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

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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

cultures, *Str. viridans*, sensitive to penicillin. July 6: soluble penicillin, 500,000 units six-hourly, begun. Relief incomplete. July 17: T. 102° F. (38.9° C.). Pain in right thigh and left foot, subconjunctival haemorrhages in both eyes, a small haemorrhage in right fundus, petechiae in neck and front of chest, axillae, and abdomen. B.P. 135/85. Spleen palpable. Blood urea 32 mg. per 100 ml. Urine: red cells, casts, and sterile culture. July 18: blood culture sterile. Penicillin continued and aureomycin, 100 mg., given for one dose intravenously, followed by 500 mg. orally four times daily for six weeks. Response was still slight and slow, fever, subcutaneous purpura, and joint swelling persisting, with, on August 15, photophobia and left head pain. During the next week, however, fever, purpura, and joint pains disappeared. On August 23, aureomycin was omitted, but penicillin was continued until October 19. The remission lasted only from August 23 to 30, when pain and tenderness appeared in the left loin and hypochondrium, not very severe, lasting 12 hours. T. 104° F. (40° C.). Urine: albuminuria, no gross blood. Splenic infarction was incorrectly diagnosed.

For a fortnight there was apparent improvement, with no tenderness or fever, but albuminuria 0.2 g. per 100 ml. No record of the blood pressure was made. On September 18 blood pressure was 200/140 in the morning. Retinae: exudates and haemorrhages. Urine: many red cells, a few white cells and hyaline and epithelial casts, and albuminuria 0.2 g. per 100 ml. In the afternoon vomiting and in the evening status epilepticus developed, needing thiopentone, 0.25 g., intravenously for control. Cerebrospinal fluid: white cells less than 1 per c.mm.; protein 30 mg. per ml. September 19: semi-comatose, mild right hemiplegia and aphasia, plantar reflexes flexor. Blood: white cells, 23,000 (polymorphs 87%). Blood urea, 38 mg. per 100 ml. September 20: conscious, and retrograde amnesia for preceding months; severe headache; B.P. 190/160. For the next fortnight intermittent unconsciousness, convulsions, fretfulness, whining, and vomiting continued, with brief intervals of improvement. Urine: many red cells and albumin 0.24 g. per 100 ml. Hypertension increased to 210/170 by October 19, with vomiting and petechial haemorrhages, and hypotensive treatment was begun (hexamethonium iodide to 100 mg. intravenously four-hourly) for four days until urinary retention and faecal impaction appeared, with the blood pressure 100/80. Oral administration was substituted and symptomatic relief obtained, though hypertension persisted (180/140). Urinary infection with *Bact. coli* was treated by streptomycin, and by November 21 the urine was sterile, with few red and white cells, occasional hyaline and epithelial casts, and albuminuria.

On November 28 renal investigation was at last begun. Intravenous pyelography showed secretion from right kidney but none from left. Urea clearance (blood urea 17 mg. per 100 ml.) 19% in first hour (103 ml.), 35% in second hour (193 ml.). Cystoscopy: normal bladder and ureteric orifices. Indigo-carmin injection: no excretion from left, good concentration in four minutes from right: urine from right kidney, few red and white cells, no casts or crystals, sterile, albumin 0.2 g. per 100 ml. Retrograde pyelography on left: normal renal pelvis. December 15: left nephrectomy (Mr. Keith Powell); removal of infarcted kidney and biopsy of left adrenal gland. B.P. pre-operatively 200/150; fell immediately after operation to 130/70; rose to 180/110 in the evening; remained during the next week 130/90, the maximum reading on discharge a month later being 140/105. On April 23, 1951, it was 150/95 and the patient resumed work (wood sorting). On March 31, 1952, B.P. was 130/90, and on March 16, 1953, 125/95. She had had psychological difficulties, being one of a "problem family," and, though she complained of fainting attacks at menstrual periods, was thought to be restored to the degree of physical and mental activity that would have been expected without her illness. She then married, and it appeared that her attacks were epileptiform.

She was readmitted to hospital on December 31, 1953. Blood pressure on admission, 135/95. Clinically, condition

unchanged. The day after admission she became inaccessible, rambling, tearful, and unable to remember her address. While her blood pressure was being taken (170/135) a major epileptic fit developed, with deviation of head to the right. On recovery, B.P. fell to 135/95 and continued between 135/90 and 160/100 during her stay, the higher readings being associated with emotional tension.

