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A joint meeting of the Heberden Society and the British Association of Physical Medicine was held at Buxton on July 25 and 26, 1959, to celebrate the Centenary of the Devonshire Royal Hospital.

Demonstrations

DR. J. H. BLAND, DR. F. D. GRAY, and PROF. J. H. KELLGREN (Manchester) presented an exhibition illustrating a review of 29 patients considered in 1950 to have generalized osteo-arthritis. This series will form the subject of a paper to be published in a future issue of the Annals.

MR. J. H. CREGAN (Buxton) presented a patient with rheumatoid arthritis complicated by cervical subluxation and paraplegia.

DR. R. HARRIS (Buxton) exhibited studies on the circulation of joints, using 20Na Sodium chloride.

MR. J. CHARNLEY (Manchester) demonstrated fluor (“Teflon”) prostheses for use in arthroplasty of the hip.

Papers.—The following papers were read by members of the Heberden Society and guest speakers:

DR. N. CARDOE (London): A Controlled Trial of G27 202 in Rheumatoid Arthritis. This paper and the discussion thereon appears on page 244 of this issue of the Annals.

DR. R. K. W. KUIPERS (The Hague): Adrenal Steroids in Combination with Pyrazolones in the Treatment of Rheumatoid Arthritis, Juvenile Rheumatoid Arthritis, and Ankylosing Spondylitis. These drugs were among the most common forms of treatment for rheumatoid arthritis, juvenile rheumatoid arthritis, and ankylosing spondylitis. As a result of its experiences his group had, from an early date, stressed the need to keep dosage as low as possible, controlling phenylbutazone treatment by estimation of blood levels, and controlling the adrenal atrophy of continued steroid treatment by regular doses of corticotrophin. With a view to further reductions in dosage he had investigated the possibilities of combined therapy with prednisone and phenylbutazone, given as compound tablets called “Deltabutazolidin” containing 1-25 and 50 mg. of these drugs respectively. An initial dosage of six such tablets a day was usually satisfactory and could often be reduced to three a day.

Gsell and Rechenberg (1953) had announced that, in twenty cases, after “boost” therapy with cortisone, they maintained good results with phenylbutazone. Dr. Kuipers’ trials of this combined therapy had been conducted in two parts:

1. A “non-placebo-controlled”, but carefully assessed trial in 300 patients with rheumatoid arthritis, eight with juvenile rheumatoid arthritis, and fifty with ankylosing spondylitis;

2. A “double-blind placebo-controlled” trial of tablets randomly allocated to 100 patients with rheumatoid arthritis.

Besides Deltabutazolidin or a placebo, both groups received 0-75 g. aspirin and 0-75 g. phenacetin daily. Prednisone had not been found to influence the excretion rate or blood levels of phenylbutazone. On the other hand, there was evidence from the investigations of Kersten and Staudinger (1957) and Korus, Schriefers, and Diroucherl (1956), that phenylbutazone might inhibit the inactivation of prednisone.

The results in the first (non-comparative) trial were very good, all but eight of the rheumatoid patients, including two who might have had co-existing systemic lupus erythematosus, received some benefit and none became worse. Comparatively large doses (in relation to body weight) were required to maintain improvement in children.

These good results were confirmed by the second (controlled) trial. Assessments were made according to the grades and criteria of the American Rheumatism Association (Steinbrocker, Traeger, and Batterman, 1949). Toxic side-effects were negligible; only one gastro-intestinal upset occurred (in a patient known to be liable to this) and oedema of the ankles appeared in six patients, but was controlled by interruption of treatment. The blood pictures remained within normal limits, and four patients who had previously developed skin reactions during or after treatment with phenylbutazone showed no such reactions during treatment with Deltabutazolidin.

