
ARTHRITIS

ARTHRITIS

Mandy Wyles
Research Associate, Office of Health Economics

Office of Health Economics
12 Whitehall London SW1A 2DY

No 104 in a series of papers on current health problems published by the Office of Health Economics. Copies are available at £3.00.
For previous papers see page 51.

Cover illustration: courtesy of Mary Evans Picture Library.

© March 1992. Office of Health Economics.

ISSN 0473 8837

INTRODUCTION

Arthritis is a common and chronic disease with over 200 conditions included in this category. It affects every tenth inhabitant of the world according to the World Health Organisation (Novosti Press Agency, 1990). Indeed, arthritis and rheumatism are the most frequent self reported condition in Great Britain with a rate of 80 per 1000 females and 40 per 1000 in males (OPCS, 1989). The Arthritis and Rheumatism Council claim a total figure in the UK of about 20 million people with some form of arthritic disorder, with between six and eight million significantly affected. Yet, it receives little public or media attention, since it rarely causes death and is often considered solely a disease of the elderly. However, 'arthritis is no respecter of age, sex, race, colour or creed' (Dieppe, 1988) – although the prevalence does increase with age (see p17) – and is the major cause of disability in this country (OPCS, 1988).

This paper will show that the morbidity of arthritis results in substantial costs to society and to the individual. It is estimated that the cost of arthritis to the National Health Service (NHS) in 1989 was nearly £500 million (see Table 11). This figure includes hospital costs of £231 million and general practice costs of £45 million. In addition pharmaceutical services absorbed £219 million, nearly one tenth of the total cost of pharmaceutical services in 1989. Furthermore, the total cost to the NHS is set to rise as the number of very elderly (over 75 years old) increases, causing costs to increase by some 14 per cent by the year 2001. However, this figure does not take account of the personal and indirect costs such as loss of earnings; arthritis accounts for nearly 11 per cent of all working days lost. The Arthritis Foundation (1982) in the United States included such areas in its calculation of the total cost of arthritis and arrived at a figure of $13.5 billion in 1982, or about £12,000 million at 1990 prices[1]. If it is assumed that the population of the United Kingdom is about one-fifth of the USA and it is conservatively estimated that the cost to the UK is only a half of that in the USA per patient it can be estimated that the total cost of arthritis in 1990 in the UK was £1,200 million. Therefore 'this expensive and chronic condition should be given far higher priority' (Agnew, 1991).

Historical Aspects

Arthritis is an ancient condition which has been identified in skeletal remains dating back to before the Egyptian pyramids were built. The first clear description of 'gouty arthritis' was provided by

1 The cost is based on the Arthritis Foundations estimates, inflated from 1982 prices to 1990 prices and converted to sterling using the mean exchange rate for 1990 of $1.79:£1.

Hippocrates (460-377 BC), although other allied conditions are not easily recognisable in the early medical texts. Throughout history arthritis has been subject to much misunderstanding as attempts to define the various diseases within this category remained vague and confusing until the turn of the nineteenth century. Past treatments have derived from views held on the causes of the diseases.

In 1000 BC Hindus in India believed that inflammatory arthritis was a deep organic malfunction. Treatment involved cleansing the body in addition to counter-irritation methods including liniments, leeches, bleeding of veins and cauterisation. Burning the inflamed area of the body remained a popular procedure for many centuries.

Hippocrates believed inflammatory arthritis was due to retained body poisons and could be relieved by draining body fluids through the skin. It was thought that all diseases were due to an improper mixture of the body fluids (blood, bile, phlegm and black bile), therefore the presumed disease invoking factors would be removed by bleeding. Draining and repeated bleedings was also common during the eighteenth and nineteenth centuries.

The treatment of rheumatoid arthritis (RA) with gold has continued to be used since its introduction by Forestier in 1928. Its orginal use was based on the premise that RA was related to tuberculosis and that gold had an anti-tubercular effect. Gold therapy was soon abandoned in tuberculosis as entirely ineffective but has remained a commonly used treatment in rheumatoid arthritis.

In the early part of this century medical remedies centred on the belief that some forms of arthritis were infectious. Thus the 'infected' parts were removed, these included teeth, tonsils and appendix. Other supposed 'cures' are mere superstition, advising people with arthritis to cover themselves in horse manure, vomit by tickling the throat with peacock feathers, carry potatoes, chestnuts, nutmegs, wear copper bracelets or even lying on the ground whilst a large brown bear tramples on their spine.

