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Indian Journal of Radiology

May 1949.

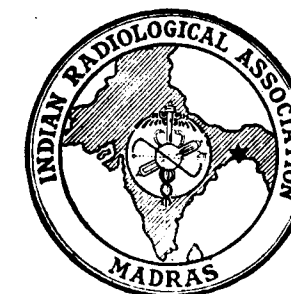


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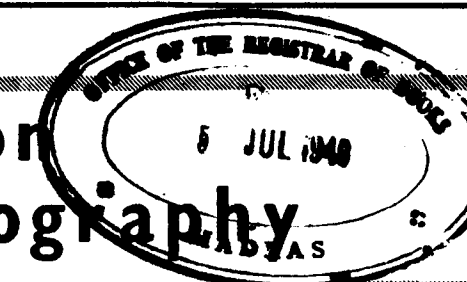
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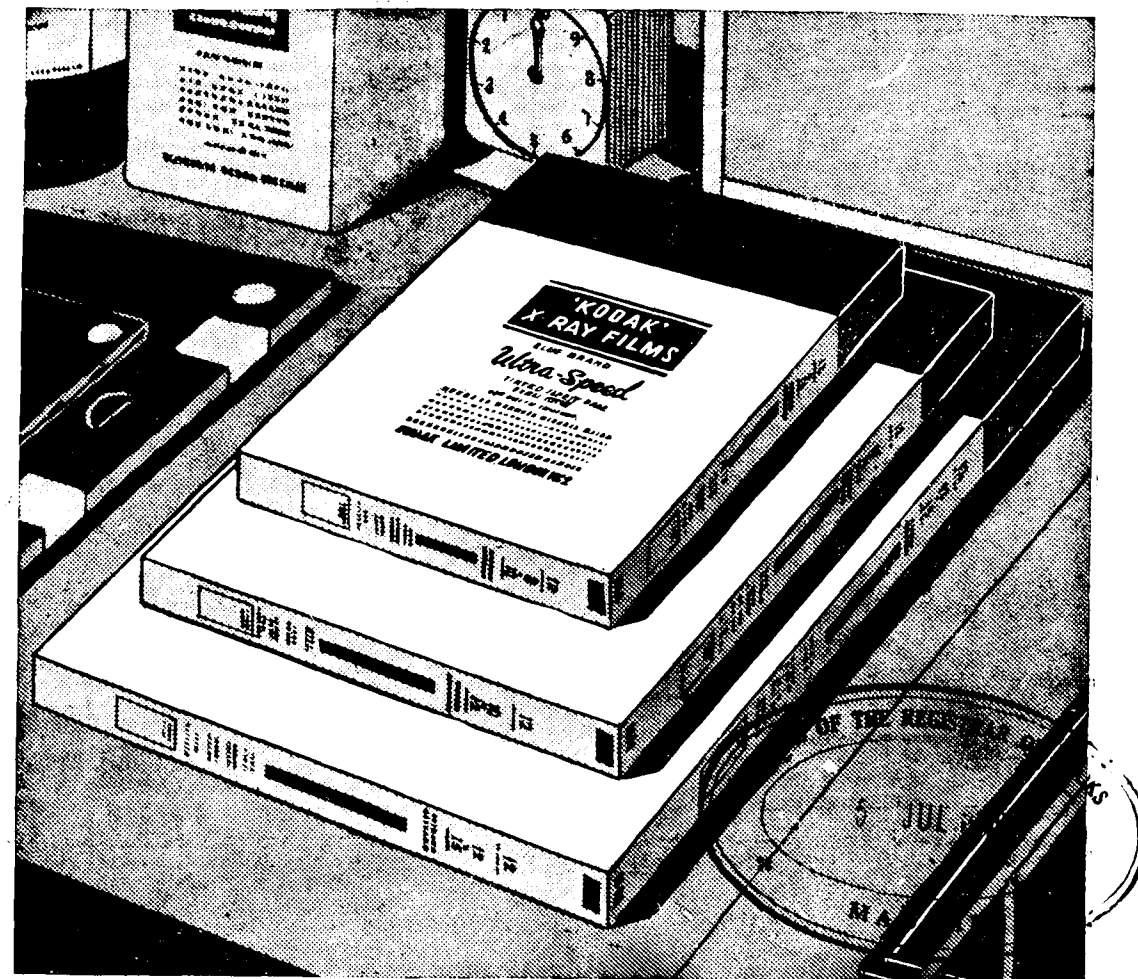
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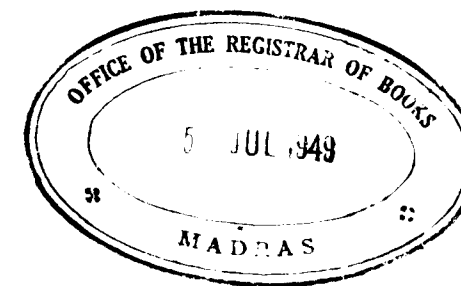
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MAY, 1949

No. 2.

A Comparative Five-Year and Ten-Year Salvage of Carcinoma Cervix-Uteri by Radical Vaginal Operation and Radiation

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SEVENTEEN years ago, during the Third International Radiological Congress in Paris, where I submitted my five-year statistical end-results of carcinoma of the cervix uteri treated with radiation therapy, I was much impressed to find continental surgeons performing the radical vaginal operation in similar cases. On further inquiry, I found that by combining this operation with postoperative radiation, they obtained very satisfactory results. The rationale of the treatment of carcinoma of the cervix is still a controversial subject. Although the general trend of opi-

nion is in favour of radiation therapy, the surgical treatment still occupies a definite place in its management. Moreover, some workers who were previously converts to radiation therapy are coming back to surgery supplemented by radiation therapy.

An analysis of world statistics shows that whatever method be followed (operation or radiation) the end-results are, for all practical purposes, the same in the hands of experts, with a small percentage of variation which may be a chance variation. The popularity of one method over the other depends not

only upon the results obtained, but also upon the easy application of the method without harm to the patient. It is the general belief that radiation therapy is much easier to apply. There might be a bit of truth in it, but it is very difficult to check and control whether radiation therapy has been properly executed or not. Experts are of the opinion that "properly performed Radium therapy is as difficult to learn as the operation itself". An irresponsible radiotherapist may carry on radiation therapy without being exposed to public criticism, because there is much less chance of primary mortality. On the other hand, it has been remarked that the "operation of carcinoma of the cervix as performed by Halban, Adler, or Stoeckel, is much too formidable an operation for general use". My only remark is: does the defect lie in the method itself or in the true make-up of a gynaecological surgeon? Should a gynaecologist be worth the name of a surgeon if he considers an important gynaecological operation formidable? I am more than conscious that everybody cannot be a Wertheim, a Bonney, or a Schauta, but can everybody be a Heyman? I believe that it is high time that the fight for the supremacy of one method over the other should come to an end. So far as cancer of the cervix is concerned, a gynaecologist who wants to take it up as a life

study must be a crack surgeon as well as a first class radiotherapist in order to work out by the process of evolution a more suitable technique in the near future, and thus to salvage more patients suffering from this fell disease.

Clinical Materials

During the last 22 years, 1463 cases of cancer cervix were examined. According to League of Nation's classification, 91 cases (6%) belong to Grade I, 321 cases (22%) to Grade II, 882 cases (60%) to Grade III and 169 cases (12%) to Grade IV. The total number of cases may be divided into 2 age period groups *i.e.*, those between 1926 to 1931 and those between 1932 to 1948. The first group were treated solely with Radium and Deep X-ray Therapy and the next group were partly treated with Radium and Deep X-ray and partly with Radical Vaginal Operation and subsequent radiation.

Complications and Technical Difficulties

The radium treatment is not without danger. Heyman gives a primary mortality of 1.19 per cent of his cases from 1914 to 1921. Out of his 6 fatal cases, one died of pulmonary embolism and the remaining ones died of sepsis resulting in either pelvic or diffuse peritonitis. Although infection can to a great extent be controlled at present by

sulfa drugs and penicillin, it is our experience that infection when once well established becomes more or less intractable. Besides infection, hæmorrhage, embolism, suppression of urine by ureteritis, perforation of the uterus and grave anæmia are not uncommon occurrences. I lost one case from cerebral embolism, two cases from acute ureteritis and several cases from combined infection and hæmorrhage. There are cases on record where there had been necrosis and sloughing of intestines necessitating resection to the extent of 6 to 8 inches of the gut. 13*

On carefully analysing these complications it appears that most of these complications are preventable. Difficulties arise from haphazard and incomplete investigation of case prior to institution of the treatment, from faulty location of the position of the pelvic organs specially the uterus, at times from inaccessibility of the cervical canal which has been either distorted or stenosed by the growth and from a gunshot type of Radium or X-ray treatment without any particular attention to individual variation.

Infection is the greatest handicap in Radium treatment and as such demands routine examination of vaginal smear for both aerobic and anaerobic growths. The precise and accurate diagnosis of the extent of the growth as well as the position of

the uterus is the next essential point. This is not always an easy job and must be done by an expert Gynaecologist, because much of the good results depend upon the accurate diagnosis of the extent of the growth and consequent suitable application of Radium. In quite a good number of cases intra-uterine application of Radium is very difficult. If not properly done and carefully manoeuvred, there will be too much mutilation of the growth, damage of the neighbouring healthy tissues, sometimes even leading to perforation. This will certainly vitiate the results of the treatment. If the cystocele and the rectocele are not properly guarded, it will lead to vesico-vaginal and recto-vaginal fistulæ. A retroverted uterus laden with Radium tandems will add to rectal troubles resulting in severe proctitis, telangiectasis and protracted rectal bleeding. A laterally deviated uterus will produce ureteric complications. That is why it has been so wisely remarked that "properly performed Radium therapy is as difficult to learn as the operation itself."

The conventional line of Radium and X-ray treatment consists of 6 to 7 thousand milligramme hours of Radium locally and about 3000r from deep X-rays at the tumour affected area as a general rule without much of individualisation. It

is certainly an empirical procedure to give Radium treatment without more scientific dose measurement. It neglects the important factors of distribution and Radium-tumour distance. Recent investigations recommend 10,000r Radium as lethal dose; and for proper implementation, distribution is to be charted by the isodose curves so that the remaining cancer affected tissues and nodes may be adequately treated by deep X-rays without much of superimposition. Dosage system for routine use without individualisation has got definite disadvantages, because it had to depend upon certain predetermined points chosen arbitrarily in relation to pelvic structures which are mobile in normal conditions and often undergo considerable displacement in multiparous women. That is why an isodose chart must have to be made in every case taking into account individual variations in anatomy. This is possible when the exact location of Radium in situ is found out either by the method of reconstructing the positions of radium sources by using the tube-shift radiograph, or by the Contour Projector method of Mayneord or by the three dimensional X-ray technique of Winternitz.

