HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY, WITH AN ACCOUNT OF TWO CASES.

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(Plates I.–IV.)

It has lately been my fortune to meet with two well-marked cases of this interesting condition, and I propose, first, to describe the features of these cases in detail, and, secondly, to give a review of the present state of our knowledge of the morbid processes upon which the disease seems to be dependent.

Case 1 is that of a boy, A. M., aged 14, who was first admitted to the Sussex County Hospital on 5th November 1901, suffering from a painful swelling, of two months' duration, above the right knee. This was incised on 12th November by Mr. Willoughby Furner, who found a periosteal sarcoma of the shaft of the femur, and consequently disarticulated the limb at the hip-joint. The boy left hospital on 27th January 1902.

He was readmitted on 29th October 1902, with what appeared to be an enlarged gland in the right groin. This was excised on 8th November, but on microscopical examination the swelling was found to consist of nerve fibres, degenerated blood vessels, muscular and fibrous tissue. There was no lymphatic gland substance present, and no evidence of malignant disease (Report of Clinical Research Association). He returned to the hospital on 14th October 1903, for swelling of the left knee and ankle, of one week's standing. The clinical notes of his case at that time state that: “Patient has had aching pains in the left knee for about a month, especially at night. The left knee is rather swollen and red. It is hot to the touch, and slightly tender on the inner side. The movements are not limited, and do not cause pain except on extreme flexion. Much free fluid in the knee-joint. The left ankle is also slightly swollen. Temperature, 99°6. January 13, 1904.—Circumference of knee, 13½ in. March 3.—Has complained every now and then of 'rheumatic' pains, and his wrists have swollen at intervals. The left border of the heart is just external to left vertical nipple line, the right border at the right sternal margin. No bruit in any area. Pulmonary second sound reduplicated. April 6.—Since last entry fingers have become clubbed. Although the terminal phalanges are bulbous, they are not cyanosed. Has been put on thyroid extract, without any appreciable benefit. Continues to have rheumatic (?) pains in elbows and wrists." An elder brother had about this time been in hospital for tuberculous testis, and the knee of this patient was at first thought to be tuberculous. The multiple arthritis which followed, which showed no destructive changes, and which was quite unaffected by treatment, put that diagnosis out of court, and the patient was consequently transferred to the medical side, under the care of Dr.
W. A. Hollis. I have to thank Mr. Furner for allowing me access to his case-books.

"April 11.—Left leg—Knee and ankle affected with a chronic synovitis; much thickening of the synovia, fluid, and pain; also grating sometimes. Left arm—Wrist in same condition as joints of leg; elbow painful on movement. Right arm—Chronic synovitis of wrist; elbow in early stage. Abdomen—Nothing abnormal. Thorax—No abnormal physical signs in lungs or heart; apex beat in fifth space in nipple line; marked clubbing of all the fingers and toes; skiagrams show thickening of these phalanges; no alteration in facies, or other indication of acromegaly. April 15.—All affected joints are tender to the slightest pressure or movement. Pain wakes him at night. April 18.—Cyanosis of finger-tips noted for first time. April 20.—Pain relieved by 30-gr. doses of aspirin." (He had previously been treated with urea, thiocol, and cod-liver oil without benefit.) "May 9.—All the original pain and tenderness of all joints have recurred, since only small doses (5 grs.) of aspirin have been given. No pyrexia. May 10.—After five 30-gr. doses of aspirin all pain and stiffness gone. May 18.—No change from Scott's dressing. May 21.—Discharged."

On 9th June he was readmitted, when I was house-physician, and I now quote from my own notes. He had been bedridden, and had never been quite free from pain since leaving hospital. On admission, both wrists were hot, swollen, painful, and tender; but there was no flushing of the skin. The distension was due in the main to fluid, but there was also considerable thickening of the lower ends of the ulna and radius, forming a prominence dorsally on the lower part of the forearm, and ending somewhat abruptly about 3 in. above the wrist. The left knee and ankle were in a similar condition, with again no flushing of the overlying skin. There was marked "floating" of the patella, and the circumference of the knee over the middle of the patella was 15 in. Both elbows were in a condition of chronic synovitis, but, unlike the other joints enumerated above, there was at present no acute exacerbation of the arthritis. Gentle movements did not elicit creaking. Both hands and the remaining foot were greatly increased in size, apparently both from thickening of the bones and from effusion of fluid into the small joints of the hands and foot. The thickening of the bones was well seen in skiagrams taken at the time (Plate I.). The skin over the affected joints was of a peculiar transparent waxy pallor, with distended superficial veins showing through it. The clubbing of the fingers has been already mentioned. It differed from any clubbing which I had hitherto seen, in that, although the distal phalanges showed the typical curved nails and clubbed enlargement of the ends of the digits, the fingers increased in size towards their base; so that the breadth of the middle finger at the base of the distal phalanx was 2·2 cms., at the base of the second phalanx 2·4 cms., and at the junction of the upper and middle thirds of the first phalanx 2·7 cms. This held good of all the five digits of both hands, and also, although to a less marked extent, of the toes. Thus about the upper part of the first phalanges there was a bulbous swelling, somewhat similar to the spindle-shaped swelling of rheumatoid arthritis, but shorter, more abrupt, and situated, not at the interphalangeal joints, but proximal to them. The
position of the swelling showed that it was not due to effusion into the joints, and skiagrams proved its cause to be a deposition of new bone along the shaft of the phalanges (Plate I.).

There was no hyperextension of the terminal phalanges. No enlarged glands were present, nor was there any enlargement of the liver and spleen. The most careful examination possible failed to detect any physical signs of tubercle or new-growth in the lungs. The position of the heart was unaltered, and the sounds healthy in all areas. Temperature, 100°.

His mother gave me the following history:—Father and mother alive and healthy. One brother has had a tuberculous testis, which was treated at the Sussex County Hospital. A first cousin has tuberculous knee and caries of ribs. There is no history of gout, rheumatism, or osteo-arthritis in any member of the family. The patient did not sweat much at home, except in the foot and hands. "These were seldom very hot, but always clammy, and the foot sometimes quite cold." His appetite had been fairly good; he had no symptoms of dyspepsia. Bowels generally constipated; no history of diarrhoea. "His pain would shift about from one joint to another. One day he would be stiff in almost every joint and be in pain, and a few days later would say that he felt right again." The hip-joint was unaffected. According to the mother, the clubbing of the fingers was noticed by her in the end of October 1903. She saw that the boy was hiding his hands from her. If her story is accepted, the enlargement had escaped notice in the hospital for some six months. There was no history of any acute infection for some years prior to the amputation. The only illnesses his mother remembers were measles and whooping-cough, "as a child's complaint"; she could not give exact dates. There had been no cough or expectoration.

June 11.—Under aspirin, 15 grs. 4-hourly, and lead and opium fomentations, swelling has diminished in all the affected joints except the knee. No cough; no sputum. June 13.—Temperature, 98°. No pain in any of the joints. No abnormal physical signs to be made out in the chest, which can now be examined posteriorly. Impaired mobility of all the affected joints. June 18.—Temperature, 99°.6. Complaining of pain and stiffness in joints again. No fluid in any, except in knee. June 21.—Temperature, 100°.2 at night. Active arthritis, both wrists. July 1.—Much better. No active symptoms. Permanent swelling and impairment of movement in the affected joints. July 14.—Went home. Since 25th June he had had Tallermann-Sheffield hot-air baths, with seeming benefit. Mobility of joints was still, however, impaired, both to active and passive movements.

