

ENCHONDROMA-LIKE FORMATIONS IN THE FEMUR, FOLLOWING OSTEOMYELITIS.

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OBSERVATIONS on pathological elongation and shortening of the long bones, associated with luxations, osteomyelitis, arthritis, aneurysm, angiectasia, infantile paralysis, and other diseases have led to interesting discussions as to the mode of growth of bones. Contrary to the experience of Duhamel, Hunter, Flourens, and others, Wolff¹ found that pins or marks inserted at measured intervals, in the shaft of growing long bones, had the distances between them slightly increased. More recently Marchand has demonstrated expansive growth of bone under certain conditions, but it is generally recognised that the chief seat of length-growth of long bones is in the intermediate cartilage.

Bidder, Jahn, Nové-Josserand, and others, have shown that injury to the intermediate cartilage in young animals by means of excision, incision, or the insertion of pins, leads to shortening of the bone, so that by injury to one side or the other (Bidder), genu varum or genu valgum may be produced at will.

Paget says: "When any of the long bones of a person who has not yet attained full stature is the seat of disease, attended with unnatural flow of blood in or near it, it may become longer than the other or more healthy bone." Helferich observed increased length-growth of bones in children from venous congestion, produced by an elastic band, and utilised this observation for the purpose of producing compensation in shortened limbs.

According to Schmidt, under the conditions which cause an early destruction of the growing cartilage in man, acute and chronic osteomyelitis, traumatic epiphysis separation, and chronic inflammation of the joints play the chief rôle.

The studies of various authors have led to the generalisation that elongation is more rare and shortening more frequent, in proportion as the irritation approaches the intermediate cartilage, whilst if it is involved an arrest of development or a less marked growth occurs.

¹ Schmidt, *loc. cit.*

The following case of healed osteomyelitis, with shortening of the femur is chiefly of interest, on account of an enchondroma-like formation situated in the shaft, and probably representing the remains of the intermediate cartilage:—

CASE 1 (Museum Preparation, No. 3211).—J. M., male, æt. 18, died in the Good Samaritan Hospital in Prague on the 8th January 1900. Post-mortem examination was made by Dr. v. Ritter on the 10th January.

PATH. ANAT. DIAGNOSIS.—*Otitis media suppurativa dextra; necrosis ossis petrosi dextri; thrombosis ichorosa venæ jugularis dextræ; abscessus metastatici pulmonum; pleuritis suppurativa bilateralis; tumor lieinis acutus; degeneratio parenchymatosa; pyohæmia; endocarditis recens ad valvulam mitralem; icterus universalis; chondroma ossis femoris dextri; hyperostosis externa et interna ossis femoris dextri post necrosim.*

ABSTRACT FROM THE AUTOPSY RECORDS.—Strongly-built, well nourished man, with jaundice of the skin and sclera. Rigor mortis is marked only in the lower extremities.

Behind the lobe of the right ear is an incision, beginning at the mastoid process and extending 5 cms. downward, in the depth of which is an area 1 cm. long, in which the mastoid process is visible and free from periosteum. The bone in this area appears rough.

The skull measures 52 cms. in circumference, and is of medium thickness. The meninges are pale. The vena jugularis interna dextra contains a putrid thrombus as far as the jugular foramen, around which, and over the os petrosum, the dura mater is greenish. The dura is otherwise normal, the sinus is nowhere thrombosed. The synostosis between the os petrosum and os basilaris is strikingly porous. The inner meninges and brain are moist, pale. The mucous membranes of the pharynx and larynx are pale; the thyroid gland hyperæmic, of normal dimensions.

In the pleural cavities is about 1 litre of putrid fluid. The pleura visceralis is covered by a fibrinous layer. Near the periphery of the lungs are numerous walnut-sized foci, filled with pus, and around these the lung parenchyma is hard and hyperæmic. The peribronchial lymph glands are anthracotic.