The transverse diameter of the mid pelvis of an average female is about 13 c.m. The central part of

the coronal plane at the level of the ischial spine or external os receives an effective dose of radium (varying from 20,000r to 6,000r), the distance covered being approximately 5 to 6 c.m. The intervening volume of tissues between this adequately treated central zone and lateral pelvic wall measuring only 4 c.m. on each side is to be treated by X-rays. The lateral 'throw' of the effective dose from the central zone should also be taken into account while calculating the effective dose of the lateral zone. The initial lesion of radiation necrosis is not so much due to direct effects of the radiation on rectum or bladder but to high dose effects at the junction of the central and lateral zones where the ureteric bend is unfortunately placed. This point is invariably ignored while deep X-rays are given subsequent to Radium treatment. Deep X-ray treatment should be given through a 4 c.m. wide portal instead of the usual 10 x 15 c.m. fields. It has become a proved fact that for effective treatment, lymph nodes require a fairly high dose. It would be practically impossible to give 3,000r. tumour dose through a small portal of 4 c.m. wide from any 200 k.v. Deep Therapy apparatus without causing considerable skin damage and great metabolic disturbances resulting from a high cumulative "integral dose". The main factors controlling the integral dose

are the size and number of the fields and the penetration of the radiation. If the full tumour dose is properly and systematically given, the integral dose averages 40 mega-gramme-r at 1,000 k.v. and produces definite impairment of patients vitality but from which recovery occurs in about 2 months. At 200 k.v. the integral dose would be over 60 mega-gramme-r, which is higher than has yet been given in any condition, and would almost certainly impair the patient's vitality to a critical degree. (Phillips). Here comes the real importance of supervoltage with a million-volt apparatus. With 200 k.v. a large portal is essential in order to have the eventual radiation which is composed of one-third primary radiation and two-thirds secondary radiations. On the other hand, secondary radiation plays a small part when millionvolt is employed. Further, with a million-volt apparatus secondary radiations that are formed do not scatter haphazardly in all directions but are driven straight forward because of the tremendous energy of the primary beam. Consequently they are added to and become part of the bundle of the primary beam. (Friedman). Thus the narrow beams of X-rays may be efficiently employed through a small portal of 4 c.m. width, without impairment of the vitality of the normal tissue or of the patient as a whole.

Statistical Analysis

For proper assaying of statistical results three things are essential, viz., operability rate, relative cure rate, and absolute cure rate. For the last twenty-two years I have been classifying my cases according to the League of Nations' formula and, since 1932, operation has been undertaken as a side-chain to radiotherapy. Materials for operation were taken not only from Grade 1 and 2, but also from Grade 3. Thus, 42.6 per cent of Grade 1, 28 per cent of Grade 2 and 5 per cent of Grade 3 cases were operated upon, and the rest were treated with radiation therapy. Although all the clinical materials were at my command, I did not insist on operation for all cases which were deemed operable. Patients were given a free choice, and operation was done only on willing patients, although I have found by careful scrutiny that 57.7 per cent of all cases could have been operated. I have operated on 165 patients, six of whom died of operation, the primary mortality being 4 per cent. The total number of patients operated upon between 1932 and 1940 is ninety-three. Five-year salvage cases totalled 35, i.e., a relative five year cure rate of 37.6 per cent. The total number of patients operated upon between 1937 and 1940 is 40, of whom 16 patients were well after five years, thus yielding a relative five year cure rate at

40 per cent. The total number of patients operated upon during 1932 to 1936 is 53, of which 19 were well after five years, and 13 after ten years, thus giving a relative cure rate of 36 and 24·5 per cent respectively. When taken separately in different Grades, the five year relative cure rates were 61·5 per cent of Grade 1, 44 per cent of Grade 2 and 15·6 per cent of Grade 3.

Against these figures and during the same period between 1932 and 1940, my five-year relative cure figures by radiotherapy are 66 per cent in Grade 1, 31·5 per cent in Grade 2, 12·6 per cent in Grade 3, and 2·3 per cent in Grade 4 (Tables I—V).

TABLE I

Operability rate	..	57·7 %
Total Number	..	165
Primary Mortality	..	4 %
Relative 5-year cure	..	37·6 %
Relative 10-year cure	..	24·5 %

TABLE II
(1932-1940)

Total No. operated	..	93
5-year salvage	..	35
Relative 5-year cure	..	37·6 %

TABLE III
(1937-1940)

Total No. operated	..	40
5-year salvage	..	16
Relative 5-year cure	..	40·0 %

TABLE IV
(1932-1936)

Total No. operated	..	53
5-year salvage	..	19

Relative 5-year cure rate	..	36·0 %
Relative 10-year cure rate	..	24·5 %

TABLE V
Relative 5-Year Cure Rate
(Radiation Therapy)
(1926-1932)

Stage I	..	53·4 %
Stage II	..	37·5 %
Stage III	..	9·4 %
Stage IV	..	1·7 %
Absolute 5-year cure rate	..	12·92 %
Absolute 10-year cure rate	..	9·4 %

TABLE VI
Comparative 5-Year Cure Rate
During the Same Period, 1931-40

	Operation	Radiation
	%	%
Stage I	.. 61·5	66·0
Stage II	.. 44·0	31·5
Stage III	.. 15·6	12·6
Stage IV	..	2·3
Absolute 5-year cure rate	..	12·9
Absolute 10-year cure rate	..	9·4

Unfortunately, I cannot give an absolute cure rate of my cases from 1932, as the patients cannot be grouped under one particular method of treatment. Result will certainly be vitiated if an attempt be made to find out an absolute cure rate of operation cases against the bulk of remaining cases who were treated by radiotherapy.

My results from radiation therapy from 1926 to 1932 are as follows : the relative five-year cure rates are a 53·4 per cent of Grade 1, 37·5 per cent of Grade 2, 9·4 per cent of Grade 3, 1·7 per cent of Grade 4. The absolute cure rate is at 12·9 per cent

after five years and 9·4 per cent after 10 years.

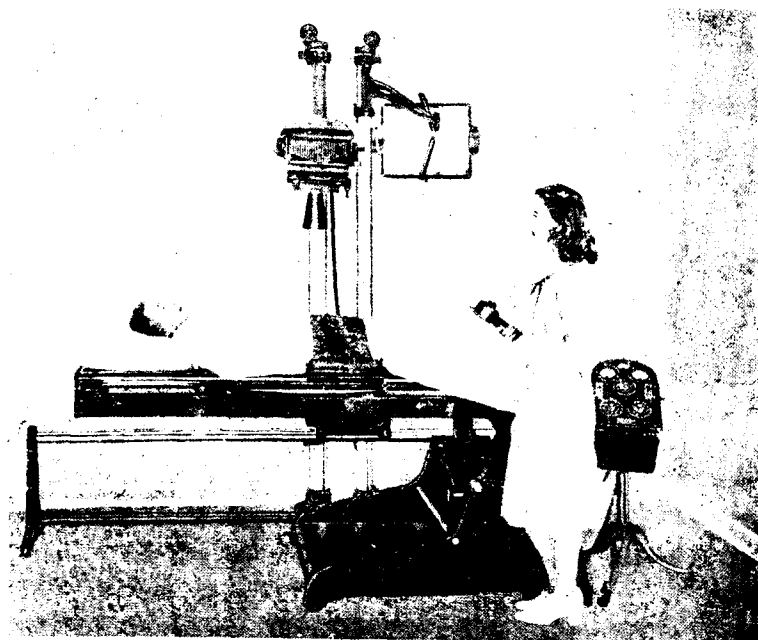
It is evident that the results of radiation are not so striking as those of operation. So far as radiation therapy is concerned, I am afraid my technique is perhaps not very accurate and precise. Besides, my hospital cannot afford to give free treatment to all patients, thereby sustaining a loss of the early cases. The patients are mostly poor and irresponsible. A good number of them (18·3 per cent) did not take the complete course of treatment, either because they could not afford it, or they thought they were cured in spite of repeated insistence to the contrary. Moreover, there being no arrangement for registration system, patients once changing their residence could hardly be traced. Lastly war, famine, flood and communal disturbances have

practically dislocated the civil life, thereby making difficult the following-up system. If I take a relative cure-rate of only those cases who have taken a complete course of treatment, the results will be 14·9 per cent, which stands comparable with the results of other workers.

Conclusions.—In conclusion, I must admit that we have as yet no remedy for advanced cases. Operable cases yield satisfactory results up to a certain limit whatever method is followed, provided treatment be given efficiently and with meticulous precision. It is only by the detection of early cases and centralization of patients in special cancer clinics that we can markedly improve our end-results and hence the right movement should be the education of lay public and special courses of cancer training for the general medical practitioners.

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Radiological Aspects of Neuropathic Joints*

DR. S. N. DEBOO, M.B., M.R.C.S. (Eng.)

HON. RADIOLOGIST TO THE G. T. HOSPITAL (Bombay),

TEACHER IN RADIOLOGY FOR D.M.R. (Bombay), AND D.M.R.E. (Bombay).

CORRECT diagnosis is the basis upon which modern medicine rests. During the past few years, clinical diagnosis has steadily improved in accuracy. Routine experiences with older methods and older technics, both clinical and radiological, steady improvements in them increasing practically every day, the correlation of medicine, surgery, pathology, and radiology, have revolutionised our conception of joint diseases.

There is no doubt that X-rays have widely extended our knowledge of the growth, development, and structure of bones and joints, normal as well as pathological. They have given us details of osseous and articular changes, and outlined them graphically. These graphic changes have frequently helped us in an accurate diagnosis, particularly when clinical signs and symptoms are indefinite, when the history is vague and unreliable, and when laboratory findings are not helpful. The use of X-rays in diagnosis has become practically routine, whenever certain diagnostic problems appear, whether it is the joints or any other part of the body.

I here wish to stress one point. It is most important that the radiologist should be furnished with clinical and laboratory data, as their assistance may give him a lead and may enable him to search specially for certain X-ray findings, which may not occur to him at the time owing to the pressure of routine work and which might thus otherwise be overlooked. Many a time cases have been referred to him for radiological examination without any clinical history or data.

Much has been said about Syphilis of the bony skeleton and about other diseases of bones and joints, but references to Neuropathic joints have been few. The literature on these joints is certainly very meagre. It is peculiar that this condition is not frequently mentioned in the radiological journals.

Necessity of an analytical scheme

A correct diagnosis of a Neuropathic joint depends on our knowledge of the significance of certain basic changes which we find in the X-ray film; otherwise correct interpretation of the film is impossible. It must be remembered that joints are complex structures, and other diseases may

* Paper on Neuropathic Joints read at the 3rd Indian Congress of Radiology at Calcutta in December, 1948.

show similar appearances, but a Neuropathic joint presents specific patterns or certain specific changes, and these must be noted by careful attention to details.

In consideration of the Neuropathic joint disease, and to interpret it accurately, it is best to proceed along a scheme. Otherwise merely interpreting it because it appears similar to one which we already saw in a previous case or one resembling a case in the literature is not a correct scientific procedure and may lead to mistakes.

The analysis of the X-ray film must thus be done taking into consideration not only all the components of the joint namely the bones synovia, soft tissues etc., but also the clinical aspects of the particular case and any clinical and laboratory data.

In such a consideration, one should have a proper knowledge of pathological changes, X-ray changes and an understanding of detailed anatomical relationship of the joints. The first step is to recognise the normal joint from the Neuropathic joint and then to review the various pathological processes which are likely to cause such abnormal changes in order to decide whether a Neuropathic joint disease is most likely to be probable or likely to be most responsible for the abnormal shadows under consideration. When one has

obtained such an information from an X-ray film pointing to a Neuropathic joint disease, then it must be correlated with the history of the case, the physical signs and symptoms, and the laboratory findings.

