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SMALLPOX IN RELATION TO PREGNANCY AND THE PUERPERIUM.

by

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INTRODUCTION.

The epidemic of smallpox which attacked Hong Kong in 1937-38 was the most virulent known in the history of the colony. More than 2,000 deaths from smallpox occurred between November 1937 and July 1938, and it is the purpose of this paper to describe the group of pregnant and puerperal women suffering from the disease who were admitted to the smallpox hospital during that period.

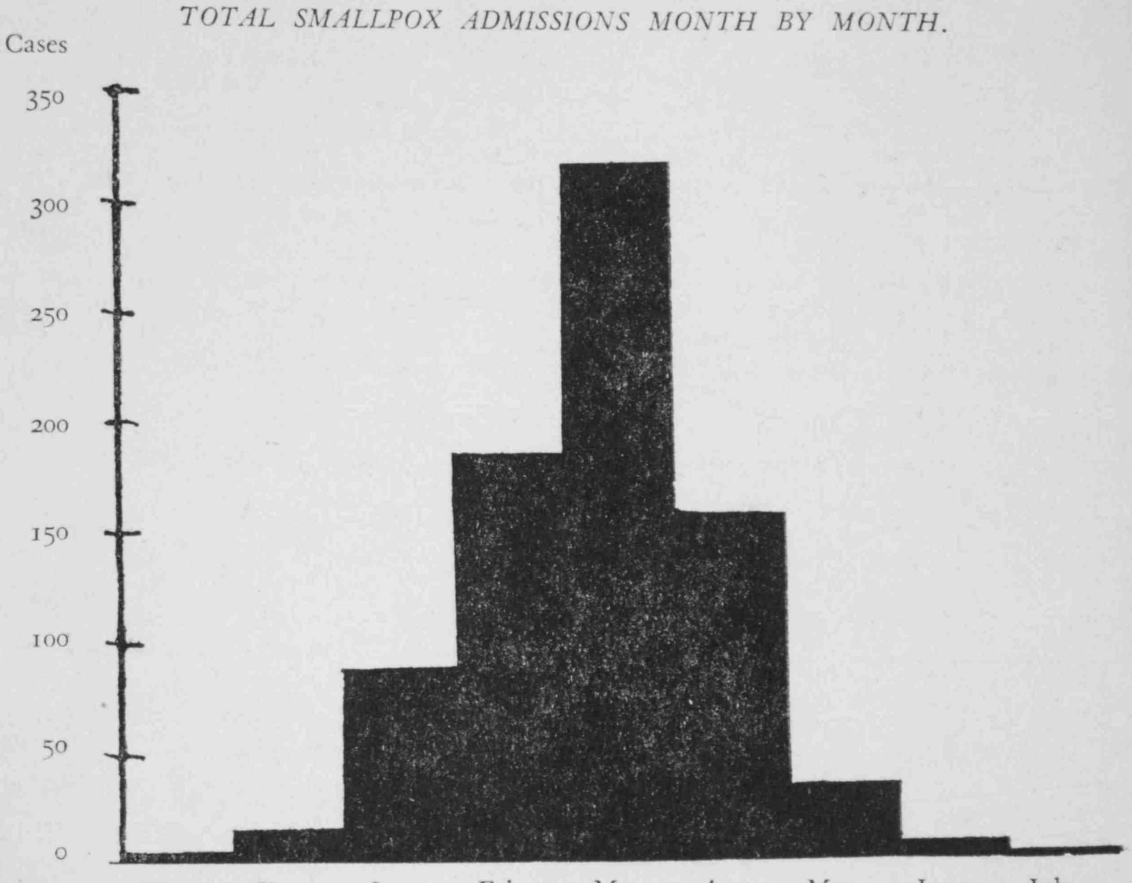
The disease first appeared in the colony in November 1937, established itself in December and assumed epidemic proportions at the beginning of 1938. The epidemic reached its peak in March and the accompanying diagrams (I and II) show the monthly totals admitted to hospital and the monthly figures of admissions for the group being discussed. The total number of cases admitted to the smallpox hospital throughout the epidemic was 810; the pregnant and puerperal women admitted suffering from smallpox numbered 44.

No attempt will be made in this paper to discuss the epidemic as a whole. Suffice it to say that it reached its peak in March 1938, one of the coldest months of the year, and that the number of pregnant women attacked increased pari passu with the intensity of the epidemic.

The 44 women included in this group have been classified arbitrarily into pregnant and puerperal cases, 27 puerperal and 17 pregnant, according to the time relation between admission to hospital and miscarriage or the birth of a child. The distinction is obviously of no value save as a means of classification because many of these patients bore their children either a few hours before or a few hours after admission to hospital, and the mere act of parturition clearly had nothing to do with the contracting of smallpox.

DIAGRAM I.

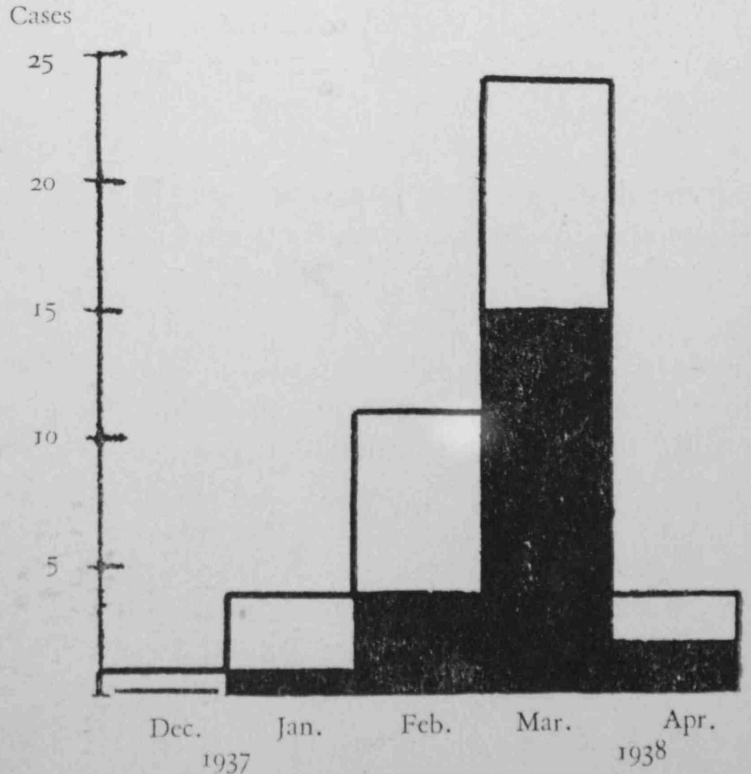
TOTAL SMALLPOX ADMISSIONS MONTH BY MONTH.



November, 1937	4	February, 1938	186	May, 1938	33
December, ,, ...	15	March, ,, ...	317	June, ,, ...	8
January, 1938	89	April, ,, ...	157	July, ,, ...	1

DIAGRAM II.

MONTHLY ADMISSIONS OF PREGNANT AND PUERPERAL WOMEN SUFFERING FROM SMALLPOX 1937-38.



Whole columns = totals for month.

Black columns = deaths.

TYPES OF SMALLPOX SEEN.

It is well known that pregnancy increases susceptibility to smallpox and also that the disease frequently tends to assume the toxic type in pregnant women. This series seems to show that women at all stages of pregnancy are liable to catch the disease and that it may assume the toxic form whether it occurs early or late in pregnancy.

The types of smallpox seen in this group were classified as follows:—

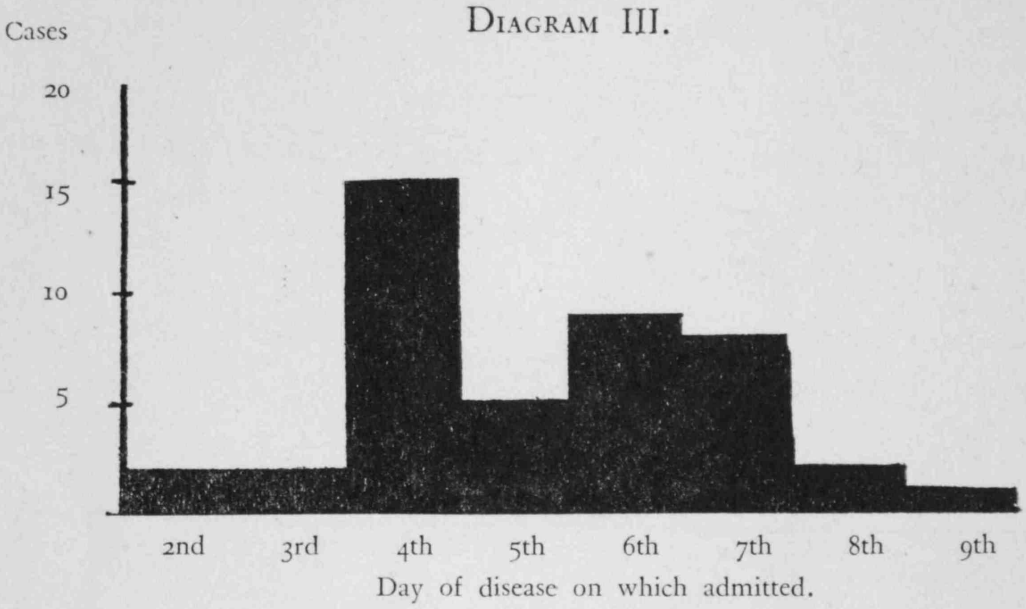
TABLE I.

Type of smallpox.	*Total.*	*Deaths.*
Toxic	16	16
Semi-confluent toxic	1	0
Confluent toxic	1	1
Confluent unmodified	3	2
Confluent modified	1	1
Semi-confluent unmodified	2	2
Semi-confluent modified	5	0
Discrete unmodified	1	0
Discrete modified	14	0
	44	22

Eighteen of the forty-four women in the group, or 40.8% suffered from toxic smallpox and seventeen died of it. This figure in itself is enough to show how greatly pregnancy predisposes to the toxic type of the disease, for of the 810 cases admitted to hospital during the epidemic only 83 suffered from toxic smallpox. In this epidemic, therefore, the toxic variety of the disease occurred four times as often in pregnant women as in the average patient. Of the other women in this group who died two were suffering from unmodified confluent smallpox, two from unmodified semi-confluent smallpox and one from a modified confluent type of the disease.

The following diagram (diagram III) shows the day of the disease on which these women were admitted to hospital.

DIAGRAM III.



The majority were admitted on the fourth day, and as was to be expected the more gravely ill the woman the earlier the disease was diagnosed. The rise in the numbers admitted on the sixth and seventh day of the illness is accounted for by the fact that most of these women were suffering from discrete modified smallpox. Their toxic phase had not been unduly severe and a definite diagnosis was only made when unmistakable focal lesions had appeared.

It has already been pointed out that the women admitted after delivery outnumbered those who were pregnant by 27 to 17, and most of these women admitted in the puerperium stated that they had borne their children within the two or three days preceding admission. That is to say, most of them had either miscarried or produced a living child at the onset of the disease when the toxic phase was at its height. One of the women in this group arrived at the smallpox hospital dead and four survived less than twenty-four hours after admission.

The longest interval noted between the birth of the child and admission to hospital was nine days.

The clinical picture of smallpox complicating pregnancy differs in few respects only from that of smallpox proper. Prodromal rashes were very much commoner in this group than in the series as a whole. Nine of the forty-four women, or 20.4%, showed a well defined prodromal rash, whereas the incidence of prodromal rashes in the series as a whole was under 5%. In five of the toxic cases the rash was petechial, the petechiae having the abdomino-femoral and axillary fold distribution; in three modified discrete and

one unmodified semi-confluent case the rash had the characteristic bathing-drawer distribution, and in two of these cases the appearance of this prodromal rash enabled a correct diagnosis to be made in a maternity hospital hours before the focal phase began.

Uterine haemorrhage occurred in 22.7% of the cases and was not a common symptom. It was seen in ten patients only, nine of whom were suffering from toxic, and one from confluent smallpox. In all the toxic cases it had been a prominent symptom from the onset of the disease. In only one case which recovered was there any abnormality of the lochia or evidence of subinvolution. This patient was suffering from semi-confluent smallpox in which the toxic phase had regained the upper hand. On admission she was covered with a profuse haemorrhagic rash. She was delivered of a premature living infant on the day following admission and her lochia became offensive during the course of the next week. All the other patients who bore children involuted normally and showed no lochial change.

The haemorrhagic phenomena seen in the toxic cases were identical with those noted in the other toxic cases in the series. Bleeding from the gums and tongue, epistaxis, subconjunctival and palpebral haemorrhages, haemoptysis, haematuria and haemorrhage from the bowel were all observed in addition to the skin haemorrhages and the uterine haemorrhages just described. No case in this group showed haematemesis however, nor were any retinal haemorrhages seen in these women, and it is pertinent to observe that these were the rarest haemorrhages in the series as a whole. Tonelessness of the facial and somatic musculature was a prominent sign in all the toxic cases and the characteristic foetor oris was almost constant. Enlargement of the liver was detected in half these women. The lobster erythema was seen in three of them, and hiccough was a troublesome terminal symptom in one. Almost all of them remained mentally clear to the end and restlessness, apprehension and a sense of substernal oppression were common symptoms. (See Figs. 2 and 4).

The cases comprised under the headings confluent and discrete smallpox differed in no distinctive way from the average case of smallpox and merit no special consideration.

ASSOCIATED DISEASES.

It is extremely common in this part of the world to find beri-beri complicating pregnancy, and all smallpox patients were asked as a routine question whether they had ever suffered from beri-beri. As the Chinese of all classes know a great deal about beri-beri their answers can be relied on.

Data on this point are not available in seven of the cases in this series. Of the remaining thirty-seven women, twenty-two stated definitely that they had never had beri-beri, but nevertheless thirteen of them showed gross reflex changes: eleven had lost both ankle and knee jerks, one had lost both ankle jerks but retained her knee jerks and the thirteenth had no knee jerks but retained her ankle jerks. The other nine women who denied having had beri-beri showed normal deep reflexes.

Fifteen women said that they had either had beri-beri in the past or had it now. Four of these women associated the disease with their present pregnancy, the other eleven associated the disease with former pregnancies. It is striking to note that fourteen of these fifteen women were multiparous, the parity of the fifteenth being unknown. In all these cases the knee and ankle jerks were unobtainable and acroparaesthesiae were present. Only one of these patients showed slight preibial oedema. It is worthy of mention that not a single woman in this series showed signs of a pregnancy toxaemia. It is true that such a toxaemia would tend to be masked by the toxic phase of smallpox, but even so pregnancy toxaemias proper were conspicuous by their absence.

Another point of interest brought out by these findings is that they show clearly how people who live on a pre-beri-beric plane pass imperceptibly from the pre-beri-beric stage to the stage of the established disease. Thirteen of the women who denied having had beri-beri showed loss of tendon reflexes.

One other point about which there is some uncertainty is whether toxic smallpox of itself can abolish knee and ankle jerks. Six of the toxic cases studied in this group stated definitely that they had never suffered from beri-beri, yet their knee and ankle jerks were absent. It seems more likely, in view of existing circumstances in Hong Kong that this loss was due to a vitamin B_1 deficiency rather than to the toxaemia of smallpox.

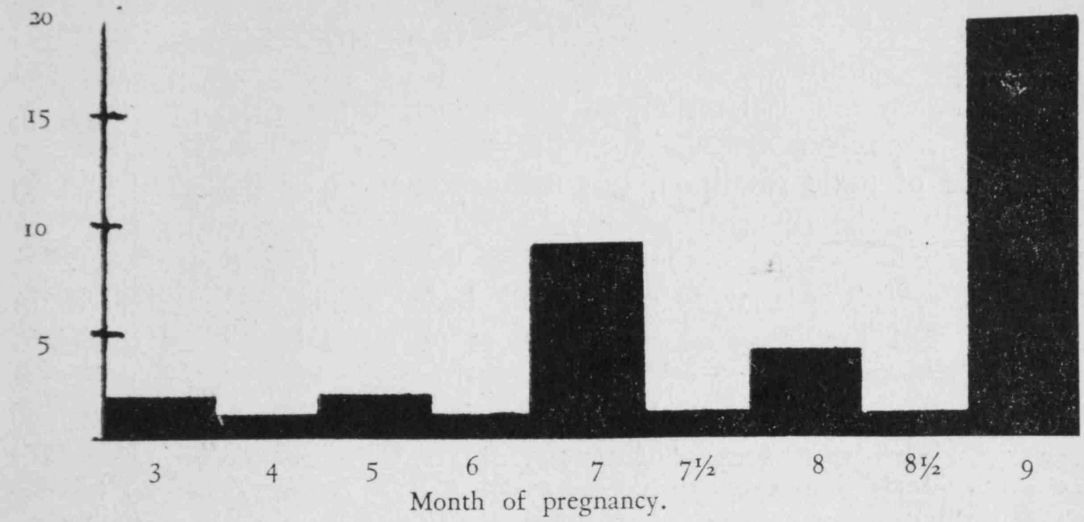
Search was made for existing or antecedent skin disease in all these patients. It is known that chronic skin diseases increase susceptibility to smallpox in time of epidemic but no definite relationship was found in this series. Only one woman was suffering from scabies when she contracted smallpox, and only two gave a history of scabies earlier in life. No other associated diseases were noted in this group of cases.

STAGE OF PREGNANCY, PARITY AND VACCINAL STATE OF MOTHER.

No case occurred earlier than the third month of pregnancy, and the majority of the patients in this group were women at or about full term, or just after delivery.

The appended diagram (IV) shows the period of pregnancy at which the women were attacked by the disease.

DIAGRAM IV.



Period of pregnancy at which disease developed.
(Data unknown in 4 cases).

No hard and fast conclusions can be drawn from these figures about the possibility of an increase in susceptibility to smallpox as pregnancy progresses, but it is perhaps significant that every pregnant woman who developed the disease at or before the sixth month of pregnancy died of it, and all the cases occurring in the first six months of pregnancy were of the toxic variety with one exception. Nor was it possible to correlate the degree of parity of the mother with any one type of the disease, or with a particular susceptibility to it.

Of the primiparae seven recovered and four died, three of the deaths being due to toxic and one to confluent smallpox. It is evident, therefore, that primiparae do not appear to run greater risks than multiparae if their pregnancy is complicated by smallpox.

TABLE II.

Parity.	*Number of cases.*	*Deaths.*
Primiparae	11	4
Para 1	3	0
Para 2	7	3
Para 3	7	5
Para 4	2	2
Para 5	1	0
Para 7	1	1
Para 12	1	0
Multiparae; parity unknown	2	1
Parity unknown	9	6

The vaccinal state of the mother was of some importance in determining the type and outcome of the disease.

The vaccinal state of these women, estimated by visible scars, was particularly interesting in the toxic group. Ten of these women showed no scars and two of them stated definitely that they had never been vaccinated, but six of them showed numerous well foveated scars, one having eight, one seven, three six and one two. It is at first blush a little startling that such well scarred women should die of toxic smallpox, but the explanation of the phenomenon is clearly that the toxaemia-modifying component conferred on human beings by vaccination disappears much more rapidly than the other components. These women had all been efficiently vaccinated in childhood but the lapse of twenty years or more had been sufficient so to reduce their power of modifying the toxic phase of the disease that they succumbed to it before the focal phase had had time to appear. The one semi-confluent woman who recovered, although the toxic phase reappeared, possibly owed her recovery to the use of large quantities of convalescent smallpox serum, but her case will be commented on in more detail under the heading Treatment. The effect of vaccination on the other groups is clear from Table I, the unmodified cases in each category representing the women who bore no vaccination scars.

The mortality for the group as a whole was 50%. This figure compares favourably with the death rate for the whole series of cases which was 44%, but it must be remembered that the epidemic was an exceedingly virulent one. The mortality rate among the women in this group who were classified as toxic was 94.4%. Thirteen of the twenty-two women who died or 59% were unvaccinated, if it is permissible to assess their vaccinal state by the presence or absence of scars. The effect of modification by previous vaccination on the death rate is shown in Table I.

FATE OF THE MOTHER AND CHILD.

Despite every effort it was impossible in three of the cases to find out what was the fate of the child. A number of mothers were admitted shortly after delivery at home or in other hospitals and many of them were too ill to give coherent histories. A number of others were admitted a few days after the birth of their child, either at home or in some hospital, and their statements about the state of their children at birth remained perforce uncorroborated.

It would be a sterile and unprofitable task to explore all the multitudinous permutations and combinations of life and death which might have occurred in this group. A simple recital of the observed facts will be more than enough.

If the patient is grievously stricken with the disease she may die with her child yet unborn. This does occasionally happen in the gravest types of the disease, and was noted in two of the toxic patients who were four and five months pregnant respectively and in one woman, eight months pregnant, suffering from unmodified confluent smallpox.

Or the mother may recover and go successfully to term, a phenomenon usually seen in modified and therefore mild cases of the disease. It was noted five times in this group, all the mothers being seven months pregnant when they developed smallpox. Four of these patients had modified smallpox, but the fifth had an extremely severe unmodified attack of confluent smallpox complicated by extensive cellulitis of the neck. (See Fig. I). She was fortunate in making a complete recovery, and intelligent enough to let us know some weeks after her discharge that she had been safely delivered of a normal full term son. The foetal heart was heard and foetal movements were detected in all cases before these patients were discharged from hospital.

Both the mother and the child may live and this conjunction of events was noted in 11 cases. It is worthy of remark that all these mothers were suffering from modified smallpox, nine of them being classified as modified discrete cases, two as modified semi-confluents. Four of these children were born in the smallpox hospital; the others had been born either at home or in some other hospital.

The mother may recover after producing a stillborn child and this happened three times. Or she may live after giving birth to a premature babe which survives a few hours only. This occurred twice. Of these five mothers four were suffering from modified smallpox, one was unvaccinated. The mother may die after producing a stillborn child. This result was seen in 8 cases. Seven of these mothers suffered from toxic smallpox, the eighth from unmodified confluent, and four of the toxic cases had not reached the seventh month of pregnancy.

Perhaps the most unfortunate of the observed possibilities is the death of a mother who has been delivered of an apparently normal child earlier in the course of the disease. This happened in 6 cases. Four of these mothers died of toxic smallpox and stated that they had been delivered of perfectly normal healthy children at home before admission. Their statements could not be corroborated and must be accepted with reserve for reasons which will be given later. The other two mothers were suffering, one from unmodified, the other from modified confluent smallpox.

Finally, the mother may die after giving birth to a premature or full term child which lives for a few hours only. This was the

outcome in 3 cases, two of them being toxic, one unmodified confluent. One of these mothers who died of toxic smallpox gave birth to an apparently healthy full term child the day after she was admitted to hospital. The child was promptly vaccinated and segregated as far as was possible. It died suddenly three days after birth without having shown any signs or symptoms. At autopsy haemorrhages were found in the thymus, the pericardium and throughout the serosa of the gut. There can be little doubt that the child had contracted smallpox in utero and that it died in the toxic phase of the disease. Its vaccination showed no signs of taking and its skin was spotless. This case makes one loth to believe that the children described in the preceding group all survived, although their mothers may have been truthful in saying that they appeared healthy at birth. Table III shows these facts in tabular form.

STATE OF FOETUS; VACCINAL SUSCEPTIBILITY OF CHILDREN.

Five living and nine dead children were born of these mothers in the smallpox hospital. In no single case was a smallpox lesion detected in either a stillborn or a living child. One of the women who miscarried produced a slightly macerated foetus but apart from this no abnormalities of any kind were noted in these children and foetus.

