(FROM THE ORTHOPAEDIC HOSPITAL, COPENHAGEN. CHIEF: P. GUILDAL, SENIOR SURGEON, AND BISPEBJERG HOSPITAL, DEPARTMENT A, CHIEF: J. FOGED, SENIOR SURGEON)

OSTEOPOROSIS AND OSTEOMALACIA COLUMNAE[1])

BY

ALFRED ZACHO

(Received by the editor March 1939)

Although the disease to be described here is usually termed osteomalacia columnae, this name is only to a certain extent justified. In most cases there is general disease of the bones, which certainly originates most frequently in the spine, where it reaches its climax, but which, if the disease progresses, gradually invades most of the skeleton. The term osteomalacia, however, is not adequate in all cases, since the disease is often osteoporosis and not actually osteomalacia in the strict meaning of the word. The matter, however, is mainly, and perhaps exclusively, one of distinction between two anatomico-pathologically different forms of the same disease with consequent differences in appearance on examination by X-rays, which will be discussed later.

Osteomalacia is usually understood to mean the classic *puerperal osteomalacia* which was formerly a frequent illness in Europe amongst pregnant and suckling women. Osteomalacia causes the bones to become soft and pliable, thus giving rise to serious deformities of the pelvis, which have been specially noted as a most characteristic feature of this disease. The main reason that changes in the pelvis come first to mind when puer-

[1]) The term osteoporosis or osteomalacia is used in the following in accordance with the definition given on page 267 or, in case of references, in accordance with the term used by the author quoted. Where no distinction is made between the two forms, the term halisteresis is employed several times to denote a decalcification of the bones.

peral osteomalacia is under consideration is chiefly that this deformity is important as a hindrance to delivery. Actually, the disease begins in the spine, where it may attain the most severe stages with compression of the vertebrae and the development of marked deformities (*Strümpel*).

This severe form of osteomalacia has gradually become a rare disease in Europe and, moreover, also in the United States of America. On the other hand, it is extraordinarily frequent in Northern India and China, where it has been made the object of intensive study during recent years (*Maxwell* 1923, *Miles & Feng* 1925, *Hannon* and co-workers 1934).

Towards the end of the Great War, and during the following years, the so-called *'Hunger-osteomalacia'* became prevalent in Austria and Germany, attacking principally elderly people, mainly women in the climacteric or postclimacteric age, although it also was found, and not infrequently, in men (*Schlesinger, Alwens, Dalyell & Chick,* and others).

In the majority of cases the disease began at about the end of 1918 or the beginning of 1919, a period during which the food supplies in the above-mentioned countries were particularly short owing to the blockade. All the patients belonged to the poorer classes and were all very much under-nourished. How widespread the disease was at that time is shown by the fact that *Alwens* in 1919 was able to collect twenty-six cases in his department during the course of four months. *Dalyell & Chick* even state that in the period from January till May 1920 six hundred cases of hunger-osteomalacia were diagnosed in one of Vienna's largest policlinics. At the same time cases of rickets and late rickets were very numerous.

The hunger-osteomalacia, like classic osteomalacia, was localized principally in the spine, thorax, and pelvis. In a number of cases only the spine seems to have been attacked, which is probably the reason for the special interest taken in the spinal changes (*Eisler & Hass, Hoffmann,* and others). The changes in the bones seem in the majority of cases to have consisted of osteoporosis, whilst actual malacotic forms have been less frequent (*Alwens*).

In the following years reports were published of more sporadic cases of osteoporosis or osteomalacia localized presumably, and sometimes entirely, in the spine.

Such cases[1]) have been described by *Baron & Barsony* 1927, *Kreuzer* 1929, and *Bohne* 1929. In 1930 *Gargill, Gilligan & Blumgart* described a case which developed after a birth and in which the disease extended not only to the spine and pelvis but also to the extremities. In 1931 *Meisels* reported a case of osteomalacia in a man with Graves, disease. In 1932 *Bauer & Marble* published two cases, one of which was due to lack of calcium in the diet, whilst in the other there was achylia with chronic steatorrhoea, and by careful examination of the metabolism of this patient negative calcium and phosphorous balances, caused by deficient absorption of vitamin D, were found. In the same year *Diechmann* drew attention to cases of incipient osteomalacia in pregnant women (U.S.A.). In 1933 *Moffat* described five and again in 1934 four cases. The majority of these were considered to be due to deficiency of calcium in the diet, whilst one case was assumed to be of hypophyseal origin. *Morville & Müller* reported a case in 1933 in a 39-year-old woman with achylia and slight goitre. In addition to the typical changes in the spine there were also marked deformities in the pelvis and diffuse halisteresis. In the same year *Schultzer* published a case of osteomalacia of the spine in a man 55 years of age, of which the cause was supposed to be a defiency in the diet (vegetarian). *Hecht-Johansen*'s patients employed in his study of plasma-protein (1933) include four cases with halisteresis and compression of the vertebrae. (These patients were included later in Meulengracht's material.) In 1935 *J. Christiansen* advocated the opinion that many patients who complain of muscular cramp and pain in the bones really suffer from osteomalacia incipiens, and that the same applies to many patients with 'gout'. In his work on fractures of the spine *Gøthgen* mentions a 55-year-old woman who sustained a compression-fracture of a vertebra when getting out of bed. The spine and the pelvis were the seat of severe halisteresis, and typical vertebral deformities were found in the spine. In 1935 *Bulmer* mentioned four cases (partly old cases reported from memory). Cases were also mentioned in 1935 by *Paviot, Enselme & Guichard* and also by *Weissenbach & Lievre*, whose two patients had kept to a strict diet for a long time. *Kleinberg*, furthermore, published a case of senile osteomalacia. In 1936 *Margitay-Becht & Gömöri* reported a well-investigated case of a 30-year-old woman with achylia. Cases were published in the same year by *Brandt* and *Imhäuser*.

---

[1]) In 1928 *E. Dam* described cases of "Zwergwuchs mit unsicherer Pathogenese bei drei Brüdern". These were possibly cases of osteomalacia.

The last-mentioned writer's patient had chronic nephritis at the same time. *Meulengracht & Rothe-Meyer* (1936) reported five cases; one of these patients had achylia and another scurvy. In 1937 *Polgár* described the results of the X-ray examination of three patients with 'presenile osteoporotic kyphosis'. Early in 1938 *Poliard, Guichard, Muller & Viallier* reported four cases of senile osteomalacia. *Meulengracht* (1938) collected eighteen cases, including the five cases previously published, of which eight had achylia and one sprue. In one of the cases the disease was thought to have been caused by excessive use of Carlsbad salts. In the same year *Secher* reported a case in a 55-year-old man whose diet had been very deficient in calcium and phosphorus. *Guérin* (1938) described an interesting case in a nun, of which the cause was supposed to be lack of sunlight. *M. Lance, Girard & P. Lance* (1938) wrote an important paper on this disease, giving a description of sixty-six cases, of which forty-four were taken from the literature on the subject. These writers differentiated between senile osteoporosis, which they placed in a special class, and the other cases, which they combined under the term 'osteoporose et malacie présenile et de l'adulte'.

As has been previously stated, this bone disease is chiefly localized in the spine; and in some cases that is the only demonstrable localization. As a rule, however, other bones are also attacked, chiefly the pelvis and thorax and, in addition, the bones of the shoulder girdle, those of the extremities being less frequently attacked, and the skull but rarely.

The changes in the bones are most frequently *osteoporotic*. By this is to be understood a change in the structure of the bones by which it becomes attenuated. The trabeculae of the spongy bone become fewer and thinner. The compact bone also becomes thinner, but the changes in this are less distinct and occur later than in spongy bone. These changes are presumed to arise from an increased lacunar absorption by means of osteoclasts, which can be seen under the microscope in increased numbers. The osteoporotic bones become brittle and fragile, and the structure of the bones may gradually become so weak that spontaneous fractures may occur from the least traumata, in this case compression-fractures of the bodies of the vertebrae, which are the first to be affected by these changes. These give rise to permanent changes in the shape of the bodies, with deformity of the spine. The osteoporotic changes are frequently

accompanied by spondylous processes with formation of osteophytes on the edges of the bodies.

