any form of treatment. After the clinical diagnosis had been fully confirmed by numerous investigations (Table I) she was treated with A.C.T.H. at intervals for the next eight months and responded extremely well. On two occasions, however, she developed fairly severe water retention, although the dose of A.C.T.H. which she was receiving at the time was not excessive (40 or 60 mg. daily) and she had previously received similar doses for several weeks without untoward effects. We attributed the attacks of water retention to the small quantities of pitressin which we believed some of the early batches of A.C.T.H. contained, and she recovered on each occasion when the A.C.T.H. was stopped for a few days and then changed to another batch. She was discharged from hospital on a maintenance dose of 25 mg. daily, but unfortunately she left the area in order to make contact with her husband, from whom she had been separated for several years, and we lost sight of her.

She was readmitted five months later suffering from severe hypopituitarism, and went into coma while we were carrying out a further series of investigations and before any form of treatment was started. During the five days preceding death there was a considerable decrease in the urinary volume and in the serum sodium, which fell from 137 to 116 mEq/l. The serum potassium also fell from 4.6 to 2.9 mEq/l. Urinary studies showed that this was due to water retention and not to urinary loss of electrolytes. There was no decrease in haemoglobin or haematocrit levels in the blood. When she was in coma the blood sugar was 65 mg./100 ml. and the rectal temperature dropped to 95° F. (35° C.). She looked bloated, and became more so as the depth of the coma increased. Measures to treat the hypothermia raised the rectal temperature to 99° F. (37.2° C.), and she was given 125 mg. of cortisone intramuscularly on the day before she died.

At necropsy the pituitary gland was found to be considerably reduced in size, weighing 0.21 g., and the thyroid gland, the ovaries, and the uterus were very small. The suprarenals were leaf-like bodies and showed severe atrophy of both cortex and medulla. The atrophy was most noticeable in the cortex, which was reduced to a thickness of some twenty cells. There was marked interstitial oedema in the myocardium, both muscle bundles and individual fibres being separated by free fluid. In the brain the Virchow-Robin spaces were distended with fluid and there was some oedema of the pia arachnoid. There was also moderate interstitial oedema in the medulla of the kidneys though not in the lungs. The pituitary was largely replaced by hyaline and fibrous tissue, only a few groups of parenchymatous cells remaining.

We feel that this case illustrates the danger of the disturbance of water balance that may exist in severe cases of hypopituitarism, and the appropriate treatment would have been to give the patient moderate doses of cortisone before she was moribund. On the other hand, Case 5 drifted into a hypothermic coma while receiving 50 mg. of cortisone daily, and recovered after cortisone was stopped and the body warmed. Recovery might have been more certain if we had also injected sodium thyroxine on the lines attempted by Summers (1953) for the treatment of myxoedema coma. In other cases of hypopituitarism the episodes of coma are caused by severe hypoglycaemia, the most effective treatment for which is the administration of intravenous glucose combined with repeated estimations of the blood sugar.

Our experience of six cases of coma encountered during the past few years, and a study of other reports, have emphasized the diversity of the functional disturbance causing the coma and the need for biochemical investigations to be carried out before treatment is started. The administration of cortisone may save life in some cases, especially when there is evidence of water retention, but in coma associated with hypothermia other measures are likely to prove more successful, and measurement of the rectal temperature, if necessary with a bacteriological thermometer, should be included in the clinical examination.

Summary

The clinical features of nine cases of hypopituitarism and the value of biochemical investigations are discussed.

Treatment with cortisone and thyroid has proved more satisfactory than with A.C.T.H. or cortisone alone. The addition of methyltestosterone is recommended in some cases.

The abnormality of water balance in hypopituitarism is emphasized, and it is suggested that water retention may be one of the causes of coma in this condition.

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OSTEOARTHRITIS AND REST

BY

F. DUDLEY HART, M.D., F.R.C.P.

*Assistant Physician, Westminster Hospital; Physician,
St. Stephen's Hospital, London, S.W.10*

MONICA WATKINS, M.B.

Medical Registrar, St. Stephen's Hospital

DENIS BURLEY, M.B.