Four of the children in Group 3 of Table III and one in Group 8 were vaccinated shortly after birth. One of them who was vaccinated on the right leg on its birthday developed a primary take six days later. This is a rare phenomenon. It is usually accepted that if a mother has been vaccinated late in pregnancy her child tends to be refractory to vaccination for the first few months of life, and there is some evidence to show that this is so. If such an infant obtains from its mother a quantity of immune body sufficient to render it refractory to vaccination, how much more likely to do so is the child born of a mother suffering from smallpox! But the phenomenon, although rare, has been recorded, if not explained.

This child was kept in the hospital during its mother's convalescence. The other four were sent home shortly after birth to be cared for by vaccinated relatives so no records relating to three of them are available. It has been possible, however, to trace one of them, and this child who was born in the smallpox hospital of a mother suffering from discrete modified smallpox is alive and well to-day.

TREATMENT.

At the height of the epidemic it became extremely difficult to provide adequate accommodation for these women, and it was quite impossible to separate the toxic cases who were obviously dying from mothers who were clearly going to recover.

TABLE III.

Group.	*No. of cases.*	*Types of smallpox.*
(1). Mother dies, child unborn	3 cases.	2 toxic. 1 unmodified confluent.
(2). Mother recovers, child survives in utero.	5 cases.	3 discrete modified. 1 confluent unmodified. 1 semi-confluent modified.
(3). Mother lives and bears a living child	11 cases.	9 discrete modified. 2 semi-confluent modified.
(4). Mother lives, child stillborn.	3 cases.	2 semi-confluent modified. 1 discrete modified.
(5). Mother lives, child survives a few hours only.	2 cases.	1 semi-confluent toxic. 1 discrete modified.
(6). Mother dies, child stillborn.	8 cases.	6 toxic. 1 confluent toxic. 1 confluent unmodified.
(7). Mother dies, child survives.	6 cases.	4 toxic. 1 confluent modified. 1 semi-confluent unmodified.
(8). Mother dies, child survives hours only.	3 cases.	2 toxic. 1 semi-confluent unmodified.

(In three cases fate of child unknown.)

The infants born in the hospital were vaccinated on the spot and whenever possible sent home to be cared for by relatives until their mothers were better. The mothers who were admitted shortly after delivery or who were delivered in hospital were given a small daily dose of streptocide as a routine measure. This was rational as haemolytic streptococci were being continually isolated from skin lesions and boils occurring during the focal phase and in convalescence. Whether the measure was effective or not is doubtful, but no puerperal infections occurred, though conditions, theoretically at any rate, favoured their appearance.

The average daily dose of streptocide given to the mothers who were not grievously ill ranged from 1.5 to 3.0 gms. daily in divided doses. Twenty-five of these patients were treated with the drug and they received total doses ranging from 1 gm. to 102 gms. Experience gained in using the drug in other cases during the epidemic made

it perfectly clear that while streptocide may, and sometimes undoubtedly does, prove life-saving in the septic complications so common in the focal phase of smallpox, it exerts no direct influence whatever on the toxic phase of the disease.

It was used in only seven of the eighteen toxic cases in this group. One of these women recovered after receiving 102 gms. of streptocide in two weeks, but as she had been given 625 c.c. of convalescent smallpox serum during the same period no conclusions can be drawn from her case regarding the efficacy of the drug. The other six women died. One of them received 7 gms. of the drug in the 23 hours following her admission and it made not the slightest difference to the course of the disease. Another received 5 gms. daily for a week but again it availed nothing.

The drug was used in over 100 cases throughout the whole epidemic and it is fair to say that never once was it found to alter the course of the toxic phase of smallpox in the slightest degree. On the other hand, it proved to be signally effective in helping to overcome the cellulitic infections, the abscesses, the sloughs and the boils which so frequently interrupt the course of convalescence from the focal phase of the disease. In this small series of women, the mother who recovered from a severe attack of unmodified confluent smallpox and carried her child successfully to term almost certainly owed her life to streptocide. She developed a spreading cellulitis at the root of the neck on the right side. Multiple incisions had to be made and haemolytic streptococci were isloated. She was gives 6 gms. of streptocide daily for a week and at the end of this time her wounds were clean and convalescence from then on was uneventful.

Streptocide, therefore, has a place in the treatment of smallpox but a minor place. It is of undoubted use in the later septic complications caused by haemolytic streptococci, and it is also to be recommended as a prophylactic against possible streptococcal infections when one is compelled to keep puerperal women suffering from smallpox herded together in a smallpox hospital taking in every type of case. It seems that the prophylactic dose in such cases need not exceed 3 gms. daily.

Convalescent smallpox serum was used during the later part of the epidemic. By that time, it had become clear that serum obtained from patients who had been suffering from smallpox for less than thirty days was useless. Attempts were therefore made to obtain supplies of serum from convalescents 30-60 days after the onset of the disease. This serum was, so to speak, home-made as the convalescents were bled before leaving hospital, and supplies were inconstant.

FIG. 1.

To show extensive sloughing at base of neck on the right side occurring in the course of unmodified confluent smallpox. The woman was thirty to thirty-two weeks pregnant but recovered and bore a living child at term.

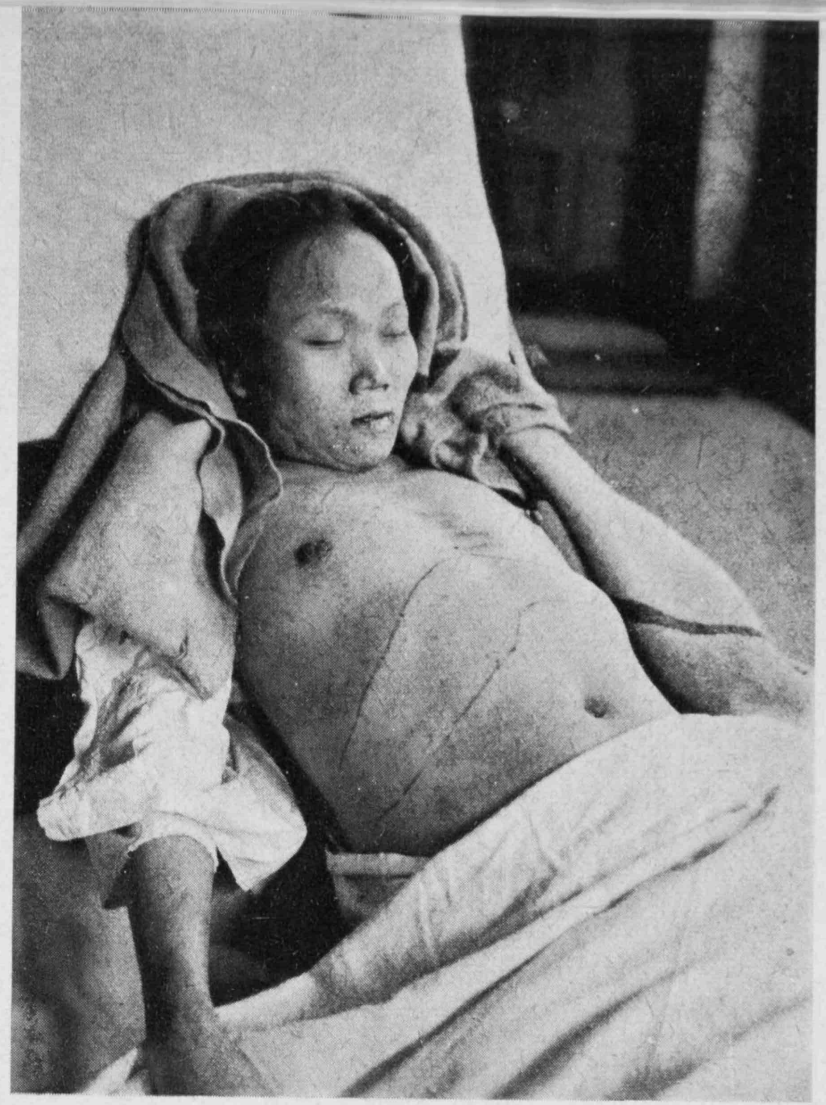


FIG. 2.

To show the massive enlargement of liver which may occur in the toxic type of smallpox. The woman was in the puerperium and died three hours after admission. The lower edge of liver, which weighed 81 ozs., and the costal margins are outlined. Note the absence of fully developed lesions and the blood clouting of the lips. Photograph taken after death.

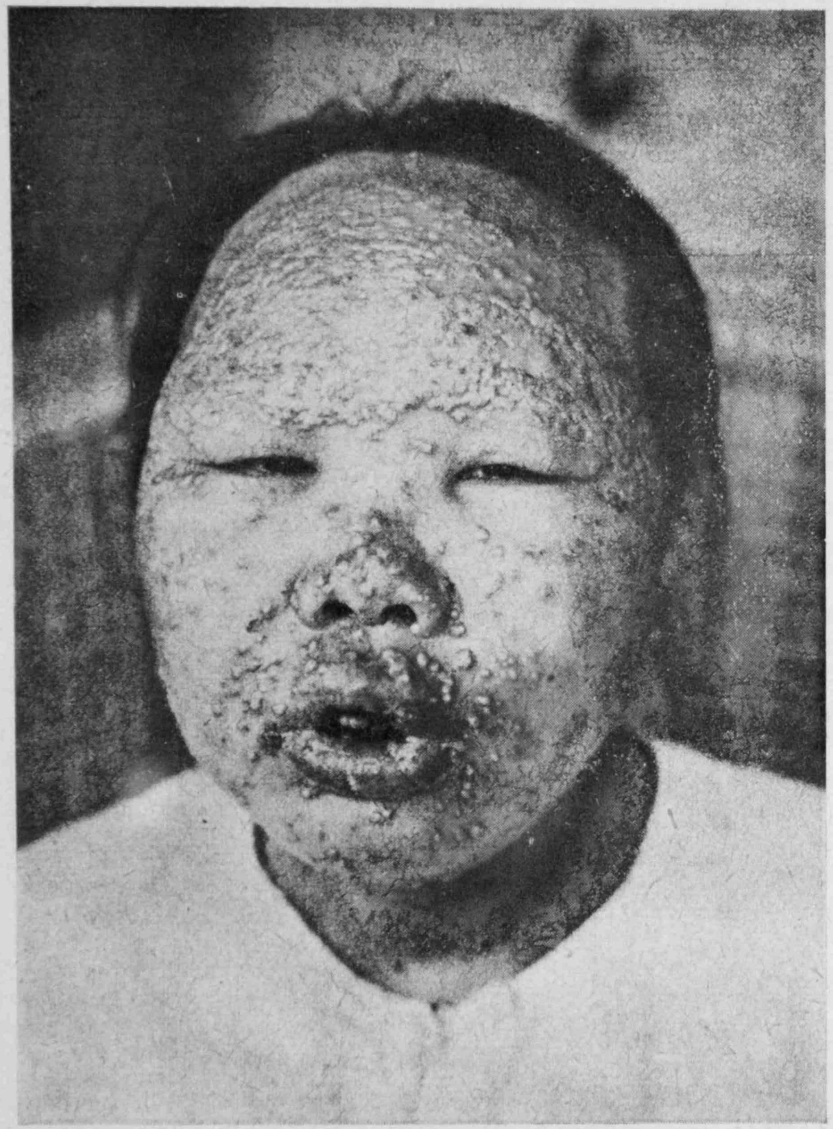


FIG. 3.

To show a woman in the puerperium suffering from a mild attack of modified discrete smallpox. She had produced a confluent patch of lesions on her forehead by applying a counter-irritant ointment to relieve headache during the period of invasion.

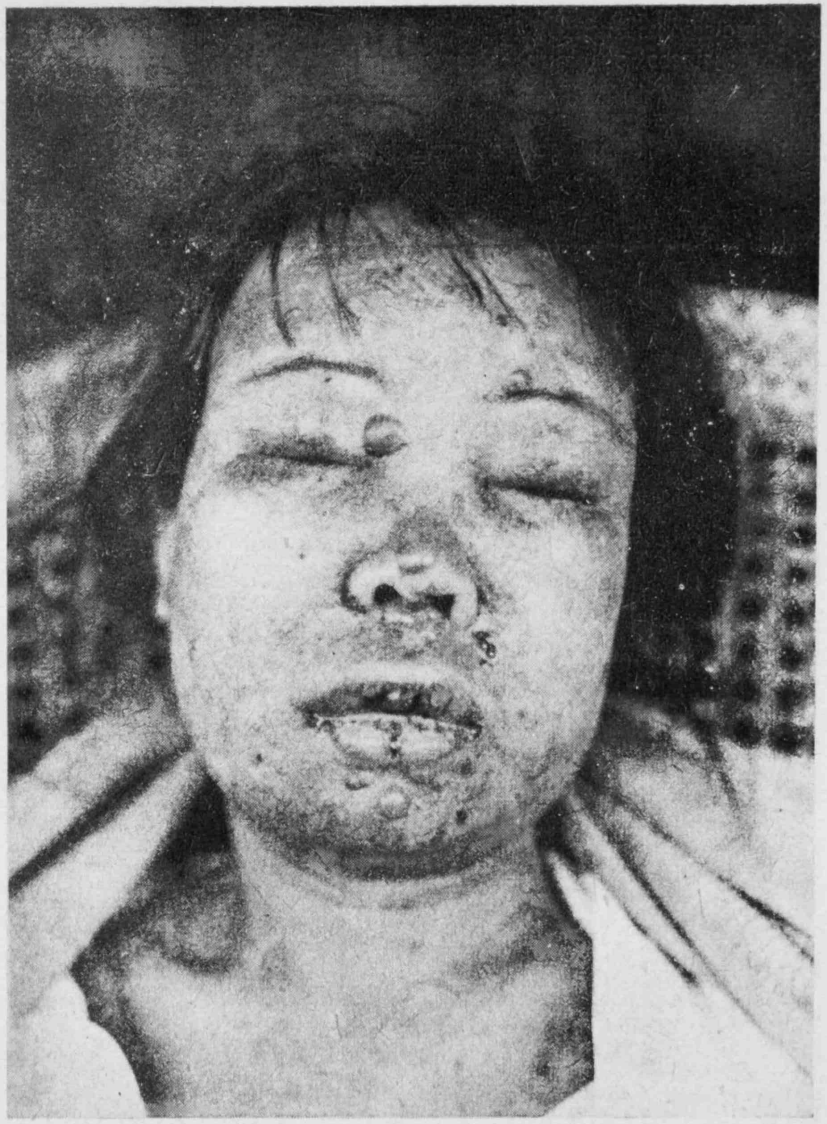


FIG. 4.

To show facial bullae filled with claret coloured fluid and blood clouted lips in a puerperal woman suffering from toxic smallpox. Note the general oedema of the face which was leaden coloured and the early lesions appearing on forehead.

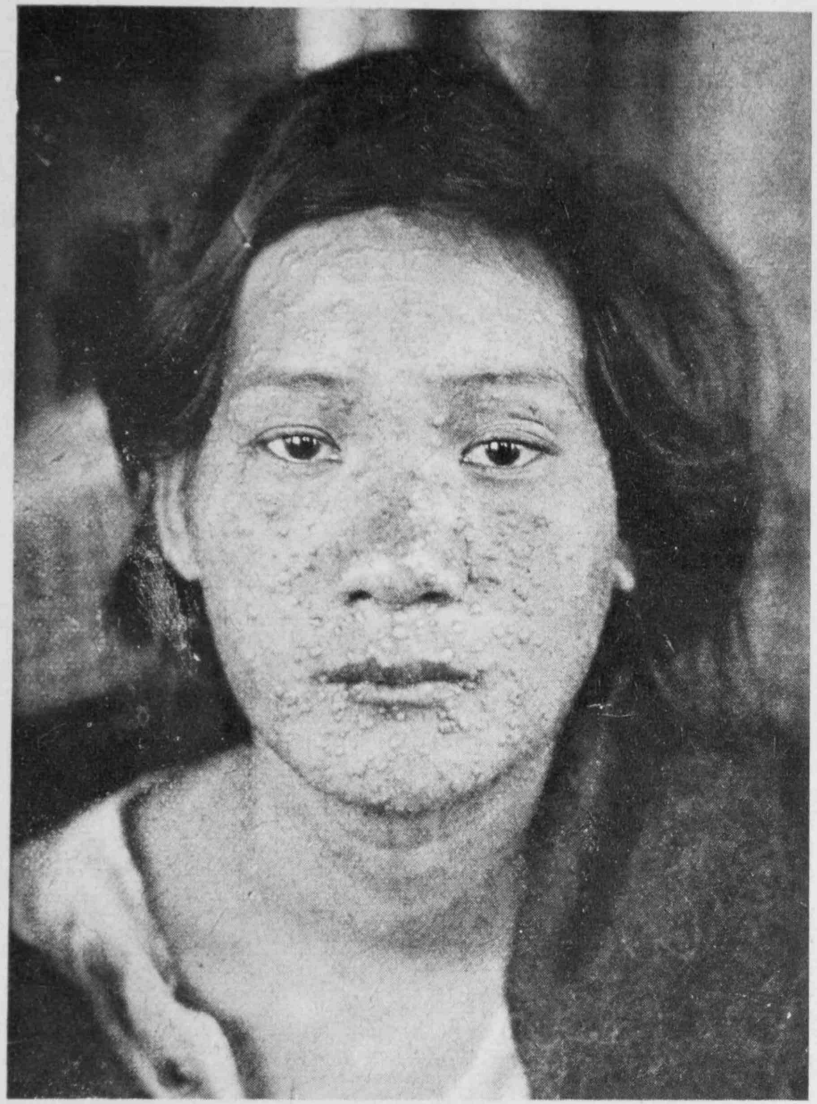


FIG. 5.

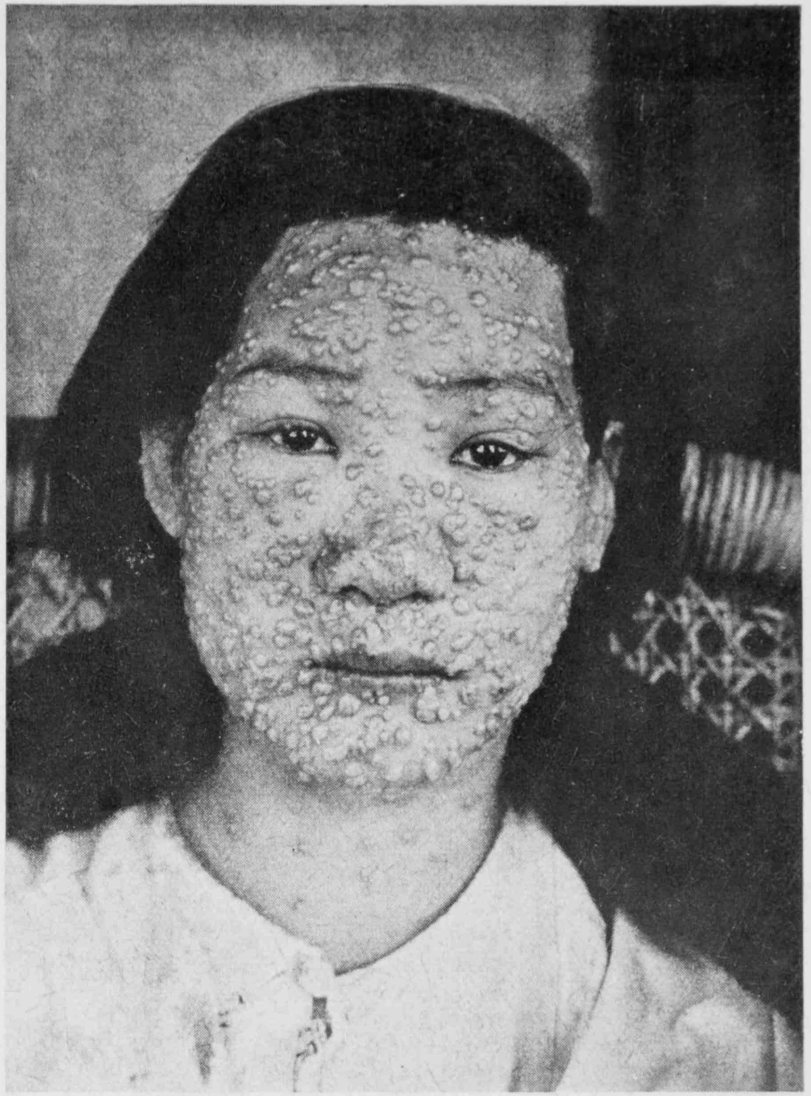


FIG. 5A.

Figures 5, 5A, 5B, and 5C show stages in the evolution of the lesions in a puerperal woman who was suffering from modified discrete smallpox. The intervals between the photographs 5, 5A and 5B were two days, while that between 5B and 5C was five days.

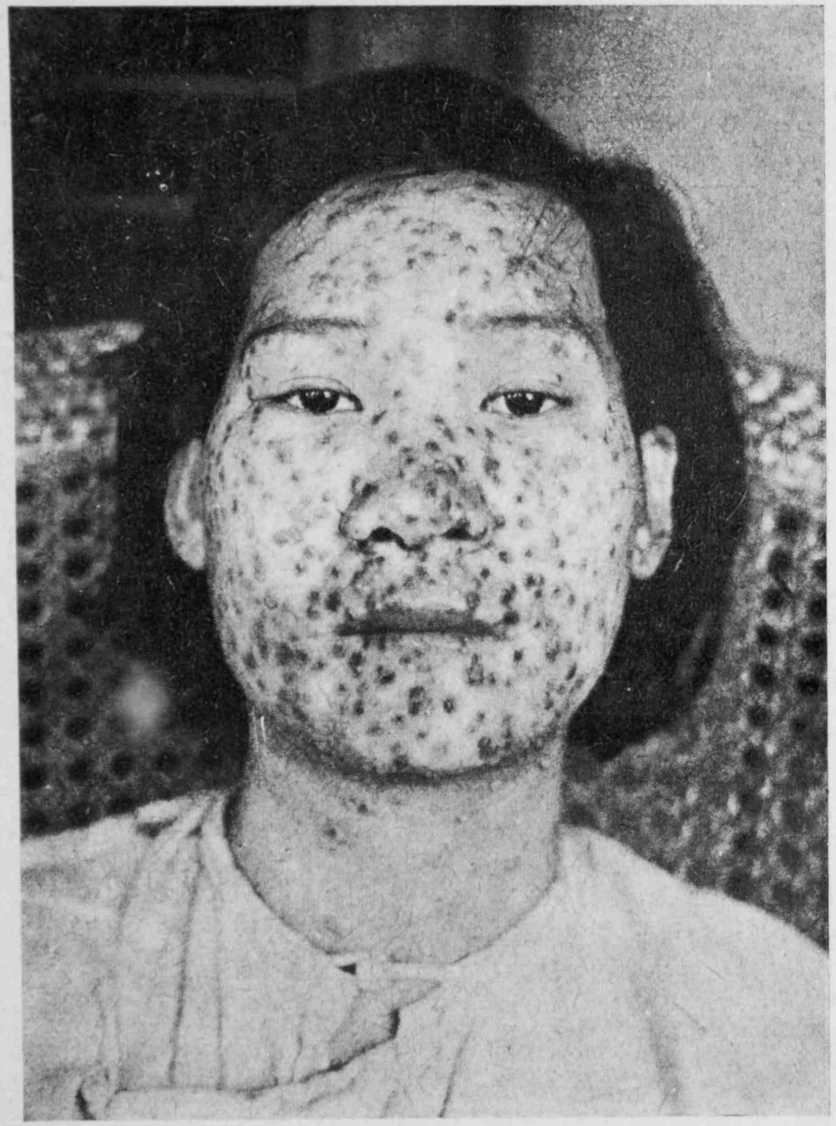


Fig. 5B.

