, in every way, as over the other two normal glands.

TUBERCULOUS GLANDS IN AXILLA.

By DR. K. BHUSHAN, L.R.C.P. & S. Ed., D.P.H. (Lond.),
Health Officer, Srinagar and Hony. Surgeon to the
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TUBERCULOSIS is unfortunately so common in Kashmir that all sorts of tuberculous tissues and organs come into the daily experience of a medical practitioner here, specially that of a surgeon. As an honorary surgeon working in the renowned Kashmir Mission Hospital, with

the kindness and courtesy of Dr. Neve, for several years past, I have had occasion to see a lot of surgical tuberculosis. I have operated upon scores of cases of tuberculous glands of the neck and axilla, with uneventful recovery. But the present case to which I want to refer in this article, has been an important and eventful one and this is my excuse for writing on it.

A woman named Rahmi, aged 35, was admitted for large lump of the size of a large orange under her right armpit with a duration of 6 months on 17th August, 1920. The tumour was moderately fixed to the tissues underneath, though by manipulations it could be moved to and fro to some degree and the skin over it was quite free. Both Dr. Roche who had seen the case on a previous day and myself who examined her on the day of operation, thought it either to be a lympho-sarcoma of the armpit or a matted up mass of tuberculous glands. We decided upon an operation, which I began to perform. Having made the usual skin incision and on displacing the subcutaneous fat and fascia on either side, I found out that it was a large mass of tuberculous glands, which had invaded not only the centre of the armpit, but the anterior and posterior folds of the axilla deeply. It was a tiresome dissection for some 45 minutes that cleared the centre and the posterior fold of the armpit of some 10 or 12 characteristic tuberculous glands, ranging from the size of a small chestnut to that of a large walnut. The main axillary artery and vein were exposed and some of the large branches passing down to the chest were cut and ligatured. On putting my finger deep under the anterior fold of axilla, I found another mass of four or five large glands, right underneath the pectoralis minor, lying on the intercostal spaces just in the vicinity of the main vessels. I made up my mind to use my fingers only and with considerable difficulty I was able to scoop out these glands, but as soon as it was done a gush of blood came from underneath the glands. This was immediately controlled by long artery forceps. My difficulty now was to tie these branches of the main vessels which had been either punctured or torn in the course of handling. Neither catgut on ordinary curved needles nor on an aneurism needle could reach them, as the bleeding vessels were quite 5 to 6 inches from the reach of my hand. I called in our chief Dr. Neve to advise and seeing the difficulty, he advised careful packing of the wound with a long-tailed sterile swab, the tail protruding through the untied stitches in the middle. Some three or four silk stitches were applied above and two or three below and the central four stitches were left open, to be tied after 24 hours, after gently pulling the swab away.

Dr. Roche had kindly taken the swab out of the wound and tied the open stitches when I arrived to see the patient next morning.

An aseptic dressing was again applied. The hæmorrhage had ceased and the wound looked very clean, there being no temperature at all for four days after the operation. We had hoped for perfect recovery when on the fifth day on my arrival in the wards, I learnt that the patient had suddenly died in the early hours of the morning. The Sister in charge of the case told me that the patient had not been taking her food well, and was feeling rather uneasy for the last two days, though there were no other urgent symptoms.

The sudden death may have been due to either chloroform poisoning (she was under chloroform for 1½ hours though that alone cannot count for it) or to embolism or heart failure from some other cause. Dr. Ernest F. Neve, who has kindly permitted me to publish this case, is of opinion that the death was probably due to pulmonary embolism.

LITERARY NOTES.

WE have to thank Dr. J. L. Todd, McGill University, Montreal, for an extract of a review of the literature on the nature of the granules formed by *Spirochata Duttoni*. The original paper was published in the *Bulletin de la Société de Pathologie Exotique*. Reference is made to the work carried out in the Runcorn Research Laboratories of the Liverpool School of Tropical Medicine, from which it was concluded in 1905 that transmission of *Spirochata Duttoni* by *Ornithodoros moubata* "is not mechanical but some developmental process is carried on in the tick." From later observations it was asserted that spirochætes may develop from granules formed by fragmentation of the chromatin and extruded especially by encysted forms.

After an exhaustive review of more recent literature the author concludes that all that is certainly known concerning the development of spirochætes cannot controvert an assertion that spirochætes multiply only by direct fission; yet, many observations make it probable that a development by a granular stage does exist in the spirochætes of African relapsing fever and in other allied organisms.

PHYSIOLOGICAL NOTES.

CARBON DIOXIDE AND BLOOD.

LOEWY gives the following figures as to the partition of Carbon Dioxide in Normal Blood:—

100 c.c. of blood yields 40 c.c. carbon dioxide distributed as follows:—

In simple solution	..	1.9 c.c.
As Sodii Bicarbonate	..	18.8 c.c.
In organic combination with protein	..	11.8 c.c.
In combination with Hæmoglobin	..	7.5 c.c.

Buckmaster has recently investigated the relations of carbon dioxide and hæmoglobin.

Arguing that venous blood has been shown by Gardner and himself to be capable of taking up and holding much larger quantities of carbon dioxide (nearly double the quantity) than the amount present in normal arterial blood; he concludes that there must be "some vehicle or vehicles for the assumption of carbon dioxide which is not already saturated." As it is unlikely that this increased absorption would depend on the conversion of sodium bicarbonate into sodium carbonate, or on an increase in the protein content, the probability is that hæmoglobin is the vehicle concerned.

The results of Buckmaster's experiments show that the total carbon dioxide which can be absorbed by defibrinated blood at a pressure of 104 m.m. of mercury rises and falls with the hæmoglobin percentages. All the proteins of the blood have the power of absorbing carbon dioxide; but none of them exhibit the feature to the degree possessed by hæmoglobin, and Buckmaster believes that hæmoglobin possesses a specific capacity for the absorption of carbon dioxide.

THE MECHANICAL EFFECT OF FLUID IN THE PERICARDIUM ON THE FUNCTION OF THE HEART.

YAS KUNO (*Journal of Physiology*, Vol. LI, Nos. 4 and 5) describes experiments designed to throw light on the effects of increasing the intrapericardial pressure. Most of the experiments were carried out on the heart-lung preparation in dogs. Ringer's fluid was introduced into the pericardium under pressure. The pressure in the pericardial cavity, aorta, and vena cava were recorded simultaneously. The following important conclusions were arrived at:—

1. "A very small amount of fluid in the pericardium causes diminution in the output of the heart. By further addition of fluid the output diminishes regularly.

2. The arterial pressure changes only slightly until the amount of fluid in the pericardium reaches a certain limit. Beyond this limit the addition of a small amount of fluid causes a distinct fall of arterial pressure.

3. The height of intrapericardial pressure which brings the circulation of blood to a standstill is just the same as that of venous pressure. If the venous pressure be raised during the standstill of the circulation, the blood-flow is restored.

4. In the intact animal, it is very difficult to raise the venous pressure by injection into the veins. An infusion of a large amount of blood causes no rise of venous pressure nor dilatation of the heart, or only a brief one. Such an infusion is therefore of no advantage to the circulation when this is hindered by pressure of fluid surrounding the heart.

5. Adrenalin diminishes the volume of the heart; the intrapericardial pressure may therefore be decreased. In the intact animal, it