Dr. Kuipers concluded that it was possible, as a result of treatment with Deltabutazolidin in a dosage of four to six tablets daily, to bring the rheumatic process to a quiescent phase without undesirable side-effects. He then described the use of measurements of urinary polysaccharide output (according to the method of Di Ferrante and Rich, 1956) in the objective assessment of rheumatoid arthritis. They had found the normal output for adult men was 4-8 mg. per 24 hrs (range

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2 to 6 to 9) and for women 3 to 7 mg per 24 hrs (range 1 to 8 to 5 to 6), higher figures being found in children. High outputs in patients with active rheumatic disease were reduced in parallel with the clinical findings after successful treatment. Using this test, there was some evidence that the compound Deltabutazolidin tablet was more effective than comparable amounts of its individual constituents.

REFERENCES

Discussion.—Prof. E. G. L. Bywaters (Taplow) said that Dr. G. Loewi had been studying the urinary polysaccharide output in patients at Taplow. There was great variation from person to person, and whilst salicylates might decrease the output in some patients, it appeared to increase in others. He felt that it was premature to use this test as an objective measure of disease activity. He also wondered if it was not a retrograde step to use a compound tablet instead of the individual constituents. It implied an unvarying ratio of dosage which might not accord with the varying needs of each patient.

Dr. Kuipers replied that with his method, which enabled him to estimate the 24-hr urinary mucopolysaccharide output in series with a constant pH, no great variations had been noted. At the beginning it was found that a wrong and inconsistent pH during the estimations gave rise to variations. The method was to be described in the thesis of one of his collaborators.

Dr. M. F. McMahon (Cork) said that he had tried Deltabutazolidin in 125 patients in the last 9 months. He thought it was "the best remedy we had yet". It appeared to be free from toxicity and, of fifty patients switched to it from phenylbutazone (without their knowledge), most felt and fared better.

Mr. J. Charnley (Manchester): Experimental Observations on the Lubrication of the Joints. This paper and the discussion thereon will appear in the next issue of the Annals.

Dr. J. J. de Blécourt (Groningen): Mobile Physiotherapy in the Netherlands. A special number of the British Journal of Physical Medicine had reported 3 years ago that home physiotherapy services, mostly voluntary, were reaching people who were unable to attend a hospital physiotherapy department in the towns and rural areas of Britain.

Many patients deteriorated quickly without physiotherapy, especially whilst awaiting a hospital vacancy or after discharge from hospital. In some of these the tragic consequences of inactivity could be averted by home physiotherapy. Patients with locomotor disorders were especially in need of such care. In the Netherlands doctors were keen to explore all means of fighting rheumatism, but their efforts had been limited by lack of facilities for the diagnosis and treatment of the large number of patients with chronic rheumatism. They were striving to found a number of centres for rheumatic diseases, attached to Universities or to teaching hospitals, and comprehensive home care (involving the co-operative efforts of general practitioner, nurse, almoner, and physiotherapist) was also becoming as an important part of the service, particularly in rural areas.

Before initiating such a service, Dr. de Blécourt had visited England, but had been disappointed to find only one doctor (a general practitioner near London) who had been at all enthusiastic about mobile physiotherapy, and several specialists in rheumatic diseases had been either not interested or positively against the idea. Most thought that no good could come of treatment not supervised by a specialist, and that proper physiotherapy could only be given in a specially organized department; anything less was considered a waste of time and money. Mobile physiotherapy, as seen at work in England, obviously reflected this lack of interest. It was insufficiently supervised, too much palliative treatment was given, and only a small part of the equipment available was in regular use. Despite this discouragement, a suitably organized mobile physiotherapy service seemed to have good possibilities, and such a service had been started in some areas of the Netherlands. A mobile unit consisted of a Ford van or Volkswagen with infra-red or radiant heat apparatus, wax baths, slings for exercising arms and legs, crutches, medical balls, and so on. There was no shortwave, ultra-violet, or other complicated apparatus. It was staffed by experienced and specially trained physiotherapists (male or female), under the guidance of a medical director. Any general practitioner or specialist could call on the services of the mobile unit in his area, and each request was assessed by the medical director by several established criteria, including inavailability of a private physiotherapist and inability of the patient to leave home. Hitherto this service had been used only for rheumatic patients.