Some forms of arthritis can be cured such as septic arthritis, whilst gout can be completely controlled by medication. Unfortunately for the most common forms of arthritis there is, as yet, no cure, although the consequences of these diseases can be minimised by medication in conjunction with the care of health professionals. In appropriate cases of arthritis surgery on the affected joints to either remove the synovium (common in rheumatoid arthritis) or a total joint replacement (in osteoarthritis or rheumatoid arthritis) offers an improved quality of life by increasing mobility and reducing pain. Moreover hip replacements are one of the most cost effective interventions (see p37). Technological innovation, particularly in replacement joint materials, has enabled for example hip, knees, fingers and wrist joints to be successfully replaced. In spite of effective

treatment many individuals with arthritis still buy and use remedies of unproven benefit. The therapeutic options are discussed more fully in a later section of this paper.

The causes of most forms of arthritis are still a mystery, although it is generally accepted that a number of factors exert an influence. Factors such as genetics, infection and the use and abuse of the joints are regarded as possible causes and are considered in this paper.

THE TYPES AND CAUSES OF ARTHRITIS

Confusion between the various rheumatic diseases can occur due to vague and inconsistent terminology. Since there are over 200 diseases within this category the term arthritis is frequently used, although it literally means inflammation of the joints. Box 1 provides a brief description of the types of arthritis considered in this paper.

There is a distinct lack knowledge about the causes of arthritis – with the exception of gout – in spite of over 100 years of intensive research. Recent research has led to interesting observations concerning the possible causes of rheumatoid arthritis and ankylosing spondylitis (see Infection), however cures are unlikely until the causes are identified.

Genetic

Some types of arthritis show an hereditary predisposition. Whilst it is not possible to generalise, studies have reported that first degree relatives of patients with rheumatoid arthritis have an increased likelihood of developing the disease of about five per cent, the risk for an identical twin rises to some 30 per cent if the other twin has arthritis.

Studies show that individuals with a genetic susceptibility to arthritis often possess a specific combination of antigens. Rheumatoid arthritis (RA) is associated with the gene HLA-DR4[2] whilst HLA-B27 is associated with ankylosing spondylitis (AS). Indeed, studies show that 90 per cent of individuals with AS possess HLA-B27 whilst the incidence is only six to eight per cent in the general Caucasian population (Arnett, 1989). However, not having the gene does not guarantee immunity from these diseases.

Infection

Since Forestier observed that some of his patients with acute tuberculosis developed inflammatory arthritis nearly a century ago, there have been reports on finding bacteria in affected joints. This idea has been supported by the identification of the bacterium, Borrelia burgdorferi, as the cause of Lyme arthritis[3].

Practically every kind of bacterium is able to produce joint problems (Barnard et al, 1984), although the relationship can be complex. For example, reactive arthritis is a delayed response to infection elsewhere in the body, often in association with enteritis

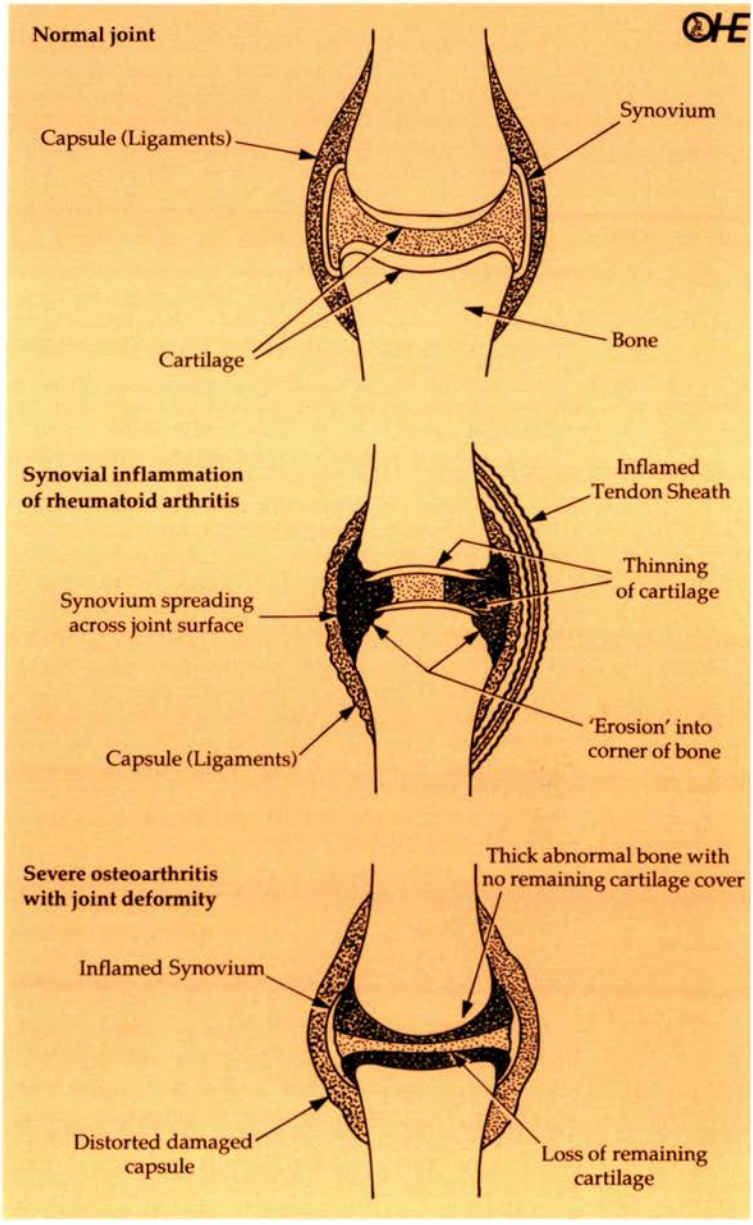
2 HLA antigens are antigens which are inherited on the surfaces of cells throughout the body. They are know as Human Leucocyte Antigens (HLA) because they were first identified on leucocytes. B27 and DR4 are antigen markers.