The heart is of normal size. On the valvula mitralis are fresh, yellowish-red, pinhead-sized, soft nodules. Foramen ovale patent, and admits a thick sound. The mucous membrane of the œsophagus is pale. The liver is large, brittle, and pale.

The spleen is markedly swollen, and on its under surface shows three hazelnut-sized infarcts. The kidneys are pale and rich in fat.

The mucous membrane of the stomach and intestines is pale.

The pancreas and adrenal glands are normal.

The right leg is 4 cms. shorter than the left. On the inner side of the lower third of the right thigh is a red coloured radiating scar, 10 cms. long and firmly grown to the bone. On the middle of the anterior surface is a second scar, 3 cms. long and about 4 cms. above the former. The distal half of the diaphysis of the os femoris feels distinctly thickened. After removal it was sawn through the middle in a sagittal direction. The upper half of the thickened portion of the os femoris is completely sclerosed, and the whole cut surface filled by dense compact bone, extending diagonally upward and forward. The sclerosed portion measures 8 cms. in the length direction of the bone, and its diameter is 44 mm. against 33 mm. just above it. The lateral diameter of the femur just above the condyles is 65 mm., in the middle of the femur 32 mm.

Near the lower end of the diaphysis is a bluish-grey mass of cartilage, situated in the spongy portion of the bone to the right of the mid-line, and

nearer the anterior than the posterior surface of the bone (Fig. 1). It measures 3.3 cms. in the length direction of the bone, and is 1.5 cm. broad. The tumour is from 4 to 6 cms. above the surface of the joint, and from 1.5 to 3.5 cms. above the line of union of the diaphysis and epiphysis, the synostosis of which is complete. It is surrounded by an area of osteoporosis, measuring about 1 cm. In the sclerosed portion of the bone are three white pea-sized areas of soft consistence on the median sagittal section. After removal of the periosteum the lower half of the femur appears extremely rough on the surface, and deep furrows are left by the blood vessels. In the middle of the posterior surface of the femur, 10 cms. above the lower articular surface, is an opening 6 by 11 mm. in diameter, which passes upward and forward in the direction of, and along the lower border of, the sclerosed portion. This opening evidently represents a former cloaca. Another small pea-sized opening on the postero-internal surface of the bone communicates with the larger perforation. The cloaca was filled by the ingrowth of cicatricial connective tissue from the periosteum, which was sclerosed and thickened over the lower half of the femur.

No clinical datum was obtainable in regard to the duration of the osteomyelitis, *i.e.* necrosis of the right femur. No evidence of remains of rachitis was to be made out on the skeleton.

Portions of the bone and tumour corresponding to the conditions noted above were decalcified in 5 per cent. nitric acid with 5 per cent. formalin, embedded in celloidin, and stained with hæmatoxylin and eosin and by van Gieson's method.

The microscopical examination shows that the cartilage is surrounded by fat marrow and by an area of extreme osteoporosis, which is broader near the centre of the shaft than on the outer side, where the tumour is nearer the substantia compacta.

Numerous multinuclear osteoclasts are present in this area, but especially at the ends of the bone trabeculae. The cartilage is of the hyaline variety, and on the side towards the substantia compacta is poor in cells and has undergone partial mucoid degeneration. The degener-



FIG. 1.—For description, see page 239.

ated portions show fine fibrillation. The cartilage cells are irregular, stellate, and many are without cell capsules. There is no evidence of a columnar arrangement of the cells. There is practically no deposition of lime, and no bone formation. The cartilage is not vascularised.

On the side of the tumour towards the centre of the shaft the cartilage is richer in cells, and here and there bone trabeculae are laid down parallel to, and along, the free surface of the cartilage, often giving the impression of metaplasia, because no line of division between bone and cartilage is perceptible. In some cases, however, osteoblasts are seen upon the free surface. The degeneration shows in places a mucoid character, in others simple disappearance of the nuclei and partial liquefaction of the matrix. Many of the cells are undergoing disintegration.

In single sections small masses of cartilage seem to lie isolated near the tumour, but in other sections are seen to have a pedicle connecting them with the chief mass of cartilage. Many of the cells are flattened and lie in hydropic capsules.