House-Physician, St. Stephen's Hospital

AND

M. T. RICHARDS, M.B.

House-Physician, St. Stephen's Hospital

Current treatment of osteoarthritis of the weight-bearing joints, if one looks into recent textbooks and articles on the subject, emphasizes the value of rest; rarely are its hazards even mentioned. The patient is encouraged to become slimmer, less active, and to reduce his activities rather than the reverse. The patients themselves have different views in most cases. Fletcher (1951) states: "Most patients are under the impression that they must use their joints as much as possible, and that if they do not they will become crippled." In Hollander's 5th edition of Comroe's *Arthritis* we read, "Many individuals suffering from degenerative joint disease are obsessed with the idea that their joints will become stiff unless they exercise them continuously; this is not true." Substitute the word "periodically" for "continuously" and it is perfectly true. Patients get to know their arthritic joints and learn how to live with them. Their statements should not be lightly discarded or dis-

credited. Buckley (1938) and Fletcher (1948) are careful to point out, though rest for the affected joint is important, invalidism must be avoided, the first author emphasizing the value of exercises to prevent contraction of the capsule of the hip. Steinbrocker *et al.* (1950), in an article entitled "Musculo-skeletal Inadequacy and Failure in the Aged," emphasize that rapid and disabling progression of symptoms may occur even with mild osteoarthritis.

While there is no doubt that overactivity and stresses and strains aggravate the condition there is also no doubt that too little activity does so also. Overexertion and excessive use of an affected joint will produce a sharp reaction within the next 24-48 hours, but immobility may produce a much longer disability. This is a lesson we have learned not from a study of patients in beds for rheumatic sufferers but in the general medical wards, where cardiac decompensation, bronchitis and bronchopneumonia, coronary thrombosis, bleeding peptic ulcers, and other diseases, usually in patients over 60 years of age, cause immobilization in bed for periods of several weeks.

At the end of such a period of bed rest not infrequently an equal period of time is spent in getting the patient on to his or her feet again. Previously complaining little or not at all of osteoarthritic hips or knees, the patient is now severely incapacitated and weeks of rehabilitation are necessary before discharge is possible. This is no small problem; several months of hospital time are taken up each year treating osteoarthritis in patients who were admitted severely ill for some other disorder. With long waiting-lists this is a criticism of our existing methods, and constitutes yet another argument for early mobilization and exercises in bed as soon as the patient's primary condition permits. Even now that we are fully alive to the problem valuable hospital time is spent treating osteoarthritis aggravated by bed rest. The following case histories are unfortunately not uncommon in the general medical wards, do what one can.

Illustrative Cases

Case 1.—A man aged 68 was admitted to hospital on October 13, 1953, with a typical history suggesting myocardial infarction; this diagnosis was confirmed on electrocardiographic examination. Twelve years previously he had had peptic ulceration treated conservatively; mild angina of effort had been present for two years. At no time had he suffered from pain or stiffness of any joint, and his gait was normal. Treatment of the cardiac disease necessitated three and a half weeks' rest in bed, although he was allowed to use a commode after the first week. Two weeks after admission he complained of stiffness and numbness of the right leg, and on examination it was found that he had considerable limitation of movement in his right hip-joint, particularly of abduction. X-ray examination at this time revealed gross osteoarthritis of the right hip and slightly of the left also. Leg exercises were started while he was in bed, but when the time came for him to be mobilized he was scarcely able to stand on his right leg. Only after four weeks' perseverance with the aid of physiotherapy was he able to walk without assistance or the use of sticks. He was discharged to convalescence on January 7, 1954, still with a considerable limp.

Case 2.—A Chinese cook aged 69 was admitted on April 24, 1953, with haemoptysis due to pulmonary tuberculosis. This required an initial period of bed rest for three months. On getting out of bed at the end of this time he complained of difficulty in walking and pain in both hips. Examination revealed gross limitation of hip movement. He had previously led a fairly active life negotiating several flights of

stairs daily, but had used a stick for two to three years. X-ray examination of the hips showed osteoarthritic changes. He was given active physiotherapy, but his discharge from hospital was delayed for four weeks, as he was unable to ascend or descend a flight of stairs until this time.

