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Further Studies in Adult Rickets (Osteomalacia) and Fœtal Rickets

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IN 1934 details of two osteomalacic pelves from China were given [1]. One of these came from a woman, a three para, who was aged 39, and in whom the active process was healed. The second was also from a multipara, aged 43, a six para in whom the disease was still active. We now add the details of a third specimen in which our previous work has again been confirmed, showing the close connexion which exists between late rickets, adult rickets, and fœtal rickets (figs. 1 and 2).

This pelvis is from a primipara in whom the disease was still active, and in whom, as an adolescent, the characteristics of a fully developed osteomalacic pelvis were already present. The fœtus also showed fœtal rickets.

Mrs. C. W. H., aged 18, a prostitute, Chinese, came to the Peiping Union Medical College Hospital on February 15, 1936. Her last monthly period was on June 12, 1935, and her expected date was therefore March 22, 1936. Labour pains had started on February 12, 1936, and she had been examined by an old-type midwife on February 13. After a very severe pain on February 14, labour pains had ceased, and the parts of the fœtus were clearly felt in the epigastrium.

She was in a state of collapse, with dyspnœa, and a hardly perceptible pulse. Free gas was present in the abdominal cavity.

Under local anæsthesia the abdomen was opened and a true Porro's operation was done, and the fœtus, placenta, and uterus removed, much free blood being found in the abdomen. The uterus had ruptured across the front of the cervix, well out into the left broad ligament, and the shoulder of the fœtus was caught in the rent. Gas was present in the broad ligament tissue.

She rallied somewhat, but in spite of transfusion, and glucose and saline, died seven hours later. Her pelvic measurements were as follows: Interspinous, 20 cm.; intercristal, 23 cm.; ext. conjugate, 18 cm.; interischial, 3 cm.; posterior sagittal, 8·5 cm.

For the previous three years, i.e. since the age of 15, she had complained of aching in the back and legs; and, for the last two years, the standing height had been noticeably diminishing. She was much dehydrated.

Blood-count: R.B.C. 4,650,000; Hb. 15·4 grm.; W.B.C. 6,850.

Blood-calcium, 8·56 mgm. per 100 c.c. of serum } (in spite of the dehydration).
Phosphorus 2·2 mgm. per 100 c.c. of serum }

Blood Wassermann positive.

A skiagram showed osteomalacia with a triradiate pelvis. The fœtus showed the signs of fœtal rickets. Its development corresponded to the thirty-fifth week of pregnancy. Its weight was 2,200 grm.

One interesting feature of the case is the age of the mother, and another is the clear signs of fœtal rickets at the 35th week of pregnancy.

Finally, to settle, from the clinical point of view, the question of the relation of adult to fœtal rickets, the following case, which we have carefully followed, presents features which fill in some of the lacunæ in our previous publications.

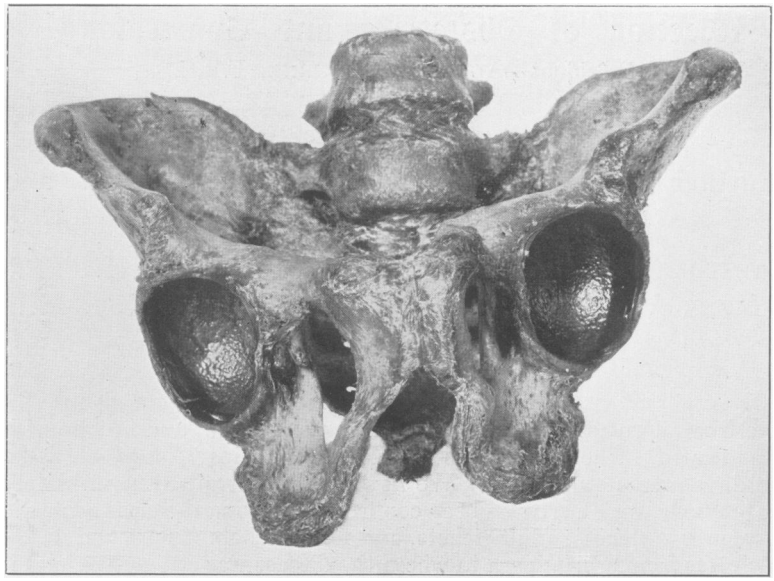


Fig. 1.