Importance of a perfect X-ray film

In order to diagnose a Neuropathic joint disease, it is absolutely necessary that the X-ray technic is perfect. Such a technic should show details not only of the bones and joints, but also of the surrounding soft tissues. Changes in surrounding soft tissues such as deposits of flocculent calcium not only within but outside the capsule of the joint might be graphic pathognomonic signs of Charcot's joint. A few points in technic may not be out of place. A long film distance from the anode is always better as it avoids excessive shadow enlargement, which by cutting off the magnification gives sharper details. The one distance most commonly used now a days is 100 cms. or 40 inches, for all joint work. The K. V. P. is here an important factor. It should vary from 40 to 70 K. V. P. depending on the joint to be X-rayed, increasing it as the thickness of the joint increases. One always uses a low K. V. but consistent with details, particularly soft tissue details and contrast. One has to avoid excessive density in these X-ray films, as it might obscure soft tissue details,

which are very important in Charcot's and other Neuropathic joints. For knee, shoulder and hip joints Potter Bucky is essential.

Importance of anatomical details

To interpret a Neuropathic joint disease, it is essential that one should be quite familiar with the anatomy of the joints. The study of the joint should include not only the bones in the region of the joint, the articular cartilages and the joint space, but should include also the bones outside the joint, the condition of the synovia, of the joint capsule, and of the soft tissue details. One sees the condition of the bones directly, but the articular cartilages, the synovia, the joint capsule, and the joint space are all transparent, and an evaluation of these points must be made by indirect evidence.

Classification of Neuropathic joints

Charcot's type of Neuroarthropathy is often referred to as arthropathy of Tabes Dorsalis, or as arthropathy of Syringomyelia or of Leprosy or even as arthropathy of Dorsal column damage. The Neuropathic joint belongs to a group of conditions like hypertrophic and atrophic arthritis, ankylosing spondylitis, gout etc. under the heading of chronic arthritis. Neuropathic arthropathy is classified by Schands¹ as follows :—

Schands'¹ Classification of Neuropathic joints

- I. Tabes Dorsalis.
- II. Syringomyelia.
- III. Following lesions of peripheral nerves. (1) Injury; (2) peripheral neuritis; and (3) leprosy.
- IV. Following lesions of the cerebrum: (1) Dementia paralytica; and (2) hemiplegia following cerebral hæmorrhage.
- V. Following lesions of the spinal cord: (1) Injury; (2) congenital malformations; (3) tumours; (4) T. B. spine; (5) acute myelitis; (6) anterior poliomyelitis; and (7) progressive muscular atrophy: (a) Aran Duchenne type; and (b) spastic type—amyotrophic lateral sclerosis.

Classification with reference to History

In 1868 Jean Martin Charcot drew attention to the joint condition accompanying lesions of the central nervous system, especially Tabes Dorsalis. He then described the clinical entity, the classical Charcot's joint of Tabes Dorsalis. The condition was at first thought to occur in Tabes and Syringomyelia only. It is true that they mostly occur in the above two conditions, and that Tabes and Syringomyelia should be first considered as

possible etiological factors. But a look at the above classification shows that they might be seen in many other conditions for example in lesions of peripheral nerves, spinal cord and cerebrum, in other words, in lesions of upper and lower motor neurone. This is an important point. Standard X-ray texts however casually mention the whole subject in their paragraphs on Charcot's joints; some even omit the subject entirely.

Pathology.—Nowhere is a knowledge of pathology more important for interpretation of X-ray films than in Neuropathic joints. The pathology and morbid anatomy findings of these joints may be divided into 2 groups: (1) lesions of upper motor neurone; and (2) lesions of the lower motor neurone. The lower motor neurone lesions may be divided into 2 groups: (1) Simple; and (2) Charcot's type. The Charcot type may be further divided into Leprosy, Tabes and Syringomyelia.

1. *Lesions of upper motor neurone*:—The cases in this group show spastic paralysis and are associated with changes in the bony skeleton. The changes are:

(a) The involved bones appear smaller with a diminution in the diameter of the bone, the cortex of the bone appears thinned and there may be osteoporosis. These are the changes associated with disuse

atrophy. Should the spastic paralysis occur in childhood, even the shape of the bones may be changed.

(b) In some cases there may be found dislocation or subluxation of the joint, and spine may show a lateral curvature. Thus subluxation may occur in the hip joint when there is marked spasticity and rigidity of the lower extremities.

2. *Lesions of the lower motor neurone and nerves*: (a) Simple; and (b) Charcot's type.

(a) *Simple*:—The cases in this group show flaccid paralysis, and follow acute poliomyelitis most commonly, or after motor nerve paralysis. Bony changes here are seen rather late in the condition. They are osteoporosis of the bones of the part, and thinning of the cortex; the bones appear smaller, and the affected limb appears also much smaller. There may be subluxations and dislocations, and even fracture may occur with a very minor degree of injury. The changes are more or less the same as those seen in the upper motor neurone lesions. But besides these there may be bone changes associated with genu valgum, or pes cavus, or coxa valga, and the spine may show a degree of scoliosis. The spine, if the scoliosis is of long standing, may show marginal lipping and secondary osteoarthritis.

(b) *Charcot's type*:—(i) Leprosy; (ii) Tabes; and (iii) Syringomyelia.

(i) *Leprosy*:—Bone changes are seen in the neural type of Leprosy; but are rare in skin type. Cases showing neurological changes with skin involvement show diffuse osteoporosis of the bones of hands and feet, and cysts resembling Jungling's disease. Pathological fractures, severe deformities, mutilations of hands and feet may be seen.

In Neural leprosy, the predominating bone changes are slow absorption, most marked in the phalanges of the hands and feet. The tips are first involved and the proximal phalanges are the last to disappear. Fractures may occur with minor degree of trauma, and usually no callus is visible. Often we see deformities, gross, extensive and mutilating.

(ii) *Tabes*:—Here the changes are seen in the synovia, ligaments, and the bones. Synovial membrane shows increase in size, hypertrophy of the villi so that the whole membrane shows a shaggy or villous appearance. The villi are not infrequently pedunculated, and are sometimes detached completely forming loose bodies in the joint (joint-mice). The synovial fluid is increased greatly in quantity and forms a hydrarthrosis.

The capsule is enormously distended, intra-articular ligaments and

tendons are often destroyed, and extra-articular ligaments are often stretched and wasted. In the later stages the synovia may be destroyed.

The effusion is rather viscid and slightly yellow, sometimes bloody, but never purulent unless it is secondarily infected from a perforating ulcer or a septic focus. The hydrarthrosis is well marked in the shoulder and hip joints.

The cartilages are worn away and from their margins spring osteophytes or ecchondroses. The bones show extreme wearing away, the head of the bone may be so worn out that the joint may show the appearance of an excised joint. Calcareous and osseous plaques may be formed in and on the capsule. Fractures and subluxation may occur. Reunion is not visible after fractures.

(iii) *Syringomyelia*:—The underlying morbid change is a gliomatous degeneration of the cord compressing and destroying the spinal grey matter in the neighbourhood of the central canal. It may occur throughout the spine, but is nearly always situated in the cervicodorsal area. The articular lesions are similar to those in Tabes, but are especially common in the shoulder and elbow. Atrophic, hypertrophic changes, and hydrarthrosis are seen. Subluxations and fractures are frequent complications. The bone

changes are seen in the bones of the fingers and other joints of the upper extremity. Owing to the frequent association of ulcers and whitlow, infections of the joints are relatively more common, but the suppuration is of a low grade virulence.

The pathological changes have been dealt with here in detail as a knowledge of these changes is absolutely essential to the proper understanding of the radiological appearances.

Etiology.—The radiologist may well ask how the lesions of central nervous system give rise to such distinct pathological and radiological entities. There are various theories, French, German etc. (Percy Delano.)² The French Theory classifies all Neuropathic joints as trophic disturbances. Charcot was a firm believer in this theory. The theory is that it is a joint destruction due to abnormal stimuli transmitted through so called Trophic nerves. This theory has been denied by some.

The German theory was put forth by Volkmann, who assumed that the spectacular joint changes merely originated in multiple subclinical injuries which were not seen to because of the insensitivity of the affected joints.

A third theory is that together with the disturbance of the central nervous system there is probably a

superadded low grade syphilitic infection of the joints. This theory can be discarded because no spirochaetes have been found at any time in Charcot's joints, and secondly these lesions occur also in Syringomyelia, and they can not be distinguished from those of Tabes radiologically.

A fourth theory has been put forward recently by Hodgson, Pugh and Young³. They believe that the bone changes are due to a type of osteomyelitis secondary to chronic infection of the contiguous soft tissues rather than neurotrophic disturbance proper. They found that an acute or chronic inflammatory process of the soft tissues in the region of the involved bones was present in all the cases of Neuropathic joints, suggesting that this may be the direct cause of the bone changes independent of any nervous disorder. They found that where feet presented neurotrophic changes, in every case there was infection of contiguous soft tissues while the disease of the nervous system was only present in a small number of cases.

Considering the pathological changes and the morbid anatomy findings, Volkmann's theory fits in more with them. He assumes that the surprising graphic changes originate in multiple subclinical trauma which were not seen to because of the insensitivity of the affected joint.

The joint thus bears an undue strain from every day life, and becomes more and more injured. The result is that the ligaments are loosened, the capsule elongates and becomes stretched, and so the bones come in undue contact and thus they rub together more forcefully than usually or normally. This results in wearing of the opposite articular cartilages, and finally even of the cortex of bones beneath the cartilages. This degeneration and partial disappearance of joint cartilages expose the bone beneath. There may be a proliferation of cartilage too and this may become converted into subchondral bone. As a repair measure there may be some bone reaction and subcortical sclerosis. The stretching of the ligaments and of the capsule may allow abnormal lateral movements of the joint. This may cause hæmorrhage, with production of flocculent calcium, subperiosteal new bone formation and even osteophytes. Continued further, the bone destruction would be extensive and would lead to almost total disintegration of the articular ends of the bones. The humeral and femoral heads may become totally lost by disintegration. In the hip a wandering acetabulum may be found. It may even result in subluxation. It may even perforate the acetabulum with the so-called central dislocation of the head of the femur.

Clinical features.—They are also important in Neuropathic joints.

Simple neuropathies like acute poliomyelitis:—Here there is history of sudden onset of fever, with stiffness of the neck, and thereafter flaccid paralysis of one or several muscle groups. Later there is muscle atrophy, flaccidity, and abnormal mobility of one or more joints, possibly associated with a deformity.

Charcot's type: Tabetic arthropathy:—Here the onset of symptoms is nearly always sudden, dramatic and painless. Sometimes the patient wakes from sleep and finds that a joint has become greatly swollen and useless. No tenderness or pain may be felt in passive movement, and movements may be permitted through an abnormal range and that also painlessly. In the knee one may obtain lateral flexion or even hyperextension. The fluid in the joint may be absorbed and the joint return to its normal condition, but more commonly the joint will show the typical changes described in the previous pages. The most important point to bear in mind is the entire absence of pain and abnormal mobility of the joint.