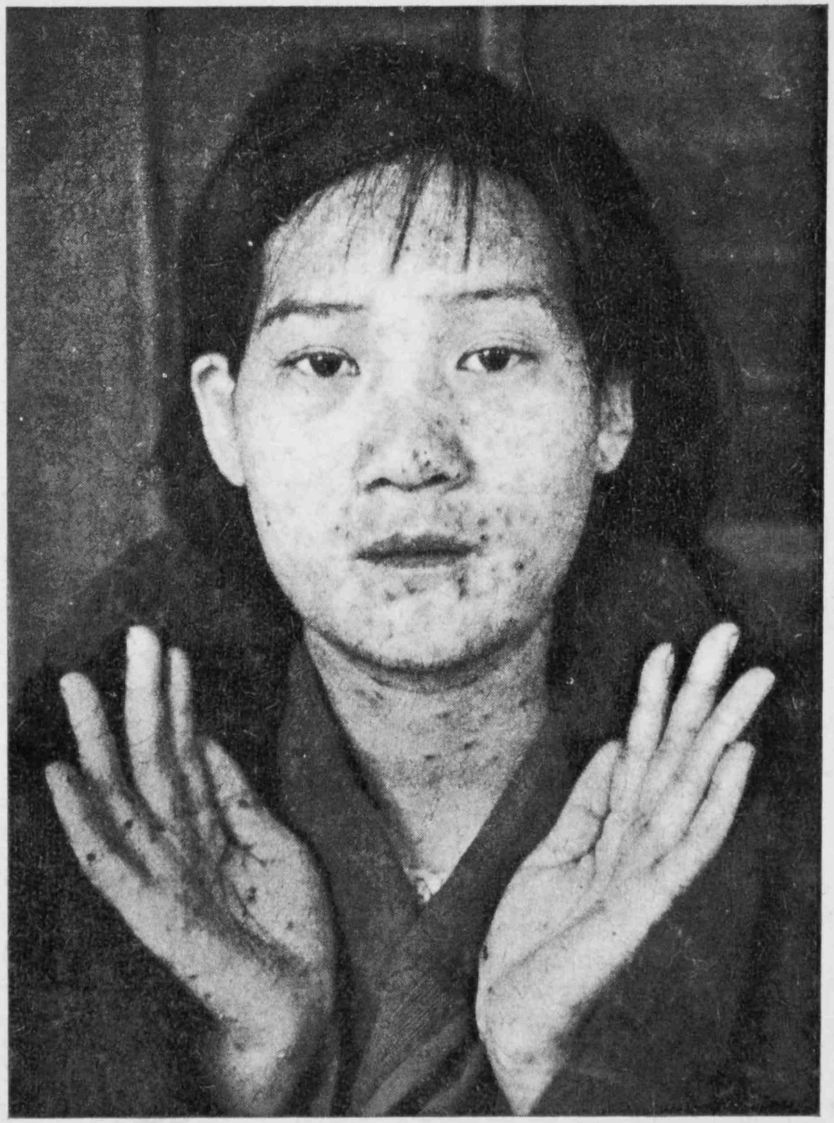


Fig. 5C.

However, enough was available to allow an extended trial of it in the last woman in this group. She was admitted to hospital on the 26th of April 1938 on the fourth day of her attack of smallpox. She appeared to be eight months pregnant, but could not give a history as she was only semi-conscious. Her temperature was 104°, her tongue dry and brown, her lips blood clouted and her body covered with a maculo-papular, poorly developed rash. Her back was a sheet of immobile purple effusion. She was given 20 c.c. of convalescent serum six hourly and after two doses her fever had dropped from 105° to 101°. This dosage was continued as far as was practicable until she had had in all 620 c.c. of serum, and despite the fact that she miscarried the day after admission and her lochia became slightly infected she recovered. The puerperal infection was treated with streptocide by mouth in a dose of 1.5 gm. daily, and douches and gave but little trouble.

No opportunity offered itself of trying the serum in other puerperal cases, but the results obtained in this case were so unexpected that the method merits further investigation.

SUMMARY.

1. A description is given of the fourty-four pregnant and puerperal women who suffered from smallpox during the 1937-38 epidemic in Hong Kong.
2. The preponderance of the toxic type of the disease in pregnancy is well shown by this group.
3. The influence of the vaccinal state of the mother on the prognosis both for herself and her child is commented on.
4. The fates of both the mothers and their children are described in detail.
5. The use of streptocide in the treatment of smallpox is touched on.

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NOTES ON A CASE OF IMPERFORATE ANUS WITH OTHER ABNORMALITIES,

by

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I. INTRODUCTION AND HISTORY.

Imperforate anus in itself is not such a rarity as to justify more than a brief note. In this case however the complete absence of an external anus, malproportion of the trunk, ectopia of the urinary orifice and skeletal abnormalities suggested the presence of deep seated developmental errors. The case to be described was obtained by the courtesy of Professor K. H. Digby. The patient was a male infant who died in the Queen Mary Hospital eight days after birth. The infant is the fourth child of healthy living parents. The first child died at nine months of unknown cause, but the second and the third are alive and well.

Unfortunately, as it will appear, there is no record of the method of feeding adopted, if natural or artificial, and no record of vomiting.

It is stated that faecal matter mixed with urine was voided by the abdominal orifice, and this point will receive comment later.

The temperature was subnormal on admission, in the neighbourhood of 95° F. The temperature rose on the 5th day to 101° F. with a rise of respiration rate to 42. From the 5th day the temperature fell gradually to 95° F. on the 8th day on which death occurred.

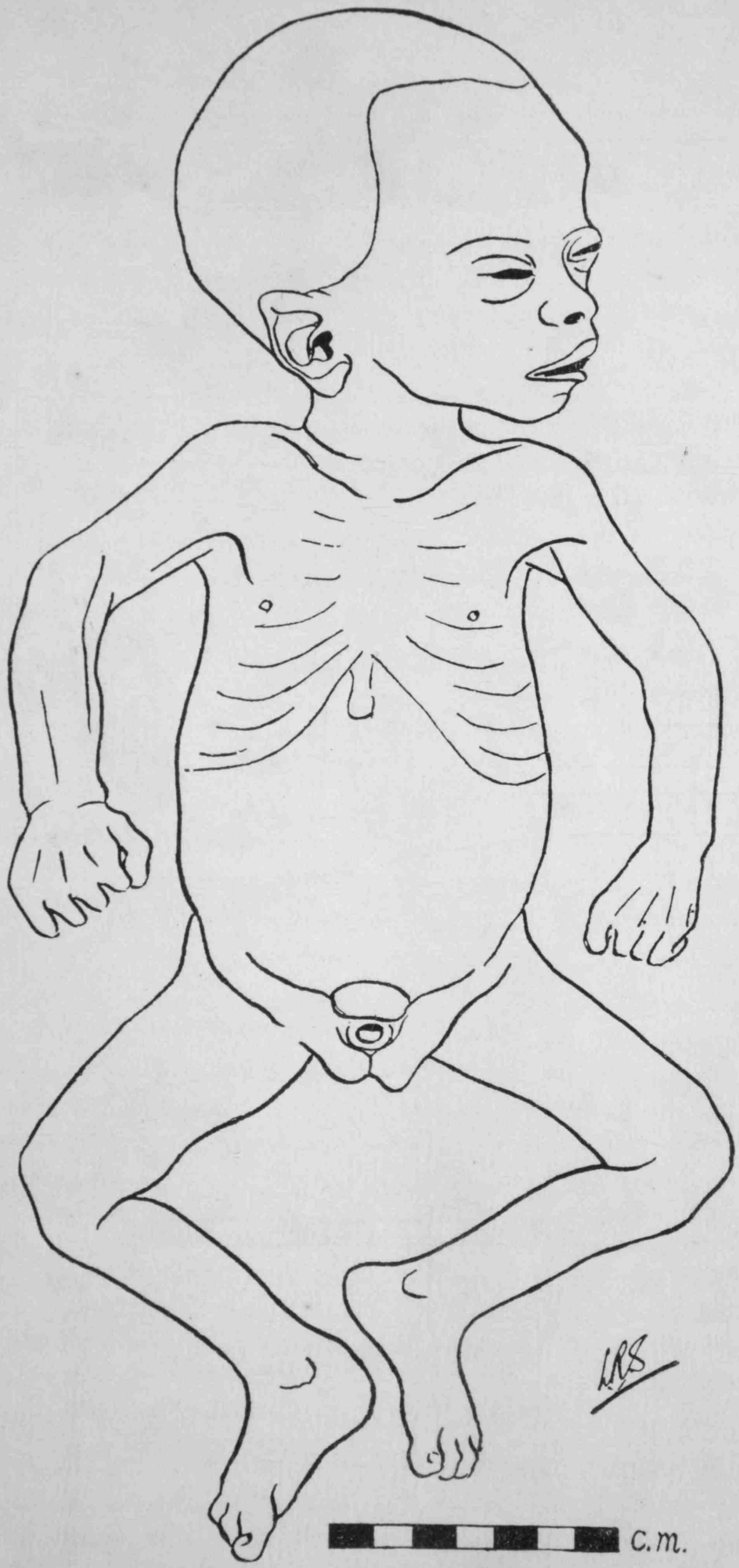


Figure I.

This figure is a drawing of the infant made from the front with the aid of the dioptograph.

The subject is somewhat undersized; but the proportions of the trunk particularly call for comment.

The sub-umbilical part of the abdomen is shortened to a very considerable degree.

The lower limbs took up the position of abduction represented in the drawing without any pressure or traction whatever.

The details of the sub-umbilical region and of the conformation of the external genitalia are shown in Figure II.

The scale at the foot of the drawing indicates centimetres.

II. EXTERNAL APPEARANCES.

The infant is thin and wasted with some degree of cyanosis. The umbilical cord is dried and shrivelled but not yet separated. The abdomen is full but not greatly distended. The most obvious abnormalities lie in the lower abdomen.

Figure 1 is a drawing made from the anterior. The observer cannot fail to notice at once the shortening of the sub-umbilical region of the trunk, with the close approximation of the external genitalia to the umbilicus.

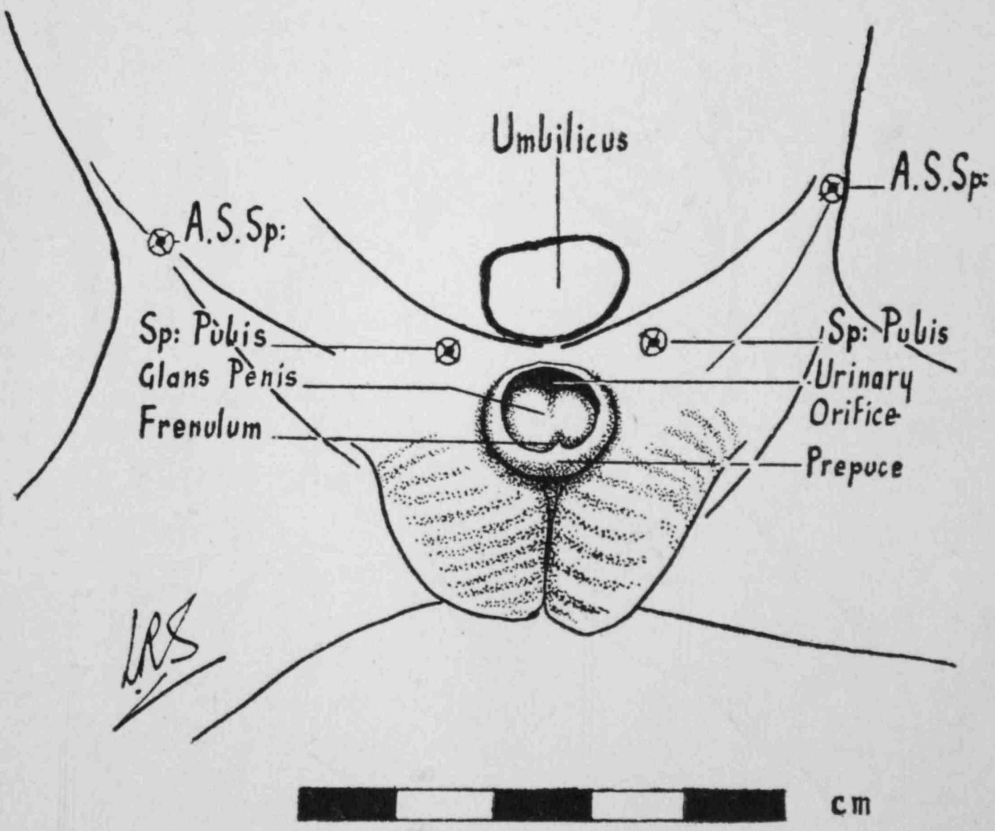


Figure II.

This is a drawing of the sub-umbilical region from the front made to scale with the dioptograph.

The positions of the Anterior Superior Spines of the Ilia and of the medial ends of the pubic bones are indicated A.S.Pp and Sp Pubis respectively. These four skeletal points could all be easily felt from the surface. A wide gap occupies the position of the symhysis pubis.

Very close to the umbilicus and below it lies the urinary orifice. It is to be noted that this orifice lies above or headward of the glans penis. The urinary orifice and the glans penis both are surrounded by an elevated custaneous ring, which is narrowest above and progressively becomes wider as it is traced downwards. This elevated ring, which is evidently the prepuce, is connected to the glans by a frenulum and also is continuous with the median raphe of the scrotum.

The scale below indicates centimetres.

Attitude.

When the body was laid on its back after rigor mortis had passed off, an attitude such as suggested in Figure I was at once assumed.

The lower limbs were scarcely flexed at all, but adopted a position of abduction and eversion with the lateral sides of the thighs lying flat on the table. This position is possibly a consequence of skeletal abnormalities which will receive comment in their place.

The External Genitalia.

Of the penis little more is to be seen than the glans, which projects from the body surface only 5 mm. The scrotum is small and slightly bifid but contains a palpable testis on each side.

Figure II shows the appearance of these organs in detail. Immediately below the umbilicus is an orifice of some 5 mm. transverse diameter through which urine was passed during life.

This urinary orifice is separated from the umbilicus by a skin ridge which projects from the body surface, surrounds the glans penis and in the perineum blends with the median raphe of the scrotum. This ridge is in fact the prepuce and the urinary orifice lies between the prepuce and the dorsum of the glans penis. A frenulum connects the glans to the prepuce on the tailward side.

The symphysis pubis is undeveloped. A gap of some two cm. can be felt between the two lateral elements of the pelvis where the symphysis would be expected to lie.

The perineal area is very small; no more than 2.1 cm. separates the glans penis from the tip of the coccyx, but this perineal area contains no vestige of the anus—not so much as a dimple can be seen or felt.

Frazer (1) remarks that at one stage the body-stalk is the caudal limit of the ventral body wall and indeed the cloacal membrane lies on the body-stalk. Separation of the body-stalk and the genitalia is brought about by ingrowth of mesoderm between them, apparently from the body-stalk itself during the fifth week.

Wyburn (2) after an exhaustive study on the development of this region confirms these statements and proceeds thus. "Extroversion of the bladder is due to mesodermal deficiency, particularly of the processes of secondary mesoderm arising from the hind end of the primitive streak, following on which there is . . . impaired development of the muscular coat of the bladder, of the symphysis pubis, and of the formation of the external genitals and infra-umbilical portion of the abdominal wall."

It seems that certain of the abnormalities in this specimen can be explained by failure of the mesoderm, (see Fig. VIII).

III. THE ABDOMINAL VISCERA IN GENERAL.

It may be convenient to the reader to offer at this stage a short general description of the abdominal viscera.

The digestive tract presents for examination a stomach with liver, duodenum and pancreas which at first sight are not highly abnormal.

Following on the duodenum is a small intestine which is remarkably short and dilated, but seemingly simple in its arrangement.

Further investigation shows that the details of its disposition relative to the mesentery and other organs present certain interesting features. The small intestine ends in the lower lumbar region in a narrow fibrous cord which connects it to a very complex visceral mass in the pelvis.

It may be remarked here that the kidneys are normal in size and position and discharge by two normal ureters into the pelvic visceral mass.

The Abdominal Viscera examined from the front.

On opening the abdomen from the front (Figure III) the most prominent feature is the gut. All of this that is to be seen has the characters of small intestine and no part of the colon is to be seen from the front or indeed is to be found by dissection.

This small intestine is large and roomy, in some parts more than in others, but in no place excessively dilated. In places, the intestine is covered with some plastic fibrinous material, indicating some slight degree of peritonitis.

Presenting its edge to the front and traversing the cavity from left to right and from above downwards, the great omentum subdivides the whole cavity into two compartments, as may be seen in Figure III.

Above and to the right of the great omentum are the stomach, liver and gall-bladder but no part of the digestive tract lower than the descending part of the duodenum. The compartment of the abdominal cavity below and to the left of the great omentum is occupied by the small intestine and the mass of the pelvic viscera.

The liver presents no unusual features except a few small accessory lobes on its inferior margin and near the gall bladder. The gall-bladder projects from below the edge of the liver and the cleft for its lodgment is normal in position but of small size.

The spleen makes contact on its visceral surface with the kidney, the stomach and the small intestine. There is no colic area and the

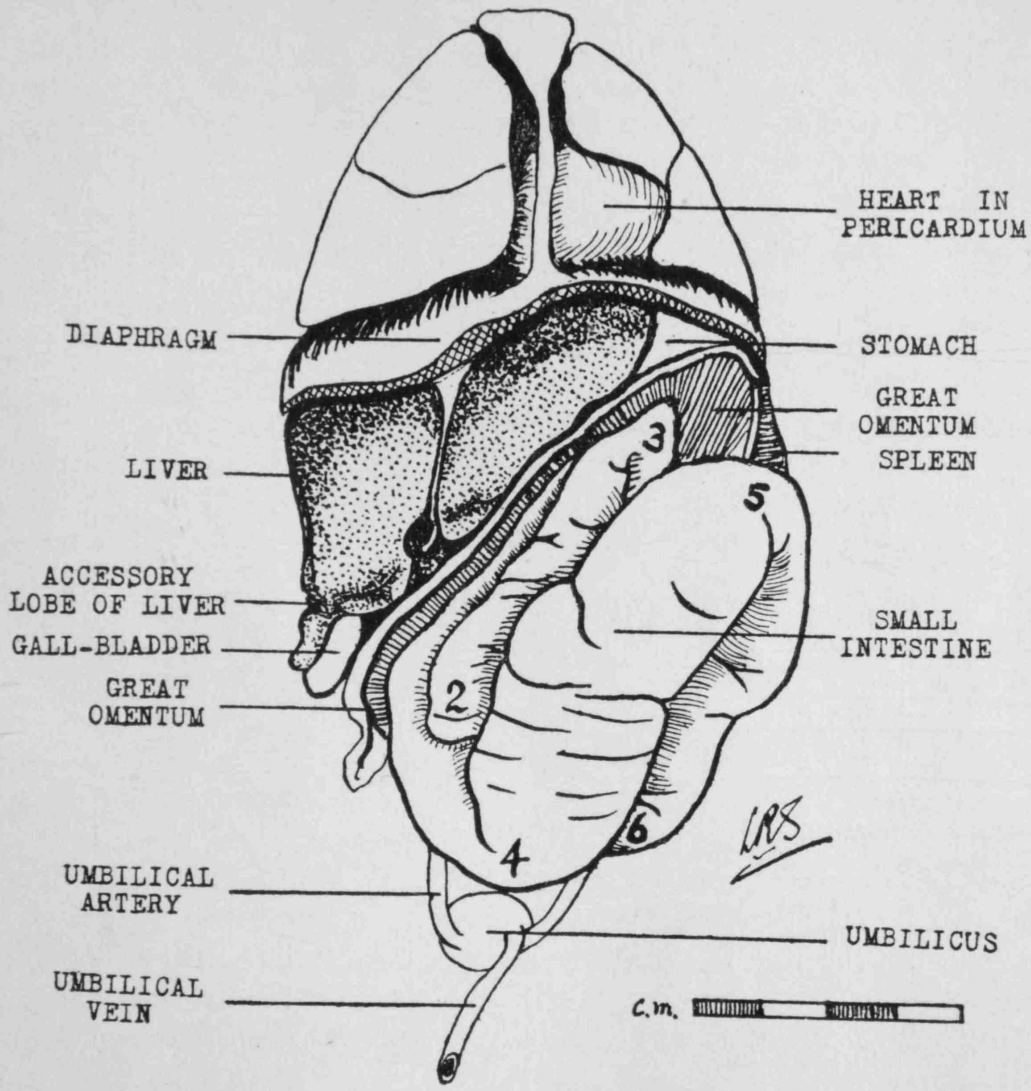


Figure III.

This is a drawing of abdominal and thoracic contents from the front. The whole of the anterior abdominal and thoracic walls have been removed. Below may be seen the umbilicus with the normal pair of umbilical, i.e. placental, arteries and the single vein. This vein has been cut and the two parts thrown apart, i.e. upwards and downwards.

The abdominal cavity is divided by the great omentum into right superior and left inferior compartments.

The right superior compartment contains the stomach, liver and gall-bladder. These organs present no striking features in front view except a few small accessory lobes to the liver.

The left inferior compartment contains a few enlarged coils of small intestine with flexure angles marked 2 to 6. The features of the small intestine will receive separate consideration in Figure IV.

It is to be especially remarked that no part of the large intestine is to be seen.

The scale of this drawing is shown below in centimetres.

only other feature for remark is plastic peritonitis in the gastric and intestinal areas. The size is normal.

On following the small intestine from below the duodenum to its termination in a fibrous cord there are found six angular bends, of which five are shown in Figure III.

IV. THE OMENTAL BURSA.

By comparison with the duodenum, which is large and capacious, the stomach is small and contracted. The peritoneal connections of the stomach, however, and the structure of the omental bursa are of interest.

In Figure IV the attachment of the great omentum is seen to extend on the right to the front of the duodenum; on the left, where it has been cut some little way from the stomach, it lies as a vertical sheet between the fundus of the stomach and the spleen.

The gastro-hepatic ligament (lesser omentum) attaches the lesser curvature of the stomach to the portal fissure of the liver, and extends further to the right than is usual, for the common bile duct lies well to the left of its right lateral edge.

When the stomach is turned upwards and viewed from below, as in Figure IV, it is seen that the stomach is attached by its posterior surface to the pancreas by a peritoneal ligament which may be called the "dorsal mesentery of the stomach." The attachment of this ligament to the stomach is very close to that of the gastro-hepatic ligament and to the pancreas, at its upper border father than its anterior surface. Between the two is the caudate lobe of the liver and the cavity of the omental bursa (lesser peritoneal cavity), which is decidedly small.