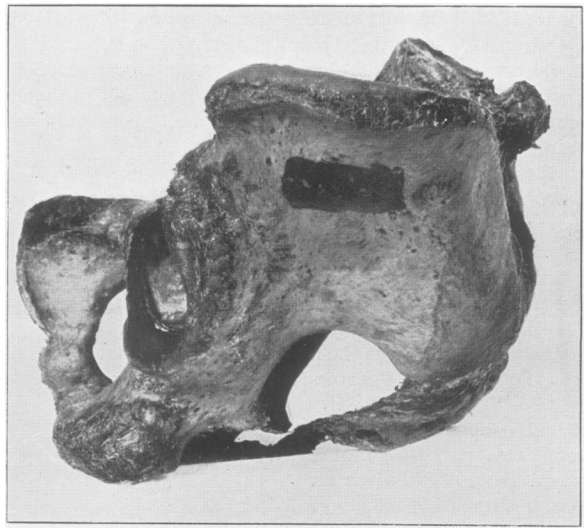


Fig. 2.

Mrs. T. L. F., aged 23, a Chinese housewife, came to the Obstetric Clinic of the Peiping Union Medical College on October 5, 1934, with a six-months' pregnancy. She was found to have a 4-plus-Wassermann reaction, was referred to the Syphilis Clinic, was properly treated with

neoarsphenamine, and without untoward reaction. The child, her third, was born at home at term and lived, but developed rickets.

She dropped out of sight, and on her next visit was admitted to hospital at or near term, on April 30, 1936. She was œdematous and had been suffering from tetany on and off for six months. Her history in detail was as follows :—

She had occasionally suffered from back and thigh pains since the age of 14, especially in the winter, with occasional numbness of the extremities. Her diet had consisted of white rice, wheat flour, and vegetables ; rarely meat, eggs, or animal fat.

She had had three children, the first did not walk till 4 years of age and had bent legs ; the second died of vomiting at the age of 40 days ; the third did not walk till 2 years of age and has bowlegs.

The patient had some albumin in her urine, but her blood-pressure was only 100 systolic, over 80 diastolic. She was spontaneously delivered of a living female baby weighing 2,450 grm., with marked fœtal rickets.

Her Wassermann and Kahn reactions were faintly positive ; the baby, however, showed no signs of syphilis, nor has she subsequently developed any sign of this disease.

On admission her blood-calcium was 4·0 mgm. per 100 c.c. of serum, and the blood-phosphorus was 2·6 mgm. per 100 c.c. of serum. She was at once placed on calcium and calciferol. The course as regards her blood picture was as follows :—

April 30	..	Ca 4·0 per 100 c.c. of serum P 2·6	May 18	..	Ca 9·75 per 100 c.c. of serum P 6·15
May 7	..	Ca 5·62 ,, ,, ,, ,, P 3·88	May 23	..	Ca 9·90 ,, ,, ,, ,, P 5·48
May 11	..	Ca 8·4 ,, ,, ,, ,, P 5·0	June 1	..	Ca 8·9 ,, ,, ,, ,, P 4·0

She left hospital well, the urine showing no abnormality.

Her blood hæmoglobin on entering hospital was 8·1 grm.%, and on discharge 10·8 grm.%. One of the interesting things about the case was the pelvic measurements. In 1934 these were as follows : Interspinous, 22 cm. ; intercristal, 26 cm. ; ext. conjugate, 19 cm. ; interischial, 10·5 cm. Arch wide.

By the time of her admission the interischial diameter was down to 8·5 cm., her pubic arch was narrowing, and there was no doubt that pelvic contraction was beginning. The remaining measurements were as before noted.

To turn to the child. At birth on April 30 it weighed 2,450 grm. and its height was 32/47 cm. Its cord blood contained only 6·4 mgm. calcium and 4·2 mgm. of phosphorus per 100 c.c. of serum. The Kline, Wassermann, and Kahn tests, were negative.

On May 9 definite carpopedal spasm, and spasm of the face, were noticed. On May 11 the blood-calcium was up to 7·46 mgm., and by June 25 it had risen to 11·95 mgm.