Investigations.—W.R., negative. Urine: sterile, with occasional red and white cells (catheter specimen); no albumin or casts. Dilution to S.G. 1002, concentration to S.G. 1020. Blood urea 30 mg. per 100 ml. Urea clearance 30% (40 ml. possible error in collection) and 80% (115 ml.). Urea concentration 2.2 g. per 100 ml. and 3 g. per 100 ml. Serum proteins, blood count, and E.S.R. physiological. Heart condition (Dr. Kemball Price): ventricular septal defect or mitral incompetence. Electroencephalogram: the alpha rhythm not very stable, showing a frequency of 10–11 c./sec. It is better seen on the right side. There is a great deal of medium voltage, 4–6 c./sec. activity, and sharp waves having a tendency to occur in paroxysms. The slow waves occur in all leads, perhaps more on the left side. The paroxysmal sharp waves occur bilaterally, but are of highest voltage over the left fronto-temporal area. Over-breathing increases the number of paroxysms and enhances the asymmetry slightly. Flicker did not add useful information. This record suggests a diffuse degenerative lesion affecting the whole brain, with maximal damage in the left fronto-temporal area. The lesion is epileptogenic (S. Kay, January 16, 1954).

During her stay in hospital the B.P. varied between 135/90 and 160/100: it was labile and the higher readings were associated with considerable emotional tension. She was discharged on routine treatment for epilepsy, and on February 22 reported one very mild attack.

Pathological Report

A specimen of the adrenal gland showed no definite abnormality (R. W. M. Johnson). The pathological report (Professor A. C. P. Campbell) was as follows:

"The kidney is considerably shrunken, weighing 70 g. The capsule has been stripped. The surface is pitted with coarse depressed scars, some of them brown-pigmented, and between the scars there is slight, fine granularity. The colour shows a patchy alteration of congestion and pallor.

"On dissection the renal artery at the point of resection just outside the hilum is shrunken and occluded by organized thrombosis. As the artery is traced into the hilum it becomes lost in a fibrous granuloma about 15 mm. in diameter, of pale-grey colour flecked with iron pigmentation. Similar smaller granulomata are seen in the hilum apparently on the course of the lobar branches of the renal artery. The kidney tissue is markedly pale (the specimen has apparently been a considerable time in formalin) and cortical pattern and cortico-medullary distinction are grossly blurred. The pelvis and calices are contracted; their mucosa appears normal.

Microscopical Examination

"Sections of the kidney show a uniform tubular atrophy of considerable degree throughout with a little diffuse fibrosis and considerable interstitial lymphocytic infiltration. The depressed scars noted above show a more complete atrophy and fibrosis indicative of areas of complete infarction. The generalized partial atrophy is compatible with partial infarction from a not quite complete ischaemia.

"The granuloma on the course of the renal artery shows an interesting picture. Segmental fragments of the artery and of its primary branches can be seen persisting within it. The granuloma is of tuberculoid type, showing small central micro-abscesses full of polymorphs, each surrounded by a zone of endothelioid cells and occasional Langhans's giant cells, outside which there is profuse lymphocyte and plasma-cell infiltration and extensive fibrosis. Within some of the abscesses there are collections of granular material partly

eosinophil and partly so basophil as to indicate calcification (confirmed by von Kossa's stain). These structures have the appearance of fungal colonies. They show a mixture of rather irregular rounded granular bodies with occasional mycelial strands. Some of the colonies show at the periphery a distinct margin of scalloped clubbing suggesting a radiate structure in the colony. The colonies are mainly Gram-negative, but some granules are Gram-positive. Possibly the negative Gram stain is due to degeneration; the irregular morphology of the individual bodies and the calcification indicate that the organism is in a degenerative state. It is unfortunately not possible to identify the fungus more exactly because of these degenerative changes.

"Throughout the substance of the kidney, mainly in the cortex, there are occasional microscopic granulomata of exactly similar structure. These do not appear to be necessarily related to arterial branches.

"The atrophy of the kidney is compatible with ischaemia produced by embolic blocking of the main renal artery. The fact that the atrophy so produced is in many places only partial could be explained by some degree of blood inflow through the capsular circulation and possibly via small accessory arteries. Histologically, the nature of the granuloma on the course of the renal artery and of those in the kidney substance is, I think, quite incompatible with a *Str. viridans* infection. One must, I think, conclude that there has been a fungous endocarditis with embolization of the renal artery by an infected vegetation. Whether the fungous infection was combined with a *Str. viridans* infection or whether the positive viridans blood cultures are a red herring I do not know.