3 Lyme arthritis was first diagnosed in Lyme, Connecticut, USA by Dr Steere in the 1970's.

BOX 1 **A summary of the common forms of arthritis**

Conditions	*Characteristics*
Osteoarthritis (OA)	OA is a condition where the cartilage covering the ends of bone becomes roughened, then thins and wears away. The bone thickens and changes shape, and 'outgrowths' form at the outer edges of joint. The lining membranes, synovial and capsule, also thicken becoming mildly inflamed particularly in the advanced stages of the condition. When the joint is severely damaged it may become misshaped and unstable, putting stress on the ligaments and other tissues outside the joint and damaging them as well (see Figure 1). Women are affected more than men (3:2). The term secondary OA describes changes resulting from a defined predisposing factor such as injury. About ten per cent of OA is identified as secondary. The most serious problems of OA involve weight bearing joints, particularly the hip and knee.
Rheumatoid Arthritis (RA)	RA is an inflammatory disease occurring more often in females (3:1), except in the elderly where incidence is equal. The synovial becomes inflamed. Fluid and cells leak out of the inflamed membrane and gradually eat in to the cartilage and bone. The membrane becomes thicker and spreads in to the joint. Eventually the bone may begin to wear away. The whole joint, tendons and ligaments may become weakened, damaged and changed in shape (see Figure 1). A characteristic onset of RA is in the wrists, hands or feet, but may progress to any joint in the body.
Juvenile Chronic Arthritis (JCA)	JCA is a term used to describe pain and swelling in one or more joints which persists for longer than three months in under 16 years old's. The most common form is termed pauci-articular arthritis which affects a few joints and continues for several years. Another kind is termed polyarthritis which can cause severe and widespread joint damage. The third, systemic (formerly Still's) disease, causes not just inflamed joints but also fever and rashes.
Gout	Gout is caused by the disposition of urate crystals in tissues and joints. Uric acid occurs naturally in blood, if insufficient is excreted in urine or too much produced, hyperuricemia occurs and crystals form. Accumulations of crystals can erode the joints, bones and periarticular structures causing frictional impairment. Classic gout attacks the base of the big toe but can affect other joints in the feet, ankles, knees, hands or wrists. It is more common among men (20:1).
Ankylosing Spondylitis (AS)	'Ankylosing' means stiffening whilst the definition of 'spondylitis' is inflammation of the spine. Inflammation in the joints between the vertebrae causes scar tissue to form, thereby stiffening the joint. The scar tissue may eventually turn to bone filling the space between the vertebrae, causing the joint to stiffen totally. AS usually affects the joints of the back and hips and occasionally the shoulders, knees and ankles.

Figure 1 **Affects of arthritis on the joints**



Source: ARC

BOX 1 *Continued*

Psoriatic Arthritis (PA)	PA is a distinct systemic disease in which psoriasis is associated with inflammatory arthritis, somewhat resembling RA but tending to be less symmetrical. Skin lesions are usually present before the arthritis appears. The arthritis can range from mild to severely erosive, affecting single or multiple peripheral joints as well as the spine.
Systemic Lupus Erythematosus (SLE)	SLE is a disease where the antibodies in the blood begin to attack the body instead of defending against infection, thereby causing inflammation. Almost any organ or tissue may be affected. SLE is more common among females (10:1) and can start with an acute or mild attack of joint pain.