On 9th September he was readmitted under Dr. Hollis, for what turned out to be the last time. A fortnight after leaving hospital had slight hemoptysis, and had brought up a few specks of blood every day since. Joints are unchanged. The clubbing of the fingers and toes, and the enlargement of the superficial veins, are slightly greater. No physical signs of tubercle or new-growth in lungs. Heart unaltered. We had now for the first time some positive evidence of affection of the lungs, and, having regard to the lapse of time from the amputation for sarcoma, tuberculosis was held to be the more likely diagnosis. I shall
now quote very briefly from the lengthy clinical notes taken during this period. September 11.—Has brought up a tiny bright red clot. Examination failed to detect tubercle bacilli or tumour cells. October 3.—There is distinct impairment of resonance and diminution of breath sounds in right lung posteriorly at level of eighth dorsal spine. The left leg has been fixed in a splint, and is now free from pain. Joints of arms giving him pain from time to time, still sweating copiously, and temperature constantly from 1° to 2° above normal. Sweating is general, but most marked on hands and foot. October 6.—Dulness slightly increased. Again slight hæmoptysis. October 14.—The right lung posteriorly is dull from scapular spine downwards, the dulness being most marked over the scapula. Over this area the vocal fremitus is quite lost, but a slight vocal resonance still remains. The breath sounds are very distinctly heard. In front and in axilla physical signs normal. October 15.—Exploratory puncture. No fluid found in right pleura. Patient is losing ground. October 29.—Same physical signs over right lung. Patient now suffers a good deal of pain in chest. About twice in twenty-four hours brings up a small blood clot. Apex beat now ½ in. external to nipple line. Losing flesh. No enlarged glands. October 31.—Severe pain in right chest this morning. Given morphia for first time. There is increased vocal resonance, a few crepitations, and impaired percussion note in third and fourth spaces in front.

November 2.—In above area breathing now is bronchial, and there are numerous medium crepitations heard. Frequent attacks of pain in chest. November 4.—Pain and cough continue. No expectoration, except the usual small bright clot. In the third and fourth spaces right lung breaking down in front. Apex beat in fifth space 1 in. external to nipple line, and diffuse forcible pulsation visible in third, fourth, and fifth spaces. A squeaking presystolic bruit in fourth space in nipple line—very localised. Pulmonary second sound accentuated. Repeated examinations of the sputum had been made by Dr. Bushnell and myself, but we had never found tubercle bacilli. It was evident now that the left lung was the seat of a rapidly growing sarcomatous tumour, or tumours. November 8.—The right lung is now breaking down rapidly, but patient still expectorating very little. No blood present in stools. Heart as before. Lower edge of liver three fingers’-breadth above the umbilicus. Right pleura explored—four ounces of blood removed. November 12.—Left border of heart now 1½ in. external to vertical nipple line. Same bruit present. Friction in right axilla. November 14.—Heart is being pushed further and further to the left. Left border now 2½ in. external to nipple line—right border at left border of sternum. Same localised squeaking bruit below nipple; blowing systolic bruit at apex; pulmonary second sound accentuated. Rough creaking friction in right axilla. Anteriorly, the physical signs of excavation in the right lung are hardly so marked as formerly; posteriorly, signs unchanged, there being normal breath sounds and resonance above the spine of the scapula. Liver lower edge two fingers’-breadth above umbilicus, but it is impossible to say whether the organ is enlarged or pushed downwards. No enlarged glands. November 15.—Increase of swelling and pain in left knee and ankle. Knee measures 15¼ in. During the last six weeks joint symptoms have been in abey-
November 16.—Constant dyspnœa and pain in chest. Patient now has morphia both by day and night. Physical signs unaltered. Wasting rapidly. November 17.—Acute dyspnœa; heart’s action weaker; temperature now subnormal—it had previously varied from 98°-8 to 100°-8. An attempt to tap the right pleura with a view to relieving the dyspnœa only resulted in the withdrawal of a few ounces of blood, partly already clotted. November 18.—Suffering acutely from breathlessness, which is only relieved by morphia. Oxygen gives no relief. Sinking. November 19.—Died at 11.45 a.m.

A post-mortem examination was made on the following day by Mr. R. A. Clapham, Third House-Surgeon, from whose notes I have abridged the following report:

Thorax.—There was a sarcoma of the right lung, apparently originating from the root and invading all three lobes, the whole of the lower and middle and most of the upper lobe. The pleura was extensively infiltrated, and the lung had to be torn from the chest wall. Throughout the growth there were numerous hæmorrhages, recent and old, and the growth was soft and extremely vascular. Below, the growth was partly adherent to the diaphragm, with a large blood cyst situated between the dome of the diaphragm and the base of the lung. Left lung—Fibrous adhesions to chest wall over lower lobe, and some recent broncho-pneumonic patches.

Heart.—Displaced to the left. Muscle pale and rather flabby. Right ventricle hypertrophied and dilated. Valves healthy. Mediastinal glands enlarged, varying in size from hazel-nut to a marble. Sternum, ribs, and vertebrae not involved in the growth. Thymus gland atrophied.

Abdomen.—Liver and spleen showed slight chronic venous congestion—no sarcomatous deposits present. Kidneys and suprarenals healthy. Stomach and intestines healthy. Glands, no obvious change.

Skull.—Cranial bones not thickened. Brain healthy. Pituitary body normal. Thyroid gland of normal size and apparently healthy.

I have to thank Dr. F. G. Bushnell, Pathologist, Stephen Ralli Memorial, Sussex County Hospital, for the following minute account of the special lesions, which he has kindly given me for inclusion in this paper:

"The autopsy showed symmetrical and general changes in the bones and joints, mainly of a chronic inflammatory character, with clubbing of the fingers and toes. There was no suppuration, no putrid bronchial secretion, no tubercle. There had been degeneration of the vascular sarcoma in the lung, with formation of a blood cyst between the diaphragm and lung, but it was extremely doubtful if septic infection of the cyst had occurred. The growth in the lung was composed of sarcoma cells of marked polymorphous character, with little intercellular substance. The heart, thyroid, pituitary body, kidneys, and intestines showed no abnormalities, and were not microscopically examined.

"Bones.—Phalanges, the articular ends of humerus, radius, ulna, femur, tibia, and fibula, and the astragalus were dissected out by me. In removing them, the hyperplasia of the gelatinous connective tissue was noteworthy, and the clubbing was seen to be due to hyperplasia of the soft parts—fatty and fibrous—of the fingers, and deposition of firm
new bone along the shafts of the phalanges. The other bones were characterised by softness of texture; the post-mortem knife could divide them easily. The periosteum was a little thickened, and there was a deposit of new bone along the shafts. No osteophytes were present.