Syringomyelic arthropathy:—The clinical features are similar to those in Tabes. There is a slow progressive atrophy of muscles, disturbances of sensation and of vasomotor function. As the favourite

site is the cervicodorsal region, the progressive muscular atrophy is most often in the muscles of the upper extremities and the shoulder girdle. A claw hand frequently develops. Usually there is a characteristic loss of temperature sense and pain sense, while tactile sensation is present. In Morvan's type of syringomyelia, even the tactile sense is lost. Often the diagnosis is only arrived at when the patient is referred for X-ray examination for swelling of the shoulder or elbow.

Leprotic arthropathy:—Ulcers on the tips of the fingers and toes, perforating ulcers in feet, dry gangrene or complete absorption of the toes and fingers are common features. Hands and feet appear mutilated, sometimes beyond recognition.

Laboratory findings.—*In simple types as in poliomyelitis*:—Blood shows slight anaemia and moderate leucocytosis. Spinal fluid is clear to opalescent, and there are increased protein and lymphocytes during active stages.

In Charcot's type:—Findings are variable. In Tabes, Wassermann may be negative. But the radiologist should not deviate from his diagnosis because the Wassermann is negative, as this is to be expected in about 50% of cases of Tabes.

X-ray findings.—*Simple arthropathies*:—In upper motor neurone

lesions, the bones in the X-ray films show two main changes. The first is thinning of the cortex, osteoporosis and a diminution in the diameters of the bones of the joint. The second change may be dislocation in a joint or scoliosis in the spine.

In lower motor neurone lesions—after poliomyelitis, similar bony changes are seen, late in the course of the disease. At first the bones of the joint show a minor but widely spread decalcification of bones. The joint space however remains normal, and the articular cartilages are normal, or may just show slight thinning. In later stages, there is marked atrophy of the muscles, and bones show marked decalcification and osteoporosis. In the growing bone, there is diminished development. The articular cartilages show more thinning. There may be dislocation of the joint, and even spontaneous fractures with a mild or minor degree of trauma may occur.

Spinal simple arthropathies:—In early cases, the films would show irregularities of the facets of apophyseal joints, slight eburnations of articular surfaces, and the intervertebral discs may show irregular or asymmetrical spacings. There may be scoliosis and kyphosis.

In the later stages, the apophyseal joints show osteoporosis and narrowing of the articular space

between the facets, scoliosis and kyphosis. Later still the spine shows secondary spondylitis.

X-ray findings in Leprosy.—According to the specifications of the International Congress of Leprosy at Cairo (1938) there are 2 types of leprosy: (1) The nerve or neural type, including all cases of benign leprosy with polyneuritic manifestations; and (2) the lepromatous type, formerly called the cutaneous type, in which are grouped all cases of malignant leprosy with lesions especially on the skin and other organs.

Of the two types of leprosy, neural and lepromatous, the lepromatous type is usually free from bone lesions, and even if they do occur, they are limited in extent. The bone changes are however more common in neural leprosy. They consist of slow bone absorption, first starting in the tips of the distal phalanges, subsequently in the middle, then in the proximal phalanges, which are the last to disappear. If serial films are taken, the gradual disappearance may be visualised. In the hands, the process seldom goes higher. In the feet, the heads of the metatarsals and the phalanges show the bone degeneration and absorption.

These bone changes are best studied by division into four groups.

1ST GROUP:—The earliest changes are the decreased density and greatly

increased decalcification or radiolucency of the bones of the hands and feet especially in the epiphyses of the phalanges, metacarpals and metatarsals. The medullary canals appear wider than in normal cases. This decalcification results in the formation of clear well defined zones, seen in both ends of the phalanges and the distal ends of metacarpals and metatarsals. Gomez and Acosta⁷ call them vacuoles. The nutrient foramina are enlarged so that they become visible on the radiographs (Murdoch and Hutter⁴). Besides rarefaction and decalcification and sometimes vacuoles, Gomez and Acosta describe zones of hypercalcification occasionally accompanied by a widening of the proximal ends of one or more phalanges, covering the adjoining phalanx like a cap. They also describe hyperostosis without hypertrophy of the bone. So that there is increased density of phalanges, metacarpals, and metatarsals especially at the diaphysis, at the same time with rarefaction of some phalanges and normal calcification of others. I have not noticed these hypercalcification or hyperostosis described by Gomez and Acosta.

2ND GROUP:—The bones of the hands and feet show at first minute defects at the tips of the distal phalanges; this is an osteitis showing absorption of bone. Then the tufts

show further erosions or excavations. At times a part of the tuft or tufts are shaved off almost diagonally, at other times the phalanx appears to be cut across transversely or obliquely, or the tip of the tuft may show a comb like appearance. The process does not stop here but invades the proximal end of the phalanx. Then the entire tuft may disappear—called the collar button appearance. The shafts may show very prominent or enlarged nutrient foramen. When the end of proximal phalanx is approached, the density there first increases and the end of the phalanx expands according to Gomez and Acosta, which they call "hooded".

3RD GROUP:—The phalanges, metacarpals, and the metatarsals may show destructive changes or cystic changes resembling Jungling's disease. These cystic areas may show fractures even with a minor trauma. One characteristic of the destructive changes at the tufts is that they never heal. Fractures usually do not unite in lepers, but Gomez and Acosta differ from this, as they have observed union of fractures with callus opaque to X-rays in 3 of their cases.

4TH GROUP:—Arthritic—both atrophic and hypertrophic types. Owing to the absence of pain, injury to bones and joints is not minded or taken notice of, and this results in

destruction of the articular ends of bones. Even fractures may not be noticed by patients. Ankylosis is not seen in these joints. Gomez and Acosta however state that in the leprotic metatarsophalangeal joint osteoarthritis which occurs in perforating ulcer of the foot, healing takes place in most cases through ankylosis.

The important point to bear in mind in interpretation of films is that whenever tips of the phalanges show partial absorption or shaving or slicing like appearance, or the collar button appearance or enlarged nutrient foramen, leprosy should be suspected.

Murdoch and Hutter⁴ in an excellent article show that the bone changes occur in cycles, showing periods of dormancy and reactivation similar to the recession and exacerbations visible in cutaneous leprosy. They say that after the initial reaction, the bone may regenerate or degenerate. One bone in an extremity may improve while the other at the same time is retrogressing. This more or less selective sprinkling of pathology in varying stages of degeneration or regeneration, they hold, is from the X-ray point of view characteristic of leprosy.

Differential diagnosis.—Cyst formations can not be easily differentiated from the cyst of sarcoids, (Jungling's disease), or other specific

cysts. The diagnosis may be actually established by accompanying clinical and skin manifestations. Bacilli may be recovered from the bone cysts.

The X-ray differential diagnosis from other neurotrophic disturbance is usually impossible. Tabes, Syringomyelia, certain forms of syphilis, chronic peripheral or spinal nerve injuries give identical X-ray pictures. The shaving or slicing like, the collar button appearance, the enlarged nutrient foramina may be helpful.

X-ray findings of Tabes and Syringomyelia.—They are considered together as both give X-ray appearances which are similar, and there are no radiographic features by which a Charcot's joint of Syringomyelia may be distinguished from that of Tabes.

Charcot's joints are usually seen in persons past 40 years in age. 2 of our cases were over 55 years. Males are usually more often involved than females. At least two-thirds are usually males. In our series all cases were males. Thus the relative proportion may be higher, than two-thirds, but the number in our series is too small. Of all patients suffering from syphilis, approximately 2 to 4% develop Charcot's joints. Amongst the Tabetics about 9 to 10% develop these changes. In Syringomyelia, the proportion is higher than 10%.

The commonest joints involved are the knee, ankle and the foot, then the hip, the spine and then the shoulder. But any joint may be affected. In our series, there is one knee, one hip, two shoulders and two feet. The joints of the lower limbs are more often affected in Tabes, and the joints of the upper limbs in Syringomyelia. Secondary pyogenic infection may occur in the foot from a penetrating or perforating ulcer. Bilateral affections may occur but unilateral affections are more common. In our series only one case showed a bilateral condition and that was in the feet. Even more than one joint may be involved in the same person, but usually the changes do not all occur together. I have not seen one case with multiple joints.

The development of a Charcot's joint is usually slow in progress. The cases seen both in hospital and private practice are advanced cases with the Charcot's joints fully developed; early cases have seldom been seen.

The main features of the Charcot's joints are excess of fluid in the joint or hydroarthrosis, irregularity and wearing out of the articular surfaces or disintegration, sclerosis of the articular surfaces, subchondral bone formations, flocculent deposit of calcium in and outside the capsule, fracture, subluxations, loose bodies

within the joint, atrophy or hypertrophy of bones.

Each of these should be studied in detail.

1. *Excess of fluid or hydroarthrosis*:—This is usually an early sign, and shows itself in the X-ray film by increased width of the articular space. Such an appearance in a joint, particularly when it is painless and after only a minor degree of trauma should make us suspect an early Charcot's joint. There may be an increase of the soft tissue shadow surrounding the joint. It may be associated with other changes of Charcot's joint.

The 2nd early sign is that the articular surfaces appear somewhat sharper, better defined and show increased density; not an actual bone sclerosis yet.

Then the 3rd sign may appear, namely that the articular surfaces may just show a regular wearing away.

All this time the joint, because of the absence of pain and abnormal range of mobility, impairment of stability and function of the affected limb, and not mindful of minor degrees of trauma, is undergoing further pathological activity, and other signs make their appearance. Yet because of the absence of pain, he may fail to see the family physician hoping the attack to be over soon.

Soon the articular surfaces show further and further wearing out, the joint space becomes wider and wider. The articular surfaces may be considerably worn out. This wearing out of the articular surfaces with increase in the joint space is very characteristic of Charcot's joint. No other joint condition shows the same wearing out.

Another characteristic, and frequently pathognomonic sign is the appearance of irregular calcium deposits, some specks, some bigger, both within the capsule and outside the capsule. Time after time, our attention is first drawn to this sign, before one evaluates other diagnostic signs. Of course one meets with such calcium deposits in other conditions as well.

There are 2 types of cases seen, atrophic type and the other hypertrophic type; in some patients the early changes are of an atrophic type, which may later on become the hypertrophic type. In other cases the hypertrophic type may precede the atrophic type.

In hypertrophic types, owing to disorganisation of the joint, the joint appears much bigger and massive, and the joint space may be twice the normal size, certainly much bigger than normal. There is irregularity of the articular surfaces, often gross, and there is considerable new bone formation. The

calcium deposits outside the capsule and inside may show organisation. New bone formation may be in the form of osteophytes or loose fragments within or outside the joint. The osteophytes may be loose detached bone fragments, probably the result of trauma. Some such fragments become attached to the bones and grow in an amorphous purposeless fashion.

If one looks at the contour of the joint, it appears quite irregular, and larger than normal. With the presence of hydroarthrosis, wearing out and considerable destruction of the articular surfaces, subluxation is frequently found, or even dislocation. In some cases the first indication of a Charcot's joint is a painless dislocation with a large effusion. In our case of Charcot's joint in the hip, the attention was drawn to a dislocation, a pseudarthrosis, which was not clinically mentioned. The painless dislocation particularly in the hip has been reported. In the knee, the tibia, fibula and the patella are frequently displaced laterally and even completely dislocated from the femur. Another region where the painless dislocation may be the first sign is the shoulder joint, and this may indicate the probable diagnosis.