The X-rays showed marked fœtal rickets, and the bone shadows were very poor. At birth the limbs were normal. On May 2, about forty hours after birth, in spite of special handling, the left femur was found fractured at about the middle of the shaft. Some time between May 10 and 20, in spite of still further care in handling, the right femur was fractured near its lower end. On May 21 a fracture was also noticed at the junction of the upper and middle thirds of the right ulna.

By June 15, 1936, the fractures were apparently healed.

Treatment was by a formula of glucose and breast milk, with orange juice 10 c.c. three times a day, and 5 drops of haliverol twice a day.

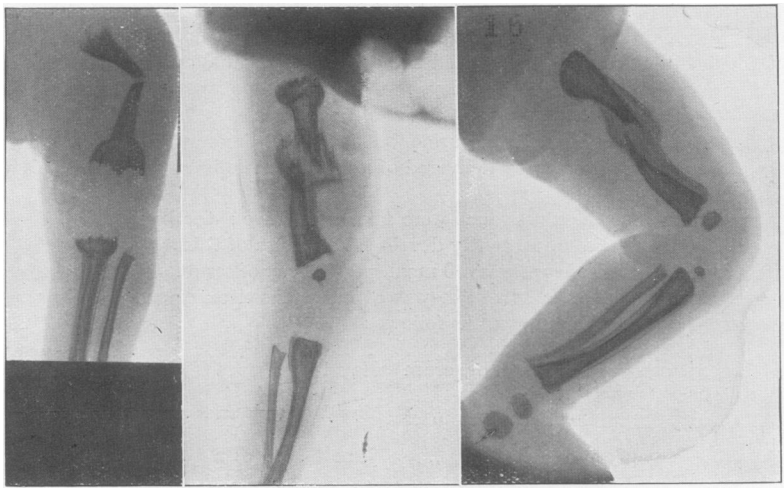
The child was slow in crawling, and in attempts to sit up. The first teeth, the lower central incisors, came through at about 1 year and 2 months. She did not walk till 2 years and 2 months of age.

One of the most remarkable things about the case was the healing of the fractures with the gradual obliteration of deformity (figs. 3 and 4).

In the mother, there are some signs of late rickets and mild adult rickets ; two of the children exhibited infantile rickets, and the last baby had marked fœtal rickets.

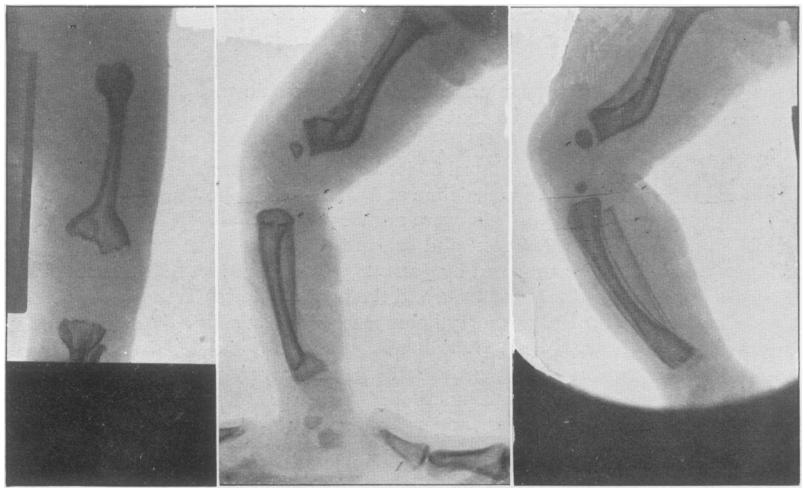
In our last paper [1] we described the changes which take place in the teeth of

children born with the evidence of fœtal rickets, and pointed out that in addition to other defects the enamel showed signs of hypoplasia, forming imperfectly, and staining badly. In the teeth of the baby from the patient with ruptured uterus, removed at the 35th week, the same process is evidently taking place (fig. 5). In this fœtus the microscopical evidences of rickets in the rib union and the ends of the long



3.5.36. 22.5.36. 15.6.36.

Fig. 3.—Baby T'ièn. Left femur. (Bone shadows have been intensified).



2.5.36. 23.5.36. 16.6.36.