"Microscopical Appearances (thirty-five sections examined).—The femur, tibia, fibula, and astragalus were decalcified with nitric acid, and phloroglucin nitric acid, and sections stained with—(a) haematoxylin-eosin, (b) eosin methylene-blue, (c) Kühne's polychrome methylene-blue, (d) by Gram-Weigert, Löeffler, and Ehrlich's methods for micro-organisms and tubercle bacilli. No micro-organisms were seen, no tubercle bacilli were present. In each case the periosteum was thicker than normal, but its external fibrous and internal cellular layers could be well made out. Osteoblasts were present in numbers, and in places were several layers deep. Perichondral ossification was limited to a mere outlining of the long bones, and endochondral ossification had resulted in an extremely rarefied cancellous tissue. Suspecting a disintegration of bone and calcified cartilaginous substance by osteoblasts, search was made in possible lacunae, but none were found. Haversian canals were present, with bone cells, the latter formed by the inclusion of osteoblasts within the bone substance. The patella showed marked horizontal and vertical fibrillation of its articular cartilage; numerous cartilage cells were present in each cartilage space from multiplication.

"Marrow.—The marrow resembled the yellow variety in all places. It was fatty and congested. Generally speaking, the marrow was of the embryonic or lymphoid character, though giant cells (zoogloeic masses) were present. Lymphoid cells, cells with a rim of protoplasm, basophil or oxyphil, or with much oxyphil cytoplasm, formed the bulk of the cells. All transitions from lymphocytes to nucleated red cells were present. No tubercle giant cells, and no caseation or micro-organisms were present in any part examined.

"Joints.—The periarticular surfaces were gelatinous. All the joints were examined by sections stained by (a) haematoxylin-eosin for general changes; (b) by Ehrlich and Kühne's methods for tubercle bacilli; (c) by Löeffler and Gram-Weigert's methods for micro-organisms; and (d) by polychrome methylene-blue and neutral orcein for mast and plasma cells.

"Knee.—Synovial membrane thickened and showed inflammatory changes, as did the periarticular tissues. Masses of round mononucleated cells surrounded the vessels, which were distended with erythrocytes as in diapedesis. Some vessels contained a few polymorphonuclear leucocytes, but not many were seen. There were many distended capillaries. The tissues were loosely fibrillated and the fibrils widely separated by oedema. Fat cells were present. The villi were swollen, their surfaces richly cellular, the cells in most cases containing large rounded vesicular nuclei with fine chromatic network and granules. The cores of the fringes were oedematous. Other cells were oxyphil, with rounded small nuclei, single or multiple, of varying functional intensity. Mast cells, so often associated with chronic inflammatory lesions, were present in great abundance, and some plasma cells which may have been derived from lymphocytes. The endothelial cells of the lymphatics possessed pale and swollen nuclei, and were proliferated
in places as described in intestinal enteric lesions. Mitoses were not specially examined for and were not seen. Other cells were those of ordinary gelatinous connective tissue. The synovial membrane showed a small coccus singly and in groups, stainable with methylene-blue, without sign of capsulation. It was not seen in Gram-Weigert sections, and as cultures were not made its nature is doubtful. No tubercle bacilli were present, nor tubercules, nor caseation.

"Ankle.—Synovial membrane was more cellular than that of the knee in places, but exhibited otherwise no difference in structure.

"Left elbow showed similar changes, but the granulation tissue had replaced the normal synovial membrane. It was composed of round cells with many thin-walled vessels, was oedematous in places, without giant cells or caseation. The synovial fringes were very vascular, swollen somewhat, and contained masses of lymphocytes.

"Left wrist showed similar changes."

**Case 2.**—C. W., aged 42, came up to my out-patient department on 27th April, and was admitted to hospital on 9th May 1905.

**Family History.**—His mother died, aged 64, from the effects of a fall; his father, aged 66, is alive and healthy. He has had two brothers and ten sisters, of whom five died in infancy; one sister died of "dropsy" at the age of 46, and one brother and five sisters are alive and healthy. **Married.**—Has three sons and two daughters alive and healthy. One daughter died of whooping-cough at nine months. Wife has had no miscarriages. No family history of chest or joint trouble.

**Personal History.**—Has worked in a brewery for twenty years past; previously was a bricklayer's labourer. Drinks his full allowance of four pints of beer daily in the brewery, and also has a pint or two outside. No spirits. Tobacco, 1 oz. weekly. As long as he can remember, fingers have been clubbed. Mother told him that he "was born that way." Remembers having had whooping-cough, measles, and chicken-pox as a child. Hands and feet, he says, remained stationary until two years ago, since when he has begun to have pain therein. No history of arthritis sufficiently acute to lay him up. When 14 years old was going up a ladder with a hod of bricks, when he had a fit of coughing and brought up a large quantity of blood. Has had slight haemoptysis from time to time since, occurring at least once a week. Last considerable haemoptysis was seven months ago. Haemoptysis always worse when he "gets a bit of a cold." When at school he had no cough that he can remember, but ever since the haemoptysis twenty-eight years ago he has had a cough and spit, worse in winter, and becoming progressively worse year by year. Spit is yellow; "when I bring a lot up it is more of a darker yellow and like the froth of beer." Sometimes sputum has "a nasty smell." At times cough has caused him to go off work for a week. Fifteen years ago had influenza, and was laid up for thirteen weeks. Cough has been much worse since then, and he has had paroxysms of coughing and expectoration in the mornings. Sweats copiously all over body, most in daytime. Sweating is most profuse on feet and hands, especially on soles and palms. No wasting of muscles has been noticed. Appetite is good. "Midddling thirsty." Bowels move daily. For three summers has had an attack of diarrhoea "each
fruit season"—attack lasting seven to nine days. No change noticed in face or ears. Legs below knee have at times "been numb in the morning until I got about on them." No pain except in joints. Has had swelling of feet for a day or two at a time, preventing him from lacing his boots tightly; now and again could not put on his boots. Has worn "eights" now for eight years past; previously had worn "sevens." No smaller size since he reached manhood. Denies syphilis or gonorrhoea.

Present Illness.—A fortnight after Christmas, had "a cold," which laid him up for six weeks. Had some hæmoptysis then. When he got up, found that left shoulder was stiff. He waited a month, and then, as the shoulder was no better, he came up to the hospital for advice, and the casualty house-surgeon, Mr. H. B. Walters, sent him on to me.

State on Examination.—He is a fairly healthy-looking man; of average development; nutrition fair only. No obvious morbid appearances, save the enlarged extremities and the spinal curvature. Height, 5 ft. 5 in.; net weight, 8 st. 3 lb. Colour good. No enlargement of facial or cranial bones. Large prominent ears, but these apparently are not pathological. Nose of ordinary size and shape. Skin healthy. Hair brown, rather dry; fair crop, no white hairs. Temperature, 98°.

Alimentary system.—Tongue clean, of normal size and shape. Some carious teeth. Gums healthy, and of good colour. Stomach of normal size. Splenic and hepatic dulness normal, except that upper part of liver dulness is obscured by the emphysematous right lung.

Hæmopoietic system.—No enlarged glands. Thyroid can just be felt.

Blood count.—Red corpuscles, 5,200,000 per c.mm.; white corpuscles, 12,000 per c.mm. Hæmoglobin (Gowers'), 87 per cent. Differential count of 100 white corpuscles. Polymorpho-nuclears, 71; lymphocytes, 20; large mononuclears, 7; eosinophiles, 2.