In 1958 the mobile physiotherapy unit at Groningen had treated 53 patients with about twenty treatments per patient, and about 151 miles had been travelled per treatment. The cost was about £1,200 and the fees received about £600. Sickness insurance funds paid the normal fee plus a contribution for travelling expenses. Private patients paid for themselves. The Netherlands Rheumatic Association met the deficit from public subscription without Government aid. Building contractors had given over £6,000 for this purpose. Four mobile units were already in operation and others were planned.

The results had been very satisfactory. Treatment was given usually twice weekly for 6 weeks, and then once weekly for up to 2 months. The physiotherapist-in-charge regularly consulted the medical director as well as the general practitioner and district nurse in the patient's home area, in-patients being interviewed before discharge from hospital. It had often been possible to shorten the duration of stay in hospital or to prevent the patient from losing the gains made in hospital, and sometimes hospital admissions could be avoided by the use of the mobile unit.

The best results had been seen in cases of ankylosing spondylitis, rheumatoid arthritis, and periartthritis of the shoulder, the results in the cases of osteo-arthritis...
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being not so good. The service had awakened much public interest and its help had also been sought for patients with non-rheumatic conditions such as poliomyelitis.

Mobile physiotherapy could be of real help to patients, and could play a very useful part in treatment. Careful medical supervision was, of course, a necessity, and without it the mobile physiotherapy service was doomed to fail.

Discussion.—Dr. C. B. Heal (Royal Free Hospital, London), who had originated the idea of mobile physiotherapy units in England, commented that such units had proved, in practice, to have certain drawbacks:

1. Low turnover, usually less than nine patients treated per physiotherapist per day;
2. A tendency for the cases treated to be of the aged hemiplegic type;
3. Loss of the stimulus and group optimism inherent in a good physical medicine centre;
4. A tendency to keep up repetitive passive placebo treatments;
5. The resulting high cost in money and personnel.

Realizing the force of these drawbacks and the urgent need for reaching rural areas with good rehabilitation, he had devised and worked out, with Morris Motors, a well-equipped Physical Medicine Department. This was based on the known experience of mobile x-ray departments and dental surgeries, and the Slough Mobile Surgery Service. This scheme, he affirmed, had the following advantages in comparison with existing mobile physiotherapy units:

1. High turnover, comparing favourably with average Hospital Departments.
2. Predominance of full-recovery cases.
3. Group recovery stimulus provided by the unit waiting-room, usually less than one minute.
4. Possible saving in total cost by relief of the ambulance service.

Dr. C. R. L'Estrange Orme (Parkstone, Dorset) had had experience of a mobile physiotherapy unit in the remote parts of Dorset. In some villages there might be only one patient to treat and he might as well be treated in his home as in a mobile department. If fit enough, such a patient should be brought to the active atmosphere of a hospital department. There was a psychological difficulty in discontinuing home therapy in many cases and such a service offered great scope for waste of valuable effort.

Dr. L. J. Barford (Redhill) said that he had been associated with a mobile physiotherapy unit for 10 years. It consisted of two vans and two physiotherapists. It ran at a loss of £1,200 a year and had to be helped by other means. He would agree with Dr. Heal that its use did not come up to expectation and that much time could be wasted in giving long courses of ineffectual treatment.

Dr. F. S. Cooksey (Dulwich) thought that it was better to treat patients in hospital and to send the physiotherapist once or twice to their houses to make sure they were continuing satisfactorily rather than to attempt treatment.

Dr. R. Harris (Buxton) felt that a little physiotherapy was of no value. If a rheumatic patient needed treatment then he needed short courses of carefully planned active treatment, not long-drawn-out occasional passive treatment.

Prof. Kellgren said that there were disadvantages in the use of Mobile Physiotherapy Units especially in England, but that the way in which they were used in the Netherlands (with strict criteria, careful medical supervision, etc.) seemed to open possibilities and to suggest that it would be useful to study the problem further.