"Endocarditis due to fungi of various sorts has been reported—for example, from monilia, erysipilothrix, leptothrix, histoplasmosis, sporotrichosis, blastomycosis, actinomycosis. The histology of the inflammatory reaction does not suggest actinomycosis, but could be compatible with several of the others."

Comment

Three points of interest are the relation of acute rheumatic manifestations to infection with *Str. viridans* or fungous infection, that of hypertension to the renal lesion, and the part played by the fungous infection.

Infective endocarditis preceded by acute rheumatism is recognized by clinicians (Friedberg, 1950), as is the occurrence in it of acute rheumatic cardiac disease by morbid histologists (MacIlwaine, 1947). The present view of pathologists is probably that the viridans infection establishes itself more easily because of rheumatic damage to tissues or disturbance of defence mechanisms, or both.

Hypertension related to human unilateral renal vascular disease has rarely been proved, the common lesion being pyelonephritis (Goldblatt, 1949; Pickering and Heptinstall, 1953). Extensive general surveys of cases showing renal infarction (Barney and Mintz, 1933; White and Porter, 1941; Hoxie and Coggin, 1940; Ormond, 1948) do not mention hypertension. Oppenheimer *et al.* (1939) found after 5,000 post-mortem examinations of hypertensive patients that though the pressure had been higher when one renal artery was notably damaged it was very difficult to bring evidence from observations on man on the incidence of hypertension. Cases which show hypertension due to renal vascular disease clearly relieved by nephrectomy are reported by Boyd and Lewis (1938), Leadbetter and Burkland (1938), Howard *et al.* (1940), Shea *et al.* (1948), Cornwell (1949), Owen and Pearlman (1951). The experiments of Goldblatt (1937) showed that in his experimental animals complete obstruction of the main renal vessel did not produce hypertension. In the present case renal artery obstruction was followed by only patchy atrophy, presumably due to partial ischaemia. Capsular and accessory vessels can, Professor Campbell suggests, provide considerable blood supply. In some patients with completely occluded renal vessels this would correspond to that provided by the partially occluded vessels necessary for hypertension in Goldblatt's experiments.

The unexpected finding of a granulomatous process in the removed kidney related to masses of fungus caused careful review of the case records. There is no doubt that blood culture on separate days grew a penicillin-sensitive streptococcus. Response to penicillin was, however, unsatisfactory, though further blood culture grew no bacteria. After aureomycin had been given as well for three weeks the fever was, in general, controlled, but occasional embolic signs occurred. The unusually severe symptoms and persistent fever in a penicillin-sensitive bacterial infection were probably evidence of an added fungous endocarditis.

Fungous endocarditis has been reported, mainly from the United States, twenty references (none from this country) being found between 1939 and 1952. A frequent cause is drug addiction, with intravenous administration (Pasternack, 1942), but such endocarditis has developed under antibiotic treatment of other infections (Cassels and Steiner, 1944; Geiger *et al.*, 1946; Zimmerman, 1950; Kunstadter *et al.*, 1952). Geiger and others report endocarditis with combined bacterial and fungous infection but with death. In the present case the long period of observation makes it seem that the unidentified fungous endocarditis was cured.

Summary

A case is reported of hypertension in a normotensive patient with infective endocarditis developing in the course of rheumatic fever, due to renal artery infarction and relieved for four years by nephrectomy. The infecting organisms were *Streptococcus viridans* and an unidentified fungus.

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"Many unmarried mothers do not know that they can adopt their own children," writes the General Secretary of the National Council for the Unmarried Mother and her Child in the July issue of *Marriage Guidance*. The procedure is the same as in adoption by a childless couple, and there is a probationary period before the adoption is ratified. This procedure has several advantages for the unmarried mother and for her child. There is no need to pose as "Mrs.," with the attendant difficulties when seeking work or making claims for dependants' tax relief or other legitimate and often much-needed financial help. The adoption certificate establishes the mother as her child's legal guardian in a way which needs no further clarification and is easier to explain to the world. The writer goes on to point out that it is wise for a mother who has taken this step, when questions are asked by the child, to be frank about the real relationship, and as time goes on to make it clear why the adoption was made.