In this type the glenoid cavity in the shoulder joint may appear dense and massive and the lower margin

may show a big cup-shaped socket. In the hip joint, the acetabular socket forms an upward extension forming a large excavation or a roof and also a pseudarthrosis. The subluxation, the wearing out of the sockets, and their extension, the excess of new bone formation, osteophytes, possibly calcium deposits, the increased density of bone can seldom be mistaken for any other condition. The sclerosis of bone beneath the articular cortex is most characteristic and this point is particularly helpful in the Charcot's spine where other characteristics may not be helpful.

In the atrophic type the bones show diminution of the extent of the articular surfaces, there is absorption and atrophy of the extremity of the bones, there is irregularity of the articular surfaces, and the size of the joint is also diminished. Deposits of flocculent calcium may or may not be visible. The important point to bear about this type is that this type resembles a surgical excision of the part of the bones.

In Charcot's joints, besides dislocations, fractures or fracture dislocation may occur. Fractures are seen in the intertrochanteric region of the femur and in the lower ends of tibia and fibula, in the femoral or humeral shafts, and in the knee where one finds marginal fractures. The one important characteristic

of these fractures is that they are apt to remain ununited. In some cases, the fractures, may precede the development of a Charcot's joint. Brailsford says that an attempt to secure union of such a joint in an extracapsular fracture of the neck of the femur by the insertion of Smith Peterson pin was unsuccessful. He says that the bone round this pin was absorbed and the pin came away and the fractured extremities of the bone presented the clear worn surfaces of the Charcot's joint.

Brailsford in his masterly description of the joints says that the appearances in Charcot's joint appear in 4 stages. According to him the first stage of hydroarthrosis appears and is seen for about 2 to 3 years. This is followed by the second stage of disintegration where the articular surfaces are considerably worn out, the joint space is increased; there are deposits of flocculent calcium within and outside the capsule. This stage lasts for 3 to 6 months, after which time the joint enters into the 3rd stage which he calls the hypertrophy stage. In this stage there are extensions of the joint socket which is twice or thrice the normal size, and the calcium deposits are undergoing organisation. He determines the duration of this stage as about 3 to 5 years. This stage is followed by the final phase which he calls the atrophy phase. Brailsford further states that these 4 stages

following one after the other appear to take place only once in the history of the Charcot's joint and this sequence is not repeated.

In Charcot's spine, the apophyseal joints may show marked eburnations, bridging, spur formation, with scoliosis, kyphosis or lordosis. The chief characteristic of Charcot's spine is destruction of the opposing bone surfaces and at the same time there is a massive new bone formation. The commonest site is the lumbar area particularly the lower lumbar spine, and sometimes also the sacral vertebrae. In such cases there may be similar changes in hip or knee or ankle or possible evidence of syphilitic heart disease which may be indirectly helpful. In other cases there may be destruction and collapse of the involved vertebrae without any new bone formation.

Differential diagnosis.—A Charcot's joint can hardly be mistaken for other lesions; there are some lesions which resemble it.

1. *Excised joint*:—The atrophic type of Charcot's joint may be mistaken for an excised joint, but the deposits of calcium within or outside the capsule, and the history of the case are helpful factors.

2. *Malignancy of bone*:—When the lesion develops in the acromioclavicular or sternoclavicular joints, a swelling is observed, and then when the patient is X-rayed, marked

destruction and osteoporosis of bones may be mistaken for malignancy of the bones. In the hip joint, a pathological fracture in the trochanteric or the subtrochanteric region may be mistaken for a sarcoma.

3. *Arthritis deformans*:—An early Charcot's joint may be mistaken for arthritis deformans, but the marked bone destruction, hydroarthrosis, calcium deposits are important differential signs.

4. *Myositis ossificans*:—After organisation of calcium deposits and subsequent new bone formation on the outer aspect of the innominate bones, or on the shaft of the femur particularly after injury may be mistaken for localised myositis ossificans.

5. *Subluxations and pseudarthrosis and fractures*:—These may be mistaken for common traumatic lesions, and suspicion of such lesions may not be had for some time, a fortnight or over, because of the painlessness.

6. *Spine conditions*:—The erosions, absorption of the phalanges and metatarsals and other signs of Charcot's joints show sometimes identical appearances as those shown by spinabifida occulta and other congenital and pathological conditions of the lower spine.

7. *Pellegrini-Stieda lesions*:—The linear calcium shadows on the side of the knee in Charcot's joint

after injury may be at first mistaken for Pellegrini-Stieda lesions.

8. *Spondylitis deformans and other spine conditions*:—The extensive destruction and new bone formation in Charcot's spine are characteristic.

9. *Tabes and Syringomyelia*:—There are unfortunately no radiographic features by which one can distinguish a Charcot's joint of Tabes from that of Syringomyelia. This is however at times a very important point. A diagnosis of mere Charcot's joint on radiological grounds only may mean either Tabetic or Syringomyelic. A diagnosis like that may condemn a patient to a syphilitic clinic. The patient may show no positive Wassermann. We know however that in 50% of cases of Tabes, Wassermann is negative. In spite of no positive Wassermann, and in spite of absence of positive clinical or neurological signs or symptoms of syphilis, or of Tabes dorsalis, he may receive a number of antisyphilitic intramuscular and intravenous injections. An examination of the patient and information concerning the history and clinical findings if possible may often help the Radiologist to be specific in his diagnosis. Here it must be remembered that even here Tabes and Syringomyelia are not the only conditions responsible for Charcot's arthropathy, as may be seen from

Schand's classification (Schand).¹ In these cases the distribution is important. The distribution of Syringomyelia is in the upper extremities, where as that of Tabes dorsalis is more common in the lower extremities.

A case quoted by Mandeville⁶ is rather interesting, where the shoulder showed signs of Charcot's joint. With the common Tabetic conception of a Charcot's joint, the patient was referred to a Syphilis clinic where in spite of a negative Wassermann and lack of positive clinical and neurological findings of Syphilis or Tabes, the patient received a number of antisyphilitic intravenous and intramuscular injections. Then the patient showed pus pouring freely from the thumb, then the loss of

tips of thumb, index and ring fingers were noticed. X-ray showed distal half of terminal phalanges missing. With these clinical findings, and X-ray findings of the bones of the hands, the specific diagnosis of Syringomyelia as the cause of Charcot's joint was then given.

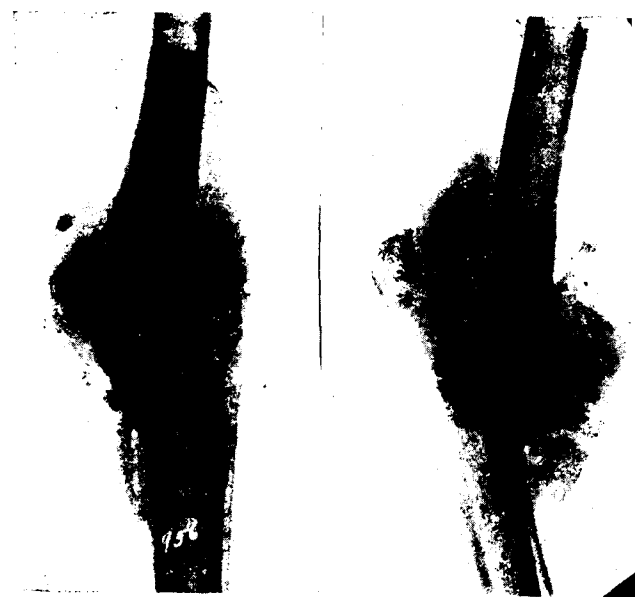
Case Accounts

Case 1.—J. N., age 55, male, complains of swelling of the left knee-joint since 3 months. Clinically there is doughy swelling of the knee-joint. No sign of fluid. Patella could not be felt. The joint shows abnormal mobility, and is painless. He shows inability to walk, occasional low fever and general weakness. He had a fall a few months back, had a sprain when the joint was greatly swollen. He went to a bone setter, and although the condition seemed apparently improved, the joint became more and more swollen. Patient denies having had syphilis or venereal disease.

Lumbar puncture shows clear fluid under tension.

Skiagrams of both knees and one ankle were taken.

Left knee shows characteristic localised



CASE 1. Left knee—A case of Charcot's joint.

paring of the articular ends of the tibia and femur. The articular ends show some increased density. The joint space is completely diminished, the bony surfaces are in closer contact. Both the condyles of the femur and the medial condyle of the tibia are completely worn down. The tibia, fibula and the patella show subluxation; patella shows lateral displacement. The soft tissues around the joint are swollen, and the pathognomonic flaky deposits of calcium are seen in an expanded capsule. These flakes of deposits are especially seen along the periosteal borders of the femoral condyles—stage of disintegration. The right knee shows no changes at all except guinea worms in the popliteal space. The ankle shows nothing abnormal.

This is a case of Charcot's knee joint.

Case 2.—G. U., age 30. Definite history of Syringomyelia. He complains of inability to raise the arm for 2 months. The film shows the characteristic erosion and absorption of the head and also the upper part of the shaft. There is marked separation of the upper part of humerus from glenoid cavity. There is a collection of flocculent calcium deposit in the expanded joint capsule. The glenoid cavity is massive and shows new bone formation and lipping. The acromioclavicular joint is also involved, showing marked destruc-

tion of the outer end of the clavicle. The joint shows the stage of disinte-



CASE 2. Charcot's joint—Stage of disintegration ending and stage of hypertrophy commencing.

gration ending and hypertrophy stage commencing.

Case 3.—M. D., disease: paraplegia. Pain in the umbilical region, and inability to walk for 4 months. He felt weak and the inferior extremity got paralysed, recovered slowly and he was going about with a stick. He gives history of syphilis 4 years back. Clinically there is incontinence of urine and stools. The lower extremities show rigidity, there is wasting of left hip, and the inferior extremity. Ankle clonus is present. Urine: nil abnormal. Blood—R.B.Cs. 2.8 millions, Hb. is 45%, and W.B.Cs. 5,800. Wassermann is negative. Cerebrospinal fluid is clear. Tension was



CASE 3. Neuropathic hip joint.

110 mms. and on pressure of jugular veins rose to 290 mms. He was referred for X-ray examination of the dorsilumbar spine for hemiplegia. The spine showed osteoarthritis. An abnormality was however noticed in the right hip joint region in X-ray of the pelvis. It showed marked subluxation of the head of femur which had completely escaped notice till the X-ray was taken. The head of femur is displaced upwards but shows no fracture, and is considerably worndown. The acetabulum shows some erosion, the greater trochanter shows considerable destruction. It shows pathognomonic

collection of flocculent calcium. There are other opaque shadows on both hip joints, probably Bismuth injection opacities.