Fig. 4.—Baby T'ièn. Right femur. (Bone shadows have been intensified).

bones are typical. In a good many of our Chinese cases the formation of osteoid is not marked, and this is probably due to the general malnutrition.

We have already written fully on the question of posture in the production of the typical triradiate deformity of the osteomalacic pelvis, and in the formation of the flat pelvis which one finds as the result of infantile rickets [1, 2]. Where a patient who

is developing an osteomalacic pelvis sits all day, she gets a fairly even, triradiate deformity. If, on the other hand, she lies mainly on one side, there is an irregular triradiate pelvis with the side on which the patient lies pressed in more than on the other side. These are severe well-marked deformities, but it is as well to remember that there are many modifications of the normal pelvis caused by mild rickets, which approximate closely to the forms of pelvis described by Caldwell and Moloy [2], the genesis of which is as yet somewhat obscure. A mild form of flat pelvis approximates very nearly to the so-called " platypelloid " type, whilst pressure resulting in a moderate yielding at the two sides will produce a pelvis which is, in all essentials, of the " android " type. Thoms [3] has already put forward the possibility of a

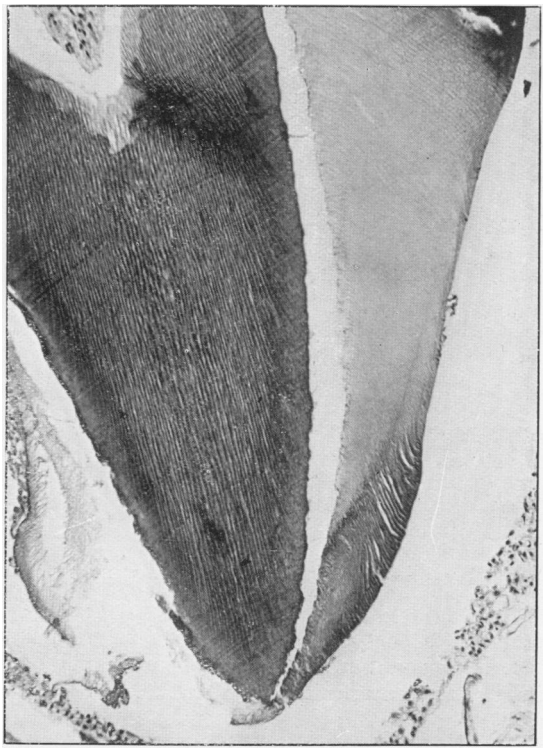


Fig. 5.—Baby C. W. H. Tooth showing enamel change. (×110).

" gynecoid " type being due to mild rickets. Undoubtedly there is a congenital element, possibly also a hormonal one, in the formation of the adult pelvis ; and the anthropoid type may be of this nature. But in our opinion mild rickets plays a much larger share in the final shape of the adult pelvis than has been conceded in the past.

In a previous paper [4] the question of instability of gait of patients in late pregnancy has been mentioned. It is a moot point whether in the vast majority of cases relaxation of the pelvic joints with instability of gait is not due to a mild degree of vitamin-D deficiency. We have never been able to satisfy ourselves that such was not the case, as we have seen the condition clear up before labour under calcium and vitamin-D treatment ; and mild conditions of this deficiency disease are by no means uncommon.

There is a further interesting fact to be noted about these cases of osteomalacia

with osteoporosis. The symphysis pubis is apt to be weakened, and unless special care is taken, may give way during delivery. We have known this to happen on several occasions, and the force needed is very slight. On one occasion Maxwell had this experience whilst doing a perfectly easy low forceps delivery in a mild case of osteomalacia, pulling with only one hand. As a rule this separation seems to take

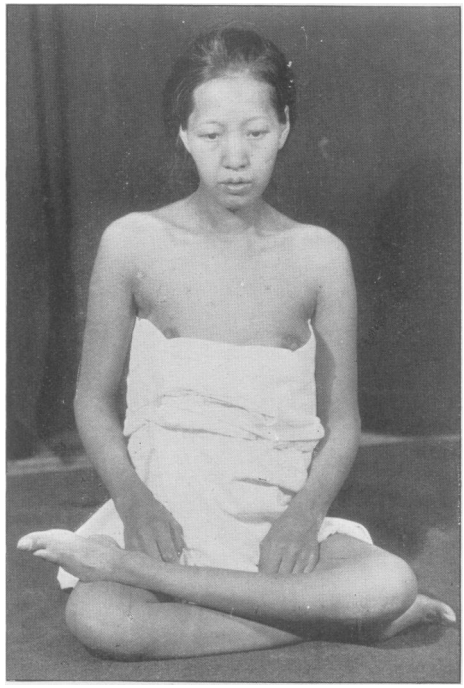


Fig. 6.—Sitting posture of Shansi woman.