The blood vessels in the osteoporotic zone surrounding the tumour are comparatively small and rather limited in number, while those near the periphery of the zone, where bone trabeculae are present, are extraordinarily dilated, filled with blood, and present in large numbers. Although their walls are as thin as those of capillaries, they are readily visible macroscopically.

The sclerosed portion of the femur is formed of thick bone trabeculae, almost as dense as *substantia compacta*, with relatively small medullary spaces filled with widely dilated blood vessels, a few fat globules, and myelocytes. There are relatively few lymphocytes present. On the walls of the medullary spaces bone is laid down by apposition, and not infrequently lacunae are present, partly empty, partly containing multinuclear osteoclasts. The directions of the bone lamellae are very irregular, but they contain well-developed corpuscles with canaliculi. A few perforating canals are present.

Although the seat of the former osteomyelitis is now at a considerable distance from the epiphysis, it is probable that at the time when it was acute the intermediate cartilage was nearer its lower border. In spite of the probable nearness of the cartilage to the focus of inflammation the bone has continued to grow, but the synostosis of the diaphysis and epiphysis have evidently taken place early, leading to 4 cms. shortening of the bone. According to Weinlechner and Schott the bone elongates in proportion to the nearness of the inflammatory focus to the epiphysis, while the majority of more recent writers conclude that lesions of the centre of the shaft produce elongation, while those near the epiphysis lead to shortening. That, however, the bone has continued to grow since the osteomyelitis began, and later ceased to grow before reaching its normal development, is shown by the cartilaginous tumour representing the approximate position of the intermediate cartilage when the inflammation was present.

I am of opinion that the cartilage mass is the result of increased proliferation of the intermediate cartilage, caused by the osteomyelitis. The following case of acute osteomyelitis, associated with an enchondroma-like hyperplasia of the intermediate cartilage, furnishes good evidence of the method by which the above tumour was formed:—

CASE 2 (Preparation No. 811, in the Museum of the Children's Hospital, Prague).—In the spring of 1896, M. L., a girl æt. 12, consulted Professor Bayer on account of a swelling and reddening of the right thigh, accompanied by fever and occasional chills. Four weeks later a purulent perforation of the skin occurred on the inner side of the lower third of the thigh, followed shortly by a second similar perforation on the outer side just above the middle. The former became a fistula discharging greenish pus, while the latter closed spontaneously.

The patient was admitted to the hospital on the 21st April 1897. The right thigh was found to be considerably thicker than the left, so that, a hand's-breadth above the knee-joint, its circumference measured 35 cms., against 29 cms. on the left side. A little above the middle of the outer side of the thigh is a white movable scar, sunken in the centre, with brownish pigmented periphery, and measuring 5 square cms. The above-mentioned fistula on the inner side of the thigh measures 1·5 cms., and upon pressure emits pus.

In the upper third of the thigh is a fluctuating area, in the neighbourhood of which the skin is diffusely reddened. The right femur is distinctly thickened in its distal half, and painful to the touch. The knee-joint appears movable.

On the 21st of May a sequestrum was sounded through the fistula on the inner side of the thigh, and a sequesterotomy was undertaken. The bed of the sequestrum was uncommonly soft, and the fistula was found to communicate with the fluctuating area representing an abscess cavity in the upper third of the inner side of the thigh. After tamponing the cavity in the bone with iodoform gauze, the wound was bound with a compression bandage. Soon after the operation the temperature rose, and on the following day was 40° C. Pulmonary symptoms developed, and the fever continued until the death of the patient on the 7th of June.

The clinical diagnosis was as follows:—

Necrosis ossis femoris dextri post osteomyelitiden; necrotomia; sepsis; pleuro-pneumonia.

The autopsy was made on the 8th of June by Dr. Kraus. Patient 13 years old.