Source: Dieppe, 1988; Golding, 1989; McCarty, 1989; ARC & Arthritis Care booklets.

caused by salmonella and certain shigella strains. Septic arthritis is caused by infection of the joint cavity of certain bacteria including streptococcus, whilst arthritis can be due also to a direct local infection or a by-product of a major infection such as syphilis and gonorrhoea. All infections need to be dealt with quickly to avoid permanent disability.

Recent research has suggested that a common gut bacterium, Proteus mirabilis, may be involved in the development of RA and Klebsiella pneumoniae possibly causing AS (Ebringer, 1991). If these results are confirmed by further studies antibiotics may be considered as an effective treatment for these diseases.

A genetic factor may influence whether the bacterial infection causes rheumatic problems, or is involved in autoimmunity (see below).

Immune System

It has been known for half a century that the immune system can attack the body's own tissues; a process termed autoimmunity. Normally an individual produces antibodies when foreign bodies such as bacteria invade the body, if autoimmunity occurs an individual develops antibodies to the body's own tissues.

However, it is unknown why or how such autoimmune destruction occurs. One hypothesis is that the immune system may be unable to distinguish between proteins on bacteria and so called stress (or heat shock) proteins[4]. Therefore when the body is fighting a bacterial

4 Stress proteins are essential for survival and are detectable in all cells after injury. Normally, they are present in low levels but when the cells are subjected to ultraviolet light, heat, chemicals, changes in pH or even alcohol, cells produce large quantities of them for protection.

infection some immune response may be misdirected to the body's own stress proteins (Clayton, 1991).

There is an element of autoimmunity in RA and other rarer forms of arthritis.

Use and Abuse of joints

Injuries to the joints can lead to arthritis. Indeed some studies show that occupational injury is an important influence on the distribution and severity of osteoarthritis (Cobb, 1971; Lawrence, 1977; Kellgren et al, 1952). Certain groups of workers such as miners and dockyard workers have a far greater rate of osteoarthritis (OA) in all age groups than groups of workers such as civil servants (OHE, 1973). New technology may also predispose an individual to rheumatic disorders, since new technology 'has led to the need for rapid, repetitive movements often limited to the upper arms or even wrists alone' (Bird, 1991).

There has been debate concerning the effect of sport on the development of osteoarthritis. Some studies have shown that competitive sports such as running and football do not render a person liable to OA of hips or ankles (Puranen et al, 1975; Adams, 1979). Conversely, Murray-Leslie et al (1977) found that 41 per cent of veteran parachutists (50-70 years) had signs of OA whilst non sportsmen (55-64 years) had a rate of only 30 per cent (Kellgren et al, 1958). Although recent research by Felson et al (1989) has concluded that prior injury to the joint may result in the later development of OA, but without prior injury most sports activity posed little risk.

Other factors

These factors are even less well understood than the other possible causes of arthritis. Instead of a single factor causing particular types of arthritis, various unrelated factors may be involved. The relevant factors may vary in different individuals, in different types of arthritis and in different social circumstances. In studies (Beighton et al, 1975; Solomon et al, 1975ab) of similar South African black populations the prevalence of RA was low in rural areas (0.87 per cent) but comparable to industrial countries in urban areas (3.3 per cent). This suggests that urban environmental factors, as yet unidentified, influence the frequency and severity of arthritis (Brewerton, 1988).

Some factors should be considered as influences on the pain endured rather than possible causes. The most common influences include the weather, diet, stress and exercise. Changes in the weather may affect perceptions of pain and stiffness. In general, cold damp climates seem to increase pain compared to warm climates.

Occasionally an allergic reaction to food can cause inflammation of the joints or aggravate some forms of arthritis, although this is

relatively rare. Being overweight may augment the pain since more pressure is put on the joints. Moreover, Dieppe (1991) argues that obesity has a strong association with knee and hand osteoarthritis. Psychological influences such as stress may affect the symptoms, such as the pain threshold, but there is no evidence that stress causes arthritis.

A hormonal role has been suspected since the 1950's when Dr Hench observed that women patients with RA often had improvements in their symptoms during pregnancy (exacerbations are possible, but rare) whilst onset and accentuation of symptoms can occur post natally or at menopause. Women with SLE not adequately controlled by medication may find the disease exacerbated by pregnancy, but exacerbations and foetal loss are rare if medication is effective. Changes in hormonal states are an important factor, but little is known of the mechanisms which causes the aggravation or precipitation of rheumatic symptoms.