place rather than fracture of the pubic rami, though where a funnel pelvis has been produced, and has been unrecognized, the disease being healed at the time of delivery, splintering of the pubic arch may occur [5].

One of the conditions modifying these deformities in North China is the posture so many women adopt when sitting on the " K'ang " (a hot platform), in their homes. When one puts one of these women into a lithotomy position, one finds an unusual power of crossing the legs, as is well shown in the illustration (fig. 6).

The Milk of the Patient with Adult Rickets

It was but natural that after investigating the calcium and phosphorus content of the blood of our osteomalacia patients, our attention should be drawn to the calcium content of the milk. As has been already pointed out, the babies of mothers with low calcium are very prone to develop tetany in the early neonatal period. Granted that they begin life with a deficient calcium content in their blood, how far is further trouble contributed to by a deficiency in the food ? Some valuable information on this subject has been given by the work of one of our colleagues, Dr. S. H. Liu, and his co-workers [6] in the Nutrition Ward of the Department of Medicine. In that paper they present a very detailed study of three cases of osteomalacia, and two cases who showed no evidence of bony decalcification. We quote from their summary : " Four

of the subjects on low calcium intake had relatively small milk yield. The negative balance in calcium was not excessive, and calcium loss through lactation accounted for a small fraction of the total output.

" Moderate addition of calcium failed to rectify materially the calcium loss, but vitamin-D administration was efficacious in reducing the stool elimination of the calcium so that a markedly positive balance was obtained.

" In one subject whose milk yield was high, the calcium intake had to be considerably increased to maintain balance even in presence of vitamin D.

" The two subjects without clinical skeletal decalcification behaved similarly to, and showed as marked response to vitamin-D therapy as those with osteomalacia, suggesting the existence of subclinical states of vitamin-D deficiency and calcium shortage in the bones."

It is a fact well known to us that the milk supply from these osteomalacic mothers tends to be very poor ; in fact, in some instances, Kuo found it difficult to get an adequate amount for analysis. This may be partially due to the fact that their calcium intake is so low. In previous papers we have shown that the calcium intake in the food is far below what it ought to be. This is also borne out by the work of Wu and Yen [7] who estimated that the diet of the ordinary North Chinese women did not contain more than 0·3 grm. of calcium daily. Liu *et al.* [6] suggest that " with low calcium intake, a safety mechanism comes into play by which milk production is reduced, so that the extent of calcium loss becomes much less than it would be otherwise, and that vitamin-D deficiency is a more important factor than low calcium intake in accounting for the mineral stress in lactation ".

The moral of this is that osteomalacia patients should have a much larger calcium and vitamin-D intake than is needed for the normal woman, in order to promote a reasonable milk yield for the child.

But is there an actual deficiency of calcium in the scanty milk supplied by these mothers ? It has been almost impossible to get twenty-four-hour specimens of milk, and the question arose whether random samples would give us a fair estimate of the calcium and phosphorus being supplied.

On examination we found that samples from the same mother, taken at different times during the day, gave results which were so near each other, that the figures given are a true index of what the bulk of the milk would contain. The following table gives the figures (average) which we have obtained. The normal figures were taken from many determinations in our wards, the quiescent osteomalacia cases were those who had been and mostly were under treatment with calcium and vitamin D ; and the active osteomalacia cases had been without treatment.

Comparison of Various Values.