In other cases, however, the changes usually termed *osteomalacia* are found. The newly-formed bone tissue (osteoid tissue) produced by apposition does not absorb calcium, for which reason, when the process has developed sufficiently, the bone tissue becomes so soft and pliable that these bones can be cut with a knife without any difficulty. The bodies of the vertebrae become flatter and broader and, at the same time, bi-concave, because the intervertebral discs bulge from above and below into the soft body, which thereby assumes the shape of a bi-concave lens.

Combined forms of these two main groups are frequently found. It is not yet clear which factors govern the development of osteoporosis and which give rise to osteomalacial changes.

Although the transition from osteomalacia and osteoporosis to *senile atrophy* of the bones appears to be gradual, the last-mentioned must, however, be considered as a separate form that is a part of the process of physiological involution in senility.

The disease is most often found in women, and here again most frequently during and after the climacteric period. It is, however, also found in men and in younger persons, although seldom in persons under thirty years of age.

The first *symptom* which brings patients to the doctor is usually pain in the back, most often in the lumbar region and frequently radiating down to the lower extremities. In addition they often complain of unbearable fatigue, especially across the loins and in the legs. In the more advanced cases the patients' disabilities may become so serious that walking is difficult, and, in especially severe cases, the patient becomes bed-ridden and can only turn over with difficulty.

Some of the patients say spontaneously that they feel as though their back had shrunk, or that their family has noticed that they have become smaller.

Sometimes the symptoms originate or are noticeably aggravated in consequence of a trauma, occasionally very slight (driving in a motor-car on an uneven road, sudden bending forwards, lifting of heavy burdens, etc.). These patients most frequently consider the trauma to be the cause of the disease, particularly since they have often felt a violent pain in the back simultaneously with the trauma. They may even have felt a crack in the back and in some cases they have actually heard a crack—'like a twig being broken'.

Patients occasionally say spontaneously that they feel better in the summer.

Objectively these patients often look considerably older than their age. They look pale and ill, their appearance not corresponding to that expected from a frequently relatively high percentage of haemoglobin.

Examination of the back occasionally shows deformation of the spine by kyphosis or scoliosis, with sometimes a gibbus. Tapping of the spinous process often reveals fairly considerable tenderness. In pronounced cases the back is also found to be short, with transverse folds of skin running outwards to the sides. The curvatures rest upon the pelvis and the extremities are disproportionately long. During the early stages the spine can move comparatively freely, contrary to the conditions found in tuberculous spondylitis. Patients are, however, afraid to bend forwards and the pains increase considerably when they do so.

A front view shows these patients to be surprisingly short in the body; the curvatures are low. In the upper part of the abdomen a transverse flat furrow or depression is found, which contrasts with the protuberant, drooping, lower part of the abdomen.

An additional characteristic of these patients is also their great aversion to standing up; as soon as they get an opportunity to sit down they immediately do so.

Diagnosis is, however, chiefly made by *X-ray examination*. As has been mentioned previously, the spinal changes are the most noticeable, although the pelvis is also frequently attacked. Deformities of the pelvis are, however, only found in the more

severe stages of the osteomalacial form. The spinal changes vary according as they originate from osteoporosis or osteomalacia.

In the early stages of *osteoporosis* only a diffused halisteresis of the vertebrae is found. The spongy bone only shows a shadow, and the contours of the bodies, the first to be attacked, can only be guessed at. Halisteresis, however, cannot be recognized until it is highly pronounced, since no normal symmetrical part is available for comparison.

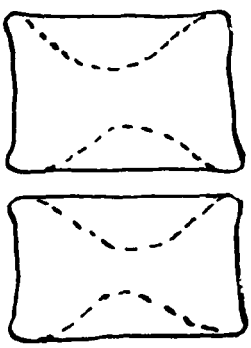

Fig. 1.
Vertebral deformity in osteoporosis.

The milder forms cannot be recognized with certainty. The next phase consists of the assumption by one or more of the lower thoracic vertebrae of a wedge-shape, with the apex turned towards the front, and of the incipient assumption by the lumbar vertebrae of the shape of fish vertebrae, caused by the pressure of the intervertebral discs and, especially, of the nucleus pulposus. The resultant nuclear 'prolapse' (Fig. 1) is, however, quite different from the true traumatic nuclear prolapse which occurs after a rupture of the intervertebral discs; but it is also different from the flat prominence which is seen in typical osteomalacia (see below). It is a necessary condition for the fish-vertebral form, however, that the nucleus should have retained its elasticity, otherwise more irregular deformities may occur (*Junghanns*). The third phase shows fracture and collapse of one or more vertebrae, most frequently of one of

the upper lumbar vertebrae. The body has become so weak in the middle, as a result of the gradual convergence of the centres of the nuclear prominences that the bone fractures on the slightest trauma. In the thoracic region the front edge of the body is more often found to be broken off, thus further accentuating the wedge-shape of these vertebrae.

The typical X-ray picture of osteomalacia shows that the bodies are flatter and broader, whilst the intervertebral discs, the thickness of which in these cases is often considerably increased, protrude convexly into the body, which consequently assumes the shape of a bi-concave lens. A combination of these two main forms is often seen.

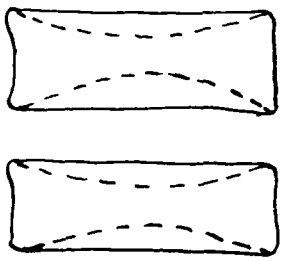

Fig. 2.
Vertebral deformity in osteomalacia.

Examination of the blood: With a disease which causes such rapid decalcification of the bones, it seems reasonable to expect that these changes should affect the content of calcium in the blood. It appears, however, that the *content of calcium* in the blood is generally normal (9.5—12 mgr.%). In some cases, however, hypocalcaemia is found. The reason why the content of calcium in the blood is thus normal in most cases is, in all probability, that there is a compensating hyper-function of the parathyroid glands. The examination, however, plays an important part in the differential diagnosis of ostitis fibrosa generalisatata (v. Recklinghausen), in which disease the calcium content of the blood is increased. *The phosphorus content* of the blood is also generally normal, but may vary considerably in different cases, being possibly above or below the average

(3—5 mgr.%). In the renal form of osteomalacia (renal rachitis) the phosphorus content in the blood is increased. It must be especially noted that the values for the content of calcium and of phosphorus in the blood, in addition to being interdependent, are liable to be affected by so many other factors that the estimation of deviations from the normal is difficult and is often only possible after intricate metabolic tests with determination of the calcium and phosphorus balances.

The plasma phosphatase is frequently slightly increased (*M. Lance, L. Girard & P. Lance*), but the figures often fall within normal limits (*Meulengracht*).

It has, furthermore, been stressed that osteomalacia is often accompanied by *relative lymphocytosis* (*Riese*) as well as by *hypocholesteremia* (*Riese, Bauer & Marble*).

Aetiology: Although *Trousseau,* as early as 1868, drew attention to the fact that osteomalacia is actually identical with rickets and can also be cured with cod-liver oil, there was a general tendency, particularly about the beginning of the present century to consider that osteomalacia is dependent on the endocrine glands. This view was mainly due to the improvement in the condition which occurred after the prophylactic ovariectomization of osteomalacial women to prevent a new pregnancy. *Trousseau*'s opinion has, meanwhile, been corroborated by subsequent investigations of osteomalacia in China (*Maxwell, Miles & Feng, Hannon* and co-workers) which have proved that osteomalacia can be compared to rickets in children and is due to a deficiency of fat-soluble vitamins in the diet, principally of vitamin D, possibly in conjunction with an insufficient supply of calcium. It is therefore now generally agreed that puerperal osteomalacia and rickets are in reality different results of the same cause. According to the studies of *Schmorl, Fomme,* and others (quoted from *Kreuzer*) rickets, late rickets, and osteomalacia are grouped together. The difference in the localities of the diseases is due to the fact that the parts of the skeleton that grow most rapidly are those most attacked. This explains why it is principally the skull that is most attacked in childhood (rickets), amongst older children preferably the long bones

(late rickets), and, after increase in height has ceased, the pelvis, spine and thorax (osteomalacia).