PATH. ANAT. DIAGNOSIS.—*Vulnus ossis femoris dextri post extractionem sequestris centralis ossis femoris dextri dies 17 ante-mortem factam; periostitis chronica femoris dextri; coxitis chronica dextra; osteoporosis eximia femoris dextri et patellæ dextræ; hyperplasia cartilaginis synchondrosis epiphysariæ inferioris femoris dextri; abscessus metastatici pulmonum; infarctus anæmici lieinis; pyohæmia; morbus Brightii acutus; tuberculosis chronica glandularum lymphaticarum peribronchialium.*

Body, 132 cms. long; strongly built frame; well-developed musculature; moderate panniculus adiposus. Body pale; a few diffuse, faint violet, post-mortem discolorations on the back. Marked rigor mortis. Visible mucous membranes pale. Thorax well developed. The abdomen is on a level with the thorax.

The brain was not removed. The thyroid gland is enlarged, and pale. The mucous membranes of the organs of the neck are pale.

Both lungs are slightly adherent. The right lung is anæmic, and contains foci of lobular pneumonia. Under the pleura, on the surface, are hazel nut-sized lobular areas of consolidation, with purulent centres, and surrounded by hæmorrhagic infiltration. The pleura of the left lung is cloudy and covered with a fibrino-purulent exudate. There is marked consolidation of the lower lobe, which contains also many commencing abscesses. The peribronchial lymph glands contain grey miliary nodules, and in places are entirely caseated.

No abnormal contents in the pericardial sac. The heart is of normal size, flaccid, easily torn. It contains a small quantity of clotted blood. (Esophagus is pale. No abnormal contents in the abdomen. The liver is swollen; the

parenchyma sallow and easily torn. The gall bladder is normal. The spleen is enlarged and covered by a fibrinous layer; the parenchyma is hyperæmic and very soft.

Both kidneys are markedly enlarged, the capsules easily removed. The surfaces show depressed scars. The parenchyma is greatly swollen, indistinctly marked, pale, and has a yellowish colour. The mucous membranes of the pelvis of the kidneys and bladder are pale. The stomach and intestines are without pathological changes. The pancreas and adrenals are normal. The genitalia are normally developed. In the left ovary is a corpus luteum.

The right lower extremity is cedematous. On the outer side of the right thigh, a little above the middle, is an old scar which is movable over the bone. On the inner side in the lower third is a granulating incision, 6 cms. long, which leads to the bone; and above it, in the upper third, is a fistulous opening in the skin, 3 cms. long, which emits pus on pressure, as does also the incision. . . . The os femoris dextri is thickened throughout, but more especially in the lower half of the diaphysis; its periosteum, as well as the neighbouring cellular tissues, is indurated and thickened. The substantia compacta, on the inner side of the lower third of the diaphysis, was removed by operation, and the marrow cavity cleaned out. The entire os femoris is markedly osteoporotic, so that it breaks very easily everywhere. The bone-marrow is very hyperæmic. On the border, between the diaphysis and the lower epiphysis, the intermediate cartilage is markedly hyperplastic (Fig. 2). It measures from above downward up to 3·8 cms. in length, and 5 cms. in breadth. It has, on the upper and lower border, an irregular lobulated margin, and consists partly of moderately soft, partly of incompletely calcified, cartilage. Along its diaphyseal border it shows a finely porous spongy tissue similar to that found in rachitis. There are several islands of cartilage lying in the marrow to the diaphyseal side of the intermediate cartilage, and apparently separated from it. The lower epiphysis and patella are also markedly osteoporotic. The articular surfaces of the right knee-joint are covered by connective tissue adhesions, and the patella is adherent to the femur. In the right acetabulum is a slightly bloody, serous exudate. The cartilage in the right hip-joint is almost entirely absent. The head of the femur is in greater part defective, and it and the acetabulum are markedly osteoporotic.

There is no evidence of rachitis in the skeleton.

A sagittal layer was removed from the lower end of the femur and decalcified in 5 per cent. nitric acid, with 5 per cent. formalin. The sections were stained chiefly with hæmatoxylin and eosin and by van Gieson's method.