Milk	Stage	Ca. (mgm. per 100 c.c. milk)	P. (mgm. per 100 c.c. milk)
Colostrum	(1) Normal	26·58	17·90
	(2) Quiescent osteomalacia ..	22·84	9·15
	(3) Active osteomalacia ..	17·35	12·02
Transitional	(1) Normal	24·31	17·63
	(2) Quiescent osteomalacia ..	24·97	15·13
	(3) Active osteomalacia ..	17·72	18·52
Late milk	(1) Normal	21·19	11·66
	(2) Quiescent osteomalacia ..	24·49	13·96
	(3) Active osteomalacia ..	17·41	13·56

It will be noted that the amount of calcium in the milk, even though concentrated and scanty, is much less in the active osteomalacia cases.

It is remarkable how rapidly a proper supply of calcium and vitamin D will affect the calcium content of the milk. We pointed out in our last paper how quickly proper treatment with calcium and vitamin D would bring the blood figures for calcium and

phosphorus up towards the normal ; and it is clear that the same holds good for the secretion of calcium in the milk, as is shown in the following case whose history has been already given in detail :—

Mrs. T. L. F.

ACTIVE OSTEOMALACIA WITH TETANY.

Treatment—Tricalcium Phosphate and Calciferol.

Material	Milk		Blood	
Post-partum	Ca (mgm. per 100 c.c. milk)	P	Ca (mgm. per 100 c.c. serum)	P
4th day	12·31	12·82	4·0	2·6
8th day	18·24	12·50	8·4	5·0
15th day	21·73	12·20	9·9	5·48
20th day	19·32	15·62		

In view of the fact that in India the relation between anæmia and osteomalacia is very close we felt it would be interesting to go more deeply into the question of anæmia in our patients, and its effect on the supply of iron to the child. One of us (Lin) has been especially working on this matter, and although more remains to be done, Dr. Lin has enough data to show that in cases of active osteomalacia the child is born seriously handicapped as to its supply of iron.

Taking first of all a number of normal deliveries in apparently normal Chinese women in our wards, the average worked out as follows :—

Cord blood of healthy Chinese babies (born of unselected mothers not apparently suffering from disease) : Average red cells 5,332,800 ; average hæmoglobin 15·34 grm. From babies born of osteomalacia mothers under treatment or old healed cases : Average red cells 4,808,500 ; average hæmoglobin 14·4 grm. From babies born of mothers with active osteomalacia : Average red cells 3,770,000 ; average hæmoglobin 11·5 grm. With regard to the last figures it must be remembered that these active osteomalacia cases come into our hands, as a rule, somewhat dehydrated and exhausted, which may possibly give a higher figure in the cord blood than is really the case.

As to the normal figure for hæmoglobin in the cord blood of newborn babies, it is generally stated as being about 21 grm.%.

Nils Faxén [8] puts the figure still higher and gives the following : Average hæmoglobin level 23·2 $\pm$ 0·25 ; red blood corpuscles 5,780,000 $\pm$ 0·130.

OSTEOMALACIA AND ECLAMPSIA

Does the presence of osteomalacia predispose to eclampsia ? On previous occasions questions have been raised as to the occurrence of eclampsia in cases of osteomalacia.

As far as is possible in North China we have made special inquiry into this. One may ask the question in two ways :—

(*a*) Do osteomalacia cases exhibit eclampsia in any unusual proportion ?

(*b*) Have any of the eclampsia cases of which one has been able to get records exhibited osteomalacia ?

In reply to both questions the answer is in the negative, with the exception of one doubtful case.

Dr. Clow (personal communication) informs us that in the Taiyuanfu Hospital, Shansi, there have been 522 recorded cases of Cæsarean section for osteomalacia, and in none of these has there been any mention of eclampsia.

From October 1929 to November 1936 in this same hospital there were records of 312 osteomalacia cases, with 213 Cæsarean sections, and no record of eclampsia in any of them.

Out of 1,800 confinements in this hospital during the same period, there were six cases of eclampsia, and in none of these was there any sign of osteomalacia.

At Pingtingchow, Shansi (personal communication from Dr. Parker), there are accurate records for 1931–6. During these five years there were 120 cases of osteomalacia, and six cases of eclampsia, but in no case have the two been combined in the same patient.

At Showyang, Shansi, in ten years there were 373 patients with osteomalacia, none of whom had eclampsia in addition.