The cause of *hunger-osteomalacia* was very reasonably attributed to insufficient nourishment. The disease was generally found to begin towards the end of winter, whilst it improved or disappeared during summer. That this was not due to the summer diet containing more vegetables was shown by the fact that supplementing the diet with extra vegetables was not alone capable of curing the patients (*Dalyell & Chick*); they were, however, cured by cod-liver oil in conjunction with ordinary mixed diet (*Alwens, Curschmann, Dalyell & Chick, Eisler & Hass*). It may, therefore, be supposed that hunger-osteomalacia is also due to lack of vitamin D, even though the majority of these patients suffered from the effects of general under-nourishment. It has also recently been shown, both in Europe and in America, by carefully testing of the calcium and phosphorus balance, that these patients have a negative balance for these substances. The administration of vitamin D may, however, produce a positive balance (*Gargill, Gilligan & Blumgart, Schultzer, Bauer & Marble,* and others). The basic factor in all these cases seems to have been deficiency of vitamin D in the diet, the function of this vitamin appearing to consist of facilitating the absorbtion of calcium from the intestinal tract as well as of retaining the absorbed calcium in the bones.

In a number of cases of loss of calcium in the bones it is, however, impossible to prove any deficiency in diet, and it must, therefore, be assumed that the calcium metabolism may be disturbed by other means too. Special attention has been paid in this respect to the conditions of absorption in the intestine. It is evident that all the factors that combine or destroy vitamin D, or which restrict the absorption of the calcium salts, may cause a negative calcium balance. Such restriction of the absorption of calcium salts may be caused by disturbances in the digestion of fats, the reason having been given that, due to the defective digestion of fats, insoluble lime-soaps are formed with the fatty acids. Amongst *Meulengracht*'s cases there is a patient with sprue whose bone disease must be supposed to have arisen in this way. Distinct deficiency of calcium in the organism may

also be caused by fatty diarrhoea due to other causes. *Sénèque* points out that *Doyon,* even in 1900, had observed osteoporosis in a dog with chronic biliary fistula. Similar observations were made by *Pawlow* in 1905 and *Looser* in 1907.[1]) In 1910 *Seidel* reported two cases of diffuse osteoporosis in patients with chronic biliary fistula. One patient became gradually weaker and died; the other recovered when the fistula was closed. *Denstad* saw a case of osteomalacia in a woman with chronic biliary fistula. This patient also showed distinct signs of vitamin A deficiency together with hemeralopia. That the deficiency in calcium was not solely due to loss through the bile is evident because the same changes in the bones can be induced by simply ligating the bile duct. These features were further investigated in dogs by *Loewy* in 1931. It is questionable, however, whether or not the lack of calcium found in connection with disturbances in the digestion of fat primarily depends on calcium being combined by the unabsorbed fat. There are many indications that the negative calcium balance is mainly due to the combination of vitamin D in the intestine. It is not improbable that a considerable quantity of the vitamin D ingested with the food escapes absorption through being held in solution and combined with the unabsorbed fat, together with which it is excreted in the manner shown by *O. Andersen* as regards vitamin A by the simultaneous administration of paraffin oil. The simultaneous lack of vitamin A in *Denstad's* case also tends to confirm this. That vitamin D is the main factor is proved by the possibility of curing osteoporosis in dogs suffering from biliary fistula by the parenteral administration of vitamin D without change of diet (*Tammann*—quoted from *Sénèque*).

As has been previously mentioned *Morville & Müller,* as well as *Margitay-Becht & Gömöri,* reported cases of osteomalacia with *achylia,* the importance of which as an aetiological factor is especially stressed by *Meulengracht,* of whose cases eight had achylia. It may be supposed that the changed conditions of absorption in patients with achylia, such as a decreased degree of acidity and abnormal formentation in the intestine in ad-

[1]) quoted from Sénèque.

dition to secondary intestinal disease, may give rise to the defective absorption of calcium salts and probably also of vitamin D. *Moffat* drew attention, in 1933, to the importance of the degree of acidity in the intestines to the absorption of the calcium salts. The reaction of the duodenal content, which is certainly particularly important, is greatly dependent upon the conditions of reaction in the stomach. The part which may be played by fermentation in the intestine was emphasized especially by *Goehlinger & Bécart,* who were of the opinion that conditions such as those caused in the intestines, for example by the coli bacillus, are very unfavourable to the absorption of mineral salts (*Delahaye*). Furthermore, *Stepp* drew attention to the vicious circle which may arise in disease of the stomach and intestines on account of the unfortunate action on the mucous membrane of the stomach and intestines caused by the reduced absorption of vitamins often found in these diseases. This, incidently, applies to vitamin A, which, as regards appearance and conditions of absorption, to a great extent follows vitamin D.

Disturbances of the excretory conditions in *kidney disease* may also cause a negative calcium balance with decalcification of the bones. Renal rickets is well known in children. Recently *Albright, Drake & Sulkowitch, Goevaerts,* and others, have spoken of decalcification of the osseous system in nephropathy. The lowering of the calcium level in the serum was supposed, in these cases, to depend on a retention of phosphate caused by the kidney disease. *Imhäuser* reported a case of severe osteoporosis of the spine in a forty-nine-year-old man with renal insufficiency. It is possible that this case of osteoporosis was of renal origin.

Attention was early directed to disturbances in *internal secretion* as a cause of osteomalacia. Since the classic osteomalacia most usually occurred during pregnancy and lactation, prevention of the progress of the disease was attempted by removal of the ovaries, thus preventing the patient from becoming pregnant again. The result of this was evident not only in the arrested development of the disease, but also in a surprising improvement. This result was, however, explained away by various authorities, who held that ovariectomy was only important because it precluded further pregnancies.

With the discovery of the part played by nourishment in the occurrence of osteomalacia and, especially, the demonstration of the excellent therapeutic action of cod-liver oil, interest waned in the significance of internal secretion as an aetiological factor.

The importance of internal secretion to the metabolism of the bones is, however, unquestionable. It is now generally known that adenoma of the *parathyroid gland* may cause severe general osteoporosis (ostitis fibrosa generalisata v. Recklinghausen). In addition to the primary adenoma of the parathyroid gland, a secondary hypertrophy may be found in various diseases which tends to lower the calcium content in the blood (*Hamilton & Highmann*). Another hypertrophy was described by *Loewy,* for example, in cases of chronic biliary fistula, and by *Pappenheimer & Wilens* and *Castleman & Mallory* in connection with disease of the kidney. In cases of basophile adenom of the *anterior pituitary* decided general osteoporosis may also be evident in conjunction with a number of other symptoms, e.g. obesity, hypofunction of the sexual glands, hypertension, and hypercholesterinaemia (*Cushing*'s disease). Some writers (*Rutishauser & Maulbetsch, Lyon* and others) believe that the secretion from the basophile cells tends to inhibit the osteoblasts and that senile osteoporosis originates on account of an increased function of these cells, possibly because of the cessation of the functions of the sexual glands (*Krause, Lyon*). *Graves' disease* is frequently accompanied by a slight decalcification of the bones and in rare cases by severe general osteoporosis (*Golden & Abbott*). It is also interesting to note that a relatively large number of osteomalacia patients have had Graves' disease or some other kind of goitre (*Schlesinger, Curschmann, Meisels, Morville*). The part played by the thymus as an aetiological factor was particularly emphasized by *Scipiades,* who claimed to have developed osteomalacia in dogs by the extirpation of the thymus. Both *Scipiades* and *Khoôr* believe they have cured osteomalacia by transplantation of the thymus. The function of the suprarenal glands also appears to be of some importance. Patients with osteomalacia often have relatively low blood-

pressure and can tolerate large doses of adrenalin (*Riese*), which is further stated to have a good therapeutic effect on the osteomalacia (*Hoffmann, Riese, Curshmann*).