Dr. J. Norrie Swanston (Toronto): New Techniques of Splint-Making. "Durafon" was a new plastic material, which made ideal supportive splints for arthritic patients. Two chemicals were mixed together and then poured into a plastic envelope, on the surface of which had been drawn the design of the splint. After a few minutes, the chemicals were hard enough for the design to be cut out. The preformed splint was then applied to the body, and maintained in position with a bandage. It finally hardened a few minutes later. The final product was a light, strong, comfortable splint. It could be lined with foam plastic, coloured by incorporating pigments, and remoulded by heat, and it was also buoyant, washable, and transparent to x-rays. Successive remouldings allowed the gradual straightening of flexed joints in stages, still using the same splint. The strength of the plastic (which had a cellular consistency) allowed it to be used for many other gadgets and appliances.

Discussion.—In answer to various questions, Dr. Swanston said that the new material cost slightly more than the proprietary plaster-of-Paris bandages and would soon be available in Great Britain. Since it could be used over and over again and there were fewer overheads, it was probably more economical than plaster in the long run. It was non-sensitizing and non-toxic.

Dr. Harris (Buxton) mentioned experiments with nylon-impregnated plaster-of-Paris for wrist supports. This substance was rather slow to harden, but was washable and could be worn by patients undergoing hydrotherapy.

Dr. Swanston later demonstrated the making of a Durafon splint.

Dr. W. R. M. Alexander and Dr. J. J. R. Duthie (Edinburgh): Studies of the Anti-Nuclear Factor in Disseminated Lupus Erythematosus. Friou (1957) had reported that serum from patients suffering from disseminated lupus erythematosus (L.E.) contained a factor with an affinity for nuclei of tissue cells. His original observations had been amply confirmed by other workers, and the anti-nuclear factor (A.N.F.) had been characterized as a gamma-globulin.

Like the L.E.-cell phenomenon, A.N.F. had so far been demonstrated only in vitro and there was no evidence that nuclei of living cells were capable of reacting with it. A.N.F. was detected by the fluorescent antibody technique of Coons (1950) which, in this application, was used as follows:

An unfixed blood film or other source of leucocytes was exposed to the serum under test for 30 minutes at room temperature and, if A.N.F. was present, it combined with the nucleus of the cell. At this stage no visible change in the cell could be demonstrated. Excess serum was washed from the slide which was then exposed for 30 minutes to rabbit serum containing antibody to human globulin. This serum had previously been conjugated, by chemical means, to a fluorescent dye.
If A.N.F. was present on the nucleus it bound the fluorescent antibody and the complex could be detected in the fluorescence microscope.

This indirect, or sandwich, technique was convenient in that only one serum had to be conjugated for screening large numbers of sera for A.N.F.

It was wished, however, to study the properties of A.N.F. in a few sera and for this purpose direct conjugation of test serum and fluorochrome was more satisfactory. The film was exposed to the direct conjugate and examined as before.

The experiments Dr. Alexander described were planned to examine the possible effect of A.N.F. on leucocytes in suspension and its relationship to the L.E.-cell phenomenon. Leucocytes were prepared from heparinized blood by differential sedimentation. The plasma was removed and replaced by conjugated serum known to contain A.N.F. and to exhibit the L.E.-cell phenomenon. Cells and conjugates were incubated at 37° C. for 2 hrs, and smears were then made. When dry, the slides were washed to remove excess serum and thereafter examined by phase contrast and fluorescence microscopy. Six individual conjugates were tested and leucocytes from healthy donors and patients suffering from disseminated lupus erythematosus were used. The results were the same in all experiments. L.E. cells were present and the inclusion bodies alone were fluorescent. There was no nuclear fluorescence of intact leucocytes or L.E. cells.

These experiments showed that A.N.F. was not bound by intact leucocytes in suspension. At some point, the nuclei of cells destined to be phagocytosed must have combined with A.N.F., but whether this occurred before or after disruption could not be said. The experiments also illustrated that leucocytes in suspension—mainly living cells—differed from dead leucocytes in air-dried blood films in their reaction with A.N.F.