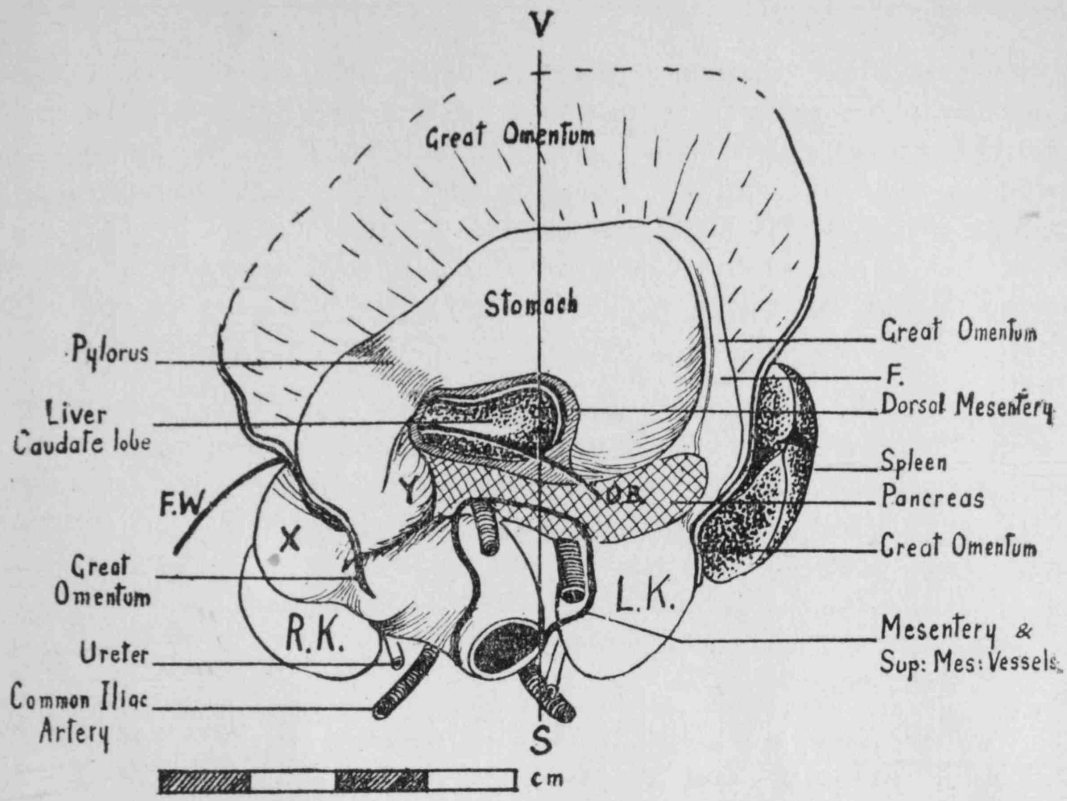
This dorsal mesentery, therefore, is the inferior boundary structure of the omental bursa, and in this sense corresponds to that peritoneal ligament which extends from the pancreas to the great omentum and contains the transverse colon in the normal.

It is hoped that the four sketch diagrams in Figure V will help to explain the topography of these peritoneal ligaments and also the interpretation put upon them.

Figure IV.

In the upper drawing the stomach and the great omentum have been lifted forwards and upwards to show the structures behind. The great omentum extends on the right to the front and right side fo the duodenum, and on the left, it intervenes between the stomach and the spleen as far as the tail of the pancreas.

From the uppper edge of the pancreas a peritoneal ligament, which it is proposed to call the dorsal mesentery of the stomach, extends to the posterior surface of the stomach near the lesser curvature. On cutting out the central part of this ligament the interior of the omental bursa and the caudate lobe of the liver are exposed. The thick black line F.W—O.B represents a curved pointer passed in



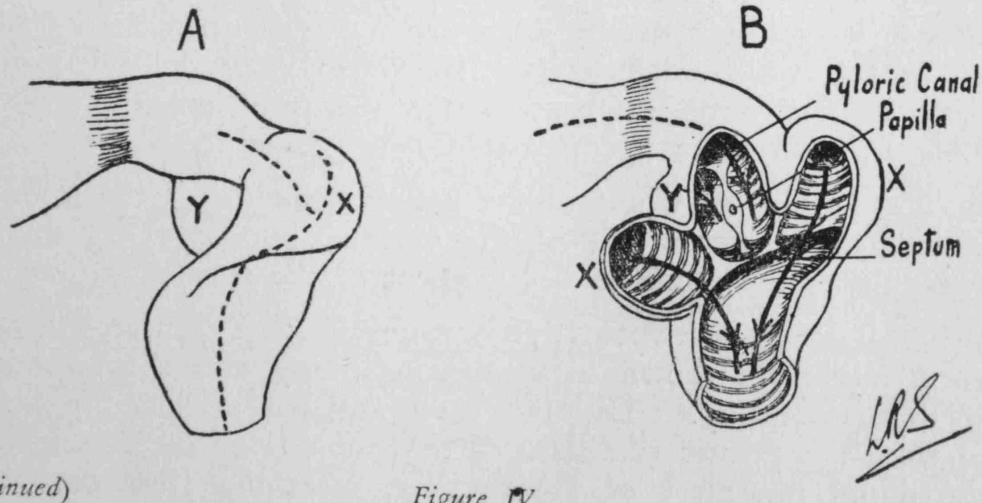


Figure IV.

(*Continued*)

through the foramen of Winslow and out through the inferior wall of the omental bursa, i.e. through the dorsal mesentery of the stomach.

The duodenum presents a sacculation to the right, marked X, and a second to the left, mraked Y. The root of the mesentery, represented by a heavy black line, surrounds this part of the intestine and the superior mesenteric vessels. The position of the common iliac arteries is to be noted.

The line V-S shows the plane of a vertical section which is represented schematically in Figure V.

The lower Figure A is a posterior outline drawing fo the duodenum showing dotted lines of section. The drawing B shows the appearance of the interior of the duodenum after having been opened with scissors as indicated and spread out.

The results show that Y is a wide diverticulum of the upper part of the duodenum which receives the pyloric canal and the opening of the common bile duct, whose papilla is well developed. A wall of mucosa closes off the upper part of the organ from the lower part, of which X is diverticulum.

Each of these diagrams presents the results of sagittal section through a plane such as is indicated by V-S in Figure IV. In each the right segment is viewed from the left, and the reader's right represents the back and his left the front. The same organs—liver, stomach and pancreas are given similar designations (L. St: P.) and in sketches B and D the colon also (C). In each diagram the heavy continuous line represents a cut of edge of peritoneum; in each the mark "X" distinguishes the caudate (Spigelian) lobe of the liver.

Diagram A presents the findings in the case under present discussion Three peritoneal structures are attached to the stomach, the great omentum (G.O), the gastro-hepatic ligament or lesser omentum (G.H) and the dorsal mesentery of the stomach (D.M). The omental bursa (lesser peritoneal sac) is enclosed by the gastro-hepatic ligament, the caudate (Spigelian) lobe of the liver, part of the dorsal wall of the abdomen, the dorsal mesentery and finally by part of the dorsal wall of the stomach.

We may compare diagram A with diagram B which represents the normal findings. The omental bursa is enclosed by like structures, except below, where the inferior wall presents essential differences. This inferior wall may be considered in two parts with reference to the transverse colon. The peritoneal ligament which connects the pancreas to the colon is usually called the transverse mesocolon (M.C). The ligament which connects the transverse colon to the greater curvature of the stomach whence hangs the great omentum (G.O) is called the gastro-colic ligament (G.C) in precise terms.

In the normal the whole of the posterior surface of the stomach enters into the anterior wall of the omental bursa. Diagram D, below B, shows the mode of development of the normal.

From a similar viewpoint at a suitable stage of growth the peritoneal layers are shown in continuous lining and the secondary fusions by cross-lining. The two layers of the peritoneum which extend from the greater curvature of the stomach to the dorsal wall, i.e. the dorsal mesentery of the stomach, form the great omentum (G.O) by partial fusion. Nearer to the dorsal wall, the true mesentery of the colon makes contact with the dorsal mesentery of the stomach

Figure V.

This figure presents a series of diagrams, each of which shows the appearances of a vertical sagittal section through the omental burma. It is supposed that in each case the reader is viewing the right half of the section from the left..

The organs are similarly distinguished in each, thus—L=liver, St=pancreas and, in B and D. C=colon. The mark X on the liver indicates the caudate lobe. P.H.=porta hepatis, G.H.=gastro-hepatic ligament, D.M.=dorsal mesentery of stomach and G.O=great omentum.

Diagram A suggests the findings in the specimen under description, of a section through the line V-S in Figure IV.

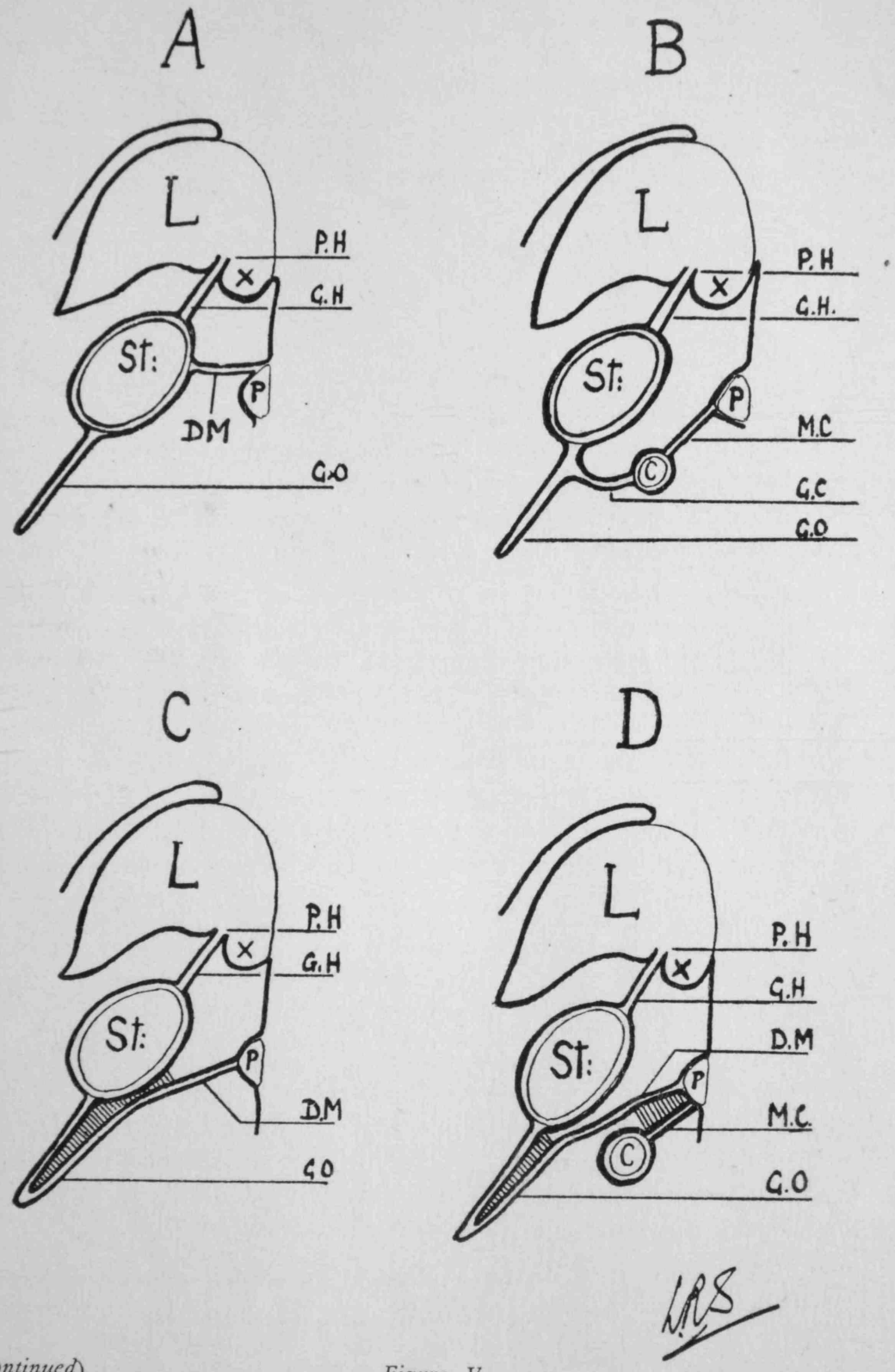


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Figure V.

Diagram B represents the findings in the normal, M.C = transverse mesocolon, and G.C = gastro-colic ligament. Other structures bear the same lettering as in Diagram A.

Diagram C is intended to furnish the explanation of the formation of the arrangements shown in Diagram A, deduced from the normal developmental plan shown in Diagram D. Fusion of peritoneal layers is shown by cross-lining.

Diagram D represents the plan of the development of the inferior wall of the omental bursa, as commonly accepted. Cross-lining indicates where fusion of peritoneal layers has taken place to produce the great omentum and the transverse mesocolon.

Further explanations are to be found in the text.

and by fusion with it forms the transverse mesocolon (M.C).

Traced from before backwards the history of the dorsal mesentery is as follows; first, its redundant layers fuse; secondly, a part remains free; thirdly and most posteriorly, it fuses with the mesentery of the colon. The most anterior part becomes the great omentum, the free part the gastro-colic ligament and the most posterior part the transverse mesocolon.

Diagram C shows the steps which we may suppose to have preceded the arrangements indicated in Diagram A and in Figure IV. The two essential modifications are these, first, the adjacent layers of the sac of the omental bursa have fused as in D, but the fusion has extended further up the posterior wall of the stomach, secondly, there has been no fusion between the dorsal mesentery of the stomach and the mesentery of any other part of the gut.

It may be remarked that in consequence of the posterior fusion of the dorsal mesentery on the stomach wall the capacity and the mobility of the stomach must have been much reduced.

V. THE DUODENUM.

Posteriorly a small vertical posterior mesentery takes the place the usually large posterior extra-peritoneal attachment. The form of the duodenum is shown from the front in Figure IV. The upper drawing shows that much of the organ lies in the transverse plane and that the familiar loop shape is lacking.

In all the drawings the place where the thick muscular wall of the stomach gives place to the thinner wall of the duodenum is shown by a band of cross-lining—the presumed pylorus.

The proximal part of the duodenum is expanded by a sacculation to the right "X" and a second to the left "Y," both of which are marked off from the distal part by a transverse flexure which is deeper on the left than on the right. Below these sacculations the distal part lies nearly horizontally. The duodeno-jejunal angle is indefinite and the suspensory muscle of Treitz was not found.

Below the main drawing in Figure IV and on the reader's left is an outline sketch "A" of the duodenum from the back, showing the sacculations "X" and "Y," with dotted lines which indicate the places of section. Scissors were passed in from below and the point engaged in the sacculation "X." This was opened up freely and then it was found that the lumen was obstructed and the second cut was made after a guide had been passed down from above through the pyloric canal. The septum across the lumen of the duodenum seems to be mucous membrane only.

The appearances after these two incisions had been made and the organ laid open are shown in sketch "B." Above the septum

is a compartment which receives the pyloric canal, the diverticulum "Y" and the common bile duct opening by a normal duodenal papilla. Below the septum is the continuation of the intestine and the opening of the diverticulum "X".

It is unfortunate that no note has been made of the results of any attempts to feed the child. Regurgitation if not forcible vomiting might have been expected.

The intestine below this obstruction contains some yellowish-green powdery material, on which Dr. E. Q. Lim has very kindly reported. This material contains no bile-salts and no more than a trace of bile-pigments. It is to be noted that "Y" is distinctly bile stained.

The occlusion of the duodenum must have taken place after the first discharge of bile into the intestine, or else the partition in reality is not quite complete, if we are to explain the presence of bile in the intestine at all.

Concerning the development of the duodenal mucosa Arey (3) writes ". . . . vacuoles appear in the duodenal epithelium of embryos between six and nine weeks old and the lumen is temporarily occluded; the remainder of the small intestine becomes vacuolated but not blocked." Frazer makes the same observation but is less positive that it is a constant occurrence (4). It is possible, therefore, that retention of an early developmental state accounts for this congenital atresia of the duodenum.

Congenital atresia of the duodenum is reported from time to time though rare. For the following references I am indebted to Bonar (5) who gives an account of a case of congenital atresia of the duodenum caused in part at least by an abnormal superior mesenteric artery, and so not comparable with this case.

Davis and Poynter (6) surveyed 392 recorded cases of congenital atresia of the intestine between the pylorus and the rectum. Of these the duodenum was the site in 134 cases.

Kulija (7) in like connection found that atresia of the duodenum accounted for 46 out of 184 cases of congenital atresia of the intestine, a proportion not very dissimilar to the other series.

When occlusion of the duodenum occurs, it seems to be most common in the region of the duodenal papilla of Vater. Cautley (8) quotes Spriggs who states this to be the case in 67 out of 92 cases of congenital occlusion at or about the duodenum.

Cordes (9) is also quoted to the effect that he recorded congenital occlusion of the duodenum above the papilla of Vater, opposite the papilla and below the papilla respectively in 20, 6 and 13 cases.

The position of occlusion in this case seems then to be the second commonest in the duodenum.

It is regretted that it has not been possible to make personal reference to all of these writers.

Specimens were taken for microscopical examination from Y above the septum and from X below it both with disappointing results.

Certainly this part of the intestine had small chance of adequate penetration by formalin. Microscopic examination showed that in both X and Y the epithelium has completely disappeared. Some traces of villi are to be found but nothing resembling the characteristic glands of Brunner.

The form of the duodenum of the 13 mm. human embryo described by Hunter (10) shows sacculations and a transverse flexure which strongly recall the features of our specimen. These features persist at least to the 22 mm. stage. It may be suggested that retention of this stage would go far to explain the present findings. Hunter gives no information as to the relative positions of the common bile duct and the transverse flexure.

The formation of duodenal glands is reported at the end of the third month by Arey, and early in the fourth month by Frazer. The 22 mm. stage is earlier than either of these dates. It is conceivable that development of the duodenum was inhibited in both its internal and external characters at some period between the second and fourth months.

For descriptive purposes at least the term duodenum must be retained, but it must be admitted that the position of the duodenum using the term in its functional sense has not been determined.

VI. THE PANCREAS.

The pancreas is an elongated structure which stretches across the upper abdomen from duodenum to spleen.

It is not differentiated into head, neck, body and tail as is normal but presents certain points of interest.

Despite the occlusion of the duodenum and the opening of its duct in the same duodenal papilla with the common bile duct it is not dilated. On the contrary, its single straight duct is narrow and difficult to follow. In this respect it differs from the case described by Bonar and referred to above in which the pancreatic duct was markedly dilated.

The peritoneal relations have received some comment. We have seen (Fig: IV) that the pancreas lies below the posterior attachment of the dorsal mesentery of the stomach, and therefore takes no part in the posterior wall of the omental bursa. This may be a result consequent on the absence of the transverse mesocolon which in the normal possibly exerts some traction on the dorsal mesentery, enlarges

the omental bursa downwards and brings about an attachment which is anterior rather than superior to the pancreas.

The position of the superior mesenteric artery is unusual but to this we can attach no great weight in view of the crowding of arterial trunks on the shortened abdominal aorta.

However, it may be remarked that normally the superior mesenteric artery lies between the body and the head of the pancreas and in part divides the two elements, dorsal and ventral from which the pancreas is developed. The dorsal pancreas normally lies headward of the artery and is represented by the body and the tail. The ventral pancreas lies tailward of the artery, and is represented by the head of the gland and provides the main duct which enters the duodenum with the common bile duct with which it developed.

The association of the pancreatic duct with the common bile duct indicates that this organ is the ventral pancreas with greater force than the position of the superior mesenteric artery proclaims it as the dorsal pancreas.

It is probable that in this case the ventral pancreas has extended across the abdomen to the spleen and so has imitated to a considerable extent the form usually taken by the dorsal pancreas.

This conjecture is to some extent supported by the finding of a small body on the back of the duodenum between X and Y which may well be pancreas, though histological findings are not conclusive. If pancreas, this is certainly the dorsal pancreas which has failed to make contact with the ventral moiety.

This small body had the size and shape, and indeed the appearance, of a split pea, but no duct was discovered. Microscopic section showed a network, or rather a spongework, of fibrous tissue which seemed to have contained alveoli of remarkably uniform circular shape and size. For the most part the spaces contained undefinable protoplasmic debris, but here and there were collections of approximately cuboidal cells, which stained readily with haematoxylin and presented a markedly granular appearance.

If degeneration had fallen lightly upon the inter-acinous fibrous tissue and heavily upon the true glandular cells the result might well correspond to pancreas in a state of extensive maceration. If this small body be pancreas it can only be the dorsal pancreas and the larger must be the ventral element despite other considerations.

The artery shown in Figure IV certainly supplies the small intestine and equally certainly the vein receives its venous drainage, therefore these vessels must be called the superior mesenteric artery and vein notwithstanding their unusual relations to each other and to the pancreas.

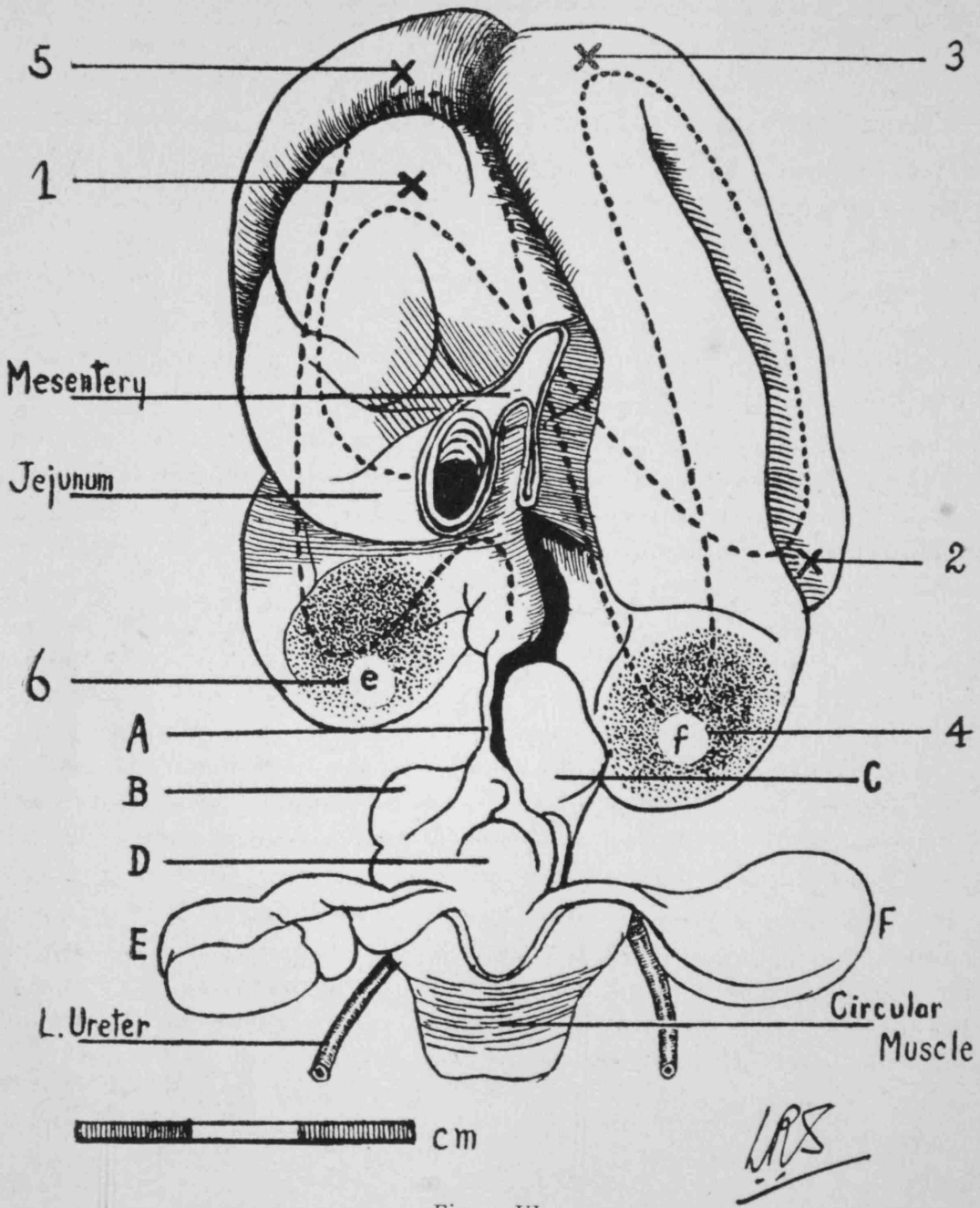


Figure VI.