It has been mentioned that the role of the ovarian function as an aetiological factor is disputed, in which connection the incidence of osteomalacia in woman with amenorrhoea (*Curschmann*) has been emphasized. It is particularly surprising that by far the majority of cases of osteoporosis and osteomalacia that have been described since the termination of the Great War in 1918 have occurred in women mainly after the climateric period (*Alwens, M. Lance, L. Girard, & P. Lance*). Even though it can thus be said that osteomalacia is not due to hyperfunction of the ovaries, there seems, on the other hand, to be a certain relation between this disease and the ovarian function. Osteomalacia in younger women is thus often accompanied by menorrhagia (*Starlinger, Riese, Kehrer,* and others). The connection is, however, hardly the same in all cases. Whilst a number of the post-climateric cases in women are probably connected with the anterior pituitary, the disturbances in menstruation observed in osteomalacia in younger women are certainly of secondary importance in comparison with the disturbances in the metabolism due to alimentary or absorptional causes (most frequently lack of vitamin D). It has quite recently been demonstrated by experiments on rats that the calcium metabolism can be affected by administering follicular hormone (*Bach*).

As shown above, osteoporosis and osteomalacia of the spine are by no means an aetiological entity. The term primary osteomalacia can in fact be applied to the alimentary form found in otherwise healthy persons who subsist on a diet lacking in either vitamin D or mineral salts, or, most frequently, lacking both. This purely primary form, which is entirely dependent on exogenous factors, may, under normal conditions (peace time), be regarded as rare in Europe. The secondary form of osteomalacia, which occurs in cases of disease of the intestinal canal, liver, bile ducts, pancreas, kidneys, or the endocrine system, must, on the other hand, be considered as a complication of these diseases. That the disease of the spine frequently seems

to be the actual disease is due to the fact that the basic disease often shows very vague symptoms, while the spinal disease generally engages the patient's attention to the fullest extent and, in the most severe stages, causes complete invalidity. These secondary forms of osteomalacia, and particularly the milder stages, are not infrequent, as has been shown by publications during the last few years. The exogenous and endogenous factors may, of course, operate simultaneously. It is evident that a secondary bone-disease with, for example, defective absorption of vitamin D and calcium salts by reason of an intestinal disease, will develop more rapidly and attain a more severe stage if the diet is also deficient in regard to these constituents. Conversely, the therapeutic results show that such secondary osteomalacia can be ameliorated by adding a surplus of vitamin D and calcium salts to the diet.

A survey of the case-records of a large number of these patients shows that a trauma is to be found in the anamnesis in a great many cases. This plays an important part in the *differential diagnosis* of certain traumatic diseases. *M. Lance, L. Girard & P. Lance* state in this connection that 32 out of 66 patients with osteoporosis or osteomalacia of the spine had attributed the cause of their disease to be a trauma. In many cases only a slight trauma was referred to, such as driving in a motor-car over uneven roads. Severe traumata in the anamneses were found in other cases (traffic accidents). It is not unusual that the spine which lacks calcium first gives rise to symptoms after a trauma. An osteoporotic vertebra may collapse even after a very slight trauma, thus causing severe symptoms, and the case is then often diagnosed as a primary fracture, the osteoporosis being overlooked or not discovered until a long time after, when the patient's symptoms do not decrease in spite of prolonged immobilisation. Conversely, a trauma, even though it need not primarily have caused a fracture, may secondarily give rise to osteoporosis (*Leriche* and others) localized at first most particularly at the site of the lesion, though it may gradually spread over almost the whole spine. It is possible that a few of the cases described as osteomalacia are actually such cases

of *post-traumatic osteoporosis.* Meanwhile, it is very necessary to discriminate between the two diseases because of the great difference in treatment, apart from the importance inherent in defining the aetiological conditions with regard to insurance claims. It is of importance for the differential diagnosis that post-traumatic osteoporosis only develops gradually, and can, as a rule, be discovered by X-ray examination at the earliest after about a month's time. X-ray examination immediately after the trauma is, therefore, of great importance. Halisteresis is most highly pronounced at the site of the lesion and is usually confined to the spine and possibly also the ribs. From the anatomico-pathological point of view the case is one of osteoporosis, the X-ray picture being characteristic of this form. Contrary to true osteomalacia, this disease seems to be refractory to treatment with vitamin D.

Of other forms of local loss of calcium in the spine, secondary halisteresis with *inflamatory conditions* must be especially mentioned, and principally in *tuberculous spondylitis.* The X-ray result in conjunction with the clinical examination will, however, generally lead to the correct diagnosis. In this respect it may be mentioned that the fixation of the spine hardly ever reaches such an advanced stage in osteomalacia as in tuberculous spondylitis.

A local secondary osteoporosis, even of a severe nature, is also often found with *multiple myelomata,* which may appear in a diffuse form and, when examined by X-rays, may have a misleading resemblance to diffuse osteoporosis of another kind. Attempts to prove the presence of Bence-Jones proteins, which has been claimed to be characteristic of this disease, often fail. On the other hand, the total amount of protein in the plasma is usually greatly increased. *Carcinomatous metastases* may also cause a diffuse loss of calcium in the spine. This applies particularly to carcinomata in the thyroid gland and mamma.

Amongst general bone-diseases that may involve some difficulty in identification from osteomalacia, the so-called *ostitis fibrosa generalisata* (*v. Recklinghausen*) must be particularly emphasized. This disease is known to be due to hyperfunction

of the parathyroid glands by reason of an adenoma in one or more of these glands. In its early stages it manifests itself on X-ray examination by diffuse halisteresis of the bones, including the spine. The cystic transparencies characteristic of this disease often appear only at a later stage. Diagnosis can be determined by the heightened calcium-content of the serum and the increased excretion of calcium in the urine, furthermore, it is possible to demonstrate an increase in the amount of parathyroid hormone in the blood (*Hamilton & Highman*). The last-mentioned phenomenon can, however, also be seen in osteoporosis due to other causes, probably as an expression of a compensatory hyperfunction of the parathyroid glands due to hypocalcaemia. Osteoporosis may also be found, amongst other symptoms (see above), in basophile adenoma of the anterior pituitary (*Cushing's disease*), and it may be particularly pronounced in the spine. General osteoporosis, with increased excretion of calcium in the urine, may be observed in *Graves' disease,* too.

In *'Paget's disease'* the spine is also early attacked. The X-ray picture is, however, fairly characteristic, the cortex thickening simultaneously with the compression of the vertebrae. *Osteopsathyrosis,* or *osteogenesis imperfecta congenita* (*Lobstein*), has so clearly defined and characteristic a pathological picture (blue sclerae, deafness, etc.) that it should be unmistakable.

Chronic arthritis deformans may be accompanied by osteoporosis often furthermore accentuated by contrast with osteophytes and thickening of the edges of the bodies of the vertebrae. It is questionable, however, whether this form of osteoporosis should not also in some cases be considered a deficiency-disease, originating from a disease of the digestive tract.

Lastly, it may be mentioned that a great loss of calcium from the spine can be noted in *myelosis. Bouchut, Levrat & Guichard* thus reported a case of halisteresis of the spine and thorax in a patient who died evincing the signs of acute myeloid leucocamia ('Myelose osteomalacique').

The prognosis of osteoporosis and osteomalacia is serious, if the disease is not treated. As a rule, the disease progresses

year by year and gradually disables the patients to such an extent that they become permanently confined to bed, and death can then ensue from general debility, often with pneumonia as the final stage.

When rationally treated, primary osteomalacia due to diet has a good prognosis, provided treatment is begun prior to the development of serious deformities, which otherwise become permanent. Slight deformities can be cured spontaneously during treatment (see *Gargill, Gilligan & Blumgart*'s cases).

The prognosis of secondary osteomalacia must be regarded as more doubtful and is to a great extent dependent on the course of the primary disease. In cases due to defective absorption from the intestinal canal the prognosis depends on the degree to which it is possible to compensate reduced absorptional ability by means of a surplus of the substances lacking in the diet (chiefly vitamin D).

Treatment: The treatment of primary alimentary osteomalacia must first of all consist of a correction of the diet in respect of the deficiencies, together with the administration of calcium preparations and vitamin D, which, as a rule, cure the disease in a comparatively short time (1—2 months).

Vitamin D must be given at the same time, in doses which, of course, must vary somewhat in relation to the aetiology of the disease. It appears therefore that smaller doses are sufficient in alimentary osteomalacia than those used for the forms due to defective absorption from the intestinal canal, in which a considerable surplus must be given to produce the desired effect. In both these forms it is necessary to remember that the patient may also show a negative balance in regard to other vitamins, such as vitamin C (*Meulengracht*'s case), or vitamin A (*Denstad*'s case and the author's case no. II), and to adjust the treatment accordingly.