Respiratory system.—Patient is round-shouldered, and thorax is asymmetrical, the left side being smaller than the right, with very marked falling-in below the left clavicle. There is a moderate degree of scoliosis present, the convexity of the curve being to the right in the upper dorsal and to the left in the lower dorsal region. Epiglottis, of normal size. Respiration, 20. Left lung.—Expansion poor. Posteriorly, the note is greatly impaired from apex to base, in parts being absolutely dull and toneless. The breath sounds are cavernous in type, with bronchophony, passing into amphoric type, with whispering pectoriloquy at the base. No accompaniments present, except at base, where there are coarse bubbling râles. In the axilla the note is also dull; the breath sounds are obscured by accompaniments, coarse consonating crepitations and grating pleural friction. Anteriorly there is a flat boxy note to percussion, of drummy hyper-resonance in places. The breath sounds in the first and second spaces are bronchial, with fine crepitations in inspiration; below, they are obscured by creaking friction and coarse crepitations. Vocal thrill and resonance increased all over. Right lung.—Expansion fair. At extreme base there are medium crepitations and bronchial breathing, which is not conducted, for the note is somewhat impaired and the vocal resonance
is increased. Many cooing and sonorous rhonchi present, especially over lower lobe. Except at base, the note all over is hyper-resonant, and the breath sounds harsh vesicular, with prolonged expiration. Sputum.—In amount varies from ¼ oz. to 2 oz. in twenty-four hours, but that is not the total amount, patient swallowing some. Faint heavy odour, not fetid at present, does not separate into layers, in the main is composed of pus, and occasionally is faintly stained by altered blood. Microscopically, pus cells are very abundant; examinations by Dr. Bushnell, the house-physician Mr. J. B. Stephens, and myself, failed to find tubercle bacilli. Dr. Bushnell isolated the diplococcus pneumoniae.

Circulatory system.—Heart.—Powerful impulse visible in left axilla over wide area. Apex beat in sixth space 3½ in. external to the vertical nipple line, and 11½ in. external to the mid-ternal line. The left border is in the mid-axillary line, the right border (?) at left border of sternum, but neither it nor the upper border can be determined with accuracy. Heart sounds all over are of good quality and intensity, pulmonary second sound slightly accentuated. Pulse.—90, regular in time and force, good volume, normal tension, artery wall moderately sclerosed.

Genito-urinary system.—No subjective phenomena. Urine for twenty-four hours has varied from 34 oz. to 57 oz. Clear amber; no deposit; acid, sp. gr. 1018; no albumin or sugar. Phosphates not deposited on boiling. Estimation by the house-physician showed 1.8 to 24 grms. P₂O₅ excreted daily. Indican, no reaction present. No abnormality of external genitalia.

Nervous system.—Intelligence rather dull. Superficial and deep reflexes all normal. No local atrophy of muscles. No tremor. Sensation normal. Optic discs healthy.

Locomotory system.—Upper extremities.—Hands.—The fingers are long, without any enlargement of the interphalangeal joints. Extraordinary clubbing of the last phalanx of all ten digits, the phalanx forming a knob at the end of the digit. Well-marked hyperextension of the terminal phalanx as originally described by Marie. The nails are broad, convex dorsally, and very incurved at their free ends, thus approximating to the so-called “parrot-beak” appearance. They show longitudinal striation, with a tendency to chipping-off laterally. The colour of the finger-tips and of the nails is good, there being no cyanosis, but the ends of the nails show the rosy hue described by Marie. Although the nails are broad, the lateral borders of each nail anterior to the root are concave, thus leaving uncovered an arc of a circle of the bed. The exposed surface is covered by epidermis, and is not “quick” (Plate II). This appearance I have never seen before, nor can I find it recorded in any of the published cases of pulmonary osteo-arthropathy to which I have had access. It is not shown in any of the photographs of other cases which I have seen, nor does Marie draw attention to it in his original description. The patient considered that it was a loss of nail substance owing to tearing of the nails by sacks, which he handles at his work. This hypothesis of the causation may be put out of court, for not only is the condition absolutely symmetrical on the thumbs and fingers, but the deficiency is present to
an even more marked degree on the toes (Plate III., Fig. 1). The fingernails do show signs of loss of substance in the splinters which are seen there, but there is no evidence of such in the toes. From the appearance of the toe-nails, I am of opinion that the condition is really due to a disproportionate hypertrophy of the parts, the pulp of the finger, and the nail bed, i.e. the part anterior to the lunula, growing more quickly than the nail itself, and the lateral portions of the matrix becoming covered with the ordinary epithelium of the epidermis as they are left unprotected by nail. The lunule is visible in all the nails of the hands. The left little finger is the seat of a deformity owing to a beer-barrel falling on it three years ago. The first phalanx is partially dislocated outwards and backwards upon the head of the metacarpal. Extension is unimpaired, but flexion is poor, especially in the first interphalangeal and \textup{in} the metacarpo-phalangeal joints. Hand proper.

The heads of the metacarpals are somewhat prominent, otherwise there is nothing abnormal to be seen. There is no enlargement of the metacarpus and carpus, as in the boy's case. No atrophy of the short muscles of the hand. There is very excessive sweating of the fingers and hands, especially marked on their flexor aspects. Wrist.—There is noticeable enlargement of the lower ends of the radius and ulna, especially of the left hand, but not nearly to the degree met with in the boy's case. In the other bones and articulations of the upper extremity there is nothing abnormal to be made out by any method of examination. The stiffness of the left shoulder is apparently purely muscular and fibrous. (The adhesions were broken down under an anaesthetic on May 18, and now, May 24, movement is fairly free.)

Lower extremities.—The toes show analogous appearances to the fingers. The narrowness of the nails as compared to their beds has been noted above. The curving of the nails is very marked; they are long, and are closely applied to the plantar surface of the distal phalanges. There is no dorsiflexion of the distal phalanges. There is distinct swelling of the foot proper, apparently in the main bony. The lower ends of the tibia and the fibula are enlarged, but there is not sufficient thickening of the lower end of the leg to give the "elephant's foot" deformity. Perspiration is even more excessive than on the hands. In the other bones and joints of the lower extremity there is nothing pathological.

I have referred to the spine under the heading of the Respiratory System, because I consider the scoliosis much more likely secondary to the contraction of the left lung, and not due to a disease of the vertebral bones or joints.

MEASUREMENTS.

Upper Limbs.

Volume of Hand up to 2 ins. above Styloid Processes of Ulna and Radius (by Volume of Water displaced)—

Right—16 oz. 4 drms. \quad Left—17 oz. 2 drms.

\begin{align*}
\text{Lower crease of wrist to tip of mid finger} & \quad \text{Right.} \quad \text{Left.} \\
& \quad 7\frac{3}{4} \text{ in.} \quad 7\frac{3}{4} \text{ in.} \\
\text{Maximum breadth of palm} & \quad \text{Right.} \quad \text{Left.} \\
& \quad 3\frac{3}{4} " \quad 3\frac{3}{4} " \\
\text{Breadth at base of fingers} & \quad \text{Right.} \quad \text{Left.} \\
& \quad 3\frac{3}{4} " \quad 3\frac{3}{4} "
\end{align*}
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Right. Left.