The *microscopical examination* showed that the finely porous zone on the diaphyseal side of the intermediate cartilage (Fig. 2) is composed of osteoid trabeculæ, which are seldom perpendicular to the surface of the cartilage, but usually parallel or running irregularly in all directions, and extending along the whole of the diaphyseal side of the cartilage. The fat marrow ends abruptly on the proximal side of the osteoid trabeculæ, while on the distal side the medullary spaces are filled with bone-marrow with practically no fat globules. The marrow is of very loose texture, and is composed mostly of myelocytes, comparatively few lymphocytes, and a considerable number of small, thin-walled, empty blood vessels. The margin of the cartilage is well vascularised, partly by thin-walled capillaries, which penetrate it often without visible accompaniment of marrow cells. Usually the blood vessels penetrate the cartilage surrounded by bone-marrow. These strands often penetrate the cartilage for a considerable distance, and the cartilage is hollowed out like a honeycomb. There is an active formation of new trabeculæ on the sides of

the spaces thus formed. There are numerous deposits of lime in the cartilage near its margin, usually in the form of fine granules in or around the cartilage cell capsules. These occasionally lie close together, but seldom form plaques. The osteoid trabeculae, formed along the free surface of the cartilage (parallel to it instead of perpendicular, as in the normal formation of bone), give the appearance of a metamorphosis of the cartilage, but are lined on the surface by osteoblasts, and become separated from the cartilage by the ingrowths of bone-marrow, osteoblasts, and blood vessels on the inside, so that, as in the former case, one can accept only the theory of appositional formation. The osteoid trabeculae are rich in cells, lying close together and in relatively large lacunae, which show no canaliculi, and no cell branches are to be made out either by the ordinary methods of staining or by Schmorl's method.



FIG. 2.—For description, see p. 239.

The process on the epiphyseal side of the intermediate cartilage is very different from that on the diaphyseal side. There is practically no new formation of bone, and the cartilage shows only here and there deposits of lime, which are usually in the form of plaques, and seldom deposited in the form of fine granules in or around the cartilage cell capsules. Bone trabeculae, containing well-developed corpuscles with canaliculi, lie either parallel to or perpendicular to the surface of the cartilage which has grown in between them, giving a lobulated character to the margins. In a few instances the cartilage has grown into medullary spaces, completely filling them. The lobulation is so marked that it often appears as though the cartilage masses are entirely independent (Fig. 2), but when followed in serial sections all of them are found united to other lobes, though sometimes only by a narrow pedicle.

Corresponding to the active formation of new trabeculae on the diaphyseal side, and the lack of osteogenesis on the epiphyseal side, the cartilage cells of the former are arranged in rows which are not parallel to the long axis of the bone but about perpendicular to the free margins of the cartilage lobules, while on the epiphyseal side there is no columnar arrangement to be made out.

The cartilage is of hyaline character and shows degeneration, especially near the centres of the lobules where the vascularisation is poor, the area being devoid of cells, and taking a deep blue stain with hæmatoxylin. Where it has reached the stage of liquefaction, fine fibrils are seen projecting into the open spaces, and occasionally large proliferating cells are found lying free in the degenerated area.

Wherever cellular marrow lies in contact with the cartilage, or where it has become vascularised, the cartilage takes a red stain with eosin, which is strongest where osteoid tissue is produced by osteoblasts. On the epiphyseal side, where the cartilage is covered by fat marrow, the eosin stains less intensely; although, throughout the cartilage where the cells lie far apart, there is a slightly reddish tone to the matrix, except where it has undergone degeneration or liquefaction.

In one of the lobules is a degenerated area filled with branched and spindle-shaped cells, which have apparently grown in from the marrow and are embedded in a finely granular or homogeneous substance, with extravasations of blood, and resembling myxomatous tissue. Numerous areas are present where the cartilage cell capsules are swollen and hydrotic, but these are most marked in the columns near the diaphysis. Many of the cells show irregular branching, and small shrunken nuclei. The epiphysis and patella are markedly osteoporotic, and are bound together by connective tissue adhesions. Few multinuclear osteoclasts are to be found. In the osteoid tissue of the diaphysis, giant cells are present in larger numbers, but resorption seems to have taken place quite as frequently through osteoblasts.