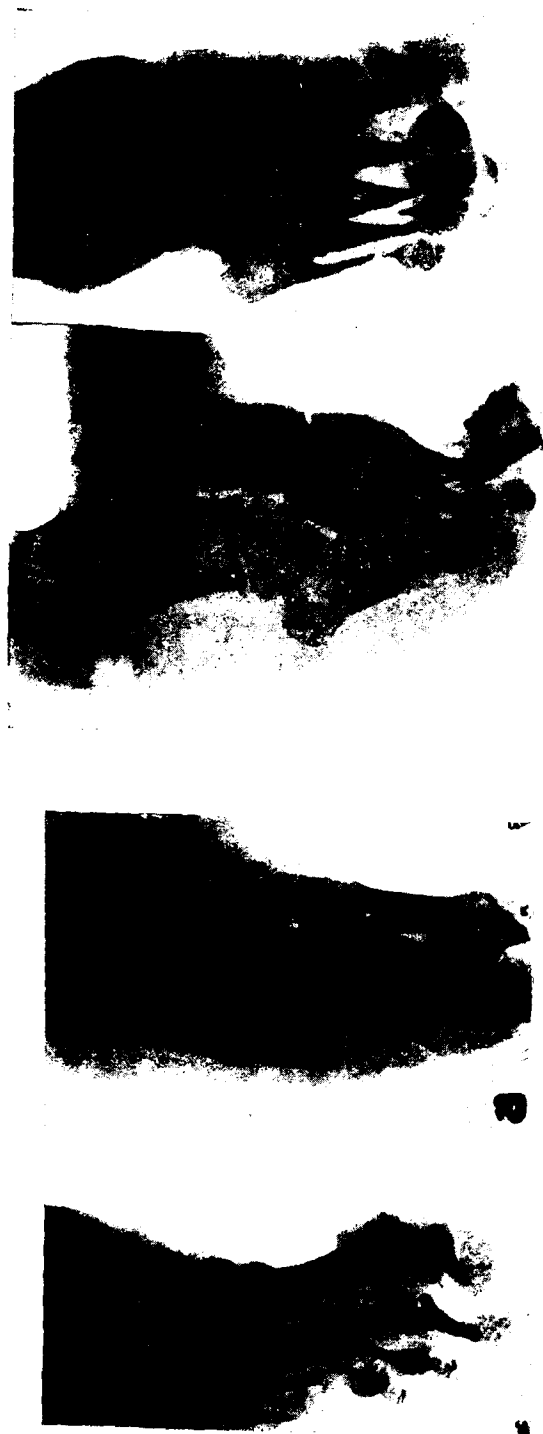
Case 4.—R. S., age 35, complains of pain in the right foot for the last 6 months. Patient denies having syphilis. Superficial sensation is not present. Patient feels sensations, such as pin prick. There is thickening of both ulnar nerves. The right foot shows deformity of the ankle with swelling and a sinus on the dorsum. There is partial loss of toes of both feet since one year. The films show marked increase in the ankle joint space.

destruction partially of the talus and of the calcaneus. There is dislocation at the mid tarsal joint. There is destruction of the cuneiforms and the cuboid and of the outer two metatarsals. No investigation was done as the patient left.

Case 5.—M. S. T. H., age 23. Patient shows trophic ulcers on the soles of both feet; duration is one year. Patient says that ulcer heals completely but breaks up again. This has occurred 6 to 7 times in one year. He complains of pain in plantar aspects of both feet. Deformity present in both the feet. He denies having syphilis. The X-ray of the right foot shows the collar button appearance of the head of the proximal phalanx of the 2nd toe; the metatarsals are pointed, with complete disappearance of the 5th metatarsal and



Case 4. Skiagrams of foot and ankle showing increased joint space of ankle, dislocation of midtarsal joint and destruction of adjacent bones.



CASE 5. Right foot and left foot showing gross changes in metatarsals and phalanges.

the little toe. Left foot shows still more characteristic appearances. All the 5 metatarsals show spontaneous multiple fractures, and pointing and thinning of the shafts. The tarsals are normal. The distal parts of the metatarsals show short sharply tapering extremities. No definite cause could be assigned; it may be a case of neuropathic joint lesions or neurotrophic lesions, as such destructive erosions occur not only in Tabes, Syringomyelia and Leprosy, but also in Psoriasis, Raynaud's disease, in Yaws, or in other pathological conditions of the spinal cord such as Spina bifida occulta, Cholesteatoma, in Trench feet etc. Provisional diagnosis: Leprosy.

CASE 6.—B. B., age 55. History of paraplegia with retention of urine. No stone in the bladder, no evidence of stricture in urethra; there is loss of power in both extremities for 2 years. Tone is increased in lower

extremities, superficial and deep sensory responses are normal. Biceps and Triceps jerks normal. Knee and Ankle jerks plus plus. No clonus. Swelling of knees; clinically suspected to have neuropathic knees. The X-rays merely show considerable decalcification of bones.

CASE 7.—N. S., child, age about 10. History of anterior poliomyelitis. The



CASE 6. Clinically suspected to be Neuropathic knee—Skiagrams show more osteoporosis.



CASE 7. Bone changes in a case of Anterior Poliomyelitis (A. P. and lat. views of knee).

radiographs of the knee show considerable osteoporosis, thinning of the bones and diminished diameter of the bones.

Case 8.—Fever, pain in chest and in limbs. Inability to move the right shoulder, for 1½ years; was given 10 injections. Right shoulder joint is completely disorganised, active movements are all limited, passive movements are present. Signs of Syringomyelia present. Deep jerks are present. Motor power and tone are normal. Sputum is negative. Urine shows some pus cells. The joint shows atypical Charcot's joint appearances.



CASE 8. Charcot's joint in a case of Syringomyelia.

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6. F. B. Mandeville—Am. Jour., Roentgenology, Aug. 1938, Vol. 40, page 230, X-ray Findings in Morvan's Type of Syringomyelia.
7. Gomez and Acosta—Bone and Joint Lesions in Leprosy, Radiology, May 1948, Vol. 50, No. 5, 6-9.

Fistula Between Ureter and Colon—Healed For Nearly Seven Years

RUDOLF TREU, M.D. (Cologne), L.R.C.P. & S. (Ed.), L.R.F.P.S. (Glasgow),
CALCUTTA.

THE following history is in many respects so extraordinary that its publication appears justified. It concerns a fistula between the urinary tract and the colon which existed for a long period and remained closed after conservative treatment for more than six years. Beyond that it is of theoretical importance as it shows that a fairly comfortable state of health was being maintained though the function of one kidney was completely abolished and the function of the other kidney being at a minimum. Throughout the time of observation there was never any rise of blood-pressure or any symptom of uræmia.

The patient, a retired Anglo-Indian male, aged 56, was seen first on 13th November, '41, after having suffered for years. The beginning was said to have been a prostatitis, later pyelitis, and since some two years there had been from time to time stool passed through the urethra and urine through the rectum. He had been treated in several hospitals and when first seen by me he had just returned from one hospital where he had been told that nothing could be done as an operation was out of the question.

His general appearance was very emaciated and anæmic. He ran a septic temperature and it was observed that stool and flatus were passed from the urethra and that his stool was watery. When the patient was given charcoal he would regularly pass a completely black urine within a few hours. In his stool urea was demonstrated chemically.

Rectal examination and sigmoidoscopy showed nothing of significance. The cystoscopy showed a very strongly inflamed mucosa of the bladder. The ureteric orifices were not clearly defined owing to the general congestion of the mucosa.

The intravenous pyelography showed a complete failure of the left kidney to excrete and an enormous hydronephrosis on the right with a very greatly dilated ureter on the right which was occluded just above the bladder. Two hours after injection of Pyelectan no urine was seen in the bladder yet.

The pyelogram failed to localize exactly the anatomical conditions of the fistula. A barium meal also failed to demonstrate the leak. It proved the presence of numerous



17-11-41. FIG. 1. 7 minutes after injection.



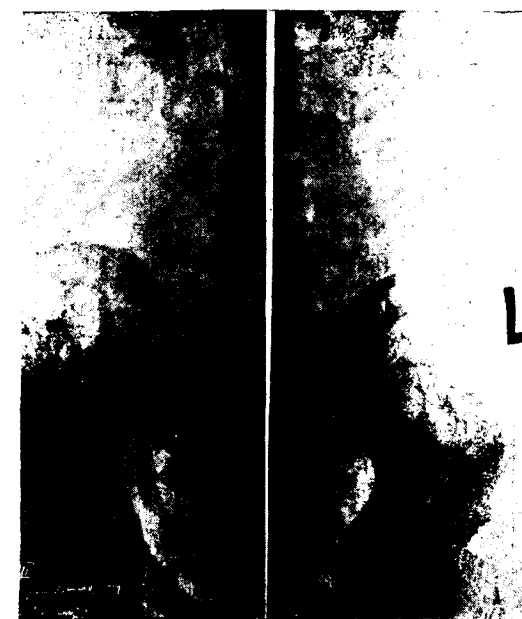
FIG. 3. 40 minutes after injection.



FIG. 2. 20 minutes after injection.



FIG. 4. 60 minutes after injection.

FIG. 5. Control, 60 minutes after injection.
Bladder filled, no excretion on left.

gone up from 45.9 kg. (7 st. 7 lbs.) to 49.1 kg. (7 st. 10 lbs.) and for one whole week there had been no more leak between the two organ systems. Enterovioform was chosen because at that time none of the insoluble Sulpha-drugs were yet available.

From January, '42, the fistula remained closed, though, of course, the pyelitis remained. The patient moved about freely again, but from time to time had attacks of exacerbation of his pyelitis with fever and large amounts of pus in the urine. These attacks were

diverticula and a hold-up in the region of the ileocaecal junction. Barium meal was retained in the ileum as long as 15 hours after meal. It is probable therefore, that all the symptoms may have arisen as sequelae to diverticulitis.

The patient's state appeared to be beyond influence by medical treatment. An attempt, however, was made to reduce the bacterial contents of the bowels by enterovioform and at the same time to reduce the intra-abdominal inflammation by short-waves. This attempt proved successful. On 19th January, '42, his weight had



FIG. 6. 2 hours after injection.



FIG. 7. 17 hours after injection.

always controlled with Suphatiazole or Sulphadiazine of which he must have swallowed during the following years many hundreds without any ill-effects whatsoever.

On September '43, when his weight was 55.6 kg. (8 st. 11 lbs.), in all 22 lbs. up, another pyelography was done, showing exactly the same conditions as previously.

On 7th June, '48, after nearly seven years of useful life, another, this time very severe attack of pyelitis began with very high fever and within two days the patient was in such a



23-12 41. FIG. 8. 15 hours after meal.

24-12 41. FIG. 9. 28 hours after meal.
Picture showing diverticula.
(This skiagram is reversed).

[R]

serious condition that removal to hospital appeared indicated. Sulpha-drugs this time were without effect, and though he was removed to hospital on 9th June, Streptomycin treatment was not begun until June 30th. At this time the patient was already very emaciated and toxic, but Streptomycin nevertheless brought about a complete change of the condition of his urine which became almost clear. However, his resistance had been

weakened by the long-standing fever to such an extent that one week after stopping the Streptomycin-treatment the old fistula-track opened up again, and he again passed stool through the urethra and urine through the rectum.

He died of general exhaustion, but not of uræmia, on 26th July, '48.

Unfortunately permission for a post mortem examination was not obtained.

INTERESTING CASE REPORT

Aneurysm of the Aorta

MAJOR S. K. BOSE, M.B.,

RADIOLOGIST, ORISSA MEDICAL COLLEGE, CUTTACK.

DEMONSTRATION of the Aneurysms of the Aorta is not very uncommon feature in the field of Radiology but the following case showing degeneration of the wall of the aneurysmal sac producing successive pouches each showing expansile pulsation is rare.