Breadth of wrist 2 3/4 in. 3 in.
Circumference of hand (maximum) 9 in. 8 1/2 in.
  " , at base of fingers 8 in. 8 in.
  " , wrist 7 in. 7 1/2 in.
  " , elbow 9 in. 9 in.
  " , forearm at middle 8 3/4 in. 8 1/2 in.
Length of thumb, palmar crease to tip 2 3/8 in. 2³/₈ in.
  " , first finger 3 in. 3 in.
  " , second 3 1/2 in. 3 1/2 in.
  " , third 3 1/2 in. 3 1/2 in.
  " , fourth 2 ³/₈ in. 2 ³/₈ in.
Circumference of first phalanx of middle finger (maximum) 2³/₈ in. 2 ³/₈ in.
Circumference of first phalanx of thumb (maximum) 3 1/4 in. 3 1/4 in.
Breadth of nail of first finger (maximum) 1 1/8 in. 1 in.
  " , thumb 1 1/4 in. 1 in.

LOWER LIMBS.

Volume of Feet up to 1 in. above Level of Tips of Malleoli (by Volume of Water displaced)—

Right—43 oz. 6 drms. Left—44 oz. 5 drms.

Anterior superior spine to internal malleolus 33 in. 33 1/₂ in.
  " , inner condyle 18 1/₂ in. 18 1/₂ in.
Inner condyle to internal malleolus 15 in. 15 1/₂ in.
Circumference of knee-joint 13 ⅞ in. 13 ⅞ in.
  " , calf 11 ⅞ in. 11 ⅞ in.
  " , ankle round heel 13 ⅞ in. 12 ⅞ in.
  " , at malleoli 10 1/₂ in. 10 1/₂ in.
  " , middle of foot 9 ¼ in. 9 ¼ in.
  " , foot at base of toes 8 1/₂ in. 8 1/₂ in.
Length of great toe 2 3/₄ in. 2 3/₄ in.
Circumference of great toe at first phalanx 3 ⅞ in. 3 ⅞ in.
Breadth of great toe at first phalanx 1 1/₂ in. 1 in.
Breadth of nail of great toe 1 1/₄ in. 1 in.

THORAX.

In horizontal nipple line during deep inspiration 17 ¾ in. 16 in.

Skagrams of this case in most respects resemble those of the boy. There is the same deposit of new bone along the shafts of the phalanges, and thickening of the lower ends of the ulna and radius, and tibia and fibula. In all these bones there was, however, less thickening than in the boy’s case, and I cannot satisfy myself that there is any thickening of the metacarpals or metatarsals. The terminal phalanges show a most interesting condition. From the borders of the phalanx, and also, although to a less degree, from the anterior and posterior surfaces, there project tufts of spicules of new bone, which thus form a solid groundwork of support for the hypertrophied soft parts (Plate IV.). This new formation is seen in the fingers and thumb only at the ungual extremity of the phalanx, but in the toes is even more noticeable at the base (Plate III., Figs. 2 and 3).

Diagnosis.—The case, then, would seem to be primarily one of
bronchiectasis: of very old standing in the left lung, in which considerable fibroid contraction and consolidation have taken place. The condition, however, is bilateral; the right lung is certainly affected, although to a much less extent, in the main being in a state of complementary emphysema.

**General Review.**—There are two striking examples of the condition first described by Marie (1) in 1890 as “Osteo-arthropathie hypertrophiant pneumique.” Since that date a great many cases have been published in this country and abroad, and a number of these have been tabulated by Janeway (2) and by Wynn (3). Marie published his monograph with a view to avoiding further confusion between these cases and acromegaly, and yet he contrived to make the mistake himself, for Arnold’s (4) post-mortem examination of the elder brother Hagner (one of Marie’s original cases) proved that his disease was really acromegaly. All the distinctive features of Marie’s hypertrophic pulmonary osteo-arthropathy were met with in my two cases; what little fresh I have to record is due, not to any omission on the part of Marie, but to our acquirement since 1890 of the X-rays.

It must always be borne in mind that this form of osteo-arthropathy is not a primary disease of the bones and joints, but a secondary process set up therein as a consequence of disease elsewhere in the body; as Marie has it, “constituant pour ainsi dire un accident au cours d’une autre affection antécédente.”

**Symptomatology.**—From a consideration of the features of the two cases described above, one gets a unique clinical picture, the like of which occurs in no other disease. To recapitulate briefly: There is a symmetrical enlargement of the feet and hands, but not only are these members enlarged, they are also deformed, the different parts not being enlarged in the same proportion. The enlargement affects hard and soft parts alike. There is subperiosteal thickening of the shafts of the long bones, effusion into the joints, and hypertrophy of the soft tissues of the terminal segments of the digits. Thus the fingers were likened to drumsticks by Marie, and the thumb to the clapper of a bell. The nails are quite distinctive, and have been fully described already. There is enlargement of the lower ends of the long bones at the wrist and ankle, with effusion into the joints. Similar changes may be met with in the knee and elbow, and much less commonly in the hip and shoulder.

Spinal curvature is common, kyphosis or scoliosis, but is usually secondary to changes in the lungs or to caries of the vertebrae, and not caused by the characteristic lesions of the disease. Marie held that kyphosis, if present, invariably affected the lower dorsal or lumbar spine, but this distinction has been proved too absolute.

The arthritic changes consist in thickening of the synovial membrane and effusion of fluid into the joint cavity, with or
without ulceration of the articular cartilage. From this and the thickening of the bones of the articulations the movements are impaired, but there is no lipping of the articular surface, far less any true ankylosis.

Pain and tenderness are variable: most cases show little of either, pursuing essentially a chronic course; in others there have been exacerbations of the process, accompanied by both pain and tenderness. Attempts have been made to show a correspondence between these exacerbations of the osteo-arthropathy and exacerbations in the primary pulmonary disease. It is important to note that there is no tendency whatever to pus formation or disintegration of the joint.

The skin over the affected parts shows excessive perspiration, on the dorsal aspect according to Marie, but on the ventral in these two cases.

Muscular wasting is usually present, but is not a marked feature. There is no reaction of degeneration, no fibrillary tremor; the wasting is general, and not localised to certain muscles or segmental groups of muscles, and is merely due to disuse, not to trophic changes. Of negative importance is the absence of any affection of the bones of the cranium and face or of the jaws, a deformity of the upper jaw described by Marie not having been confirmed by later observers.

Various writers have reported other pathological conditions of every system of the body, but none of these are even approximately constant, and would seem to have been mere coincidences. All I need refer to here is that in several cases there has been noted enlargement of the acromial end of the clavicle, of the acromion process and spine of the scapula, and of the crest of the ilium.

The two cases I have described above may be taken as illustrating the two types of the disease as regards its clinical course, the boy being a subacute case, the man a case of the much commoner chronic type. The boy, indeed, was a nearer approach to an acute case than any which I have read or seen,—within six months of the first symptoms of the disease he was practically a helpless cripple, and during the last year of his life he had again and again attacks of subacute arthritis, in which he suffered great pain. In his case the arthritis, in the man's case the osteitis, was the more prominent.

Diagnosis.—The diagnosis rests upon the presence of the characteristic features of the disease enumerated above, and the presence of a primary disease—usually in the lungs. The osteo-arthropathy was present in the boy's case for nearly a year before we were able to diagnose the disease of the lung. In the majority of cases, however, the osteo-arthropathy does not appear until the primary disease has been well established. The man's story that he was born with clubbed fingers may be discredited, but in all probability the condition dates back to infancy or very early
childhood, and the bronchiectasis may have been first set up by whooping-cough or measles. At the same time there is always a possibility of the bronchiectasis being congenital.