The effect of exposing damaged leucocytes in suspension to conjugated sera was next investigated. Damage was produced by exposure to ultra-violet light—a technique which did not cause morphological changes in the cells. Leucocytes from a healthy donor were irradiated in suspension in the donor plasma. Conjugated serum containing A.N.F. was then substituted for the plasma and incubation continued for 2 hrs. No L.E. cells were found—presumably because irradiation had rendered the cells incapable of phagocytosis—but, perhaps more surprising, nuclear fluorescence was not detected. Thus, although the cells were damaged, their nuclei did not bind A.N.F.

Preparations of leucocytes from patients suffering from disseminated lupus were similarly treated. In most experiments the results were the same as those obtained with irradiated leucocytes from healthy donors, but on two occasions a striking difference was observed. L.E. cells were found in the preparations and there was fluorescence of inclusion bodies in L.E. cells and of nuclei both of intact leucocytes and L.E. cells.

At first sight it was difficult to reconcile this result with those of the previous experiments. How might a leucocyte sufficiently damaged to combine with A.N.F. yet be capable of phagocytosing other nuclear material to form an L.E. cell? The answer, it was thought, was that these L.E. cells were formed before the leucocytes were exposed to the fluorescent sera. It had since been demonstrated that L.E. cells could indeed have been formed in the 30 minutes during which the leucocytes were being separated from the donor plasma. The subsequent uptake of fluorescent A.N.F. might be explained by postulating that the leucocytes, including the pre-formed L.E. cells, suffered some further change during their exposure to ultra-violet light in the presence of plasma containing A.N.F.

If this hypothesis could be substantiated it would imply that, in certain circumstances, damage to living cells in the presence of A.N.F. might be followed by uptake of A.N.F. If this could be demonstrated in vivo—and experiments had been begun to determine this—it would raise the status of A.N.F. from that of a serological oddity to a substance of possible importance in the pathogenesis of disease.

REFERENCES


Discussion.—PROF. E. G. L. Bywaters asked if irradiation could destroy complement which was said to be necessary for phagocytosis.

DR. H. A. Valkenberg (Leyden) agreed that complement was necessary for phagocytosis.

DR. Alexander replied that he had not tested the effect of ultra-violet irradiation on complement, but that he had a strong suspicion that complement was unlikely to survive the process of conjugation.

PROF. J. H. Kellogg (Manchester): Report of the Joint Committee of the Empire Rheumatism Council and Nuffield Foundation. Prednisolone Anaesthetics Trial. This report and the discussion thereon appears on page 173 of this number of the Annals.

DR. A. St. J. Dixon and DR. H. Duncan (Post-graduate Medical School, London): Familial Hyperuricaemic Nephropathy. A young man of 19, investigated for high blood pressure and arthritis, had been found to have gout and severe renal disease. The serum urea was 106 mg. per cent. and the uric acid 19 mg. per cent. The question whether the gout was secondary to renal failure to excrete urates, or whether this was a severe example of gouty kidney, was investigated by examining the patient’s family. Some 20 per cent. or more of first-degree relatives of a patient with gout should have a high serum uric acid, but this finding would hardly be expected in the family of a patient with primary renal disease. The family study disclosed that all the boy’s five surviving siblings and both his parents were hyperuricaemic. This was strong evidence that gouty hyperuricaemia was the primary defect, passed with 100 per cent. penetrance from parents to offspring. However, it was also disclosed that all but two members of the family (including a sister who had died) showed some degree of renal disease, and none of the others had...
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gouty arthritis, although one subsequently developed it. The sister who had died had had chronic pyelonephritis clinically, and a review of the histology of the kidneys showed some features suggestive of gout. The mother and the remaining two sisters had all had "pre-eclamptic toxaemia" and had been left with evidence of residual renal damage. Two other brothers were well, but had raised blood urea, and one had albuminuria. The father and youngest brother had no evidence of renal disease—so far.

Thus, in this family hyperuricaemia existed without renal disease, but renal disease did not exist without hyperuricaemia, and it therefore appeared that hyperuricaemia was the primary defect and renal disease the complication. Dr. Dixon had called this condition "hyperuricaemic nephropathy" and had gone on to examine other causes of hyperuricaemia to see if similar renal damage could occur.