U. Patel, male 50 years, sent for X-ray examination of the chest for a vague discomfort over the cardiac region, occasional hæmoptysis and gradual loss of weight. Clinical history of the case did not show anything very particular in either of the lungfields; a bruit was present over

the upper part of the cardiac area conducted towards the root of the neck. W. R. strongly positive; other blood examination report—within normal limit. A. F. B. in sputum absent on repeated examinations. Pulse on two sides showing marked variation, right side faintly perceptible.

Skiagrams taken in usual positions showing aneurysmal dilatation of the arch of the Aorta with a second sac protruding on the left side. Expansile pulsation over both these regions well marked. A small pouch arising from the upper part



FIG. 2.



FIG. 1.

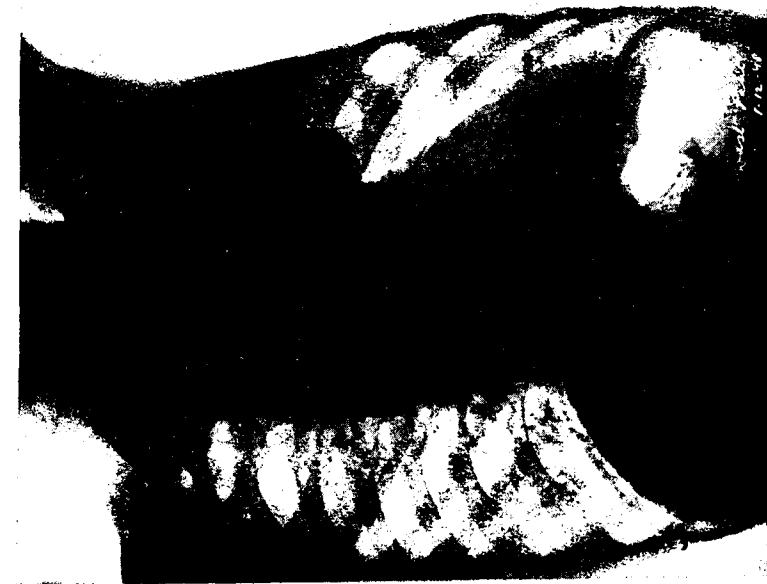


FIG. 4.



FIG. 3.

of the second sac on the left, showed expansile pulsation as well. Marked displacement of the Trachea towards the right side well visualised. On Barium swallow marked deviation of the oesophageal outline detected. Obliteration of the aortic triangle and calcified plaques in the wall of the sac well marked. Cardiac shadow in the postero-anterior view did not reveal any gross abnormality except that it is displaced downwards.

Besides the above findings well developed cervical ribs detected on both sides.

Unfortunately the patient left the hospital against medical advice before any adequate treatment being given.

Extension of the aneurysmal sac on the right side and its pressure on the main vessels leading to the right arm may explain the cause of variation in the volume of the pulse on the right side.

Abstracts From Current Literature

Cancer of larynx.—Max Cutler, *Radio-logy*, Oct. 1948.

Before 1922 Surgery constituted the only form of treatment for laryngeal cancer. During the last 25 years the effectiveness of radiotherapy has improved to a degree that its use in properly selected operable lesions is now generally accepted. Two main problems arise (1) The choice between surgery and radiotherapy for early carcinoma of the true cord amenable to laryngofissure. (2) The choice between laryngectomy and radiotherapy for operable laryngeal cancer beyond the scope of laryngofissure.

Laryngofissure is a well established procedure yielding 80-85 per cent cures in carefully selected cases of cancer of the vocal cord. The results of radiotherapy in this group are at least as good as surgery with the advantage that generally the voice after radiotherapy is better. Laryngofissure yields the best results in lesions limited to the middle or anterior two-thirds of one cord with complete mobility. Once the lesion

involves the posterior extremity of the cord, crosses the midline, extends subglottically, involves the false cord or becomes partly fixed it is no longer suitable for laryngofissure. If the histologic structure as indicated by the biopsy, shows a highly malignant anaplastic carcinoma (Grade IV, Broder's), most laryngologists consider laryngofissure inadequate. Laryngofissure has a long record of favourable results and should not be abandoned; at the same time adequate recognition must be given to the increasing evidence that radiotherapy yields equally good results with a better voice. Recurrences following laryngofissure do not respond as well to radiotherapy as does the original lesion. Exploratory laryngofissure should not be undertaken.

Regarding laryngectomy, it has been said that radiotherapy is a much more efficient substitute for laryngofissure than for laryngectomy. This is only partly true. Lesions unsuitable for laryngofissure by virtue of site, extent or histological structure, but

still not completely fixed by carcinomatous invasion are ideally suitable for radiotherapy with a curability equal to that of laryngectomy (which is about 60 per cent.) Cutler now limits laryngectomy to cases of intrinsic cancer in which there is total fixation of the laryngeal structures, provided that the fixation is due to carcinomatous invasion and not to inflammation. In border line cases, irradiation may be tried; if the response is favourable, the treatment is completed; if the chances of success are remote laryngectomy is undertaken. It is now established that previous correct irradiation does not interfere seriously with subsequent laryngectomy.

The following criteria are suggested as a guide in choosing between surgery and radiotherapy:

(1) *Extent of the lesion*:—Lesions which are extrinsic by origin or extension are generally regarded as inoperable. Presence of cervical lymph node metastasis usually indicates inoperability. When the lesion is so advanced as to be inoperable and apparently incurable by radiotherapy irradiation should be designed with a view to palliation.

(2) *Site of origin*:—Lesions arising in the ventricles, false cords and base of the epiglottis are generally more radiosensitive whereas subglottic lesions are usually more radioresistant. The radiosensitivity of lesions of the true cord depend largely on the extent and degree of infiltration.

(3) *Gross character of lesion*:—Generally speaking papillary lesions are radiosensitive in infiltrating lesions relatively radioresistant.

(4) *Mobility of laryngeal structures*:—Mobility of laryngeal structures generally indicates that the lesion is early, or if more advanced that it is papillary and not infiltrating. From the point of view of irradiation such a lesion is radiosensitive and to a considerable degree curable, the curability

being directly related to the extent of involvement, the degree of malignancy, general condition of patient and efficiency of treatment.

Partial or complete fixation is often due to inflammation caused by secondary infection and not to carcinomatous invasion. Such fixation occurs especially after one or more biopsies. It generally begins to diminish and disappears soon after irradiation is begun and in cases of doubt this response is a useful sign in interpreting the cause of fixation. Rapid increase in mobility also occurs after a small amount of irradiation when the lesion is highly radiosensitive. From a practical point of view the early and rapid response is an indication of radiosensitivity and probable radiocurability.

(5) *Microscopic structure*:—Many believe that the more undifferentiated carcinomas have a worse surgical prognosis. Some are influenced by the histological grading in their choice of treatment. Cutler believes that it is hazardous to draw conclusions from the histologic structure of a biopsy specimen except in a general way. There are two types regarding which he gives positive opinion (1) when there is marked anaplasia especially with the formation of spindle cells the lesion is nearly always highly malignant and offers an unfavourable prognosis regardless whether surgery or irradiation is used (2) "Transitional cell carcinomata" are generally radiosensitive and when not too advanced are curable by adequate irradiation. This type is usually found in lesions originating in the false cords, the ventricles, the aryepiglottic folds and the epiglottis. S.V.

Artificially prepared Radio-active Isotopes as a means of administration of Radiation therapy—E. H. Reinhard, *American Journal of Roentgenology*, December, 1947.

(2402)

Of the various Radio-active Isotopes, only two have been used extensively in the treatment of diseases and proved to be of real value.

Radio-Active Phosphorus

When the nucleus of stable phosphorus is bombarded with low energy deuterons Radio-active phosphorus (P. 32) is obtained. By giving the emission of an electron or beta ray the Radio-active phosphorus becomes stable sulphur. The maximum range of penetration of these beta rays is approximately 0.7 cm. and the half life of P. 32 is 14.3 days.

Investigations showed that when P. 32 is administered to animals or human beings it is selectively drawn from the blood by certain tissues and cells. The tissues in which P. 32 accumulates in largest amounts are those which are primarily involved in Polycythemia vera, Leukæmias and Lymph node diseases. Studies have shown that P. 32 is taken up much more rapidly and in two to three times larger amounts by the lymph nodes and spleens of mice with Lymphatic leukæmia than in the corresponding organs of the normal mice. In human beings the distribution of P. 32 in leukæmia is found to have greatest concentration in the bone marrow, lymph nodes, spleen and liver.

Polycythemia vera :—There is substantial agreement that excellent clinical and hæmotologic remissions can be obtained in the majority of patients with the use of P. 32. The dose recommended is 6 to 8 millicuries for the first course. Certain features regarding the response of patients to P. 32 need to be emphasised. (1) There is a marked variation in the response of different patients to the same dosage of Radio-active phosphorus. The exact amount required to restore the blood counts to normal in any particular case cannot be predicted in advance. (2) The erythrocyte count never shows a signi-

ficant decrease in less than 3 weeks and the latent period is frequently much longer. It is usually 2 to 3 months after treatment is started that the red-cell count becomes stabilised at a new lower level. The remissions both hæmotologic and symptomatic have lasted anywhere from 5 months to more than 4 years; the average interval before a second course of treatment is required is about 13 months. It is not possible to draw any conclusion with the available data regarding the effect of P. 32 on the duration of life of the patients. To compare roentgen therapy with P. 32, the follow-up is not yet sufficiently long.

Myeloid leukaemia :—All investigators have agreed that P. 32 is of little value in the treatment of acute myeloid leukæmia. There is agreement that in chronic myeloid leukæmia P. 32 will restore the W.B.C. count to normal or approximately normal levels and that there is a rise in the R.B.C. count at the same time. P. 32 prolongs life by only a few months. There is no significant difference between this and X-ray therapy from this point of view. Both definitely prolong the period of useful and relatively symptom free life.

Lymphatic leukaemia :—Most people agree that P. 32 is effective in the majority of cases of chronic lymphatic leukæmia in restoring the W.B.C. count to normal levels reducing the size of enlarged lymph nodes and spleen and temporarily relieving symptoms. P. 32 is less effective in chronic lymphatic leukæmia than in chronic myeloid leukaemia.

In any type of chronic leukæmia, X-ray therapy is more effective in some cases than P. 32 in bringing about a rapid reduction in the size of the spleen or lymph nodes. Therefore roentgen therapy should be used whenever prompt reduction of nodes or spleen is necessary to relieve symptoms or

remove pressure on some vital organs. Roentgen therapy may be used to supplement P. 32 for this purpose.

Other diseases :—P. 32 has not been found of any value in the treatment of acute forms of Leukæmia. Various people have used it in Hodgkin's disease, Lympho-sarcoma and Multiple Myeloma. In the experience of the author none of these conditions respond as favourably as they do to X-ray therapy. A few cases of Lympho-epithelioma, Malignant melanoma, Carcinoma of the gall-bladder, Carcinoma of the breast and Ewing's tumour, Mycosis fungoides and Xanthomatosis have been treated with P. 32. There is no convincing evidence that this treatment is of any value in any of these neoplastic diseases.