In the Peiping Union Medical College Hospital between 1921 and 1937, there were 7,567 labour cases with 151 cases of eclampsia and 124 of osteomalacia. In only one of these have the two diseases been combined, and in this case the diagnosis was doubtful. It was an atypical post-partum eclampsia, occurring about twelve hours after Cæsarean section, the patient making a good recovery.

So that one can definitely say that osteomalacia does not predispose a patient to develop eclampsia.

At the time of our last paper we were already aware of certain degenerative processes which are occasionally seen in connexion with osteomalacia. One of us, Pi [9], took up the study of cataract in our osteomalacia cases, and in relation to what is known as "Cataracta tetanica." As cataract has hitherto never been especially associated with osteomalacia the subject is important, and also because the term "cataracta tetanica" is, in some cases at least, obviously a misnomer.

This cataract has to be looked for, as subjective ocular symptoms are slight; and as the majority of our osteomalacia patients are illiterate, it is only when the disease has considerably advanced, that they are likely to complain of loss of vision. What is the incidence of the trouble in osteomalacia cases? We have records of 124 osteomalacia cases admitted to the Peiping Union Medical College Hospital, and Pi, who examined about half of these, found 13 cases of this form of cataract amongst them. So it is probable that the incidence is about 20%. Under appropriate treatment these cataracts undoubtedly improve but do not disappear.

We have observed three other degenerative processes in cases of osteomalacia. These are not peculiar to osteomalacia, but are, rather, manifestations of avitaminosis in patients who are known to be deficient in vitamin D; and it is probable that where they occur in connexion with osteomalacia the underlying cause may be a mixed vitamin deficiency. They are: (*a*) Keratomalacia; (*b*) a peculiar form of failure of nutrition of the nails; (*c*) keratoses on the cheeks and over the elbows.

(*a*) *Keratomalacia.*—One of us [10] has previously reported a case of fœtal keratomalacia in an infant born from an osteomalacic mother. In this case it was impossible to get roentgenograms to determine whether the baby was also suffering from fœtal rickets. This case was kindly seen and verified by Dr. Pillat.

But the combination of rickets and keratomalacia is probably not so uncommon in this region as might be supposed. During five years there were as in-patients in the hospital in Peiping at least 10 cases of keratomalacia and proved rickets in infants under 2 years, and of these 10 cases, three were under 3 months of age and two under 4 months. Granted that these infants might not have shown roentgenological evidence of rickets at the moment of birth, it is not unlikely that if it had been possible to get sections of the epiphyses there might have been microscopical evidence of commencing rickets, and they are a striking commentary on the supposed absence of rickets in North China. And it is clear that where one gets deficiency of one vitamin, one is very likely to get a deficiency of more than one.

(*b*) *Nutritional disturbances of the finger nails.*—Two of our osteomalacia cases especially showed certain nutritional defects in the finger nails.

The first case came in on December 26, 1932, with very low blood calcium and marked osteomalacia. A Cæsarean section had to be done on January 13, 1933, tetany of the uterus having supervened. She made a good recovery. On April 21, 1933, her

finger nails were found to show certain nutritional defects. A second photograph was taken on June 9, 1933, showing marked improvement. There was no attack of abdominal pain in this patient.

The second case came in on March 4, 1933, with tetany and pregnancy.On April 13 she began to run a temperature and developed a subacute dysentery (Shiga). The first photograph was taken eight days later and shows the affection of the nails well established. By April 29 the dysentery was over. On May 21 a Cæsarean section was done, and she was discharged well with her baby on July 10. On July 21 the second photograph was taken, showing that the morbid process had been quickly arrested. Whether or not the dysentery played any part is doubtful.

The exact cause of these nutritional defects is not clear ; the rate of growth of the nail in this second case was about 0·15 cm. per month.

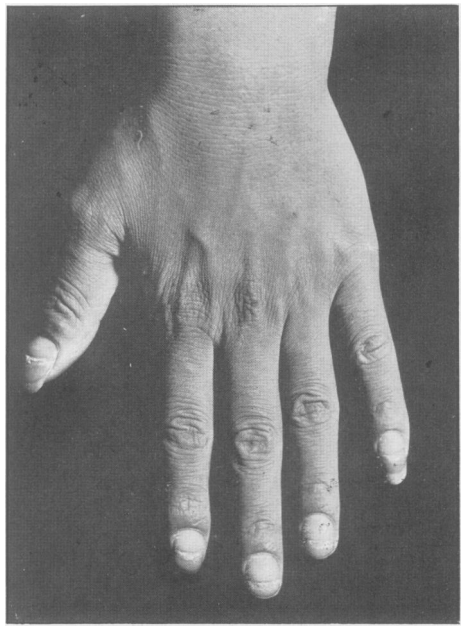


Fig. 7.—Nutritional disturbance of nails.