One difficulty was the diversity of renal lesions known to accompany primary gout. Nevertheless, the association with gout was common and rose with duration of disease to 100 per cent. in an autopsy series. Experimental urate infusions both in man and in animals, were directly damaging to the kidneys. Renal damage could complicate secondary gout: examples had been seen in chronic leukaemia, polycythemia, congenital haemolytic anaemia, and myelomatosis. It also occurred in acute and chronic leukaemia and myelomatosis with hyperuricaemia but without gout. In an attempt to find out whether unsuspected hyperuricaemic nephropathy was the cause of otherwise unexplained hypertension, Dr. Duncan and Dr. Colin Dollery had studied about 100 patients attending a hypertension clinic. A large number were found to have hyperuricaemia, but this proved to be the result of the antihypertensive drugs in use. All these drugs, including chlorothiazide (and also pempidine and reserpine, not previously known to have this effect) could raise the serum uric acid without raising the blood urea.

In summary, it seemed possible that hyperuricaemia might damage the kidneys, and the concept of a hyperuricaemic nephropathy (either inherited or acquired) may explain the liability to renal disease of patients with both primary and secondary gout, and may also explain some of the renal disorders associated with blood diseases and myelomatosis. There was also a possibility that an inherited hyperuricaemia might in some patients cause otherwise unexplained renal disease, hypertension, toxaemia of pregnancy, or even symptomless albuminuria. These considerations had ceased to be of merely academic importance since potent uricosuric drugs were available to attack the underlying metabolic abnormality.

Discussion.—Dr. G. D. Kersley (Bath) asked about the effects of the uricosuric drugs on renal function in this case. He had had three cases of gout with renal disease who showed improvement in renal function with uricosuric drugs.

Dr. Dixon said that this was a case of severe renal disease. They had tried probenecid, but both the serum uric acid and the blood urea fell, but not to normal levels. One sister who had pre-eclamptic toxaemia had been given probenecid, but the effect on renal function in her case could not be assessed.

Prof. S. J. Hartfall (Leeds) asked if the progressing chronic nephritis in this case was not linked with the aetiology. With the high level of blood urea one might expect a rise in serum uric acid.

Dr. Dixon replied that, in his experience, the chronically failing kidney was not associated with high serum urate levels. In acute renal disease, in eclampsia, and in the conditions dealt with by the artificial kidney, high levels of serum uric acid were seen, but in chronic nephritis the serum urate seldom rose above 6 to 7 mg. per cent.

In reply to another question, he stated that the parents of the index case were not related.

Dr. A. Aronoff (Montreal) said that he had seen two cases of gout precipitated by the use of anti-hypertensive drugs. The first, a patient aged 53, had been given several g. nicotinic acid daily plus chlorothiazide, and had developed an acute attack of gout in the ankle with a rise in serum uric acid to 15 mg. per cent. The second patient, a woman aged 58 with carcinoma of the cervix and essential hypertension, had been started on reserpine and was later given chlorothiazide. This had a marked effect on the hypertension, but the serum uric acid rose from 5.5 to 9.3 mg. per cent. and she developed an acute attack of gout in the right hand.

Dr. Dixon replied that these appeared to be some of the first reported cases of gout caused by treatment with anti-hypertensive drugs. In his experience, chlorothiazide, mecamylamine, pempidine and reserpine had all caused hyperuricaemia, the least effect being from the last. A common pharmacological action seemed likely. In answer to Dr. Dudley Hart, he stated that the anti-hypertensive drugs did not increase the blood urea.

Prof. E. G. L. Bywaters said that it would be interesting to know more about Dr. Aronoff’s patients. He believed that the miscible pool of uric acid had increased enormously above normal before clinical gout occurred. He wondered whether patients with hypertension had been accumulating uric acid over the years, or whether they had developed gout by a different mechanism, without much deposition of uric acid in the body.