Arthritis has no single underlying cause rather a number of contributing factors. Recently, research has led to important observations concerning the possible causes of RA, nevertheless more research is necessary to increase understanding in all rheumatic diseases which may eventually lead to effective treatments.

DIAGNOSIS

Early, and accurate, diagnosis of arthritis and allied conditions is important since the long term prognosis of the disease may be influenced by the prompt initiation of appropriate therapy. The diagnosis is usually made by the general practitioner (GP) or rheumatologist.

The diagnosis of rheumatoid arthritis is based first on establishing whether or not the joints are inflamed, secondly on documenting the pattern of joints involved and whether the involvement is symmetrical, and thirdly on the results of various blood tests and X-rays. The family history and past medical history are also essential parts of the diagnostic information.

The diagnosis of osteoarthritis (OA) and juvenile chronic arthritis (JCA) is aided by the criteria indicated in Box 2, in addition to the criteria generally used in large population studies for rheumatoid arthritis (RA). Once the diagnosis is established the extent of the disease can be determined by the ability of the patient to perform daily activities, examination and X-rays whilst the progression can be monitored using radiographic findings.

Some non specialists cannot easily distinguish between non inflammatory musculoskeletal disorders (Wright et al, 1979), moreover, newly qualified doctors rarely examine the joints and so miss many conditions that are likely to have an impact on patients morbidity (Doherty et al, 1990). Therefore it would seem prudent for GPs to refer more doubtful cases to specialists.

Clinical diagnosis is modified by laboratory and radiographic findings. The blood can be tested for the erythrocyte sedimentation rate (ESR) which indicates the presence and extent of inflammation, in addition it can be tested for a substance called 'rheumatoid factor' (RF). RF is detectable in up to 80 per cent of individuals with rheumatoid arthritis (RA) but only about five per cent of the 'normal' population (Dieppe, 1988). X-rays may show erosion of the bones at the edge of the affected joints and disease progression.

Osteoarthritis (OA) may be confused with other forms of arthritis because pain, stiffness and limitation of movement are common features of all these related disorders. Differential diagnosis is further complicated by the high radiographic prevalence of OA in the general population that often bears no relation to the disease of a given patient. Diagnosis is simple in most cases but atypical disease presentation and behaviour may require different diagnostic considerations. History, examination and X-rays play a central role both in the diagnosis and in the assessment of the severity of osteoarthritis whilst blood tests are usually normal.

The lack of familiarity of GP's with juvenile chronic arthritis may lead to some missed or incorrectly diagnosed cases, therefore doctors

BOX 2 **Rheumatoid Arthritis Diagnostic ‡ Criteria (ARA 1958 Revision)**

1. Morning stiffness.
2. Pain on motion or tenderness in at least one joint.*
3. Swelling (soft tissue thickening or fluid, not bony outgrowth alone) in at least one joint.*
4. Swelling in at least one other joint.*†
5. Symmetric joint swelling with simultaneous involvement of the same joint on both sides of the body.*† Terminal phalangeal joint involvement does not satisfy the criterion.
6. Subcutaneous nodules over bony prominences, on extensor surfaces or in juxta-articular regions.*
7. Radiographic changes typical of rheumatoid arthritis (which must include at least bony decalcification localized to or greatest around the involved joints and not just degenerative changes).†
8. Positive agglutination (anti-gamma globulin) test.†
9. Poor mucin precipitate from synovial fluid (with shreds and cloudy solution).
10. Characteristic histologic changes in synovial membrane.†
11. Characteristic histologic changes in nodules.†

Categories‡	*No. of criteria required*	*Minimum duration of continuous symptoms*	*Exclusion***
Classic	7 of 11	6 weeks (nos 1-5)	any of listed
Definite	5 of 11	6 weeks (nos 1-5)	any of listed
Probable	3 of 11	3 weeks (one of nos 1-5)	any of listed

* Observed by physician
† Refer to original references for further specification
** Refer to original references for listing of exclusions
‡ Criteria mainly used in large studies

Source: Ropes et al, 1957; Ropes et al, 1959.

1987 Revised American College of Rheumatology criteria for RA

Criterion No.	*Criterion description*
1	Morning stiffness of at least 1 hour's duration
2	Arthritis of at least 3 joint groups with soft tissue swelling or fluid observed by physician
3	Arthritis involving at least one of the following joint groups: proximal interphalangeal, metacarpophalangeal and wrists
4	Symmetrical arthritis
5	Subcutaneous nodules
6	Positive rheumatoid factor test
7	Radiographic changes typical of rheumatoid arthritis

Criterion numbers 1 through 4 must be present for at least 6 weeks duration.