(c) *Nutritional disturbances of the skin.*—It has also been noticed that in some of these osteomalacia cases there is a marked dryness and scaliness of portions of the skin.

We have seen more than one example of this disturbance of nutrition, and think there is little doubt that it is due to an avitaminosis, though one hesitates to say which of the vitamins is involved.

It is clear that our original contention that osteomalacia was not a disease *sui generis*, but rickets in the adult, was correct. It is also clear that fœtal rickets, where adult rickets is present in the pregnant woman, is not uncommon, and that it presents clinical and radiological symptoms rendering it easy to diagnose; that where the affection has not progressed to the point of definite clinical symptoms, microscopical evidence may be present and that there is a definite tendency to the development of tetany and infantile rickets in the newborn child.

It is also clear that the cord blood in these cases of fœtal rickets presents definite deficiencies in calcium, phosphorus, and iron. Premonitory symptoms of adult

rickets such as thigh and back pain, excessive movements of the fœtus *in utero* and instability of gait should be kept in mind in antenatal examinations and the appropriate treatment given for the trouble.

Finally, it should be borne in mind that a mother with the signs of adult rickets is likely to be iron-deficient, and this deficiency, unless corrected, will be transmitted to the newborn child. The tendency of patients with adult rickets to suffer from opacities in the lens of the eye is an additional reason for careful and early treatment of the disease in general.

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Discussion.—Mr. V. B. GREEN-ARMYTAGE said he had first seen osteomalacia in Calcutta thirty years ago, and since then, although he had seen and collected a consecutive series of 500 osteomalacic patients in Calcutta, not once, despite their anæmia, their albuminuria, and their blood metal shortage, had the state of toxæmic eclampsia occurred. This was a point of some importance just at the moment when the Peoples National League of Health was spending quite a lot of time and money upon an experimental diet and vitamin supply to hospital antenatal cases. Would this experiment prove anything in view of the facts also recorded by Professor Maxwell? One point of great import did evolve from this paper, namely that in the depressed areas and where nutrition was at its lowest, one might expect to find in the rising generation a large increase of android or funnel pelves which might be missed by a too-casual or careless antenatal clinic. These cases were commoner all over the world to-day and there could be little doubt they were related to a vitamin calcium phosphorus-deficiency in the food.

Prof. F. J. BROWNE said so far as he knew this was the first clinical demonstration that the experimental work of May Mellanby was applicable to human beings. We usually emphasized the importance of vitamin D and calcium in the diet of the expectant mother in order to prevent dental caries in the child, but there had lately been a tendency to stress the influence of heredity in the causation of dental caries and to minimize the importance of the mother's diet. Professor Maxwell's work seemed to prove that whether or not heredity played a part in dental caries the diet of the mother was also important.

Dr. KATHLEEN VAUGHAN said that in Kashmir the boat women and others who lived in the open air on the coarsest food never had the disease. Osteomalacia was considered to be hereditary by many Kashmiris themselves, but was really due to the social custom prevailing in high-class families of observing "purdah" or the seclusion of women. This usually entailed the women living in dark ground-floor rooms, the only light coming from small windows near the ceiling, placed there so that no man passing by could look in. Branches of the same family living in the country and going out freely were free from it, and sometimes a woman developed it only when her husband moved to a more densely populated part of the town and shut her up strictly in semi-darkness.

Although the softening of the bones was caused, just as rickets was caused, by absence of sunlight, yet the deformity produced was determined by the posture habitually assumed by the patient; thus sitting on the floor with arms clasping the legs produced a pelvis with an elongated conjugate, whereas sitting on chairs in European fashion tended to increase the transverse diameter. Later on, with further softening, the walls of the pelvis seemed to fall in, in all directions, and we had the Y-shaped pelvis typical of acute osteomalacia.