Dr. F. Françon (Aix-les-Bains): Osteo-Articular Reactions after Prostatectomy. This operation was safer than it used to be, and thus more frequently performed; post-operative complications were thus more often seen, either in the form of infective conditions, including osteitis which varied in severity from mild inflammation to frank suppuration, or in the form of gout.

Among the infective complications, osteitis pubis was the most common. It appeared 1 or 2 months after surgery, especially if the Terence Millin suprapubic approach had been used, and if the patient had had an infected urine. There was pubic and ischial pain, and slight fever (to 100° F.). Radiographic changes usually (but not always) followed, and appeared as bone condensation, or more rarely as rarefaction and erosion. Synostosis was exceptional. The lesion took from 4 to 6 months to heal, less if a retropubic abscess could be found and evacuated, more if the infection were complicated. Nelson (1957) had managed to avoid osteitis...
pubis in 410 prostatectomies by taking special precautions to prevent infected retropubic haematoma. In rare cases the intravertebral disks (about 3 months post-operatively), the hips (about 5 months post-operatively), and the tarsus could be involved by the same process. An example of the latter occurring 10 days after prostatectomy was cited. *Escherichia coli, Staphylococcus aureus*, and *Pseudomonas aeruginosa* were the organisms most frequently implicated.

Gout had also been seen following prostatectomy. An example was cited of a man aged 70 (with a previous history of chronic tophaceous gout) who had had a most severe attack of acute gouty arthritis in the knee 13 days after operation, which signally failed to respond to colchicine.

The following papers were read by members of the British Association of Physical Medicine and guest speakers:

**BOOK REVIEW**


This volume, which comes from the University of Barcelona, will bring back memories to those who served in the Mediterranean campaigns of the last war. Brucellosis is still endemic in Spain, and the author was able to collect 174 cases between 1927 and 1955. The ten chapters, each with an English summary, cover all the likely osteo-articular complications of the disease, and illustrate how it may mimic a variety of rheumatic affections. The diagnosis was confirmed by blood culture in 58 per cent. of cases, but serum agglutination was positive in 98 per cent. and complement fixation in 94 per cent. The erythrocyte sedimentation rate is frequently normal.

To the British reader it will appeal as a work of reference.

David Preiskel

**SOCIETÀ ITALIANA DI REUMATOLOGIA**

*Acqui Prizes, 1959*

The result of the Fourth Acqui International Prize Competition, for which 28 entries of great scientific merit were received, was announced on September 20, 1959.

The Prizes were presented by Senator Piola, the Financial Under-Secretary, at the end of a scientific session at which papers were given by the foreign members of the panel of judges, Dr. J. Forestier and Prof. P. P. Ravault.

The other members of the adjudicating committee were:

- Prof. L. Antoggetti, Director of the Medical Clinic of the University of Geneva;
- Prof. G. C. Dogliotti, Director of the Medical Clinic of the University of Turin;
- Prof. F. Quaglia, Representative of the Medicinal Springs of Acqui-Terme;
- Prof. A. Robecchi, Vice-President of the European League against Rheumatism;
- Prof. L. Villa, President of the Italian Society against Rheumatism and Director of the Medical Clinic of Milan.

The prize of 1,200,000 lire for original and unpublished work was divided as follows:

- G. Sala, G. Ratti, and E. Cirla (Medical Clinic, Milan): Researches on the Metabolism of Uric Acid in Normal and Gouty Subjects (500,000 lire).
- R. Garelli (Rheumatism Centre, Turin): Histological Study of the Structural Changes of the Joint Capsule occurring during Adolescence and Old Age (300,000 lire).
- A. Marmont (Medical Clinic, Gênes): Critical Review of the Neurovegetative Theory of the Pathogenesis of Scleroderma. Analysis of the Significance of the New Serum Protein Factors (Rheumatoid Factor, L.E. Factor, etc.). (300,000 lire).

The prize of 500,000 lire for a monograph published during the year 1958 was awarded to the book entitled *The Shoulder in Rheumatological Practice*, by S. de Sèze, A. Ryckewaert, and M. Maître (Hôpital Lariboisière, Paris).