The articular cartilage is covered with loose connective tissue, enclosing numerous cells with a large amount of protoplasm and granules. The cartilage is interrupted in one place, and seems to have sunk into the epiphysis. The transposed cartilage shows proliferation upon the surface towards the joint, and formation of osteoid tissue, with deposition of lime on the surface towards the intermediate cartilage. It is separated from the surface by fat marrow and bone trabeculae, upon which lies connective tissue. The articular cartilage is distinctly fibrillated, and specimens stained by Weigert's method contain a few elastic fibres.

The substantia compacta of the diaphysis is osteoporotic, and shows osteophyte formation under the periosteum, which is markedly thickened. Portions of the bone near the osteomyelitis cavity show cells embedded in a calcified matrix, very similar to those found in sclerosis of bone obliterating the medullary tube in tumours, etc., the cells being irregularly shaped, occupying a relatively large lacuna, and possessing no processes or canaliculi.

One can, of course, call this an enchondroma, but more exactly hyperplasia of the intermediate cartilage, of which it still retains many of the characteristics, such as the position and bone formation, by apposition on the diaphyseal side, and its absence upon the epiphyseal side. Olliers¹ calculated that from twelve to twenty times less bone is laid down on the epiphyseal side than on the diaphyseal side of the cartilage, which would not be too high an estimate for this case.

The cartilage is no longer uniformly continuous with the articular cartilage being partly cut off by the interposition of bone trabeculae and marrow. It is therefore improbable that its growth represents a corresponding increase in the length of the bone. Paget calls attention to irregular, stellate, branched nuclei or cartilage cells, similar to those present in this case, which, he says, do not occur in normal cartilage; he considers that they are characteristic of cartilaginous tumours.

Klebs considered that the lobulated structure in enchondromata is due to unequal proliferation in different portions, and often in close relation with the ingrowing blood vessels. No evidence of unequal proliferation was to be observed in this case, although the ingrowth of blood vessels and marrow seems to have played an active part in producing the irregularity of the margin on the diaphyseal side, while, on the epiphyseal side, the lobulation was produced by the

¹ Schmidt, *loc. cit.*

proliferation of the cartilage downward, and the depressions were caused by the resistance of the bone trabeculae in the epiphysis.

The lesions suggest in some respects those found in rachitis—the production of osteoid tissue, the evident poverty in lime, the height of the cell rows, and the irregularity of the ossification, upon which Virchow laid especial stress in the production of enchondromata in rachitis. While Virchow considered that occasionally, in the development of bone, fragments of the primitive cartilage remain unossified, and form the starting-point for the development of a tumour, he reported several cases of rachitis in which portions of cartilage became isolated in the irregular ossification of the intermediate cartilage, forming enchondromata. More recently Zeroni, Colley, and others have reported similar cases of rachitis, in which islands of cartilage lay at a considerable distance from the line of union of the diaphysis and epiphysis; and both Virchow and Chiari have reported cases in which numerous exostoses and ecchondroses protruded from the sides of the bone, and which they traced back to previous connection with the intermediate cartilage.

Weinlechner and Schott found in pathological elongation of bones a considerable proliferation of the cartilage cells, accompanied by active ossification. *In the production of the enchondromata in these two cases the essential point of difference lies in the defective ossification.*

According to Schmidt, in cases of elongation there is mostly inflammatory, certainly always arterial, hyperæmia; and Loetsch, who made a careful study of the literature, came to the conclusion that lesions which do not affect the bone directly are equally important in the production of elongation as diseases of the bone itself.