A complication which must also be reckoned with is that the lumbar spinous processes often come to rest upon each other on account of the compression of the bodies. Pains may thus be caused (*Baastrup*) which may disguise a possible improvement

in the condition. In milder cases the pains can be eliminated by injections of novocaine between the spinous processes. In more severe cases it may perhaps be necessary, when the osteomalacial process has been cured, to chisel off part of the edges of the spinous processes, thereby increasing the distance between them.

As regards osteomalacia due to diet, the cure is permanent provided that lasting deformities have not occurred and that the diet is always adequate. Cases of the secondary form in which the primary disease is incurable require, however, the maintenance of a constant addition of a considerable surplus of vitamin D and of calcium salts, otherwise the disease of the bones recurs.

Three cases of osteoporosis and osteomalacia, localized principally in the spine, are described below. The cases were under observation at the Orthopaedic Hospital in Copenhagen (nos. I and II) and at Bispebjerg Hospital Department A. (no. III).

I. Journ. no. O. H. 5667/38. Woman aged 53 years. Healthy in childhood and whilst growing up.

Menses from 13th year, always regular, normal. Menopause in 36th year, for no external cause, and accompanied by severe climateric discomfort. Never pregnant.

1907 (23 years old) operated on for oophoritis, and at the same time a benignant growth was removed from the left breast.

1909 operated on for adhesions after the first operation. A couple of years later an attack of ileus, cured without operation. *Total lack of acidity in the gastric juice was apparently ascertained at that time.*

1922 treated for enteritis, and achylia diagnosed at the same time. Kept diet 1 year. Never previously suffered from dyspepsia.

1923 treated in a nursing-home for 'muscular rheumatism' in the back. Has since been subject to *'fatigue in the back'*.

1924. Broken rib after fall with cycle.

1926. Rheumatic fever, mainly in the joints of the feet. Sent to hospital where she received, amongst other treatments, massage, because she had pains in the back at the same time. *During massage of the back fracture of a rib is said to have occurred.*

1929 again broken rib after fall with cycle.

1930. Articular rheumatism, chiefly in the feet.

1931. Fracture of the right radius after slight trauma 'tried to prevent herself from falling with her hand'.

1937 in hospital for cysto-pyelitis which disappeared after treatment with amygdalic acid mixture.

The patient herself states that the present disease commenced in January 1934 in the following manner: Whilst in the act of lifting a wash-tub full of clothes, she suddenly felt a violent pain in the lower part of her back, 'as though a stick broke inside her'. She could not hold herself erect, and, after having stayed in bed at home for a couple of

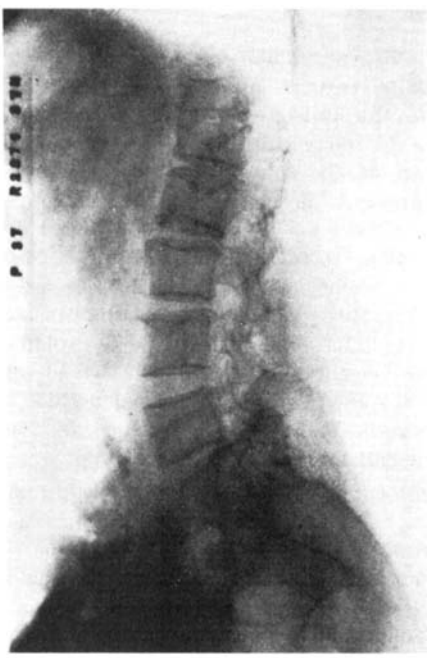

Fig. 3.
Case no. 1. Columna lumbalis.

days, she was sent to hospital, where X-ray examination a week later revealed a fracture of the first lumbar vertebra. (Study of these pictures shows also considerable halisteresis of the spine). Treated by lying in plaster of Paris for 4 months and then with supporting corset, but with no noticeable improvement. Pains in the back continued, worst in sitting-position and very particularly when she had to bend forwards. Could at the utmost walk half-an-hour with a stick. Was totally unfit for work.

Examination by a surgical specialist in August 1936 showed: Pt. pale. Looks older than her age. Walks carefully. Back straight. Direct tenderness corresponding to lumbar vertebra I. No indirect tenderness. Slight

projection of the spinous process above. Movements somewhat restricted, but no particular stiffness of the diseased region. The loin muscles rather tender, but not tense. A small scar on the left breast from the above-mentioned operation. No formation of tumours. Further physical examination discovered nothing abnormal. Toilet urine: ÷A, ÷S, contained numerous leucocytes and bacteria. X-ray examination: Compression-fracture of lumbar vertebra I with slight displacement. Entire spine extremely deficient in calcium. One shin-bone, one fore-arm, and the skull also deficient in calcium.

Towards the end of August 1937 the condition suddenly became worse, but without preceding trauma, and 13/9/37 the patient was admitted to the Orthopaedic Hospital. Although the pains had previously been mainly confined to the lumbar region, she now also complained of pains in the thoracic part of the spine and in the shoulders, as well as of weakness in the arms. A physical examination showed practically the same conditions as in 1936, but there were now added direct and indirect tenderness corresponding to thoracic vertebra VIII. No deformity. X-ray examination of the spine showed a healed compression-fracture of thoracic vertebra VIII (even in 1934 it was slightly wedge-shaped) and wedge-shaped deformity of lumbar vertebra I. Prolapse of the nucleus pulposus in lumbar vertebrae I, II, and IV. Considerable diffuse halisteresis of the whole spine. The pelvis showed nothing abnormal beyond considerable halisteresis. The thigh-bones, hands, feet, and the skull also showed pronounced halisteresis.

Other examinations: Sahli: 72—87. Sedimentation reaction: 9—6 mm./1 hr. W.R.: ÷. G.R.: ÷. Serum-Ca.: 11.00 mgr. %. Plasma-P.: 3.63 mgr. %. Ewald: 0/30 in 90 ccm. Kemp: ÷ retention. Faeces: ÷ Benz. (4 times). X-ray of stomach and duodenum: nothing abnormal. Urine: ÷ alb. Nothing of note from microscopic examination.

From detailed questioning about the patient's diet it appeared that this had been good and ample. Contained ample meat, fish, and vegetables, as well as fresh fruit. On the other hand, since 1922 the patient had neither drunk milk nor eaten milk-food. Apart from the year 1922 the patient had not kept to a diet. Except for short intervals, had taken hydrochloric acid drops or citric acid tablets since 1910. Stated that she 'could stand the food better' when she took them. Consumption of tobacco and spirits negligible.

The patient was primarily treated with decamin and later on with spinatin. Folliculin preparations were given at the same time. The result was, however, very doubtful. From 1/2/38 the treatment was changed to the injection of Holtz' A. T. 10 (2 ccm. twice a week) together with intravenous injection of calcium gluconate, the calcium content of the serum being frequently checked. The patient then improved rapidly and could be discharged from hospital 29/3/38. During the first part of the

treatment the patient lay in plaster of Paris. She was subsequently allowed up with a supporting corset, and was discharged from hospital with this. Her condition on discharge was considerably better. Pains in the back only with particular movements. After leaving hospital she continued with decamin fortior 15 drops a day. On post-examination

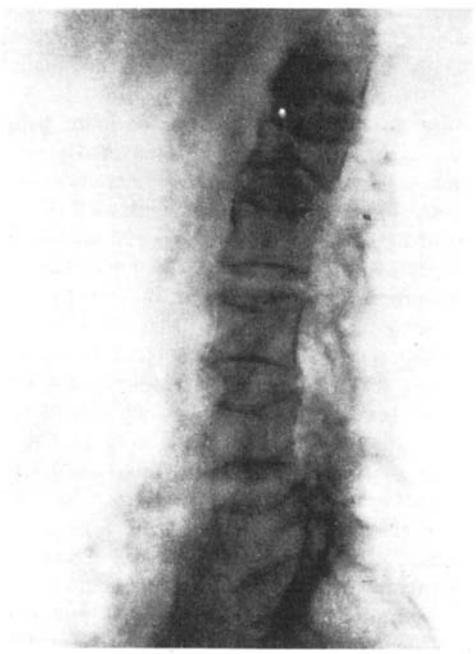

Fig. 4.
Case no. II. Columna lumbalis—(the contours of the bodies have been touched up with pencil).