This is a drawing of the small intestine and the pelvic viscera removed en bloc and viewed from the back.

The small intestine has been cut high up as shown in Figure V. It will be noted that the mesentery is very small and limited in its attachments.

The course of the intestine is shown by a heavy dotted line superimposed on the outline. In all the small intestine makes six flexures numbered 1 to 6 in this drawing. The general tendency of the intestine is to lie in longitudinal loops.

Most of these flexures are shown in anterior view in Figure III.

The stretch of intestine 2-3 has been moved a little to the right to show its anterior position, and this accounts for a slight difference between this drawing and Figure III.

Below flexure 6 the intestine narrows, first into a small globular portion and then into a fibrous cord distinguished as A. Below A and lying mostly in the pelvis is a complicated structure presenting diverticula at B and C, lying respecively left and right, at D in the middle adn at E and F more remotely left and right.

There is no mesentery to the gut other than that indicated, nor to A; the pelvic mass is also without mesentery. E and F lie in the posterior abdominal wall and have peritoneum only on their anterior and superior surfaces. E made a contact with flexure 6 at e, and F with 4 at f, both through the peritoneal cavity.

The ureters are shown below and also some circular muscle fibres.

The scale below is in centimetres.

VII. THE SMALL INTESTINE.

Figure III, which is an anterior view, shows that the small intestine is short and of wide calibre. In general it lies in a series of loops in the longitudinal axis, with a corresponding number of flexures numbered 2 to 6.

Figure VI shows the intestine removed *en bloc* and viewed from the back. The course of the intestine is shown by a heavy dotted line and may be followed from the commencement of the jejunum to its termination in an impervious cord.

The small intestine was cut just distal to the place resembling the duodeno-jejunal angle; from here the small gut has an upward course to Flexure 1, which is the most posterior part of the small gut. Flexure 1 is hidden from the front and so is not seen in Figure III; the course of the gut from here is downwards and forwards to Flexure 2, which appears in Figure III. The stretch 2-3 makes contact with the great omentum and the inferior edge of the liver. In making the drawing shown in Figure VI, 2-3 has been drawn a little to the right in order to show its position anterior to 3-4. In this respect there is some lack of correspondence between Figures III and VI. The stretch 3-4 is attached by a narrow medially placed mesentery near Flexure 1; its general direction is nearly vertical behind 2-3, but it is placed directly against the structures of the posterior abdominal wall. The largest and longest stretch 4-5 is anterior throughout, is parallel to 2-3 and at 5 attains the left side and makes a contact with the spleen. Below this point 5-6 is separated from the posterior abdominal wall by Flexure 1 and the commencement of the jejunum and the mesentery. Below 6 the gut becomes much narrower. A portion of about 5 mm. length lies in the mid-line, below the root of the mesentery, and then gives place to a fibrous cord which connects with the irregular lobulated mass which lies in the pelvis.

The description of the pelvic structures will follow but it may be well to state that they also are shown in outline in Figure VI. The structures E and F have been turned downwards and backwards in making the drawing. In fact "E" made a contact on the back of flexure 6 at "e," and "F" on the back of 4 at "f," both structures being separated from their gut contacts by the potential space of the peritoneal cavity.

The termination of the small gut, the fibrous cord and the pelvic mass are all enveloped in peritoneum but lack a mesentery. The main median pelvic mass is extra-peritoneal in its anterior attachment and the structures E and F in their posterior attachments.

The whole of the small intestine is dilated but is very far from being filled; in no place is the wall "ballooned" and thin as one may meet it in intestinal obstruction.

The interior is normal in the arrangement of the mucosa in circular folds.

VIII. THE BLOOD VESSELS OF THE ABDOMINAL VISCERA.

Figure IV shows the common iliac arteries emerging from behind the pancreas and thus is indicated a very high division of the abdominal aorta. The length of the whole abdominal aorta is only about 2 cm., but it gives rise to three median ventral and three lateral pairs of arteries, which are therefore crowded in the present of such shortening.

The artery which supplies the small intestine arises 2 mm. above the bifurcation. It has a strong downward slope behind the pancreas and if only on account of its distribution, must be called superior mesenteric. At the same level the two renal arteries arise from the aorta, and at a very slightly higher level the two testicular arteries.

A pair of suprarenal arteries arise 1 cm. above the bifurcation.

The hepatic artery arises in the mid-ventral line 1.7 cm. above the bifurcation. This artery besides supplying the liver gives off the splenic and the gastro-duodenal vessels.

A median vessel arises 2.2 cm. above the bifurcation in a position which is really in the posterior mediastinum. This vessel lies in the areolar tissue around the lower end of the oesophagus and gives branches to the lesser curvature of the stomach, the diaphragm and the suprarenal glands. This artery is probably to be identified with the coeliac and its high position is associated with the peculiar arrangement of the stomach which lies partly in the thorax.

Figure IV shows a fold of peritoneum "F" which stretches from the great omentum, between the tail of the pancreas and the spleen, to the greater curvature of the stomach and encloses a small fossa. This fold "F" contains gastric branches of the splenic artery. No left gastro-epiploic artery was found.

Figure VII

This figure presents three sketches of the pelvic viscera. The top left shows the pelvic viscera from the right lateral aspect, the top right from the left lateral aspect and the lower sketch shows the pelvic visceral mass opened in the mid-dorsal line, spread out and viewed from the dorsum.

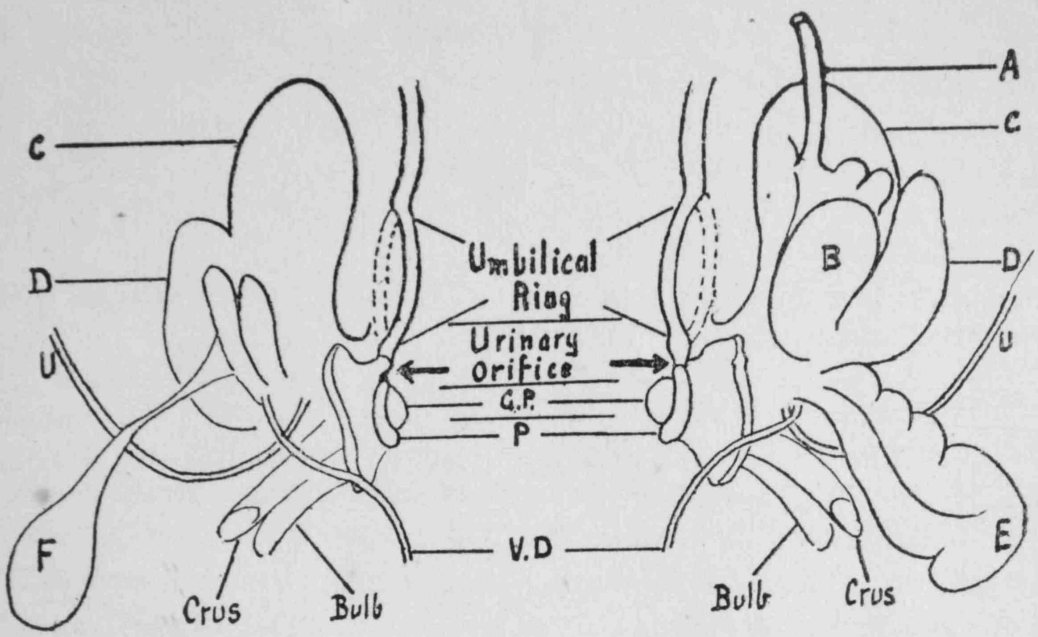
The annotations in the three drawings are the same as in Figure VI.

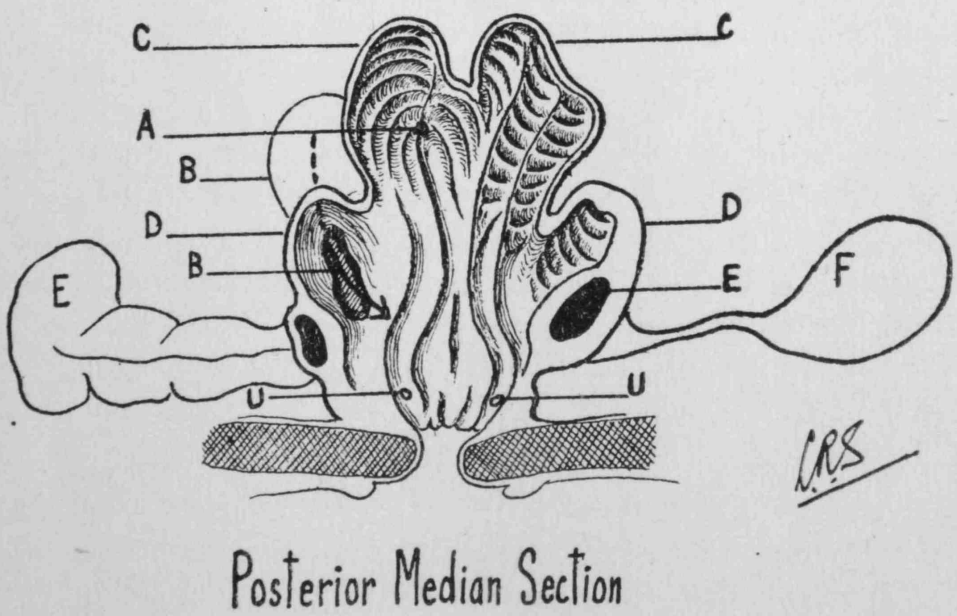
Thus: "A" is the termination of the small intestine, "B", "C", "D" are lobules of the central mass; "E" and "F" are more distantly attached. In addition are shown the position of the fibrous ring of the umbilicus and the urinary orifice.

G.F = glans penis. P = prepuce. U = ureter. V.D = vas deferens.

In other respects it is hoped the sketches are self explanatory.

The lower drawing shows the appearance from the dorsum after section in the mid-dorsal line. The interior contains a few prominent longitudinal ridges and small transverse ridges. D is a posterior diverticulum of the main (cloacal) cavity but communicates freely with it.





(*Continued*)

Figure VII.

E lies in the posterior wall of D, and crosses the mid-line to end blindly on the right side. Thus E lies transversely but is completely shut off from the main cavity.

B opens into the main cavity by a large opening. A small dimple A marks the place where the blind end of the small intestine was attached.

The two ureteric orifices are marked U.

F has no opening into the cloaca and leaves no trace of the place of its attachment in the interior.

At the foot of the rdawing is shown in criss-cross lining the bulb or corpus spongiosum.

There is no inferior mesenteric artery and the pelvic viscera are entirely supplied from the internal iliac.

The position of the superior mesenteric vein has already received comment in Section VI. The portal vein is normal in position and receives normal tributaries except for the inferior mesenteric vein which is absent.

Except for a high commencement the inferior vena cava presents no abnormalities.

IX. THE PELVIC VISCERA.

The reader will have seen in Figure VI the complicated lobulated structure which occupies the pelvis and whose nature must be indicated by such facts as these—

(*a*) It receives the termination of the small intestine such as it is.

(*b*) It receives the ureters.

(*c*) It is in communication with the testes by means of the vasa deferentia.

Therefore, the nature of this structure must be at the same time intestinal, urinary and genital. In general terms, there is a median lobulated mass with two prominent lateral offshoots, which give to whole a multilobular appearance. This arrangement may justify the use in the heading of the plural rather than of the singular "viscus."

The mass has a posterior lobule which is marked D in Figure IV. On either side are lobules B and C, lying respectively on the left and on the right. Of these C is considerably the larger.

The front of the median mass is in contact with the anterior abdominal wall, but separated by peritoneum above the lower edge of the umbilical ring.

In fact, B, C and most of D lie behind the fibrous annulus of the umbilicus, the lower edge of which is at just the level of the indication line D in Figure VI.

A is completely invested with peritoneum; so are B, C and D on their lateral and posterior surfaces.

E on the left side, by its sacculations and a longitudinal stria gives some suggestion of the form of the large intestine. F on the right side differs from E in that its communication with the median mass is a tenuous cord seemingly empty and devoid of a lumen. The globular end of F is hollow, thin walled and entirely closed.

Figure VI shows E and F after having been drawn backwards and downwards in order to give a better view of the other pelvic viscera. E and F are, in fact, attached to the posterior wall of the abdominal cavity, and were invested by peritoneum on their anterior surfaces only.

Figure VII presents three sketches of the pelvic viscera viewed from the right, from the left and also from the dorsum after having been opened in the sagittal plane.

The upper left drawing presents the appearances from the right. The glans penis with the prepuce is represented lying just below the fibrous ring of the umbilicus. Separated from the anterior abdominal wall by a part of the peritoneal cavity is the prominent structure C.

There is a strong but diffuse fibrous connection between the median mass and the lower part of the umbilical ring, but no real urachus. It may be again remarked that the two umbilical (i.e. placental) arteries and the single vein were present and normal, except in the proportional length of the arteries.

Behind C and on the right are a few lobules which lie in the long axis. It is to the most posterior of these lobules that the impervious stalk of F has an attachment. From the base of the same lobule the vas deferens arises, but makes no discoverable entry into the internal cavity of the visceral mass.

The right ureter ends its course from the posterior abdominal wall by passing below the stalk of F, thence below the proximal end of the vas deferens to enter the pelvic mass a short distance in front of the vas.

The upper right sketch presents the arrangements of the same structures viewed from the left. Many of the structures indicated in this sketch are the same as in the foregoing and bear like annotations. B is a sacculation on the left of C and intermediate in size between C and D.

In this sketch we see A, the termination of the small intestine, attached to the left side of the larger sacculation C above the level of B. Posterior to B and C, D is partly separated by a deep transverse fissure. To the base of D stretching laterally is E, which, as we have remarked, presents some of the superficial characters of the large intestine. Below B and anterior to the attachment of E are the terminations of the left ureter and of the left vas deferens. These two structures, though bearing a normal relation to each other some little way to the side of the pelvic mass, lie so very close to each other at their pelvic end that it seems that indeed they have a common opening. Certainly only a single opening into the median viscus is to be found.

The lower drawing in Figure VII presents the features of the greatest interest. The hollow pelvic mass has been split in the mid-dorsal plane as far as its anterior wall; the two halves have been thrust apart and the interior is viewed from the dorsum. The right of the drawing, as the reader views it, is the right of the specimen, and the left of the drawing is the left.

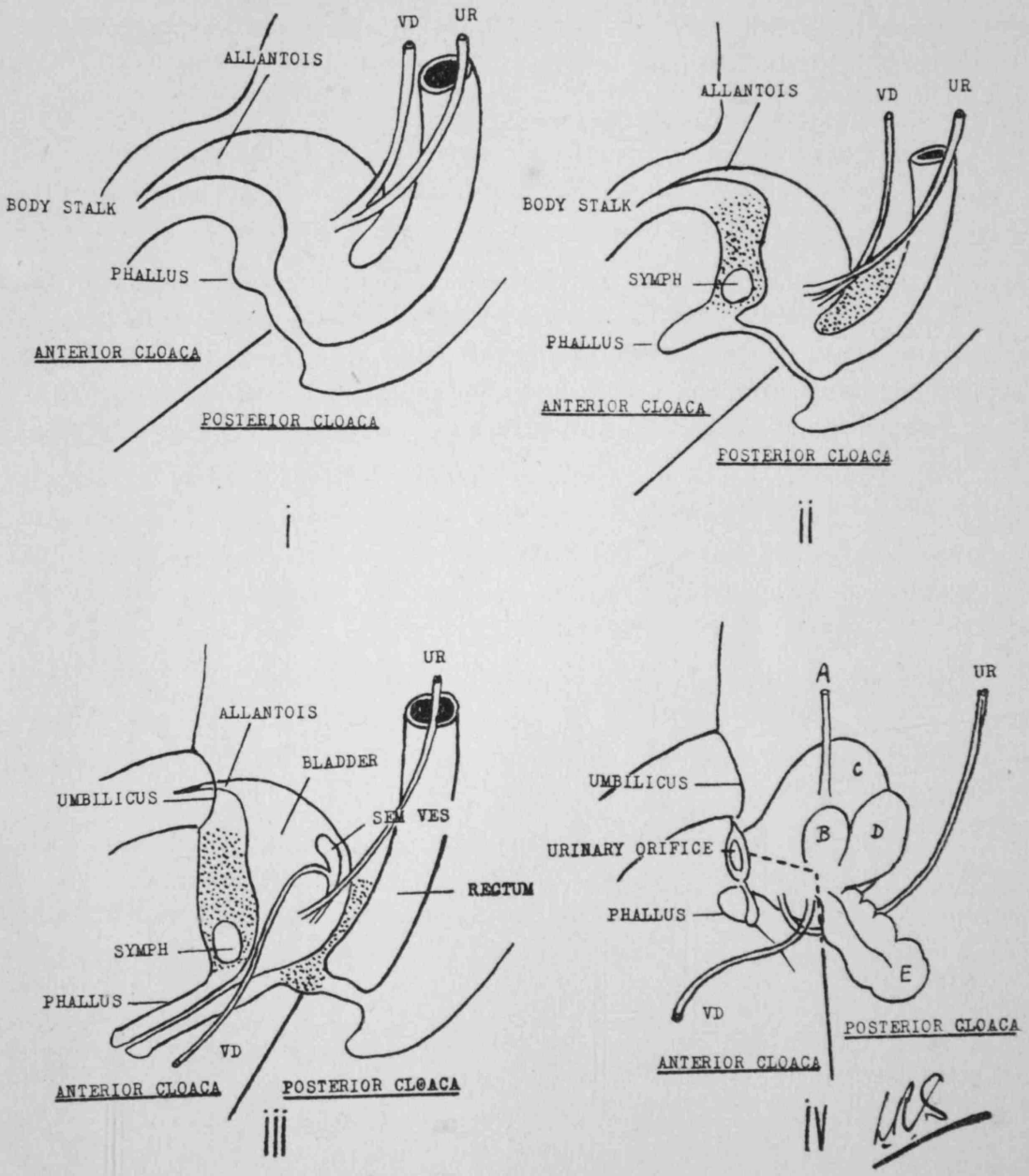


Figure VIII.

Four sketch diagrams which illustrate in outline the development of the cloaca. Very early stages are shown in I and II. In III the cloaca is divided in a normal manner into an anterior genito-urinary part and posterior intestinal part or rectum. Sketch IV reproduces the features of one of the sketches in Fig: VII. The stippled areas in II and III are those which have failed to develop in our specimen IV.

The subdivision of the cloaca is represented in all these sketches. Anterior Cloaca and Posterior Cloaca lie at the foot of each sketch in front and behind an indicating line. The first represents the genito-urinary part of the cloaca and the second the intestinal part.

The interpretation of these in IV rests on histological findings and are contained in the text.

UR = ureter. V.D = Vas Deferens SYMPH = symphysis Pubis.

This dissection has done much to clear up the nature of the structure E. It now appears that the cavity of E is partly contained in the posterior wall of D, where it crosses the mid-line and ends blindly on the right. A very interesting feature revealed, however, is this; E is entirely closed off from the cavity of the rest of the hollow pelvic viscus. It may perhaps be suitable at this stage to state that microscopic section has provided satisfactory evidence that E has the structure of the large intestine. It might not be an incorrect usage of terms to designatic the whole pelvic mass the "cloaca." The interior of C and of the cloaca immediately above the urinary orifice is marked by prominent longitudinal ridges. Secondary ridges lie transversely for the most part between the more prominent longitudinal ridges, and are shown in the drawing in a diagrammatic fashion; they are far more regular than the ridges ordinarily found in the interior of the bladder.

The ureteric orifices lie between the most posterior of the longitudinal ridges and the partition of the cloacal wall which cuts off E.

Histological methods were applied to the other cloacal adnexae with varying success. In most places the material, and especially the epithelium, was very poorly preserved. The nature of F is not to be stated with any assurance whatever. When opened, F presented a surface which seemed somewhat velvety, but microscopic section showed no more than a clearly defined longitudinal muscle layer, an outer circular muscle layer, an adventitial coat and no recognizable epithelium whatever.

The fibrous end of the small intestine can be seen in the interior of C in a dimple. From microscopic methods it is impossible to state more than this,—the structure of C is unlike bladder and like intestine. Perhaps this is to be expected in view of its connection with A, which is certainly intestinal. D was also subjected to microscopical examination; again intestine is suggested by the findings.

A portion of the cloaca just above the urinary orifice was removed for section. Here, no doubt by reason of better formalin penetration, the results are unequivocal. A section shows a skin-bladder junction with a quite typical transitional epithelium on one side. Here alone is typical bladder structure found.

Deficiency of the mesoderm is shown in its failure to subdivide the cloaca completely, into genito-urinary and intestinal parts, (see Fig. VIII).

It has been stated that the orifice in the anterior abdominal wall voided faeces and urine. If we are not mistaken, this can only be explained on the supposition that intestinal contents reached the cloaca before occlusions occurred and that the urine gradually washed out an accumulation.

It is to be doubted if the term "ectopia vesicae" should be applied in this case. If so, it is of the slightest degree, for though there is a skin-bladder connection at the top of the urinary orifice, the bladder epithelium is not extruded nor is visible from the surface.

The urethra is non-existent, but the urinary orifice is open on the dorsum of the rudimentary penis. To that extent the term "epispadias" is justified, though the position of the urinary orifice between the glans and the prepuce must be most unusual.

X. THE PERINEAL STRUCTURES AND THE TESTES.

The two upper sketches in Figure VII show a median bulb, which is also represented in the lower sketch, and lateral crura.

The perineum and the pelvic floor were not examined in very great detail, but these facts can be stated with confidence.

The crura are much smaller than is the bulb or the corpus spongiosum. Embryologists inform us that this structure is developed as a pair of structures which fuse in the mid-line around the urethra. In this case there is no urethra, and the corpus spongiosum, a prominent structure enough, lies below the urinary orifice and the cloaca.

If we accept current embryological teaching we would expect that the corpus spongiosum would either remain in its paired condition or fail to differentiate at all.

The divergent crura were connected by transverse muscle fibres which also enclosed the bulb. These muscle fibres must be identified with the superficial perineal muscles, undifferentiated. The circular muscle fibres shown in Figure VI are certainly on the headward or abdominal side of the bulb and the crura, and are most likely to be the representative of the levator ani muscle sheet.

The Testes.

The testes are both normally developed and fairly well preserved. A large pale cell with a distinct nucleus is very frequently found throughout the substance of both testes. The left testis which opens into the cloaca presents an extensive small round cell infiltration throughout. The other testis which has no discoverable opening has many fewer of these small round cells, which may perhaps be an indication of an ascending infection from the cloaca.

XI. THE THORACIC VISCERA.

Except for the lungs and the oesophagus, in the details of their structure, dissection of the thorax has revealed no abnormality of importance.

The pericardium, heart and aorta and main branches are all normal.

The lungs.

There are some patches of consolidation in the left lung and there is some adhesive exudate in the pleural cavity especially near the left apex. The bronchi are filled with a mucous secretion.

Broncho-pneumonia and bronchitis probably accounted for the rise of temperature and respiration rate noted on the fifth day of life.