Among the cases of elongation of bones following dilated blood vessels, Schmidt mentions those of Nicoladoni (phlebarteriectasia), Israel (arteriectasia), Broca (traumatic arterio-venous aneurysm); and Loetsch reported a case of elongation associated with varicose veins and *ulcus cruris*. The other causes of elongation which have been reported are osteomyelitis, necrosis, luxations, fractures, intermuscular abscesses, *ulcus cruris*, chronic arthritis, caries, and infantile paralysis (see Loetsch, Tillmanns, Schuchardt). Many of these are such as would lead to hyperæmia. As is well known, an increased flow of blood is accompanied by an increased production of bone. If, then, there is not a concomitant increase in the growth of the intermediate cartilage, it would soon become ossified, leading to shortening of the bone. We must therefore accept, that hyperæmia causes not only an increase in the growth of bone but also of cartilage. Cartilage is, however, a tissue which is notoriously capable of growing with a comparatively poor blood supply; and it is not excluded that, under certain conditions, it might assume a special activity without being directly dependent upon an increased supply of blood. The vitality of cartilage is shown by the fact that when a piece of cartilage with

bone becomes broken off in a joint, the bone dies, while the cartilage continues to live as a free body in the joint (Bollinger). On the other hand, the softening and degeneration of the centres of large enchondromata is a matter of common observation.

Nové-Jossierand mentions several cases of shortening of bones associated with enchondromata, but according to him the bones are usually elongated. Cases of enchondromata, associated with vascular disturbances, are not rare in the literature, such as those with angiomas, reported by v. Recklinghausen, Steudel, and others.¹

Klebs reported a case of symmetrical enchondromata of both humeri, associated with a considerable development of the blood vessels, which he considered the cause. Nélaton² reported an enchondroma with hyperæmia of the surrounding spongiosa.

Virchow believed that the persistence of cartilage islands was the result of poverty of vascularisation, which, upon restoration of the circulation, become calcified. Zeroni took the opposite view, namely, that the proliferation and isolation of the cartilage is due to hyperæmia, and the calcification follows narrowing or obliteration of the blood vessels. v. Recklinghausen suggests that the defective calcification which was present in his case might have been due to aplasia of the blood vessels.

Virchow, while admitting the production of enchondromata following fractures, suggests that they form in some cases a predisposition to fractures. In the two cases reported above, the osteoporosis was not the result of the enchondromata, for it is in one case also present in the patella, although we know that the growth of cartilaginous tumours may lead to the destruction of a whole bone. It is in my opinion to be attributed more to the defective deposition of salts, and in Case 1 probably to defective vascularisation. As Marchand has pointed out, the osteoblasts are capable of dissolving the bone substance, the presence of multinuclear osteoclasts being by no means essential. The small number of osteoplaxes present is therefore not sufficient indication to warrant a positive statement. This much is certain—that in many cases of enchondromata fractures occur, such as in those reported by Virchow, v. Recklinghausen, and Colley; and in Steudel's case there was an abnormal softness of the whole skeleton, accompanied by multiple fractures. In how far the osteoporosis, by removal of resistance, might allow the growth of cartilage into a tumour is, however, uncertain. Ollier and Seeligmüller believed that the elongation in infantile paralysis was due to absence of pressure

¹ The other cases reported are by Hoestermann, "Enchondroma hæmatodes," Bonn, 1868; Hutchinson, "A Case in which Congenital Angiomas and Enchondromata coexisted in the Upper and Lower Extremities of the same side," *Arch. Surg.*, London, 1891, (2), vol. iii.; Langenbeckmann, "Enchondroma teleangiectaticum," Würzburg, 1890; and Mafucci, "Di un caso di enchondroma ed angioma multiple Movimento," Napoli, 1881.