The differential diagnosis presents no difficulty. The main points of difference from acromegaly were laid down by Marie as follows. Acromegaly is a primary disease dependent upon disease of the pituitary body, and running a continuous course to a fatal termination. Osteo-arthritis is secondary to disease of the lungs, its course is liable to intermissions, and it is not fatal in itself. In acromegaly the various parts of the hands and feet are enlarged in the same proportion; in osteo-arthritis the main enlargement falls upon the fingers and toes, and especially on the terminal phalanges. As Marie says, the hands and feet are not only enlarged, but also deformed. In acromegaly the nails are unaffected. In acromegaly joint affection is rare: in osteo-arthritis, common. In acromegaly, the face, tongue, lower jaw, genitals, and the cartilages of the ears, nose, eyelids, and epiglottis are enlarged; they are unaffected in osteo-arthritis. These data are quite sufficient to make the differential diagnosis perfectly clear. One cannot, I think, insist upon enlargement of the carpus and metacarpus being peculiar to acromegaly (cf. boy's case), or give diagnostic importance to the seat of any kyphosis present.

Pathology and etiology are best considered together. By far the most exhaustive account in the literature of this country will be found in Wynn's paper. Marie's name is cumbersome, but it is at least expressive: he bases it partly on the history of his cases, partly on examination of the lungs, and states that "Pneumique comprend ici pour moi non seulement le poumon, mais aussi ses annexes, bronches et plèvres." He thus includes all cases due to bronchiectasis and empyema. His view of the pathology of the condition was that there was first production of toxins in the lungs, followed by absorption of these toxins into the general circulation, and finally selective actions of the toxins on certain bones and joints. This view was supported by von Bamberger(5), who held that the essential cause of the osteo-arthritis was the absorption of chemical toxins from phthisical or bronchiectatic cavities. He endeavoured to produce the disease artificially in dogs by rectal injections of foul bronchiectatic sputum, but a six-weeks' trial of the method proved unsuccessful. So far as I know, his are the only experiments which have been made. Must there then be an additional factor present, predisposing the bones and joints to chronic inflammatory changes?

Godlee(6) "regrets the absence of reference to the possible complication with syphilis in the cases recorded," and suggests that some of the cases recorded as osteo-arthritis were really examples of that form of osteo-arthritis which is associated with chronic septic suppurations, such as gonorrhoea and leucorrhoea, complicated with independent clubbing of the fingers. The
question whether syphilis may be a factor in the disease has been carefully investigated by Wynn, who could find only one undoubted case, that of Schmidt (7), whose patient developed osteo-arthropathy during the tertiary stage, and improved somewhat under potassium iodide. There was no evidence whatsoever of syphilis in my two cases. As regards Godlee's second consideration, I would point out that in chronic gonorrhoea and leucorrhoea, drainage of pus, as compared to that pent up in vomicæ or in the pleural cavity, is free. During last winter we had in the wards of the Sussex County Hospital under Dr. Hollis, a labourer, aged 40, with a general gonococcal infection. This patient had gonococcal pleurisy and polyarthritis of the most persistent character, the arthritis, moreover, affecting both hands and feet; but although we had the man under observation for seven months, there was not the slightest evidence of osteo-arthropathy, nor was there clubbing of the digits.

Wynn believes that infectious diseases “play an important part in predisposing the joints and bones to osteo-arthropathic changes.” He cannot, however, show any precise evidence in favour of this hypothesis. Infectious diseases were certainly not a factor in Wynn's own cases, or in mine. A past history of infectious disease, i.e. of one or more specific fever, is no doubt present in most cases of osteo-arthropathy, but so it is present in most cases of any other disease, and for that matter in most healthy individuals. There does not seem, judging from the summary of cases given by Wynn and my own investigation of the literature, to be a single recorded case in which an acute specific fever, occurring in the course of a chronic pulmonary disease, was followed by osteo-arthropathy. In Soltan's case there was a history of influenza eighteen months previously, no lung disease was found; in Harris's case there was also a history of influenza followed by phthisis, and the latter by osteo-arthropathy.

Thorburn (10) suggested the name “tuberculous polyarthritis,” on the assumption that the disease was “in reality a tuberculous affection of a large number of bones and joints, but that it is of benign type, having no tendency to caseate.” The majority of published cases showed no clinical signs of tubercle, and the same holds good of the reports of autopsies, so that Thorburn's view lacks support.

There is no real evidence that the nervous system plays any part in the production of this form of osteoarthropathy. Möbius (11), in a patient with bronchiectasis and clubbed fingers, saw an extreme enlargement of the ends of the two ulnar fingers follow an ulnar neuritis. This is quite an isolated case, and may have been due to vasomotor changes. Farquhar Buzzard (12), in a case reported originally by Walters (13), found no degenerated fibres in the median, ulnar, sciatic, and anterior crural nerves examined.
post-mortem. The disease differs essentially from the arthropathies and osteo-arthropathies of nervous origin, in being hypertrophic, not destructive in nature. In Hirschfeld's (14) three cases there was marked alcoholic neuritis of the legs and arms, but Hirschfeld considered that the enlargement of the extremities was due to vasomotor influences—"dermato-hypertrophia vaso-motoria."

Several post-mortem examinations have disclosed changes in the thyroid gland. Thorburn and Westmacott (15) found old tuberculous lesions in the right suprarenal body, Thorburn and Buzzard changes in the pituitary, and Erb (quoted by Marie) described substernal dulness, but there is no record of enlarged thymus being found post-mortem. There is thus no proof of the condition being caused by disease of the ductless glands, no constant changes being present in any.

There is, however, strong evidence that hypertrophic osteoarthropathy does not always depend upon pre-existing disease of the respiratory system alone. Thayer (16), Janeway, and Wynn have drawn up lists of cases tabulated according to the predominant primary disease. Thayer found such disease to be in the lungs or pleurae in forty-three out of fifty-five cases, Janeway in sixty-five out of ninety-three cases, and Wynn in sixty-eight out of one hundred cases. In Wynn's series there were thirty-eight cases "who had no pus at any time, and thirty of these had no lung disease. Therefore, if the disease be produced by the action of toxins, either these toxins can be manufactured in the absence of pus, or different toxins having an identical action can be produced by very various processes, and it becomes necessary to search for other factors." Most of these other factors which have been brought forward, I have already discussed: it only remains now to consider what part can be played by the urinary, alimentary, and circulatory systems, for I cannot hold with Wynn that "injury too, though rarely mentioned in the reported cases, should be able to play a like predisposing part."

Urinary system.—Chatin and Cade (17) have published a case in a man the subject of chronic interstitial nephritis, who showed symptoms of uremia. The patient had had several attacks of acute rheumatism, and was very thin and pale. Unfortunately the writers do not give a detailed account of the state of the heart and lungs, and, considering the nature of the case, one can hardly accept as strictly accurate the bald statement that there was "rien au cœur, ni aux poumons." It is interesting to note that they describe an appearance of the free ends of the distal phalanges of the fingers identical with that of my second case. This they picturesquely compare to the appearance of a bar-magnet which has been plunged into iron-filings. A radiogram is given, and their two cases (vide infra) and my own are the only such which I have found in the literature. Chatin and Cade, how-
ever, do not attribute the osteo-arthropathy in their case to the renal disease, but consider it more probably due to the antecedent attacks of acute rheumatism.