Apart from this: the lungs present anatomical abnormalities. The left lung has a third lobe, apparently a subdivision of the upper lobe. The right lobe also has a subdivision of its upper lobe, and so has four lobes in all.

The Oesophagus.

The oesophagus presents a fusiform dilatation about 2 cm. in length just above the diaphragm. On opening it in its length, it was found that the lining membrane of the dilated part is thrown into prominent folds which resemble the longitudinal folds of the stomach and indeed merge into them without any break of continuity.

Above the dilatation the lining membrane changes its character and becomes smooth for a short space; above this again the appearance is that of the normal oesophagus.

It seems that the stomach has been to some extent included within the thorax, though no hernia of the diaphragm is present. Microscopic examination confirms this supposition. Sections typical of the oesophagus and of the cardiac end of the stomach were obtained from above the dilatation and from the dilatation itself.

The Heart and Great Vessels.

No abnormalities are to be reported other than a patent ductus arteriosus of small size, which in an infant of this age is hardly a matter for comment.

XII. THE SKELETON.

The outlines of the pelvis together with the femora and the lumbar part of the vertebral column, still for the most part in the cartilaginous state, are shown in Figure IX, in anterior and posterior views.

The Pelvis.

A very conspicuous feature of the pelvis, appreciable even before dissection, is its flattening in the transverse plane. So extreme is this flattening that the anterior superior spines and the crests of the two ilia lie in almost the same plane.

The pubic bones are separated by 1.9 cm. This gap is greater than the interval between the two ischial tuberosities, which is only .6 cm.

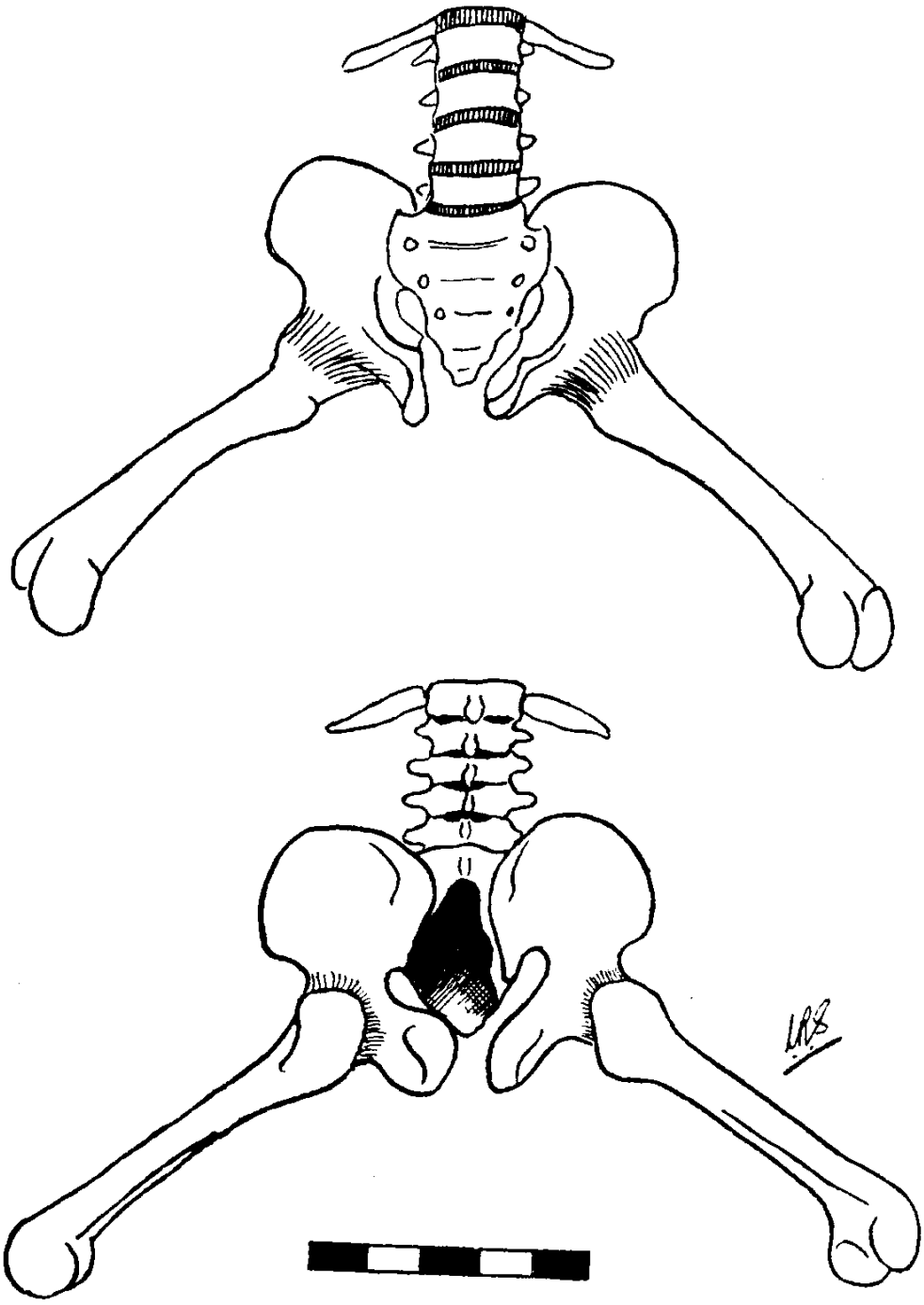


Figure IX.

In this figure are two drawings made to scale of the skeleton of the lumbar column, pelvis and femora. Above is the anterior view and below is the posterior.

It will be noted that there are only four lumbar vertebrae, i.e. the 24th vertebra has assumed sacral characters and articulates with the ilium.

The sacrum is asymmetrical, The right ala being larger than the left, the right sacro-iliac articulation is rather the larger of the two. There are only three anterior sacral foramina on either side and it seems therefore the sacrum is reduced to four elements. There are seven recognizable elements in the whole sacro-coccygeal skeleton, of which four must be called sacral and three coccygeal vertebrae.

The gap between the pubic bones is very conspicuous. Comparison of the upper drawing with the lower will show that the inter-pubic gap is actually greater than that between the two tuberosities of the ischia.

The lower, posterior, drawing shows that the dorsal arches of the vertebrae from the 24th downwards are lacking and a wide gap is present.

The scale is in centimetres.

The sacrum lacks its dorsal arch in its lower four constituents The dorsal spinous processes of the lumbar vertebrae below the third are decidedly under-developed. Dissection showed that the mid-dorsal gap was widest at the third sacral vertebra, where it measured 1 cm. transversely. The gap was covered in by a fibrous membrane which concealed a cauda equina which was normal in every respect.

The number of thoracic vertebrae is twelve, but the 24th vertebra, normally the fifth lumbar, enters into the sacro-iliac articulation. This condition is common enough and is best called "sacralization" of the 24th vertebra.

On both sides the lateral elements of two vertebrae only enter into the sacro-iliac joint. Both lateral alae of the sacrum are under-developed. The right is slightly larger than the left and presents three anterior sacral foramina; the left ala has only two foramina. On both sides these foramina are separated from the sacro-iliac joint by a skeletal bar which is decidedly undersized.

The Femora.

The femora have adopted a position of abduction and partial flexion. This was their position before preservation and hardening of the body.

It seems probable that this attitude has some relation to the flattening of the pelvis and to the inter-pubic interval. The body of a young infant, on hardening in "natural" position, far more commonly adopts the foetal attitude of flexion of the lower limbs than marked abduction.

XIII. GENERAL COMMENTS.

The abnormalities recorded in these notes would make a very formidable list if set out in tabular form.

The most significant features are to be found in the intestinal tract.

The intestine is occluded in three places; the small intestine is greatly shortened; the large intestine is almost completely suppressed, and the cloaca has failed in its normal differentiation.

From the outset, it must be inferred, the endodermal gut-forming material was insufficient for requirements; in other words, the organism lacked the wherewithal of essential endoderm for the development of an adequate gut and cloaca.

It is likely that non-development of the cloaca removes the stimulus to the local mesoderm to furnish the material for the genitalia, lower abdominal wall, perineum and symphysis pubis. Mesoderm in general is by no means lacking, to judge by the general development of the skeleton and of the muscular system, to take two examples.

If one could imagine whole body segments lost, as stitches are dropped in knitting, one could better understand the intestinal omissions. In the presence of such changes one might be prepared for gross disorder in the segmental arrangement of the body, either by elision or by non-differentiation and fusion, evidenced by mal-arrangements of the vertebral column or of the segmental nerves.

But, except for sacralization of the 24th vertebra there is little evidence of such events; and this condition is common enough apart from all visceral abnormality. The only suggestion of the sort is in the shortening of the aorta and the crowding of its branches.

Certainly no skeletal elements have been dropped. Organs much more liable to gross abnormality such as the stomach, spleen, liver and kidneys have no considerable defects. The testes are normally developed and this suggests that deficiency of the sex-gland hormone, if such there be at this early age, is not a primary cause of the whole train of events.

It is difficult to offer further comment on the internal viscera; the gross effects of misdirected growth stand obvious before us and no hint of causation has come to light.

The lower part of the intestine, though occluded, joins the anterior and superior part of the cloaca, while the posterior and inferior part has undergone traceable transformation into intestine. A small part only near the urinary orifice, lacking definite boundaries, has taken on the characters of bladder in the formation of a urinary epithelium (see Fig. VIII).

It is obvious that no surgical measure could have been of avail. The good judgment that refrained from interference is to be applauded. Had laparotomy been performed it is possible that ileostomy, or perhaps duodeno-jejunostomy, might have been attempted and either procedure would have been foredoomed to failure.

XIV. SUMMARY.

This communication contains notes on a male child who died on the 8th day after birth.

From the exterior the most notable abnormalities include imperforate anus, shortening of the subumbilical part of the anterior abdominal wall and underdevelopment of the external genitalia.

The gastro-intestinal tract is much reduced in length not only by shortening of the small intestine but also by suppression of the large intestine.

The intestinal tract is occluded in the duodenum and at the termination of the small intestine.

The cloaca is scarcely differentiated at all into genito-urinary and intestinal portions. The non-differentiated cloaca opens to the exterior by an anterior sub-umbilical orifice, there being no rectum, anal canal or anus.

There is a gap between the pubic bones in the place of the symphysis. The sacrum is underdeveloped in its alae, in the sacroiliac articulation, and in the dorsum.

Abnormalities are found in the duodenum and the pancreas. The lungs are excessively lobulated and the cardiac end of the stomach occupies in part the position of the thoracic oesophagus.

The arrangement of the main arteries and of other structures is highly unusual in some respects.

XV. ACKNOWLEDGEMENTS.

I am greatly indebted to Professor K. H. Digby who allowed me to examine a case of such interest.

My thanks are also due to others who have given me help during the investigation.

Dr. E. Q. Lim of the Depratment of Physiology, kindly examined and reported on the intestinal contents.

Dr. A. L. Tsai, of the Department fo Anatomy, has given invaluable help in the preparation of many histological specimens. Such information as I am able to give as to microscopic structure and identification of certain organs rests upon this work, but the information is scanty in proportion to the labour ungrudgingly spent on unsatisfactory material.

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THE INTRADERMAL TUBERCULIN TEST IN CHINESE,

by

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INTRODUCTION.

Although tuberculosis is rampant in Hong Kong no statistics are available regarding the incidence of the disease in the child population as a whole. This investigation was undertaken in an effort to determine wheher there is the same uniform increase in the incidence of positive tuberculin reactions in growing children in Hong Kong as there is in other countries.

We employed the intradermal method of Mantoux and worked with a 1/10,000 dilution of Koch's old tuberculin. The subjects of the investigation were chosen at random from the childrens' ward, the medical wards and the nursing staff of the Queen Mary Hospital, and all were Chinese.

METHOD USED AND CRITERIA OF A POSITIVE REACTION.

0.1 c.c. of a 1/10,000 dilution of Koch's old tuberculin was injected intradermally. The reaction, if any, was read 24, 48 and 72 hours later. A positive reaction is indicated by the development 24-48 hours after injection of an area of erythema with a central oedematous zone surrounded by a slightly indurated margin. The area of ervthema should measure at least 1 cm. in diameter and care should be taken not to overlook the possibility of a delayed positive reaction.

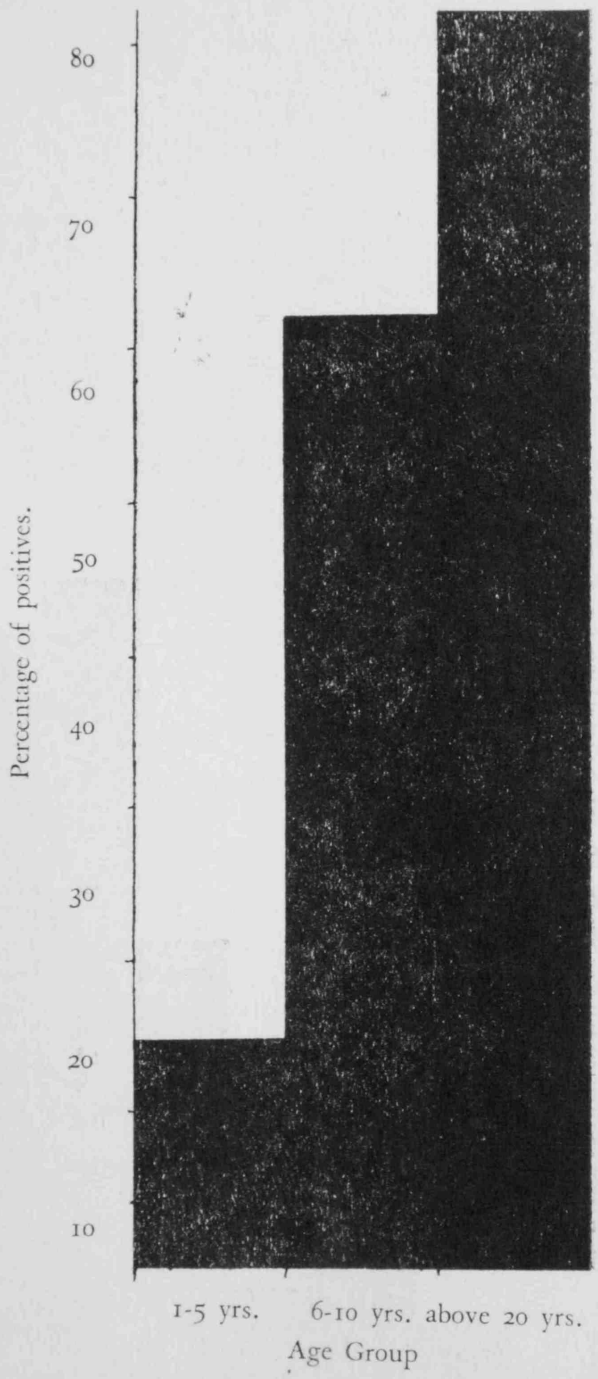
RESULTS.

69 children, 23 adults and 94 nurses were the subjects of the investigation. Our results show clearly that the number of positive Mantoux reactions increases pari passu with age. In the 1-5 year age group the incidence of positive reactions was 15%, in the 6-10 year group 62% and in the group over 20 years 82%. When the results for the first five years of life are examined year by year the same type of increase can be noted and is well shown in the appended bar diagrams.

Similar findings were obtained in Mantoux testing a group of of 94 nurses and dressers who were all in the 20-25 year group with three exceptions whose ages were 26, 26 and 30 respectively. The percentage incidence of positive Mantoux reactions in this group was 60.6%.

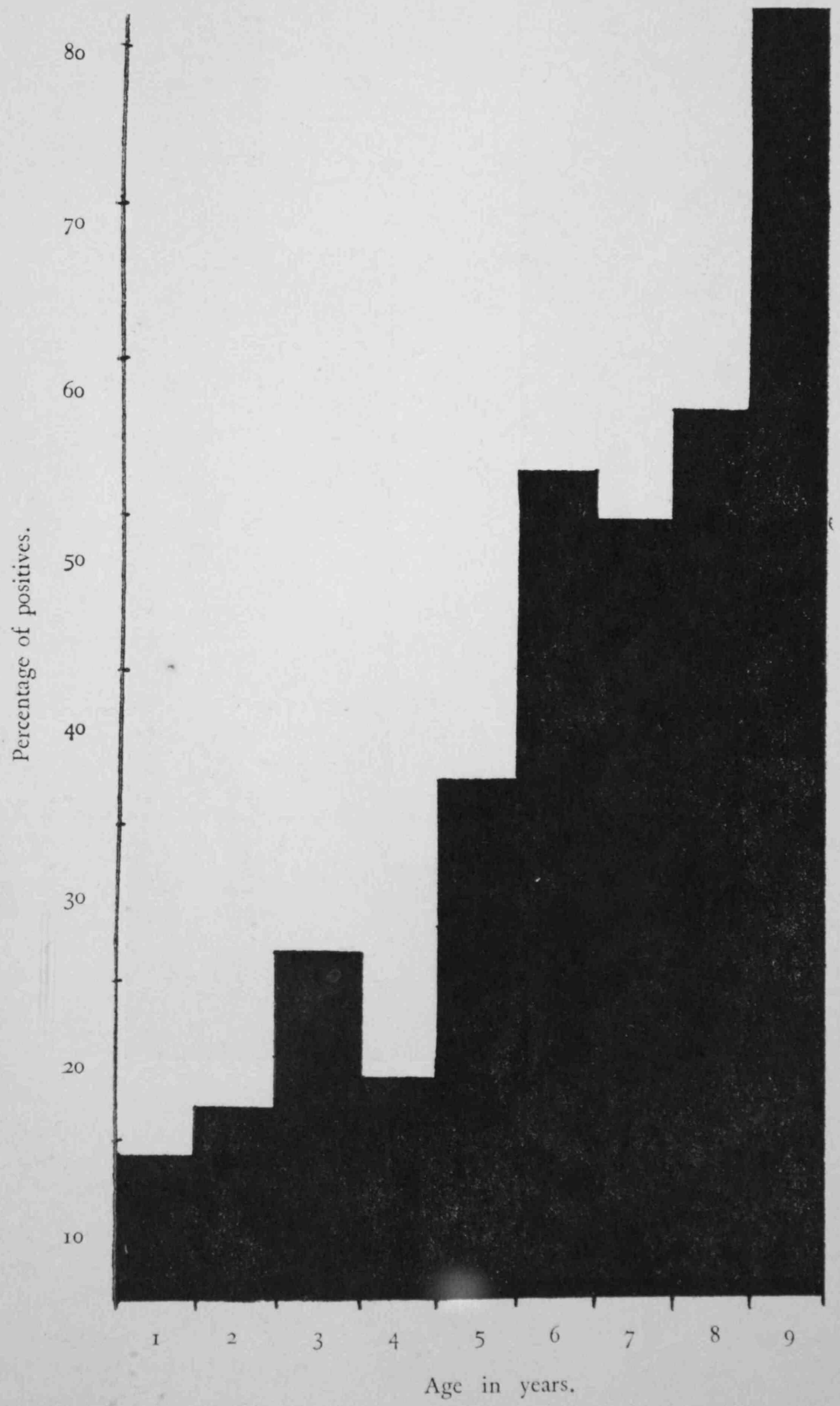
It is, therefore, obvious that in this extremely tuberculous community from 60-80% of those who reach the age of 20 have a positive Mantoux reaction. This simply means that these positive reactors

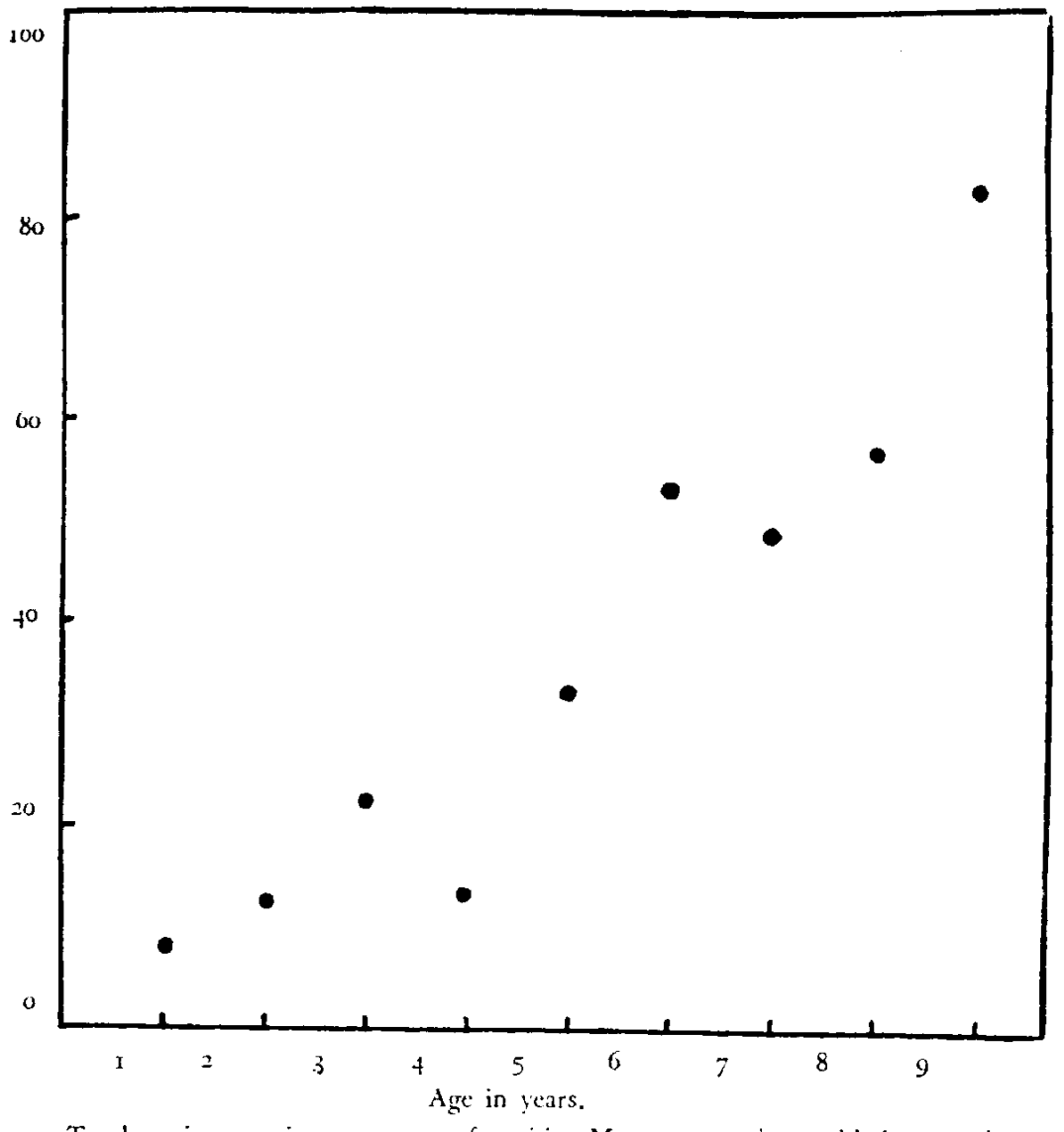
PERCENTAGE POSITIVE MANTOUX REACTIONS.



have a tubercular focus somewhere in their body. Naturally the younger the patient the more likely the focus is to be an active one, for there will have been less time for healing. But by the time the age of twenty has been reached healing will almost certainly have occurred and the lesion will probably be either latent or obsolete. It follows from these considerations that all infants under two years who are Mantoux positive should be treated as actively tuberculous. It follows equally that Mantoux positive reactors in the 20-25 and 26-30 year age groups may be regarded as people who have a healed or

PERCENTAGE POSITIVE MANTOUX REACTIONS.