Case 3.—A woman aged 68 was a known case of diabetes mellitus of four years' duration stabilized on diet alone. She was well and fully mobile until two weeks before admission, when she went to bed at home with an upper respiratory tract infection. This was followed by malaise and vomiting, and two days before admission she fell out of bed and injured her right knee. She was admitted for stabilization of her diabetes on August 15, 1953. On examination she was grossly obese, and an effusion was present in the right knee. On diet alone her diabetes was satisfactorily controlled within a week. The right knee was treated by rest and immobilization with passive movements for one week, and an attempt was then made to mobilize her. On getting out of bed, however, she complained of severe pain and stiffness in both knees, and an x-ray film showed gross osteoarthritic changes. She was given active physiotherapy, but required another three weeks in hospital before she was fit to be discharged.

Case 4.—A woman aged 79 was admitted to hospital in a collapsed state with a history of an attack of abdominal pain and vomiting. Her general condition necessitated a period of four weeks' rest in bed. Towards the end of this period she complained of pain and stiffness of the left knee. The latter was slightly swollen, with some fluid in the joint. Movements were painful and limited to about 90 degrees of flexion. X-ray examination showed degenerative changes in both knees and hips. She stated that while in hospital two years previously she had had a similar attack which involved both knees, the right one being more affected than the left. She was treated then with heat and exercises, and her condition improved, leaving her with only minor degree of pain and stiffness. On the present occasion heat in the form of a kaolin poultice was applied to her left knee and at the same time her activities were gradually but rapidly increased. She was discharged to convalescence 10 days later, walking well, stiffness and swelling having disappeared, and with minimal pain only.

Comments

While most current textbooks on the subject are careful to recommend only short periods of rest in the treatment of osteoarthritis, few emphasize the real dangers of prolonged immobility. Perkins (1953) states that osteoarthritis has two main pathological features—first, the faulty nutrition of the cartilage, which fibrillates and "wears out"; and, secondly, contraction of the capsule so that the range of movement diminishes. "When a person is trying all the time to move a joint beyond the range allowed by the capsule, the capsule is subjected to repeated stretching. The symptoms of osteoarthritis are in the main caused by the reaction of the capsule to this repeated stretching." Kellgren and Samuel (1950) and others have emphasized the importance of the capsule as a cause of pain; it has an abundant nerve supply, unlike the synovial membrane.

Lloyd-Roberts (1953) considers the development of osteoarthritic changes in a hip-joint to be somewhat as follows: the cartilage, fibrillating, throws off small particles which are engulfed by the synovial membrane, which becomes hyperplastic. Sustained synovial hyperplasia is followed by capsule fibrosis, particularly in the most dependent postero-inferior pocket of the joint. Pain on certain movements is caused by stretching of the capsule, which gradually shortens with increasing fibrosis as the synovial membrane also fibroses. Restriction of movement of the hip causes a smaller area of the femoral head to come into apposition with the acetabulum and aggravates the degenerative changes in the small area of degenerating or degenerated cartilage bearing the body weight. He writes: "It

has already been suggested that shortened and thickened capsule becomes tense prematurely and so restricts the range of hip movement. It is now suggested that if movement continues beyond this limit imposed by the shrunken capsule, and if at the same time body weight is added to the movement, a considerable traction force is likely to fall upon this abnormal ligament, thereby producing pain and protective muscle spasm." He explains the characteristic position of the osteoarthritic hip—adduction, flexion, and lateral rotation—in terms of capsular fibrosis and shortening; this position, at first the painless one, becomes in time the fixed and permanent one.

Harrison *et al.* (1953) made the interesting discovery, in their thorough and painstaking study of the osteoarthritic hip, that the earliest degenerative changes in the cartilage are not in the weight-bearing areas but in the non-weight-bearing areas. They found that daily use preserved rather than wore out articular cartilage, and state "... inadequate use is the commonest cause of cartilage degeneration," and again "... the necessity for use and compression of cartilage in order to maintain its continued health strongly suggests that mechanical forces are concerned with cartilage nutrition."

The above findings all help to explain the deterioration which occurs in a patient's weight-bearing osteoarthritic joints during a period of complete bed rest. In bed the hip tends to be held in flexion and lateral rotation with the knee flexed; therefore a very vigorous policy of routine exercises for all patients confined to bed in the ward is required several times a day.