No constant changes in the urine have been observed. *A priori,* in a disease such as this, in which there is increased bone-formation, one would expect during the periods of activity a diminution in the excretion of phosphates, but the results of estimations are at variance. Schittenhelm (18) in one case saw, between May and July 1901, moderate diminution in the size of the hands. Measurement of radiograms showed that the retrograde change involved the soft tissues almost exclusively. In a metabolism experiment with known phosphorus-nitrogen ratio in the food, he found during a period of fourteen days a normal phosphorus excretion in the urine.

**Alimentary system.**—Gilbert and Fournier (19), and shortly afterwards Frederick Taylor (20), described a form of cirrhosis of the liver associated with great enlargement of the spleen, occurring in children, and apparently not caused by alcohol. In this "spleno-megalic cirrhosis," clubbing of the fingers, and, according to Gilbert and Fournier as usually quoted, enlargement of the bones and effusion into the joints, are met with very similar to what is seen in Marie's hypertrophic osteo-arthropathy. According to Rolleston (21), "such lesions are extremely rare in biliary cirrhosis, and are not dependent upon pulmonary lesions." Taylor (22) states positively that in his case the clubbing of the fingers was due not to a change in the bones or joints, but in the tissues, and he publishes a skiagram in proof thereof. With this type of hepatic cirrhosis I am unacquainted except by reading, and I do not find a perusal of the French cases at all convincing. In Gilbert and Fournier's original series, clubbing of the fingers was present in three of the seven cases. Of these three cases, in the first only was there joint effusion—in the knee. The child had had measles at two years, but on examination there was nothing abnormal made out in the lungs. In Case 2, the child had a chronic cough, and there were physical signs in the left lung suggestive of tubercle. Case 3 had congestion of the lungs in infancy, but the present condition of the lungs is not given. In none of these cases was bony enlargement confirmed by radiography, and the writers state that they would be tempted to suggest that the clubbing was due to upward displacement of the diaphragm by the enlarged abdominal organs, and consequent diminution of the respiratory surfaces, were it not that in Case 3 the liver and spleen were not greatly enlarged. Gilbert's second paper, in collaboration with Lerebouillet (23), is even less convincing. Case 1, when first under observation, had no clubbing of the fingers, but, seen later, having had respiratory troubles in the interval, clubbing was present. Radiograms are given, and the sole evidences of bony enlargement are an exostosis of the terminal
phalanx of the right index finger, and a slight enlargement of the ungual extremity of the distal phalanx of the left thumb, which "does not seem to exceed notably what is seen in radiograms of the normal hand." Of this case the writers conclude: "Les lésions osseuses sont en tout cas minimes." In Case 2, they found by post-mortem dissection of the parts that there was only a hypertrophy of the soft tissues, the bones being absolutely normal. In Chatin and Cade's second case, the patient—a man of 50—had cancer of the head of the pancreas, and secondary obstructive jaundice from pressure on the common bile duct. During the course of the disease a moderate degree of clubbing of the fingers developed, and skiagrams showed the free end of the distal phalanx of the index middle and ring fingers studded with osteophytes. These cases, and there are others in the literature, would seem to prove that hypertrophic osteo-arthropathy may be caused by the absorption of toxins formed elsewhere than in the lung, and especially is this the case if one accepts the view that simple clubbing is the precursor of the former condition.

Wynn suggests that in nearly all the recorded cases of hypertrophic osteo-arthropathy there is a common factor present, namely, auto-intoxication from the alimentary canal. In my second case there may be such auto-intoxication, for the man habitually swallows part of his sputum, of which his alimentary canal would seem to be remarkably tolerant.

The circulatory system.—The majority of well-marked cases of hypertrophic osteo-arthropathy would appear to occur in bronchiectasis, chronic phthisis, and empyema, all diseases in which there is increased strain thrown upon the circulatory system. In hypertrophic cirrhosis of the liver there must be increased work thrown upon the heart in maintaining the circulation through that organ, and consequently, unless there is sufficient reserve power in the heart, the general circulation must in turn suffer. Granted, then, that the circulation be embarrassed, sluggishness of the blood current will be especially found in those parts furthest distant from the heart, and, since the circulation is slowed there, it is on these parts that toxins in the blood would have most prolonged action, and would be able to exercise most effect, provided the tissues were susceptible to their action. One thus arrives at a plausible explanation why the ends of the fingers and toes are especially affected. To my mind this seems the most rational explanation of the pathology which can be offered at present. It is confirmed by the close relation between simple clubbing and hypertrophic osteo-arthropathy. No sharp line can be drawn between these two conditions. They are met with in precisely the same class of diseases. In osteo-arthropathy the fingers and toes are invariably clubbed, and in some cases the clubbing has been seen to precede the bone and joint changes. Others which on ordinary examination appeared to be cases of
simple clubbing, have been found post-mortem, or on radiographic examination, to have thickening of the bones (e.g. Cases 8, 9, and 11 of von Bamberger's series). Thus hypertrophic osteo-arthropathy is intimately connected with the clubbing of congenital heart disease. This was recognised by Marie, who evades the difficulty rather ingeniously, as the following quotation shows:—

"On remarquera cependant que la cyanose congénitale se termine fréquemment par la tuberculose pulmonaire." But all cases of congenital heart disease with clubbing do not die of pulmonary tuberculosis, nor is there any constant relation between the cyanosis and the clubbing. There may be clubbing without cyanosis, and cyanosis without clubbing. Cyanosis is not characteristic of the fingers and toes in hypertrophic osteo-arthropathy. Yet the presence of the latter condition in diseases in which there is prolonged venous congestion seems to me to be more than a coincidence. Bécère (24) has reported the case of a patient suffering from aneurysm of the third part of the right subclavian artery. The left hand was perfectly normal. The fingers of the right hand were clubbed. The pressure on the veins was not sufficient to cause oedema or change of colour when quiet, but on use of the arm the hand became cyanosed. Wynn, who quotes other cases, is of opinion that the enlargement of the fingers was oedematous, and not true clubbing. In clubbing, whether there be bony enlargement or not, the greater part of the swelling is usually due to hypertrophy of the soft tissues, and particularly to an increase in the fat, in itself an evidence of diminished oxygenation. My first case is a manifest exception to the rule laid down by Wynn, that even where bony enlargement is present, "the terminal phalanges are much longer than the others which have greater increase of bone." Freytag (25) has described great distortion of the capillaries in the bed of the nail. In one of Thorburn's three cases of hypertrophic osteo-arthropathy the boy had mitral stenosis and clubbing of the fingers. There was no disease of the lungs, and the thickening of the lower ends of the long bones at the wrist and ankle was confirmed post-mortem. Samuel West (26) has made a special study of clubbing of the fingers, and holds that, although it is often associated with disease of the chest obstructing the circulation, nothing is known of the cause in general. He cites cases occurring in individuals apparently quite healthy, and other cases in which the onset was so sudden that the enlargement must have been vascular in origin. West considers that osteo-arthropathy may be divided into two groups. His first group he makes up of cases in which, with clubbed extremities, there is swelling in or round the joints, but no bony lesions. These cases he compares to gonorrheal rheumatism, and thinks that they are probably due to absorption of toxins. In his second class the cases show extensive changes in the shafts and articular ends of the bones, and are, he says, so
rarely associated with chest lesions that such association may be merely accidental. He inclines to think that the true cause may be tubercle or syphilis—thus coming into line with Thorburn and Godlee, to whose views I have already referred. Godlee, in Fowler and Godlee's work on the lungs (27), goes even further, and holds that "it is impossible to distinguish many cases of pulmonary osteo-arthropathy" from tubercular or syphilitic bone and joint lesions and osteo-arthritis. While one is bound to respect the opinion of such eminent authorities, my sympathies are with Marie in his view that the changes in osteo-arthritis are distinct from those of any other affection.