29/8/38 the following entry was made in the journal 'very satisfied with the back, no complaints, feels well'.

II. Journ. no. O. H. 1446/38. Man, aged 58 years, operated on 25 years ago in nursing-home for supposed disease of the stomach. Nothing abnormal appears to have been discovered by the operation, of which it has not been possible to obtain further information. The pains, however, were relieved after operation but since then he has periodically suffered from attacks of dyspepsia. During last 25 years he has repeatedly been given test meals and has each time been informed that there was 'slight catarrh of the stomach', on account of which he has kept to a diet for short periods. For the last 5 years he has suffered from distension of

the stomach, has been troubled with eructation, sour belching, and periods of flatulence. He has generally been constipated, with periods of diarrhoea.

Apart from the periods of diet, which seem to have been of fairly short duration, his diet has consisted of ordinary mixed food, with milk-food, cereals, and ample meat and fish. He has always eaten butter, but has not drunk milk. Vegetables and fruit, however, have been eaten in only small quantities. He has not smoked tobacco for the last 3 or 4 years, but previously smoked 7 or 8 cigars a day. Occasionally drinks a little red wine at meals but has not taken any spirits for the last 6 years.

For many years he has suffered from periodic pains in the back. These pains have nearly always been situated in the middle part of the spine and have not radiated. The pains gradually increased year by year, but became suddenly worse 2 years ago after bending forwards. 28/4/37 admitted to a medical side for this disease. X-ray examination of the spine showed that lumbar vertebra III was lower than the others, and this was considered to be the result of an old fracture. He was treated with heat and massage, and at the same time his dyspepsia (colitis) was treated with laxative puree diet, bismuth, and laxantia. His back did not improve, however, and two months previous to his being admitted to the Orthopaedic Hospital his condition became considerably worse, for, whilst bending forwards, he felt something snap in his back and simultaneously had severe pains across the loins. Lay in bed a fortnight, during which he was treated with heat and massage, but without result.

On admission to the Orthopaedic Hospital 25/2/38 the physical examination showed the following: The patients complexion grey-pale, elderly appearance (temperature, which had been normal before admission, was, strangely enough, 40.3° C. on admission, but was afterwards normal and remained so throughout his stay). The objective examination revealed nothing else abnormal excepting slight tenderness in the left lumbar region. X-ray examination of the spine showed a rather considerable flattening and bi-concave deformation of the upper lumbar vertebrae, and, to a lesser degree, of the lower thoracic vertebrae, which were more wedge-shaped. The lower lumbar vertebrae had slightly fish-vertebral form. The spinous processes were very close to each other in the lumbar group. In addition, mild spondylosis was found. The whole spine was acutely osteoporotic the pelvis to a lesser degree. The hip-joints were normal. The remainder of the bone system also seemed to be rather lacking in calcium, although to a considerably lesser extent.

Blood-test: Sahli: 105. Index: 1.16. Red corpuscles 4.54 mill. White corpuscles 7440. The red blood picture was normal. Differential count:

49 % neutrophile leucocytes
5 % eosinophile leucocytes
43 % lymphocytes
3 % monocytes

Formogel reaction: ÷. Blood pressure: 155/95. Urea in blood: 28 mgr. %. Blood sugar (fasting): 0.065 %.

Ewald: Congo 45, phenolphthalein 65. Faeces: ÷ Benz. (3 times), ÷ mucus, ÷ fat. Urinary diastase: 32.

The serum-Ca. was determined many times and varied from 9.6 to 11.0 mgr. %. Plasma-P.: 4.87—5.76 mgr. %.

Ophthalmological examination revealed nothing abnormal beyond a marked reduction in distinguishing-power (Clemmesen—Edmund). This disturbance, however, was eliminated within a week by daily injections of vitamin A (A-vital).

The patient was treated with light diet rich in vitamins, charcoal tablets, Holtz's A. T. 10 (parenterally at first and then perorally), the serum-calcium being checked frequently, vitamin A and D preparations (Decamin and A-vital), carbon arc-light, and a plaster of Paris jacket. In addition, injections of novocaine were given between the spinous processes in the lumbar region. He improved greatly during the treatment. Three months after admission he was allowed up, with a supporting corset. Could then bend forwards without pain and 'the man, who when admitted had aged greatly in appearance, now looks much younger'. Discharged 21/6/38.

III. Journ. no. B. B. H. Department A. 3177/38. Woman, 72 years old. Has given birth to four children, the last born 43 years ago. Menopause at the age of 45. During last four years has periodically suffered from phlebitis in the legs, together with undefined rheumatic attacks (pains in left knee and across the loins). Otherwise the patient has previously been quite well.

17/8/38. The patient slipped in the street, fell down on her left side and felt pain in right side of the chest as a result. On admission to Bispebjerg Hospital, 2 days later, X-ray examination showed that the fifth rib on the right side was fractured. There was considerable halisteresis of all the ribs. X-ray examination of the spine also showed severe halisteresis of the whole spine with rather serious deformity of several vertebrae. Some of the thoracic vertebrae were very much flattened, either wedge-shaped or slightly bi-concave. Somewhat pronounced nuclear prominences were found in the bodies of thoracic vertebra XII and lumbar vertebra II. There were, in addition, slight spondylitic changes. The pelvis was also halisteretic, althoug there did not appear to be halisteresis of the extremities.

She reported that she ate ample mixed food (though never drank milk). No dyspeptic attacks. Always very constipated. For the last 3—4 years she had taken Kruschen salts in the morning.

Tabl. calc. lact. gr. 1 × 3 daily, and vitamin D preparations (Ultranol)

20 drops, twice daily, were prescribed, but the patient ceased to take these medicines immediately she left hospital 29/8/38.

Re-admitted 3/11/38 on account of a genital prolapse that was treated with a pessary, which, however, had to be removed as it caused her discomfort. Continually complained of pains across the loins. The pains

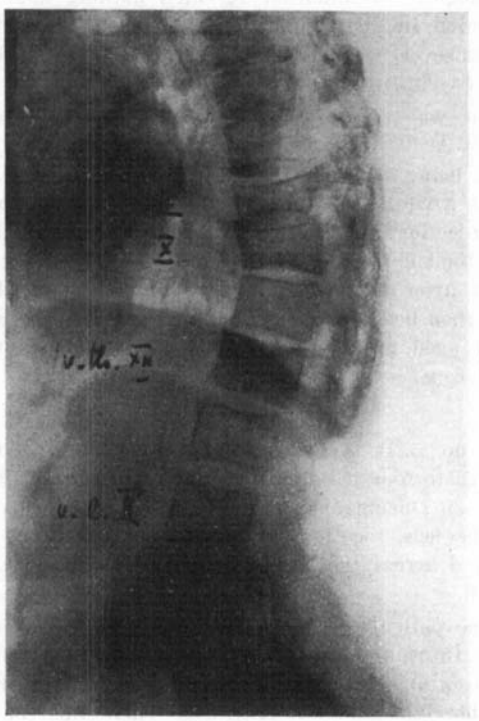

Fig. 5.
Case no. III. Columna thoraco-lumbalis—(the contours of the bodies have been slightly emphasized with pencil).

were most pronounced in the right lumbar region and were felt particularly when rising from a sitting position. X-ray examination of the spine showed the same conditions as when previously admitted.

Other examinations: Serum-Ca.: 10.1 mgr. %. Plasma-P.: 6.6 mgr. %. Plasma-protein: 6.96 %. Plasma-phosphatase: 99 units/100 cm. Sahli: 105. Index 1.17. Red blood-corpuscles 4.24 mill. White blood-corpuscles: 4200. Differential count:

Neutrophile leucocytes	48 %
Eosinophile leucocytes	1 %
Lymphocytes	40 %
Monocytes	5 %
Transitional forms	6 %

Blood pressure: 150/80. Sedimentation reaction 24 mm./1 hr. Urine: ÷ A, ÷ S. Microscopic exaniination: nothing abnormal. Ewald: 31/52 in 35 + 47 ccm. and 29/48 in 19 + 13 ccm. Apart from the first test, the stools showed only traces of blood for some days (coincident with test meals), only once traces of catalase and no mucus, on the other hand drops of fat were constantly found on microscopic examination (medium diet, without meat). There were, however, no macroscopic fatty stools.