Source: Adapted from Arnett et al, 1988.

BOX 2 *Continued*

Grading System for Radiological Osteoarthritis

Grade 0	None	No features of OA
1	Doubtful	Minute osteophyte, doubtful significance
2	Minimal	Definite osteophyte, unimpaired joint space
3	Moderate	Moderate diminution of joint space
4	Severe	Joint space greatly impaired with sclerosis of subchondral bone

Source: Kellgren et al, 1963.

Diagnostic Criteria for Juvenile Chronic Arthritis

Ansell & Bywaters, 1959	*Eular WHO, 1977*	*ARA, 1977*
1. Age of onset before 16 years	1. Age of onset less than 16 years	1. Age of onset less than 16 years
2. Involvement of 4 or more joints (minimum period of 3 months) with either pain and swelling, pain and limitation of movement, or limitation and swelling	2. Duration of arthritis for 3 months	2. Arthritis in 1 or more joints
	3. Classification by onset: systematic, polyarticular or pauciarticular	3. Duration of disease 6 weeks
3. If fewer than 4 joints involved, biopsy of synovial membrane should show histological changes compatible with RA	4. Exclude other diseases as far as possible up to 1 year from onset of symptoms	4. Type of onset of disease during first 6 months classified as polyarticular (5 joints or more), pauciarticular (4 joints or fewer) or systematic disease (arthritis & intermittent fever)
4. Exclusion of other diseases		5. Exclusion of other forms of juvenile arthritis

Source: Bywaters, 1968; Wood, 1978; Brewer et al, 1977.

should ensure that they take an adequate case history from either child or parent in addition to a musculoskeletal examination.

The cost of diagnosis in terms of GP's time is sizeable. An estimated 835,645 new cases were presented to GP's in 1989 (OHE, 1989) at a cost of £8.6 million[5].

5 The proportion of new cases specifically for osteoarthritis and allied conditions, gout, rheumatoid arthritis and allied conditions was applied to the total expenditure on the General Medical Services in the UK in 1989 (OHE, 1989).

Outcome

The clinical features of the various types of arthritis are familiar, yet the course of individual cases is unpredictable. Many forms of arthritis do get better on their own, given time – this is termed natural remission and has nothing to do with treatment. According to several studies (Ustinger et al, 1985) the relationship between disease duration and outcome largely reflects the natural history of the disease. The natural history can be observed by examining patients in the early stages of the disease and following the progress of the disease. However, many studies have based their conclusions on patients with an established condition, thus presenting an unfavourable view of arthritis by omitting patients who have the disease only mildly or who have gone in to early remission (Jacoby et al, 1973). Moreover, many of these reports appeared before the availability of 'remission inducing' (also known as second line, long acting, disease modifying) medication used for rheumatoid arthritis and psoriatic arthritis which may alter the relationship observed between disease duration and outcome. Generally, the longer arthritis remains active the more likely it is to cause severe difficulties.

In the early stages of RA – particularly the first two years – there is a high incidence of sustained remission. It is estimated that between 20 and 40 per cent of all people diagnosed with RA have only one or two episodes of arthritis and then go in to 'spontaneous' remission[6] (Melvin, 1989). Some clinicians (Harris, 1985) report that the incidence of spontaneous remission may be as high as 80 per cent when diagnosis is made early, that is within the first two months of the disease. In addition to spontaneous remission stronger anti-rheumatic medications (for example, gold, salazopyrine, penicillamine and azathioprine) may induce a nearly complete remission of symptoms. Therefore, early treatment and therapy with monitoring for potential side effects may be effective in preventing deformities or limitations.

Remission can last for several years or may be indefinite. Indeed, the prognosis for the majority of patients with RA is good. About 20 to 30 per cent have a variable or intermittent course of RA with alternating periods of remission and exacerbation, for others the disease is slowly or rapidly progressive. Whilst a small minority (three to five per cent) have a serious progressive disease which is not responsive to current medication (Harris, 1985). It has been recognised that pregnancy may induce remission (see Page 11).

The prognosis for osteoarthritis is generally more favourable as it may be confined to a single joint and usually only affects a few joints.

6 Spontaneous remission is a term used when the reason for remission is not understood. This can occur in all major rheumatic diseases.

It is not necessarily progressive and often improves. The greatest disability occurs in the latter stages of the disease when weight bearing joints are affected.

Juvenile chronic arthritis includes several different subsets which have different prognoses. Generally, the outlook for children is rather better than for adults, since the majority will have complete remission and around 70-80 per cent will regain 'normal' function, whilst mortality is between two to seven per cent (Calabro, 1985; Ansell et al, 1976; Ansell et al, 1983).