To show increase in percentage of positive Mantoux reactions with increase in age

a latent tubercular focus. Whether any special steps should be taken with regard to them is uncertain but it is abundantly clear that Mantoux testing young adult Chinese is a time-wasting procedure, for one may be certain before starting that the results will be 60-80% positive.

It is of interest to note that the incidence of adult positive reactions was 82% in the hospital classes but only 60.6% in the group of nurses who come from a better social stratum.

CONCLUSIONS.

1. Intradermal tuberculin tests carried out on an unselected group of forty Chinese children of the hospital class show that the incidence of positive reactions increases from 8% in the first year of life to 33% in the fifth.

2. Twenty-three adults of the hospital class aged 20 and over show an 82% incidence of positive reactions.

3. A group of ninety-four hospital nurses in the 20-30 year age group gave a 60.6% incidence of positive reactions.

4. It seems that the Mantoux test applied to young adults in Hong Kong is neither very informative nor of great practical value, and these conclusions agree with those obtained in many other countries.

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OSTEITIS DEFORMANS OR PAGET'S DISEASE OF BONE IN A BOY AGED 12,

by

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INTRODUCTION.

Paget first described this disease in 1876. He wrote three papers on it and collected twenty-three cases, all his patients with one exception being over 40 years of age at onset. He himself thought, and the view was maintained until as late as 1915, that osteitis deformans was a rare disease, and Osler stated in 1912 that only 1 in 10,000 hospital patients suffered from it. The introduction of routine skiagraphy into the investigation of diseases of bone has enormously extended our knowledge of the condition, and it is now recognised that osteitis deformans far from being a rarity is probably one of the commonest of chronic diseases of bone. It is also recognised nowadays that the disease may occur very much earlier in life than was thought at first, and it is the purpose of this note to describe an undoubted case of the disease in a Chinese boy of 12.

As his history shows, the condition began to manifest itself at the age of 9, and has steadily and slowly progressed since then. The disease appears to be rare in Chinese, at any rate in South China, for no case has been recognised in the Surgical Unit during the last 20 years.

CASE HISTORY.

L. Y. aged 12, came to the out-patient department in February this year, complaining that for three years past the left side of his skull had been gradually growing larger. The enlargement first manifested itself in the left temporal region and in the face over the left zygomatic arch, but it was unaccompanied by pain or headache nor was any impairment of ocular movements or vision noted.

Four months ago he found he was becoming deaf in the left ear and this deafness was progressive. There had been no tinnitus.

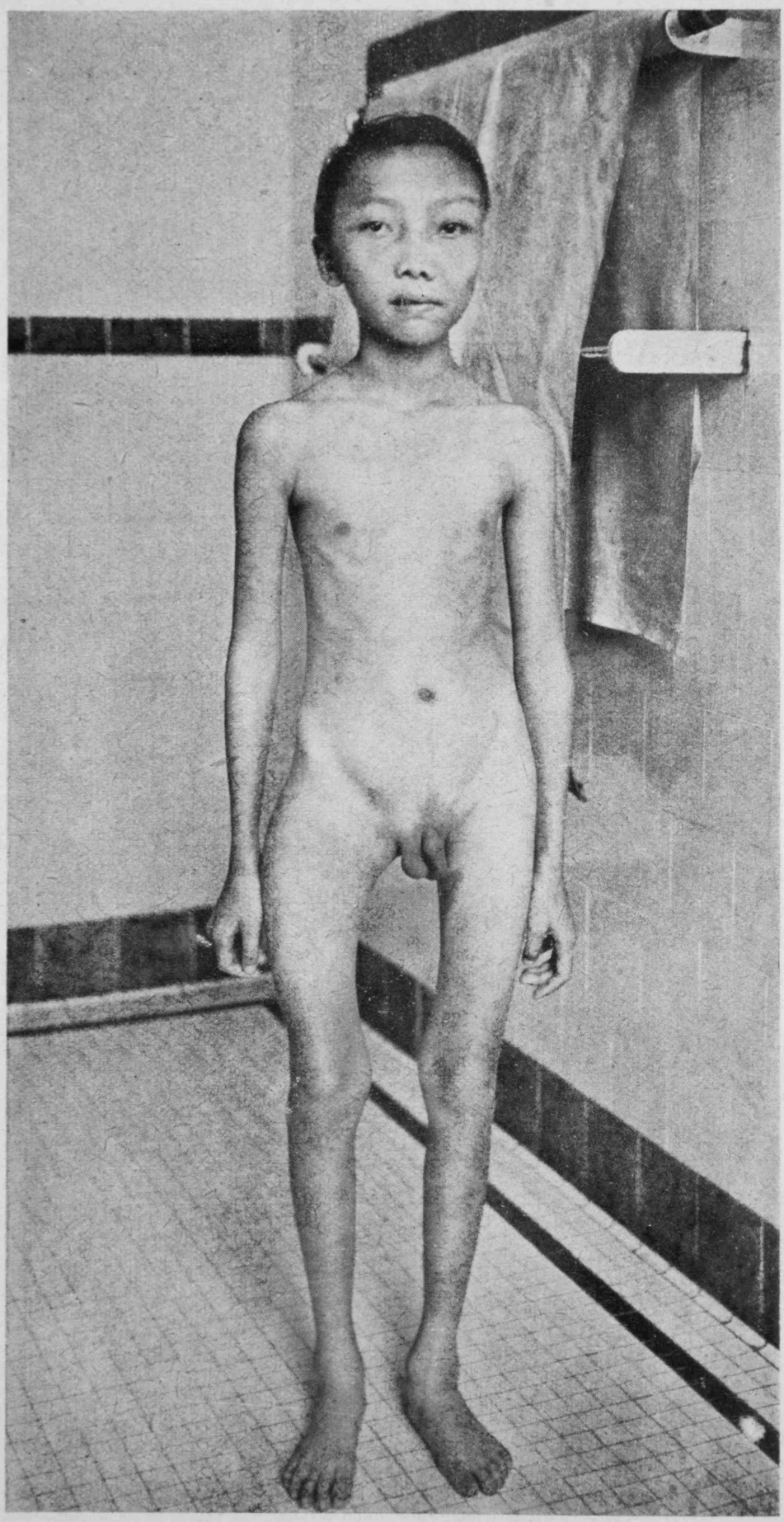
He also stated that during the last three months he had had occasional dull aching pain in the right thigh and he had noticed that it was changing in shape. The pain was intermittent in character and had never been severe enough to keep him awake. The deformity had advanced far enough to make his gait limping and the upper third of the right thigh was obviously bowed outwards

and forwards. He complained of no eye symptoms and no loss of sight. He had had no fits nor had any mental change been noted. There was nothing of significance in the child's past history, and his mother said that no other member of the family was similarly afflicted. There was nothing in the family history to suggest a high incidence of familial tallness, obesity or diabetes mellitus.

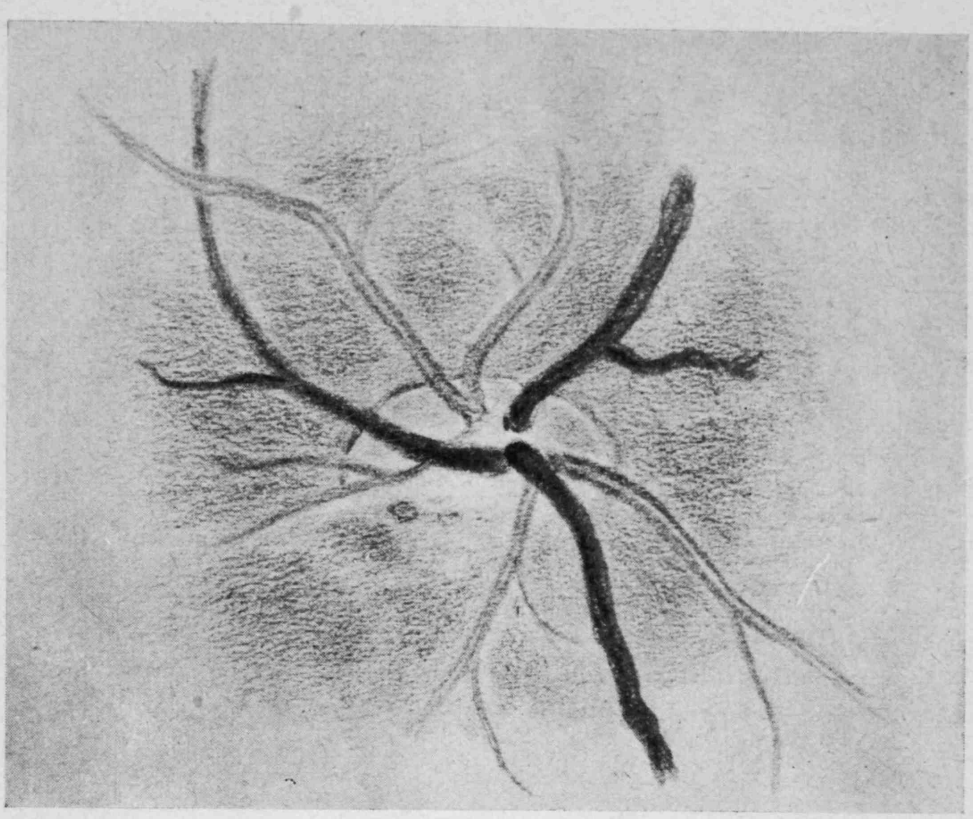
On examination it was apparent that the boy was slightly deaf and his manner was thought to be a little simple and childish for his age. His head was grossly asymmetrical, the asymmetry being due mainly to hyperostosis of the left frontal bone. The enlargement was smooth and regular. Measurement of the skull showed that the horizontal distance from nasion to inion was 10½" on the right side and 12½" on the left. The distance between the medial canthi was 1-7/8", the average for a Chinese child of his age being 1¼—1½". His face was trapezoid in outline and obviously broader than normal. The palpebral apertures were equal but there was was a slight tendency to left sided proptosis.

The arms and shoulder girdles appeared to be normal and no asymmetry or change was noted in the clavicle, ribs or vertebral column. A pigmented area 4" × 2" was noted on the medial aspect of the right thigh just below the inner end of Poupart's ligament. The skin in this area was café au lait in colour and slightly thickened, and part of the patch is shown in the photograph of the child. The right femur was bowed outward and forward in its upper third, a change well shown in the photograph, and as a result of this the pelvis was tilted and the gait was limping. The left leg was bigger throughout than the right, the difference being apparently due to slight generalised disuse atrophy of the musculature of the right leg. Measurements showed shortening of the right thigh, the distance from anterior superior iliac process to adductor tubercle being 14½" on the right side, 15½" on the left. The circumference of the right leg (4" below the tibial tuberosity) was 9", that of the left 9½". There appeared to be an increase of local heat over the middle third of the right tibia, but no changes other than this could be seen or felt in the bone. The boy's respiratory, cardiovascular and alimentary systems presented no abnormalities on clinical examination. On examining his cranial nerves, the only abnormalities apart from the slight left sided proptosis were found in the optic and auditory nerves.

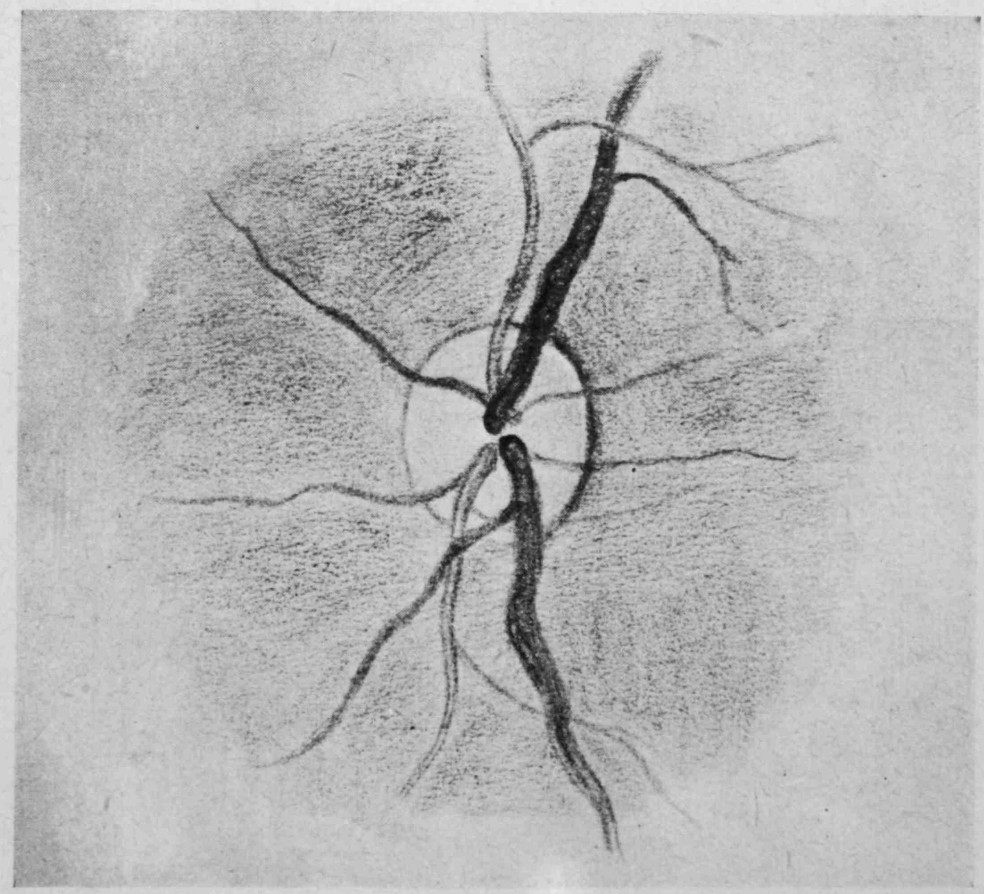
The nerve head on the left side was definitely atrophic, the disc being pale grey in colour. The right optic disc showed marked distortion. The normal circular or ovoid outline of the disc had disappeared and had been replaced by a flattened hemi-ellipsoid which was pearl grey in colour. The appearances, which are well shown in the attached sketches, suggested that the nerve head might be undergoing progressive compression in the vertical plane, a condition



To show the deformity of the left half of the skull and the right femur. Note the pigmented patch in the left groin.



RIGHT FUNDUS



LEFT FUNDUS

Sketches of the fundi made by Dr. S. Bard to show the flattening and deformation of the right optic disc.

Facing p 151.

which could be readily explained by assuming that the optic foramina were becoming constricted by deposition of new bone. Unfortunately the extreme density of the new bone made it impossible to obtain X-ray photographs of either optic foramen.

Visual acuity was 6/9 in the left eye, 6/12 in the right. The visual fields showed a slight degree of peripheral constriction in the outer two quadrants, but no central or paracentral scotomata could be demonstrated, nor was it possible to show any enlargement of the blind spots. The field charts are shown in the reproductions.

On examining the ears it was clear that the child was suffering from deafness which was more marked on the left side. Tests showed that he could hear "ordinary" conversation at 20″ on the right side, 2″ on the left, a watch at 24″ on the right, 6″ on the left, and whispering at 6″ on the right and 1″ on the left.

Bone conduction was greater than air conduction on both sides and greater on the right than on the left. The membrana tympani showed no gross abnormality, but the left one was partially obscured by the irregularity of the meatus, an irregularity presumably due to deposition of new bone. It was evident that there was a high degree of middle ear deafness on both sides with greatly exaggerated acuity of hearing through bone, and it is to be noted that bone conduction on both sides was very much greater than in the normal human being.

The area of cardiac dullness and the heart sounds were normal, and the blood pressure was 108/54. The electrocardiogram, however, showed some abnormal complexes in all three leads, one left ventricular extrasystole was seen in lead III and one auricular extrasystole in lead II. The P-R interval was within normal limits and there was no evidence of right or left axis deviation. S_2 and S_3 both showed flattening and a tendency to notching.

LABORATORY FINDINGS.

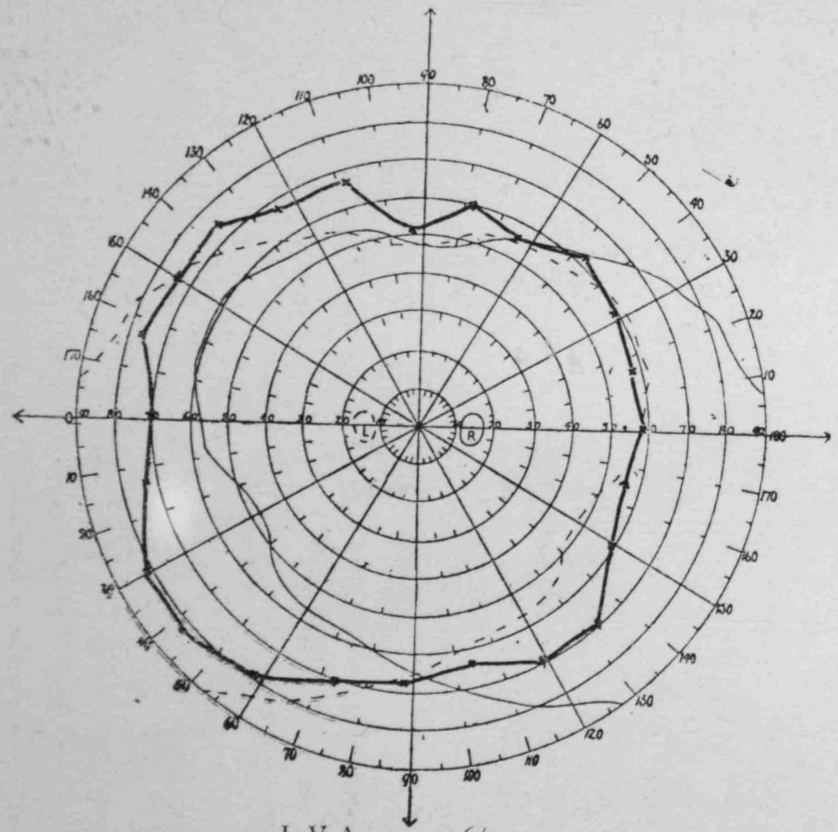
The biochemical investigations revealed no abnormalities other than an increase in the blood phosphatase, a change which is found constantly in osteitis deformans according to O'Reilly (1932).

The results of the various tests are appended in tabular form:—

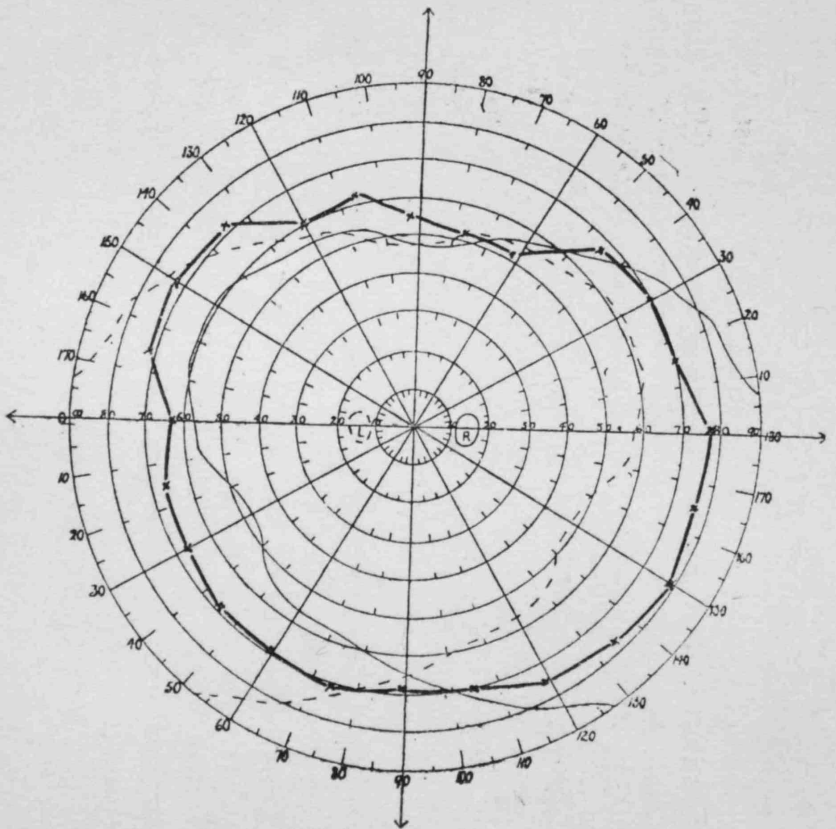
Blood urea	21	mgm%.
Blood cholesterol	1.30	mgm%.
Blood creatinine	1.37	mgm%.
Blood sugar (resting)	104.25	mgm%.
CO_2 combining power of plasma	54	Vol. %.
Blood pyruvic acid	0.78	mgm%.
Serum albumen	4.18	gm%.
Serum globulin	2.88	gm%.

VISUAL FIELDS

Left.



L.V.A. 6/9
Colour ..... White
Size ..... 2 mm.
Illumination ..... Daylight.



R.V.A. 6/12
Colour ..... White
Size ..... 2 mm.
Illumination ..... Daylight.

Right.

Serum calcium	8	mgm%.
Inorganic phosphorus	3.12	mgm%.
Blood phosphatase	6.1	blue units per c.c.
(Normal controls)	1.8	,, ,, ,,
	2.0	,, ,, ,,

The cerebrospinal fluid showed the following results on examination:—

Fluid clear and colourless.
Pressure 162 mm. of water.
Queckenstedt—immediate rise (on both sides).
Pandy.....negative.
Cells.....2 per cu. mm.
Sugar.....74 mgm%.
Chlorides.....707.7 mgm%.
Kahn.....negative.

The urine contained no sugar, albumen or Bence-Jones albumose.

The faeces contained no ova and showed no abnormalities.

X-RAY EXAMINATION.

Skiagrams were taken of the skull, the vertebral column, the thorax, the pelvis and the long bones of the limbs. The changes characteristic of the disease were best seen in the skull, the right femur and the right tibia. Early changes were also noted in the ilia. The vertebral column, thorax and arm bones appeared to be uninvolved.

The excessive absorption of bone which occurs in the early stages of Paget's disease leads to irregular osteoporosis, and it is at this stage that deformities appear. The new bone laid down later in the disease is described by the radiologists as being either "spongy" or "amorphous", the terms being merely descriptive and without pathological significance. In the "spongy" form the new bone is laid down in coarse striae which usually run in the direction of the normal lamellae; in the "amorphous" form the skiagram shows merely an opaque deposit. The two types frequently co-exist and do so in this patient.

The base of the skull in the anterior and middle fossae shows the generalised opaque deposit which characterises the "amorphous" type. The normal structure of the bones is replaced by a homogeneous shadow and where individual elements can be made out, such as the clinoid processes, they show marked thickening. The antero-posterior view of the skull shows clearly that this "amorphous" change has

progressed more on the left side than on the right and that it is most advanced in the left half of the anterior fossa.

In the occipital region, on the other hand, the "spongy" type of change with gross widening of the bone is apparent.

In the upper third of the right femur changes are conspicuous. The bone is bowed outwards and forwards and the neck has become almost horizontal. The diameter of the affected portion of the femur is increased, and irregular calcification has produced a slightly mottled appearance in the outer zone of the cortex. The affected area merges gradually into normal bone in the middle third of the femur.