Radio Active Iodine

There are two isotopes I-130 and I-131. The former has half life of 12.6 hours and the latter 8 days. Both are prepared by bombarding of metallic Tellurium by deuterons. Both I-130 and I-131 emit beta and gamma rays. From the standpoint of the effect on tissues the beta rays are more important. It has been found in rabbits that normal thyroid collects up to 80 times the quantity of iodine compared to the amount collected in the general body tissues; the hyperplastic thyroid collects upto several hundred times the quantity found in other tissues. It is found that in human beings the same principles apply.

Hyperthyroidism :—After I-130 therapy about 70 percent of the patients were no longer thyrotoxic and they remained so for 3 to 5 years after treatment. The thyroid decreased in size in all these patients. Several patients who had not responded well to Thiouracil or routine lugolization subsequently responded well to Radio-active iodine. No undesirable radiation effects were observed. Some workers have used I-130 and others I-131.

Carcinoma of the thyroid.—The malignant portion of the thyroid, has no significant capacity to take up Iodine; the cancerous tissue takes up very little and the secondary metastatic tumour much less. The more malignant and anaplastic the thyroid cell becomes the more completely it loses the ability to take up and utilise Iodine. However an occasional thyroid cancer does utilise Iodine. So it should be kept in mind that therapy with Radio-active iodine is effective in only one, rather a rare form of thyroid tumour and the vast majority of thyroid cancers are totally unaffected by Radioactive-iodine.

Chromic Phosphate prepared from P. 32

On injection of this drug into laboratory animals it has been found that the material is selectively concentrated in Reticulo-endothelial cells of the body. The highest concentrations were found in the liver, spleen and bone marrow. These studies suggested the possibility of using such a preparation in the treatment of Hodgkin's disease, Reticulum cell sarcoma and other tumours arising in the Reticulo-endothelial system. Since then radio-active chromic phosphate as well as radio-active iron phosphates have been given to a few human patients with various Lymphomas with equivocal results. By infiltrating radio-active chromium phosphate around the periphery of Rodent ulcers in human beings satisfactory results have been obtained. The workers who used this method for these tumours emphasise that these tumours can be readily cured by surgery or X-rays and the use of radio-active chromium phosphate was purely experimental.

Radio-active Manganese

The use of Radio-active Manganese has been tried by intravenous administration of colloidal suspensions of manganese dioxide containing the radio-active manganese in

the treatment of Hodgkin's disease, Chronic leukaemias and Lympho sarcomas. The work is still in the preliminary stage and the early results have shown promise.

Radio-active Calcium and Strontium

Strontium is physiologically interchangeable with calcium and an isotope of strontium which emits energetic beta rays can be prepared with relative ease. This has been employed in the treatment of bone tumours. The radio-active strontium is accumulated in large amounts in those parts of the bone where active cell multiplication is taking place, such as epiphyses and also at the site of the fractures. Osteogenic sarcoma

tissues will take up some of the isotopes but the clinical results have been very disappointing and this type of therapy has been abandoned.

Radio-active Sodium (Na. 24)

It has half life of 14.8 hours and emits both *beta* and *gamma* rays. It can be easily prepared. There is no selective uptake by bone or bone marrow. The isotope is distributed evenly to the tissue fluids throughout the body and treatment with this corresponds to general body roentgen radiation. It is of some use in Chronic leukaemia where it shows the same response as in general body roentgen irradiation.

S. V.

6th International Congress of Radiology

The 6th International Congress of Radiology will be held in London from July 23rd to July 29th 1950. The headquarters of the Congress will be at the Central Hall, Westminster, which will also house an extensive Scientific Exhibition. The Technical Exhibition of apparatus will be located in the Halls of the Royal Horticultural Society nearby.

The Officers of the Congress are as follows:

<i>President:</i>	DR. RALSTON PATERSON, Manchester.
<i>President-Emeritus:</i>	DR. A. E. BARCLAY, Oxford.
<i>Vice-Presidents:</i>	
Diagnosis:	DR. S. COCHRANE SHANKS, London.
Therapy:	PROF. B. W. WINDEYER, London.
Biology:	DR. F. GORDON SPEAR, Cambridge.
Physics:	PROF. W. V. MAYNEORD, London.
<i>Treasurer:</i>	DR. H. GRAHAM HODGSON, London.
<i>Secretary-General:</i>	DR. J. W. McLAREN, London.

The subjects chosen for the main scientific meetings are:

1. General Congress Scientific Meetings

- "Radiological Achievement, 1937-1950."
- "Mass Radiology of the Chest."
- "Supervoltage Radiotherapy."
- "Radiation Hazards."

2. Diagnosis Section Symposia

- "Skeletal Changes in Blood Diseases."
- "Radiology of the Small Intestine."
- "Arthrography."
- "Angio-cardiography."

3. Therapy Section Symposia

- "Method of Presentation of Results of Treatment."
- "Radioactive Isotopes."
- "Cancer of the Larynx."
- "Cancer of the Breast."

4. Biology Section Symposia

- "Radiation Histology."
- "Radiation Chemistry."
- "Radiation Genetics."
- "Mode of Action of Ionizing Radiations."

5. Physics Section Symposia

- "Acceleration of Particles and the Generation of Ionizing Radiations."
- "Radiological Units."
- "Radiotherapeutic Physics."
- "Production and Physical Properties of Radio-Isotopes."

Selected speakers will be invited to contribute the major portion of these symposia. Other sessions will be devoted to the reading of papers preferred by authors on their own topics.

A varied and interesting social programme is being arranged for members of the Congress, and special attention is being paid to the entertainment of Associates accompanying Members.

During the week preceding and the two weeks following the Congress demonstrations will take place in the radiological departments of a number of London Hospitals. Planned tours to centres of interest in Great Britain and Ireland are being arranged to follow the Congress; they will include excursions to the neighbouring countryside by coach, visits to buildings of historical interest, demonstrations at hospitals, and a full social programme.

Those wishing to attend the Congress as Full Members (£7. 7s. 0d.) or as Junior Members (under 30 years of age on January 1st, 1950—£4. 4s. 0d.) must be members of a radiological society, or sponsored by a radiological society. Ladies and children accompanying Members can be registered as Associate Members (£3. 3s. 0d.). Associate Membership is also open to technical staff of radiological departments and laboratories, or of the X-ray industry. A late fee will be charged to those registering after April 1st, 1950.

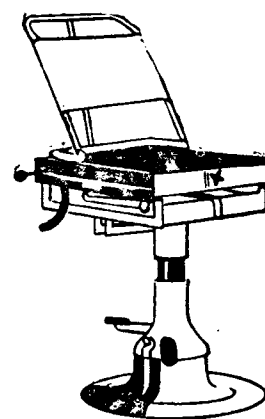
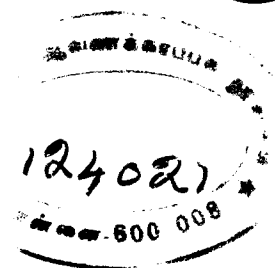
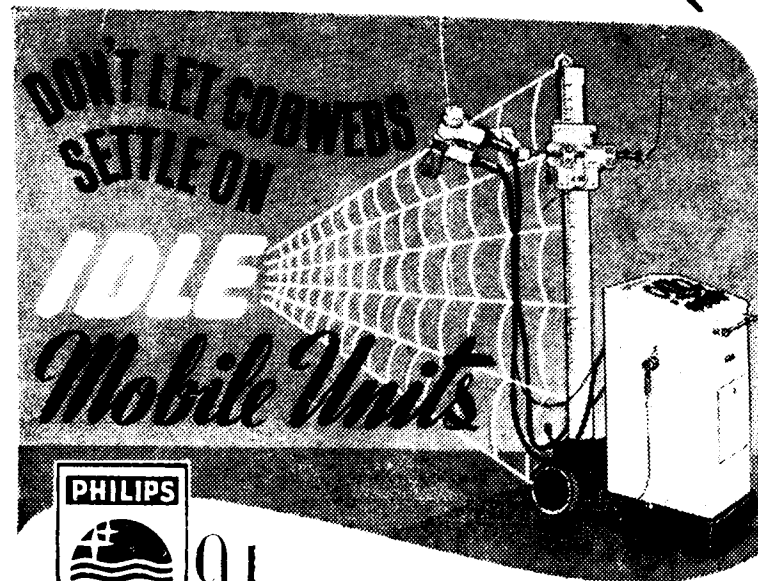
Members of the Congress may make their travel and hotel reservations through any office of Messrs. Thos. Cook & Son, Ltd., (or their associated Company, the Cie Internationale Des Wagons-Lits) who have been appointed the official travel agents for the Congress.

It is planned to issue a detailed programme with registration forms to the members of radiological societies in April 1949. All communications in connection with the Congress should be addressed to the Secretary-General at 45, Lincoln's Inn Fields, London, W.C. 2.

NOTE:—It has just been learnt that H. R. H. the Princess Elizabeth hopes to open the Congress.

NOTICE

Any contributor to Diagnosis Section Symposia should immediately communicate to Dr. K. P. MODY, Victory Court, Queen's Road, Bombay. He will get in touch with the Secretary and send on the name. Selection, according to the circular, will be limited as there are only 6 speakers in each symposium. The matter should be treated as urgent.



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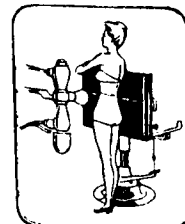
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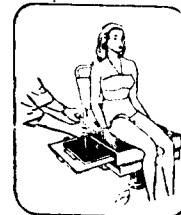
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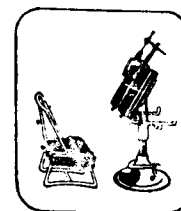
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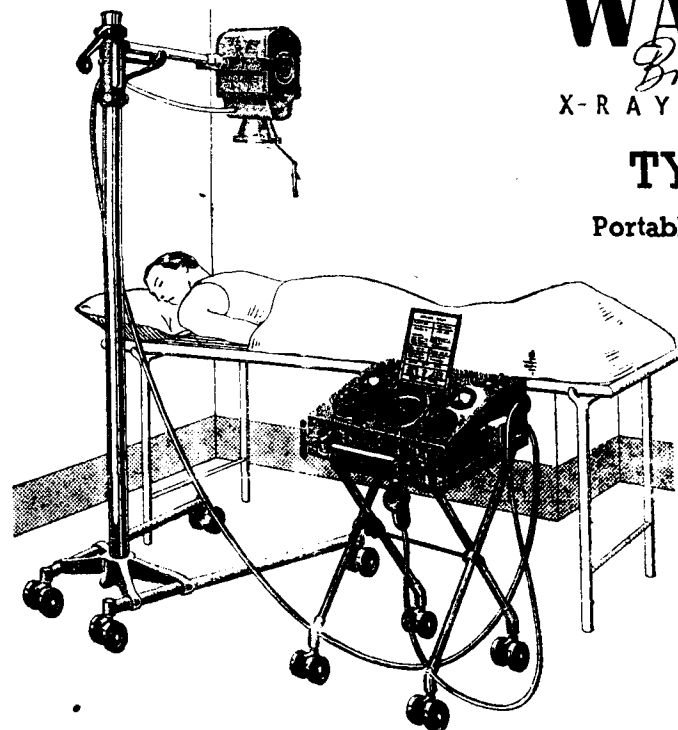
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