It is important to have an accurate diagnosis since the various diseases require different treatment regimes. Indeed, diagnosis should be established beyond doubt before a patient is committed to long term treatment.

EPIDEMIOLOGY

The epidemiology of arthritis is complex since it encompasses so many differing, yet allied conditions. Arthritis is common, with some 20 million people believed to be affected in Great Britain (Khaligh et al, 1986). More accurate statistics concerning the number of people with arthritis in this country will become available following the completion of the Arthritis and Rheumatism Council's survey in Norfolk. This study is the first in the world to include all individuals visiting their GP and not solely those referred to hospital due to their arthritis.

Arthritis and rheumatism is the most frequent self reported condition in Great Britain (OPCS, 1989). It is mentioned by 80 per 1000 females compared to only 34 and 33 per 1000 for the next most common categories of asthma and hypertension. It is also the top category among males, but with a considerably lower rate of 40 per 1000. The rate increases with age in both sexes, rising in females from 25 per 1000 aged 16-44 years to 282 per 1000 aged 75 and over.

A more accurate picture can be formed if the various diseases are considered separately. Table 1 indicates the number of individuals with various types of arthritis in the United Kingdom. Whilst it is not possible to appraise all forms of arthritis in detail, a few of the more common forms have been selected, namely rheumatoid arthritis, juvenile chronic arthritis and osteoarthritis.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is common in all parts of the world. In the United Kingdom as many as two to three per 100 people (ARC Handbook) have some evidence of the disease, although in many

Table 1 **Number of individuals with arthritis in United Kingdom in 1990**

Type	*Percent*	*Number*
Osteoarthritis	9%	(2.35%*) 5,163,840 (1,348,366)
Rheumatoid Arthritis	4.3%	(0.56%*) 2,467,168 (321,305)
Gout	0.27%*	154,915
Ankylosing Spondylitis	0.1%	57,376
Systematic Lupus Erythematosus	0.0065%*	3,729
Psoriatic Arthritis	0.1%	57,376
Juvenile Chronic Arthritis	0.065%	7,111
TOTAL		7,911,515

*These rates are from the OPCS (1986) morbidity data and for a variety of reasons may underestimate the rates. However, the rates for RA, OA and gout are used in the costings since it is these individuals who will probably use most of the NHS resources.

cases it may be extremely mild. To ensure comparability between RA studies authors may utilise a modified version of the American Rheumatism Association (ARA) criteria (see p13). When studies use different methodology, definition or severity the data cannot be compared.

Incidence[7]

Only a few RA incidence studies exist. This may be due to the large populations required to derive the number of new cases, and/or the recognition and definition of RA may be difficult at its onset.

Table 2 **Average annual incidence rates for rheumatoid arthritis (RA)**

Place and type of rate	*Rate per 1000 population*
Rochester, Minnesota	
Probable and definite RA in adults	
Males	0.43**
Females	0.85**
Rotterdam, Netherlands	
Probable and definite RA in adults	0.87*
Hiroshima & Nagasaki, Japan (1958-64)	
Definite RA in adults	
Males	0.17**
Females	0.59**
Hiroshima & Nagasaki, Japan (1965-67)	
Definite RA in adults	
Males	0.38**
Females	1.05**
Sudbury, Massachusetts	
Probable and definite RA in adults	2.90±1.60*
Oregon†	
RA as diagnosed by clinician-patients 40-49 years	
Males	1.00
Females	1.5