Tabl. calc. lact. 1 gr. daily and vitamin D preparations (Ultranol 20 drops twice daily), were again prescribed. Discharged 17/11/38. The pains in the back were then quite unchanged.

Post-examination 17/12/38, 'has taken her medicine regularly. The pains in the back have almost disappeared. Looks healthy and well. Spinal movement normal for her age. No pains when bending forwards'.

A considerable deficiency of calcium in the spine was common for the above-mentioned patients. Regarding the first two patients, in whose cases the remainder of the skeleton was also clearly halisteretic, the X-ray picture proved to be practically equivalent to the previously described picture, which is characteristic of the osteoporotic changes, whilst in the case of the last patient, where the changes were mainly confined to the spine and the pelvis, the deformations of the vertebrae characteristic for the osteomalacial form were also found. Although the patients were not so thoroughly and so systematically examined as might have been desired, the diagnosis may, however, be considered as correct.

The diet of all three patients showed no noticeable deficiencies. Certainly, none of the patients drank milk, but their food was, on the whole, good and ample, quite on a par with the average food-standard in this country. The presence of other aetiological factors in the case of all three patients must, therefore, be suspected.

In regard to the first patient, attention was immediately given to the patient's achylia, which appears to have been

diagnosed as early as in 1909. Her dyspepsia became worse in 1922, and, after having kept to a diet for a year, she began to get pains in her back. Subsequently she suffered from a series of fractures, of which at least one, the fracture of the rib, must be described as a spontaneous fracture, since it was caused by such a slight trauma as massage of the back. It is difficult to say anything about the importance of the menopause which occurred at as early an age as 36 years. In comparison with previously published cases of osteomalacia in connection with amenorrhoea, there is a suspicion, also in this case, of a certain relation between the two phenomena (see above).

The second patient had no achylia, but this patient, too, had suffered for many years from attacks of dyspepsia, which had become especially severe during the last five years. In this case, too, the intestinal disease must certainly be regarded as the cause of the spinal disease, on account of defective absorption of vitamin D and calcium. It is noteworthy that this patient had, in addition, pronounced A-vitaminosis.

As regards the last patient, it has been impossible to show any definite cause of her osteomalacia. No actual food-deficiency could be proved. Except for constipation there were no dyspeptic complaints. There was no achylia. The plasma-phosphorus was increased, but the calcium content in the serum was normal. The phosphatase value was very high, but the importance of this does not seem quite clear. Microscopic examination of faeces constantly showed drops of fat, although there were no fatty stools visible macroscopically. It is possible, however, that defective digestion of fat may have contributed to the onset of her disease. On the other hand, proof of any definite aetiological factor has not been forthcoming, and the case must probably be classified amongst the as yet mysterious cases of senile osteomalacia.

The quantity of calcium and of phosphorus in the blood was not decreased in any of these cases. It seems as though the concentration of these substances in the blood is upheld at the expense of the osseous system. With reference to the last two patients, differential counts of the white blood-corpusles were

carried out, and, in accordance with earlier records in the literature of the subject, relative lymphocytosis was found in both cases.

The beneficial effect of treatment with vitamin D and calcium salts is characteristic for all three patients. In spite of the subjective improvement in condition, X-ray examination failed to show a correspondingly increased calcium content in the spine. Incidentally, it must be taken into consideration that a very pronounced deposit of calcium in the osseous system must occur before it can be revealed by means of X-rays.

RÉSUMÉ

Il est fait une description de la maladie ordinairement connue sous les noms d'osteoporosis ou d'osteomalacia columnae. Après avoir fait mention des caractéristiques et des symptômes, l'étiologie de cette maladie est plus minutieusement étudiée. Dans sa forme primaire, cette maladie consiste presque toujours en une déficience diététique (le plus souvent avitaminose D), tandis que dans sa forme secondaire, on peut en faire remonter l'étiologie en partie à des troubles d'absorption (maladies des voies intestinales, du foie, des voies biliaires et du pancréas), en partie à des troubles d'excrétion (maladies des reins) et enfin à des troubles de la sécrétion interne. Dans la forme due aux troubles d'absorption, l'absorption défectueuse des vitamines D et peut-être aussi des sels de chaux en sont les facteurs les plus ordinaires.

Après avoir brièvement décrit le diagnostic différentiel, le pronostic et le traitement de la maladie, il est rendu compte de trois cas d'osteoporose et osteomalacie, localisés principalement dans la colonne.

I. Femme âgée de 53 ans. La maladie était accompagnée de ménopause apparue très tôt (à l'âge de 36 ans), ainsi que d'achylie qui semble être la cause de la maladie osseuse.

II. Homme âgé de 58 ans. Ce malade souffrait simultanément d'une avitaminose A (diminution de la faculté de percevoir)

guérie à la suite de l'administration de vitamine A. La cause de la maladie osseuse semblait être une dyspepsie (gastritis et enterocolitis) de longue durée.

III. Femme âgée de 72 ans, avec des altérations ostéomalaciques typiques dans la colonne. La cause de celles-ci ne fut pas entièrement éclaircie, mais elles s'expliquent peut-être par une digestion défectueuse des graisses.

SUMMARY

A description is given of the disease usually termed osteoporosis or osteomalacia columnae. After mention of the characteristics and symptoms, the aetiology of the disease is more fully dealt with. This, in the case of the primary form, nearly always consists of a defect in diet (most frequently D-avitaminosis), whilst the aetiology of the secondary form may partly depend on disturbances in absorption (in diseases of the intestinal canal, liver, bile ducts, and pancreas), partly in excretion (kidney-diseases), and, finally on disturbances of the internal secretion. In the form due to disturbances of absorption, defective absorption of vitamin D and possibly also of calcium salts, is the most usual factor.

After a short description of the differential diagnosis, prognosis, and treatment, a report is given of three cases of osteoporosis and osteomalacia, principally confined to the spine.

I. Woman, 53 years old. The disease was accompanied by early incidence of menopause (in 36th year) together with achylia, which is presumed to be the cause of her bone-disease.

II. Man, 58 years old. This patient had a A-avitaminosis simultaneously (decreased distinguishing ability) which became normal after the administration of vitamin A. The cause of his bone-disease was supposed to be dyspepsia (gastritis and enterocolitis) of long duration.

III. Woman, 72 years old, with typical osteomalacial changes in the spine. The cause of these was not quite clear, but they might possibly be explained by defective digestion of fat.

ZUSAMMENFASSUNG

Es wird eine Beschreibung der Krankheit gegeben, die gewöhnlich als Osteoporosis oder Osteomalacia columnae bezeichnet wird. Nach Besprechung ihrer Eigentümlichkeiten und Symptome wird die Ätiologie dieser Krankheit genauer behandelt. Diese besteht bei der primären Form fast immer in einem Diätmangel (meist D-Avitaminose), während die Ätiologie bei der sekundären Form zum Teil in einer Störung der Absorption (in Krankheiten des Intestinalkanals, der Leber, der Gallenwege und des Pankreas), zum Teil in einer Störung der Exkretion (Nierenkrankheiten) und schliesslich in einer Störung der inneren Sekretion bestehen kann. Bei der Form, die auf einer Absorptionsstörung beruht, ist eine mangelhafte Absorption von Vitamin D und vielleicht auch von Calcium-Salzen der gewöhnlichste Faktor.

Nach einer kurzen Erwähnung der Differentialdiagnose, Prognose und Behandlung wird über drei Fälle von Osteoporosis und Osteomalacia berichtet, die hauptsächlich auf die Wirbelsäule beschränkt waren.

I. Frau, 53 Jahre alt. Die Krankheit war von einem verfrühten Eintritt der Menopause (im 36. Jahre) begleitet, in Verbindung mit einer Achylia, die vermutlich die Ursache ihres Knochenleidens ist.

II. Mann, 58 Jahre alt. Dieser Patient hatte gleichzeitig eine A-Avitaminose (verminderten Unterscheidungsvermögen), die nach Verabreichgung von Vitamin A beseitigt wurde. Man nimmt an, dass die Knochenerkrankung durch eine Dyspepsie (Gastritis und Enterocolitis) von langer Dauer verursacht war.