Dr. John Thomson writes to me that his belief is that pulmonary osteo-arthritis is an entirely different affection from clubbing of the fingers, but until a case is found of osteo-arthropathy without clubbing, or until a definite and distinct causation is found for the two conditions, it seems to me right, in view of their intimate clinical relation, to regard them as different stages of the same process. Clubbing is not edema, and is but seldom associated with edema. I cannot see, however, that this negatives the point at issue, as some authorities maintain. Complete stasis is much less likely to cause the lesions of hypertrophic osteo-arthritis than a sluggishness of the circulation, which continues to bring fresh supplies of toxins to the parts, and at the same time affords them, when there, more time to exert their special action upon the tissues. The result of that action is hypertrophy of the tissues, and venous congestion, artificially produced, has been used in surgery to promote regeneration and hypertrophy.

These are the main facts that can be brought forward in an attempt to prove that obstruction of the circulation is a factor in the causation of hypertrophic osteo-arthritis. Should the results of the blood-count from the finger-tip of the second case be confirmed by later examinations which I intend to make, they will afford important confirmatory evidence. Such an explanation of the pathology cannot, however, hold good of all cases, for in many there was no disease of the heart found either clinically or post-mortem, and in some there was no apparent cause of embarrassment of the circulation, nor was there any evidence of such embarrassment. We cannot at present determine accurately the exact pathology of the disease.

Pathology of my own cases.—In Case 1 the true pathology can be only a matter of conjecture. The latest views of the metabolism of malignant tumours do not support the hypothesis that morbid products are introduced therefrom into the circulation. Yet the case is typical of hypertrophic osteo-arthritis, although there was no pus or decomposing or necrotic tissue present in the body, and although at the time of onset of the symptoms there was cause of obstruction of the circulation, the heart being healthy.
and in its normal site, and the soft fleshy tumour in the interior of the lung forming no embarrassment to the circulation. The case may have been further complicated by rheumatism, but no bacteriological examination of the blood was made, our pathological laboratory not being in existence at the time. I would again draw attention to the fact that the beginning of his illness was the periosteal sarcoma.

In Case 2 the pathology to my mind is clear. The case corresponds exactly to Marie’s original description. There are numerous badly drained vomicae in the right lung from which toxic infection of the blood ensues, and I would suggest that the obstruction to the circulation by the displacement of the heart and the condition of the lungs is sufficient to account for the main localisation of the lesions at the extremities.

Nomenclature.—Marie’s name, although unwieldy, gives us in three words the essentials of the disease, as usually met with clinically. “Secondary hypertrophic osteo-arthropathy,” the term first suggested by Massolongo (28), and adopted by Thayer and Wynn, is no shorter and is less definite than Marie’s original title, but it gets over the difficulty that in some cases no pulmonary disease has been found. Other names which have been suggested are “Hyperplastic osteo-artiitis” (Walters); and “Secondary hyperplastic ostitis” (Arnold), and “Toxigenic ossifying osteoperiostitis” (Sternberg (29)), neither of which includes the changes in the joints. Owing to the site of the primary disease in my two cases, I have preferred to publish them under Marie’s original name.

Treatment must of course be directed towards the primary disease. Gillet (30), Moizard (31), Moussons (32), Godlee, and others have reported cases where simple clubbing diminished or disappeared after successful treatment of the primary disease, usually empyema. It may be that the bony changes, in their early state, are also susceptible to treatment, but once well established they are permanent. In one case special treatment would seem to have been attended with a measure of success. Demons and Binaud (33) have observed considerable improvement in a patient treated by subcutaneous injection—during several months—of tissue extract from the lungs of healthy sheep. The rationale of such treatment does not seem to me sufficiently clear to warrant its adoption. During the periods of exacerbation treatment of the osteo-arthropathy itself may give relief to the patient’s pain, but is palliative only, and cannot in any sense be held to be curative. As a rule, the bones and joints give little trouble, their symptoms being of quite secondary importance compared to those of the primary disease.

I have to thank Dr. W. Ainslie Hollis for his kindness in allowing me to publish the first case. My thanks are also due to Mr. Geoffrey Bate, M.B., for the two photographs of the second case.
PALMAR surface of left hand and wrist from Case 1. Shows enlargement of fingers, metacarpus, carpus and wrist, as described in the text. The nails are visible on thumb and index finger. Note subperiosteal sheaths of new bone on middle and proximal phalanges, metacarpals, ulna and radius. The carpal bones are separated by effusion of fluid into the small joints. The epiphyseal lines are shown.

PLATE II.
Right hand and wrist from Case 2.

PLATE III.
Fig. 1.—From a photograph of right foot of Case 2, to show the toes and nails. Note the uncovered portions of the nail-beds.

Fig. 2.—Radiogram of left hallux from Case 2—plantar surface, natural size. To illustrate especially the new bone formation in the distal phalanx. There is also some thickenings of the first phalanx.

Fig. 3.—Skiagram of one finger from Case 2. Shows the bulbous hyper-extended distal phalanx, the outline of the nail, and the bony thickening.

PLATE IV.
Skiagram of palmar surface of left hand from Case 2. Shows the length of the fingers, deposits of new bone at ungual enlargement of distal phalanges, and along shafts of middle and proximal phalanges, especially the latter; also enlargement of lower ends of radius and ulna. Contrast metacarpal bones and small joints of carpus with those of Case 1 shown in Plate I.

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THE IMMORTAL MEMORY OF HARVEY.

By Charles E. Underhill, M.B., F.R.C.P.Ed., President of the Harveian Society.

The Harveian Festival was celebrated at the Royal College of Physicians on the 9th June 1905. There were present the President, Dr. Charles E. Underhill, Sir Halliday Croom, Professor A. R. Simpson, Dr. Clouston, Professor Chiene, Dr. Crawford Renton, Dr. Playfair, President, Royal College of Physicians; Dr. McBride, Dr. George A. Gibson, Dr. Dunsmure, and about fifty members of the Society. The immortal memory of Harvey was eloquently proposed by the President in the following terms:—

I now come to what is really the toast of the evening, namely, "The Immortal Memory of William Harvey." It is but to very few men that this honour is accorded. So far as I know, John Hunter is the only other man in our profession on whose name a Society has been founded, and such a toast is annually proposed; while at the annual dinners of the Edinburgh Obstetrical Society one of the toasts is always the Immortal Memory of Sir James Simpson.

Now it seems to me to be rather a solemn thing to stand here, and, looking backward over the centuries, to ask you to drink to this toast to one of our greatest professional heroes. Hero-worship, and this is hero-worship, is an ennobling pursuit so long as our hero is a worthy one; and who will venture to say that Harvey is not worthy? It lifts us out of the dull and dusty track of daily life, and raises us to a higher plane and a purer air. It makes us forget for a while the trials and the troubles, the sorrows and the sins, with which we have to contend day after day and hour after hour, as we fight the battle of professional life.