In orthopaedic wards of military hospitals overseas during the war routine exercises were carried out at set intervals to prevent renal calculus formation, the so-called "kidney drill"; a similar routine is necessary for the elderly bed-fast osteoarthritic patient in civilian medical wards. To prevent deterioration in osteoarthritis of weight-bearing joints certain common-sense measures may be taken. The commode is usually preferable to the bed-pan, and, unless patients must not or cannot be taken from bed, this time out of bed can be used also for exercising hips, knees, and ankles. Elderly osteoarthritic patients should be mobilized as soon as possible—a measure which will also lessen thrombo-embolic complications, and an active programme of exercises in bed should be undertaken from the time they are admitted to the ward if their general condition permits. Patients able to do so can avoid the pitfalls of the flexed hip and knee by lying flat in bed, with pillows and back rests removed, twice a day. Selected patients with deformities already present can be induced to spend equivalent periods in the prone position.

Analgesics should be used freely over such active periods if pain prevents proper co-operation, for effective analgesic "cover" for exercises is invaluable. Phenylbutazone ("butazolidin"), with its prolonged action, would be most useful were it not for its toxic side-effects, the most important in this group of patients being gastric irritation and salt retention with resulting oedema and precipitation of congestive cardiac failure; in certain carefully selected patients, nevertheless, it may prove very useful. Intra-articular hydrocortisone may be used in cases in which the knee is at fault; unfortunately, injections into the hip are far less effective (Hollander, 1953) and much more difficult. An injection into the knee of 25–35 mg. will often help in rehabilitation, and Hollander *et al.* (1951) found the results better in osteoarthritis than in rheumatoid arthritis, as the effects appeared to last longer. Injection of a local analgesic into ligamentous attachments is useful in mobilizing stiff and painful knees. None of these measures may be possible in severely ill patients, but emphasis should be on as early mobilization as is possible compatible with their medical condition. This has been emphasized by many writers in the past—for example, Asher (1947) in an article entitled "The Dangers of Going to Bed"—but we feel that the subject needs even further emphasis in this ageing community.

Summary

While graded rest is quite correctly the basis of orthodox treatment of osteoarthritis of the weight-bearing joints, there has been too little emphasis in the past on the dangers of immobility and excessive rest in this disease. Aged patients admitted into the general wards of our hospitals are not infrequently severely disabled by aggravation of their pre-existing osteoarthritis by complete bed rest. Attention is drawn to this problem and certain therapeutic measures are suggested.

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NEPHRECTOMY IN HYPERTENSION DUE TO RENAL ARTERY INFARCTION WITH SUPERINFECTION OF STREPTOCOCCAL ENDOCARDITIS BY FUNGUS

BY

W. A. BOURNE, M.D., F.R.C.P.
Physician, Brighton General Hospital

Of 46 cases of hypertension due to unilateral renal disease and cured by nephrectomy, Langley and Platt (1947) found renal artery infarction recorded as the cause in one only. A similar case, observed before hypertension developed and for three years after great relief by nephrectomy, is here described. It seems also to be one of the earliest recognized cases of fungous endocarditis in Britain. No record has been traced in this country, and very few elsewhere, in which this infection complicated bacterial endocarditis.

Case Report

A single girl aged 17 was admitted to Brighton General Hospital on June 21, 1950, with a six-weeks history of joint pains and fever which resisted salicylates and "cremomerazine." Past history: a weak heart diagnosed aged 8, but no previous rheumatic disease. Family history: non-contributory. No epilepsy. Clinical examination: T. 101° F. (38.3° C.). Synovitis of small joints of right foot only. Heart: not enlarged; systolic murmur maximal midway between apex and lower sternum; B.P. 120/80. Urine normal. Blood: Hb, 60%; red cells, 3,510,000; white cells, 7,700 (normal differential); E.S.R. 63 mm. in first hour; haematocrit 28%, adjusted 32 mm. Nose swab: *Staph. albus* and scanty *Staph. aureus*. Throat swab: no growth. X-ray examination: heart, lung, and vascular shadows physiological. E.C.G.: P-R interval 0.24 second. Treatment with salicylates to the limit of tolerance did not reduce the fever.

July 2: B.P. 120/80. Urine: many red cells with granular and epithelial casts, albumin 0.07 g. per 100 ml., and sterile culture. Retinoscopy: superficial haemorrhages in both fundi and blurring of right disk. July 4 and 5: blood