III. Frau, 72 Jahre alt, mit typischen osteomalazischen Veränderungen der Wirbelsäule. Deren Ursache war nicht ganz klar, sie sind aber vielleicht durch eine mangelhafte Fettdigestion zu erklären.

BIBLIOGRAPHY

1. *Adams, M., Boothby, W. M. & Snell, A. M.:* J. Am. Med. Ass. 104, 252, 1935.
2. *Albright, F., Drake, T. G. & Sulkowitch, H. W.:* Bull. John Hopk. Hosp. 60, 377, 1937.

3. *Alwens:* Münch. med. Woch. 66, 1071, 1919.
4. *Baastrup, Chr. L.:* Fortschr. Geb. Röntgen. 48, 430, 1933.
—: Journ. rad. d'electrol. 20, 78, 1936.
5. *Bach, E.:* Klin. Woch. 16, 280, 1937.
6. *Baron, A. & Barsony, Th.:* Fortschr. Geb. Röntgen. 36, 377, 1927.
7. *Bauer, W., Albright, F. & Aub, J.:* J. Clin. Invest. 8, 229, 1930.
8. *Bauer, W. & Marble, A.:* J. Clin. Invest. 11, 21, 37, 1932.
9. *Bohne, O.:* Zeitschr. orthopäd. Chir. 50, 764, 1929.
10. *Bouchut, L., Levrat, M. & Guichard, A.:* Le Sang 8, 925, 1934.
11. *Brandt, H.:* Bruns Beitr. klin. Chir. 164, 354, 1936.
12. *Bulmer, E.:* Lancet 228, 741, 1935.
13. *Castleman, B. & Mallory, T. B.:* Am. J. Pathol. 13, 553, 1937.
14. *Christiansen, J.:* Tidsskr. norsk Lægefor. 55, 1167, 1935.
15. *Coryn, G.:* Presse med. No. 13, 228, 1938.
16. *Curshmann, H.:* Deutsch. Arch. klin. Med. 129, 93, 1919.
17. *Dalyell, E. J. & Chick, H.:* Lancet 201, 842, 1921.
18. *Dam, E.:* Acta med. scand. 68, 574, 1928.
19. *Denstad, T.:* Nord. med. Tidsskr. 15, 514, 1938.
20. *Dieckmann, W.:* Am. J. Obst. Gyn. 23, 478, 1932.
21. *Eisler, F. & Hass, J.:* Wien. klin. Woch. 34, 55, 1921.
22. *Goehlinger, H. & Becart, A.:* Presse med. No. 24, 452, 1938.
23. *Gargill, S. L., Gilligan, D. R. & Blumgart, H. L.:* Arch. Int. Med. 45, 879, 1930.
24. *Golden, R. & Abott, H.:* Am. J. Roentgen. Rad. Therap. 30, 641, 1933.
25. *Govaerts:* Le Scalpel Jan. 1938. Cit. by M. Lance. L. Girard & P. Lance.
26. *Guérin, R.:* J. Med. Bordeaux 115, 405, 1938.
27. *Gøthgen, E. W.:* Bibliot. Læger 127, 39, 1935.
28. *Hamilton, B. & Highman, W. J.:* Nord. med. Tidsskr. 15, 441, 1938.
29. *Hannon, R. R., Liu, S. H., Chu, H. J., Wang, S. H., Chen, K. C. & Chou, S. K.:* Chinese med. Journ. 48, 623, 1934.
30. *Hoffmann, R.:* Wien. Arch. inn. Med. 4, 91, 1922.
31. *Horwitz, J. B.: J.* Missouri State Med. Ass. p. 169, 1938.
32. *Hunter, D.:* Lancet 228, 1025, 1935.
33. *Imhäuser, W.:* Deutsch. med. Woch. 62, 677, 1936.
34. *Johansen, E. Hecht:* Bibliot. Læger: 125, 379, 1933.
35. *Jung, M. A.:* Rev. Chir. No. 7. 473, 1937.
36. *Junghans, H.:* Arch. klin. Chir. 165, 303, 1931.
37. *Kehrer, E.:* Zeitschr. Geburth. Gyn. 116, 141, 1938.
38. *Khoôr, O.:* Endokrinologie 2, 40, 1928.
39. *Kleinberg, S.:* Arch. Surg. 30, 30, 1935.
40. *Kreuzer, H.:* Zeitschr. orthop. Chir. 51, 463, 1929.
41. *Lance, M., Girard, L. & Lance, P.:* Rev. d'orthoped. et de chir. 25, 385, 1938.

42. *Leitch, J.:* Nutrition Abst. & Rev. 6, 553, 1936—37.
43. *Loewy, G.:* Presse med. Nr. 89, 1627, 1931.
44. *Lyon, E.:* Am. J. Digest. Diseases & Nutrition 4, 332, 1937.
45. *Margitay-Becht, A. & Gömöri, P.:* Klin. Woch. 15, 939, 1936.
46. *Maxwell, J. P.:* Chin. med. Journ. 37, 625, 1923.
—: Nutrition Abst. & Rev. 4, 1, 1934.
—: Proc. Roy. Soc. Med. 23, 639, 1930.
—: Proc. Roy. Soc. Med. 28, 265, 1935.
47. *Meisels, E. L.:* Fortschr. Geb. Röntgen. 43, 735, 1931.
48. *Meulengracht, E.:* Ugeskr. Læger 100, 633, 1091, 1936.
49. *Meulengracht, E. & Meyer, A., Rothen:* Ugeskr. Læger 98, 961, 1936.
— — —: Acta med. scand. 92, 584, 1937.
50. *Miles, L. M. & Feng, C.:* J. Exp. Med. 41, 137, 1925.
51. *Moffat, B. W.:* J. Bone & Joint. Surg. 15, 679, 1933.
—: Arch. Surg. 28, 1095, 1934.
52. *Morville, P. & Müller, P.:* Hospitalstid. 76, 970, 1933.
53. *Møllgaard, H.:* Ugeskr. Læger 96, 565, 1934.
54. *Paliard, F., Guichard, A., Muller, B. & Viallier, J.:* Lyon. Méd. 160, 360, 1938.
55. *Pappenheimer, A. M. & Wilens, S. L.:* Am. J. Pathol. 11, 73, 1935.
56. *Paviot, J., Enselme, J. & Guichard, A.:* Lyon Méd. 156, 745, 1935.
57. *Polgar, F.:* Zeitschr. Geb. Röntgen. 56, 208, 1937.
58. *Riese, H.:* Münch. med. Woch. 80, 600, 1933.
59. *Rosenfeld, W.:* Med. Klin. 26, 421, 1930. Cit. by Riese.
60. *Rutishauser, E. & Maulbetsch, A.:* Beitr. pathol. Anat. u. z. alg. Pathol. 94, 332, 1934—35.
61. *Schlesinger, H.:* Wien. klin. Woch. 32, 336, 1919.
62. *Schultzer, P.:* Hospitalstid. 76, 417, 1933.
—: Ugeskr. Læger 95, 23, 1933.
—: Am. J. Med. Science 186, 532, 1933.
63. *Scipiades, E.:* Zeitschr. Geburtsh. Gyn. 81, 156, 1919.
—: Zentralbl. Gyn. 48, 1885, 1924.
64. *Secher, K.:* Acta med. scand. 97, 335, 1938.
65. *Seneque, J.:* Presse med. 36, 516, 1928.
66. *Starlinger, W.:* Deutsch. med. Woch. 53, 1553, 1927.
67. *Stepp, W.:* Münch. med. Woch. 83, 1119, 1936.
68. *Strümpell:* Lehrb. spez. Pathol. Therap. inn. Krankh. Vol. II. p. 164, 1926.
69. *Wagoner, G.:* Am. J. Med. Science 191, 511, 1936.
70. *Weissenbach & Lievre:* Soc. med. hop. 12/7—1933. Ref. in J. Am. Med. Ass. 106, 55, 1936.
71. *Wilder, R. M. & Wilbur, D. L.:* Arch. Int. Med. 57, 422, 1936.
72. *Wright, S.:* Applied Physiology. p. 212. Oxford Med. Publications 1934.