The upper halves of the ilia on both sides also show irregular circular patches of mottling. In the middle third of the right tibia changes have occurred over an area about 4" long. In this region there is a fusiform swelling in which the cortex of the bone is thickened. Three small cracks can be made out running obliquely across the bone at the upper and lower ends of this swelling. It was impossible to obtain accurate pictures of the optic foramina as the "amorphous" type of change had progressed so far that the bone was completely opaque.

DISCUSSION.

Although our views about this disease have been much modified during the sixty odd years which have elapsed since Paget's description, we are still uncertain about the aetiology of the condition.

The earliest hypothesis put forward was that of an infective osteitis. Hutchinson, who championed this view, maintained that osteitis deformans was simply an infective osteitis and that the disease spread by infection from one bone to another.

Other observers claimed that micro-organisms could be isolated from the bones, but these claims have not been confirmed by later workers. There is equally little reliable evidence to support the view that the disease is a trophic condition secondary to changes in the postero-median columns of the spinal cord. Those who still support the toxi-infective hypothesis say that the histological changes found in the affected bones tally with those found in chronic inflammatory lesions, and attribute the origin of the toxins to some perversion of metabolism without, however, adducing any evidence to support their view. That there is some inherited tissue proclivity which predisposes to the disease seems likely as there are at least seven instances on record of the disease occurring in more than one member of the same family.

Recently there has been a growing tendency to link osteitis deformans with other bone diseases such as leontiasis ossea and osteitis fibrosa, and efforts have been made to prove the existence of some disorder of parathyroid function in the disease.

Lawford Knaggs writing in 1925-26 drew attention to certain histological similarities between osteitis deformans and osteitis fibrosa, and he suggested that a hypothetical toxin of metabolic or intestinal origin might produce either disease in a predisposed human being, the final issue being determined by the age of the patient. He maintained that in susceptible young people with insufficient resisting power osteitis fibrosa would develop, whereas susceptible individuals endowed with sufficient resisting power to stave off osteitis fibrosa in youth might succumb to osteitis deformans later in life when their resistance had been undermined by loss of vitality or disease. He supported his argument by saying that osteitis fibrosa was a disease of young people and that the subjects of osteitis deformans usually showed marked evidence of generalised arterial degeneration. Unfortunately one case of the sort described in this paper is almost enough to blow such a theory sky-high, and our case is not unique.

Hamburger and Nachlas in 1926 recorded a case which suggested to them that at any rate some cases of leontiasis ossea are in reality cases of Paget's disease of bone. In their patient, a woman of 28, the disease began with the alveolar bony changes characteristic of leontiasis ossea and progressed to involve the vault of the skull, the clavicles, vertebrae and femora. The bones involved later in the disease showed the X-ray changes typical of osteitis deformans.

Ivimey in 1929 reported an extraordinary case of bone dystrophy occurring in a boy of 11. The child's face had begun to enlarge when he was one year old, and by the age of 11 his right arm and right leg had also undergone enlargement. Both optic discs were atrophic and skiagrams of the skull showed marked hyperostosis of the facial bones, while the vault showed the changes characteristic of early Paget's disease. The skiagrams of the vertebrae and the long bones of the right limbs, on the other hand, suggested osteitis fibrosa cystica. The author leaves the aetiology of the condition in doubt but diffidently suggests that the case affords evidence in favour of a relationship between the three diseases, and she argues in favour of a transition of the type:—

Leontiasis ossea. ⟿ Osteitis fibrosa cystica. ⟿ Paget's disease of bone.

In any case, the term leontiasis ossea as it is used to-day certainly includes all three diseases, and doubtless many of the earlier cases of leontiasis ossea were actually young cases of Paget's disease.

Berman in 1932 put forward the view that the disease was due to a dysharmony between the functions of the parathyroids and the adrenal cortex. He stressed the well-known pathological sequence seen in osteitis deformans; excessive resorption and osteoporosis, osteoid formation, deposition of inorganic salts in osteoid and hyperostosis, and he suggested that in the early stages where osteoporosis

and softening were marked an excess of parathormone was being produced.

This parathyroid overfunction was, according to this view, followed by a stage in which hyperplasia of the adrenal cortex occurred with excessive production of cortin and a subsequent lowering of serum calcium with marked hypercalcification and hyperostosis. Again, the hypothesis is not supported by the facts for cases of osteitis deformans show no significant variations in serum calcium at any stage of the disease, nor have changes in the parathyroid glands been demonstrated in the few cases where an examination has been practicable.

O'Reilly and Race in 1922 published a detailed investigation into 22 cases of the disease. They agree with all recent observers in stating that the disease is commoner than is usually thought and they point out that it may be present for a long period without producing signs or symptoms. They found no constant association with arterial degeneration or hyperpiesis but confirmed the well-known association with osteosarcoma.

The most important feature of their paper was their detailed biochemical investigation of the disease. The only constant biochemical finding was an increase in the plasma phosphatase. The serum calcium and plasma phosphorus figures were normal in all cases, and there were no changes in the blood sedimentation rate or blood picture, apart from an occasional monocytosis. Nor has any significant abnormality been found in studies of the calcium balance. These writers state with some emphasis that there is no evidence of parathyroid involvement and that the only constant findings in the disease are the skiagraphic appearances and the increase of plasma phosphatase.

The facts which emerge from this brief survey of the views on the aetiology of osteitis deformans are few and depressing. The first is that quite clearly we do not know what the cause of the condition is, though we can say definitely that it is not inflammatory in origin. Second, the disease is obviously a constitutional one, although only one bone may be involved. Third, it is common to find the disease associated in elderly people with vascular degenerative changes, and it is also associated with osteosarcoma in a significantly high proportion of cases at this age period. Fourth, the disease is commoner and has a much wider age incidence than was thought at first. Fifth, there is no convincing evidence to show that the parathyroids are implicated in the aetiology of the condition or that it is due to a disorder of calcium metabolism. Nor is the disease related in any way to generalised osteitis fibrosa.

The skiagraphic appearances and the marked increase of the blood phosphatase show that our case is indisputably one of Paget's disease of bone. The most interesting features of the case are the involvement of the optic and auditory nerves. Optic atrophy and other eye changes have been recorded before. Paget himself described retinal haemorrhages in four of his cases, but it is almost certain that they were due to concomitant arterial degeneration.

Optic atrophy was recorded in two cases of the disease by Wyllie in 1923. His first case showed a paracentral scotoma and a constricted field on the right side. Visual acuity was reduced to 6/12 in both eyes and both discs were pale. Coupled with these changes was a right-sided impairment of hearing. Bone conduction was greater than air conduction as in our case. His second case showed bilateral constriction of the visual fields with bilateral temporal pallor of the optic discs and a reduction of vision to 6/24 in the right eye and 6/15 in the left. No scotomata were present and there was no impairment of hearing.

In the same paper Wyllie reported two cases of paraplegia supervening in the course of the disease as a result of cord compression brought about by progressive narrowing of the spinal canal. It is fairly obvious that the optic atrophy seen in this disease is due directly to compression of the optic nerve and that this compression is caused by progressive narrowing of the optic foramina. It is clear from the skiagrams that the whole base of the skull in our case has undergone marked hyperostosis and that this is enough to account for the ophthalmoscopic appearances and the reduction in vision.

Deafness has long been recognised as a symptom of Paget's disease of bone and in 1923 Jenkins drew a comparison between the changes found in the capsule of the labyrinth in otosclerosis and in osteitis deformans. In both conditions he found the predominant change to be osteoporosis, and in both diseases bone conduction is increased, as compared with the normal.

SUMMARY.

1. A description is given of Paget's disease of bone occurring in a Chinese boy of 12.
2. The onset at nine years of age, the bilateral optic atrophy and the bilateral deafness were striking features of the case.
3. The skiagraphic appearances were pathognomonic of Paget's disease, the bones most involved being the skull, the right tibia, the ilia and the lumbar and sacral vertebrae.
4. The one abnormal biochemical finding was an increase in the blood phosphatase.
5. The available literature has been briefly reviewed.

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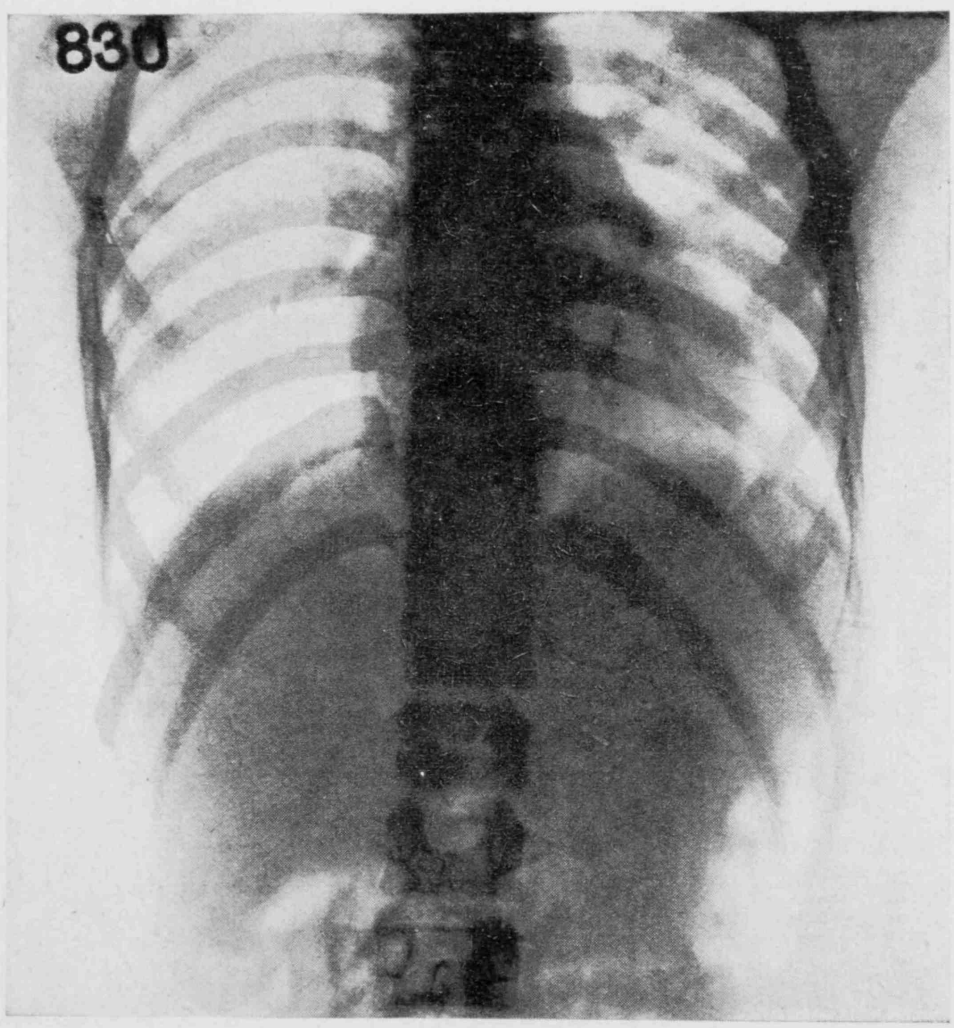
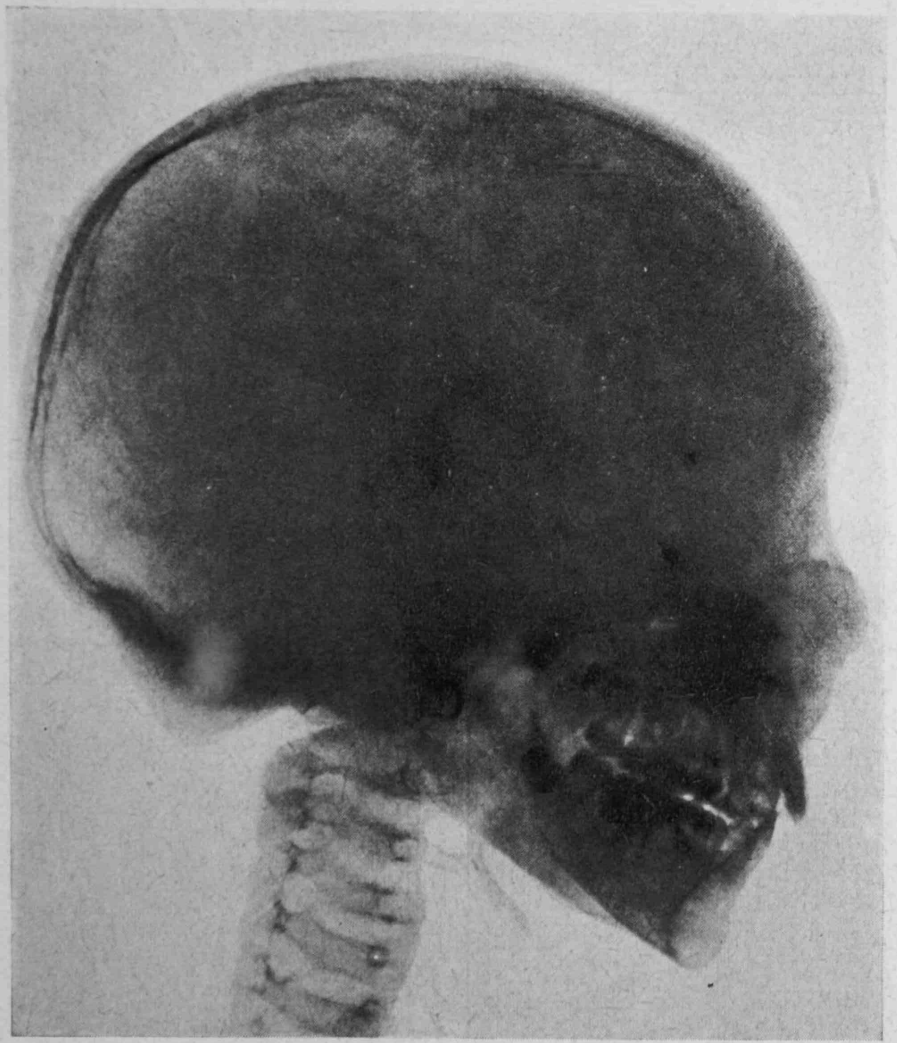
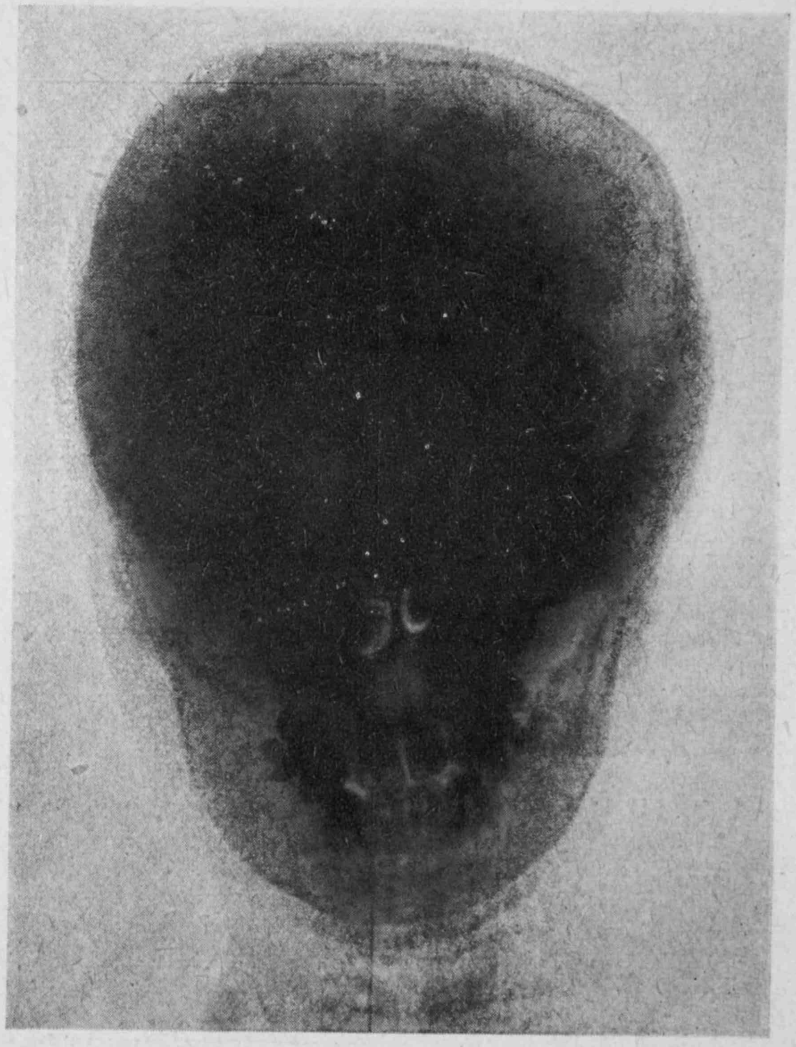


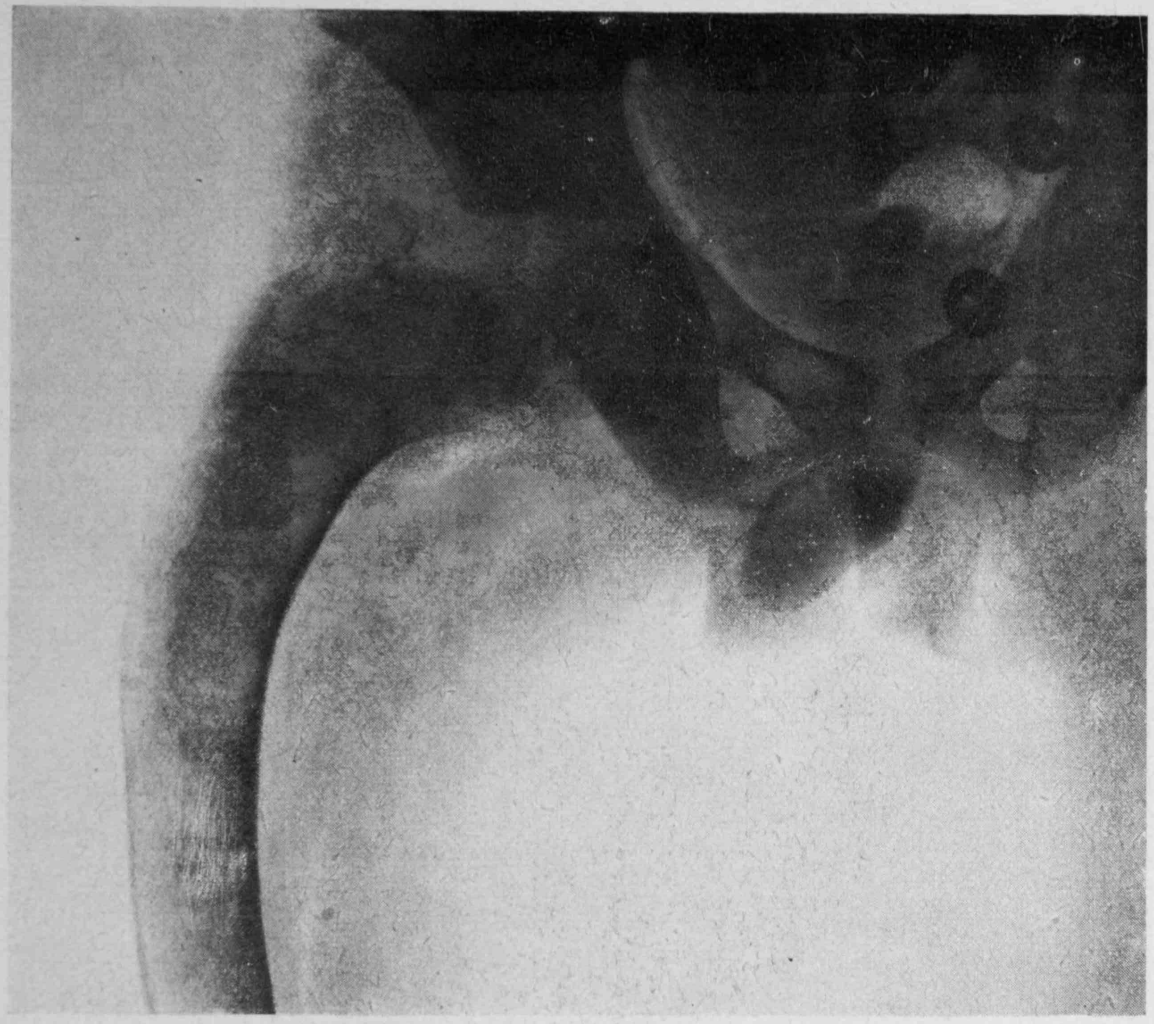
To show the normal thorax and vertebral column.



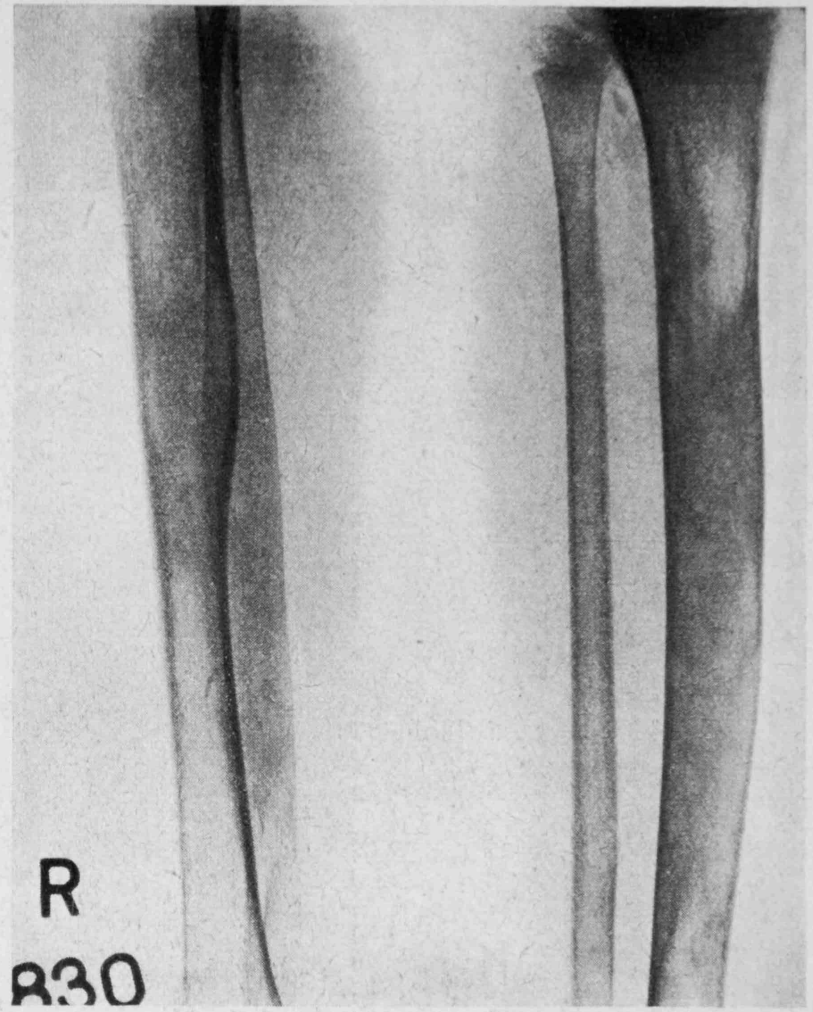
Lateral view of the skull showing the generalised opaque deposit in the middle midanterior fossae.



Antero-posterior view of the skull.



To show the changes in the upper third of the right femur. The neck of the femur runs almost horizontally.



To show the changes in the right tibia.

Acknowledgments.

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