² Zeroni, *loc. cit.*

on the diseased bone, permitting the intermediate cartilage and bone to grow more easily before becoming atrophic. Also, in rachitis, it is easy to think that the removal of pressure might play an important rôle in the growth of the cartilage, and in the formation of enchondromata. Such a theory would have to admit a difference in etiology of enchondromata in bones and those in soft structures. Virchow advanced the theory of embryonic inclusions, and, as is well known, cartilaginous tumours of the parotid gland and especially of the testes are often traceable to trauma. Weber,¹ in collecting statistics upon enchondromata, found thirty-four in sixty-two cases, in which the tumours were attributable to previous injury. Further, one finds in the literature cases of exostoses and ecchondroses affecting several members of the same family, and both Virchow and Paget report cases of enchondromata which were apparently of hereditary origin. Cases are, however, not infrequent in which there is hereditary predisposition to osteomalacia or fractures. Fractures are followed by an increased flow of blood, and in healing are often accompanied by a considerable production of cartilage, or lead to the formation of enchondromata.

As one will readily see from the above, there is a lack of unity in regard to the etiology of enchondromata in different parts of the body. There is, however, a striking similarity in the causes which lead to abnormalities in the length-growth of bones, and those which lead to the formation of enchondromata arising from the epiphyseal cartilage. There fails, however, more positive evidence of the association of inflammatory lesions with enchondromata in bones. The two cases reported above are, as far as we know, the only ones which are recorded in which enchondromata followed acute and chronic osteomyelitis. According to Virchow, enchondromata of the soft parts occur very commonly in connection with chronic inflammations, which in turn are often of traumatic origin. He has also reported enchondromata in the lower end of the femur in two cases of white swelling of the knee, and in one case found the intermediate cartilage with a transparent, bluish, swollen appearance, and interrupted in numerous places by the *substantia spongiosa*, in a young girl with congenital syphilis, caries genu, osteomyelitis fibrosa, and multiple gummata. Nové-Josserand quotes the case of Ollier and Vincent, who published a case of tuberculous osteitis, under the name of "*Rachitisme tardif inflammatoire*," in which they found, at the level of the intermediate cartilage, defective ossification characterised by the disposition of the cartilage to form irregular islands. Colley reported a case of chronic tuberculosis of the leg and condyle of the right femur, accompanied by abscesses and fistulous ulcers of the thigh and groin, osteoporosis, rachitis of the ribs, and hyperplasia of the epiphyseal cartilages of the femur and tibia. He attributed the last to

¹ Perls, *loc. cit.*

rachitis, but it is quite probable, in view of the cases reported above, that the inflammatory process played an important part in the growth of the cartilage, the rachitis being an evidence of poverty of the system in lime. Tillmanns refers to numerous authors who have shown that, by a diet poor in lime, lesions similar to rachitis can be produced in young birds and dogs.

Attention has already been called to the similarity between the cartilage in rachitis, and in Case 2, reported above, although in neither case was any evidence of rachitis to be observed upon the skeleton. In view of the pathological evidence and the similarity of the causes of pathological elongation of bones, and the causes of enchondromata proceeding from the intermediate cartilage, it seems to me justifiable to conclude that the determining factor in the production of one or the other is the presence or absence of lime. It is not improbable that the mass of cartilage, isolated by the irregular vascularisation, and losing its physiological function, might later take the character of an autonomous growth.

I wish here to express my gratitude to Prof. Chiari for his kindness to me during this work, and for many valuable suggestions which I have received from him.

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DESCRIPTION OF FIGURES IN TEXT.

FIG. 1.—Sagittal section of the femur in Case 1, showing the medullary tube partially obliterated by dense compact bone, containing three soft pea-sized areas. Near its lower margin is a cloaca, which was filled by the ingrowth of connective tissue from the periosteum. Below the sclerosed portion is an irregular mass of cartilage surrounded by an area of osteoporosis. ($\frac{2}{3}$ nat. size.)

FIG. 2.—Sagittal section of the femur and patella in Case 2. The femur shows hyperplasia of the intermediate cartilage with apparent isolation of small portions, which, however, in other sections are seen to be continuous with one another. Along the diaphyseal margin of the cartilage is a finely porous zone of osteoid tissue. The diaphysis, epiphysis, and patella are markedly osteoporotic. The intermediate cartilage is in some sections (as in this) no longer continuous with the articular cartilage. (Partly schematic, nat. size.)