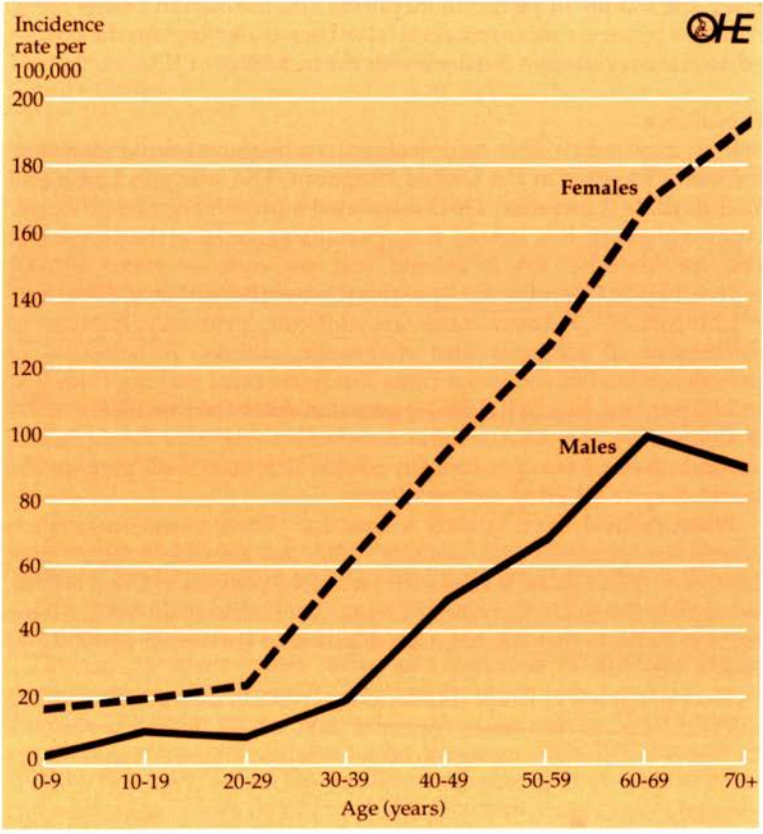
* Crude rates
** Age-adjusted to 1960 US white population
† No criteria applied in case selection.
Source: Adapted from Linos et al, 1980.

7 There is some confusion over the terms incidence and prevalence: incidence refers to the number of new cases within a given population in a given time (usually a year), prevalence is the number of cases present in a given population at a given time.

Studies in the UK have failed to produce a reasonable estimate of the incidence of RA, although studies in other countries (see Table 2) show age adjusted estimates ranging from 0.17 to 1.05 per 1000. The only study to use the American Rheumatism Association classification of 'probable' and 'definite' RA reported a rate of 67 per 100,000 (Linos et al, 1980). All the studies found a higher rate among females, but the rate rises with age in both sexes (see Figure 2).

An interesting suggestion is that the incidence of RA has fallen over time. Incidence rates decreased substantially from 1964 to 1974 in the Rochester study (Linos et al, 1980). Whilst the female rate fell substantially over the period, the male rate remained relatively stable. This corresponds to the results of a recent Seattle study (Dugowson et

Figure 2 **Incidence rate of RA per 100,000 Rochester, Minnesota, 1950–1974**



Source: Linos, 1980

al, 1989) and data from the United Kingdom (Silman, 1983). The Royal College of General Practitioners (Wingrave, 1978) reported the incidence of RA among women who used oral contraceptives was about 50 per cent less than non users. This directly supports the trend in Rochester as the decline in incidence occurred in the late 1960's and 1970's after the introduction of oral contraceptives and the more widespread use of oestrogen in postmenopausal women. However, a later study conducted in Rochester of current or prior use of oestrogen in women with and without RA showed no differences (Del Junco et al, 1985). This has lead Masi et al (1989) to suggest that a sex related host factor determines the onset and severity of RA, with males possessing a protective factor that may be lost at older ages; although this is not widely recognised. An alternative explanation has argued that RA is an autoimmune condition related to streptococcal infection. This infection attacks the joints and heart valves in acute rheumatic fever, the kidney in glomerulonephritis and the skin in scarlet fever. All these hypersensitive reactions have been in decline[8] for the past 25 years and may explain the decline in the incidence of RA.

Prevalence

Statistics are scarce since no prevalence studies have been undertaken for some 20 years in the United Kingdom. The study in Leigh and Wensleydale (Lawrence, 1961) calculated a prevalence rate of 1.1 per cent for 'definite' RA. Indeed, it is generally agreed that the prevalence rate for 'definite' RA is around one per cent, or about 500,000 individuals in Great Britain have this disease (Banard et al, 1984).

Comparisons between rates are difficult, primarily because of differences in methods and diagnostic criteria. A selection of prevalence studies shown in Table 3 indicate rates varying from 0.35 to 2.08 per cent. Mitchell (1985) argues that when the prevalence of RA is defined by the ARA criteria for classical/definite RA a rate of around one and two per cent (in adults) is found in all parts of the world despite differing methodologies.

When defined more broadly – American Rheumatism Association criteria for 'probable' and 'definite' – RA has a variably higher rate. Lawrence (1961) found a combined rate of 4.3 per cent in Great Britain, lower than the Rochester rate of 7.3 per cent (Linos et al, 1980). All the surveys indicate that RA has a predilection to females of about 2.5 to one.

The prevalence of RA at different ages reflects the age of onset, the duration before remission or in a few cases death. Prevalence increases with advancing age in both sexes (see Figure 3) but is higher

8 There is some evidence of a resurgence of these conditions in the United States associated with a resistance to antibodies.

Table 3 **Studies of prevalence data in RA**

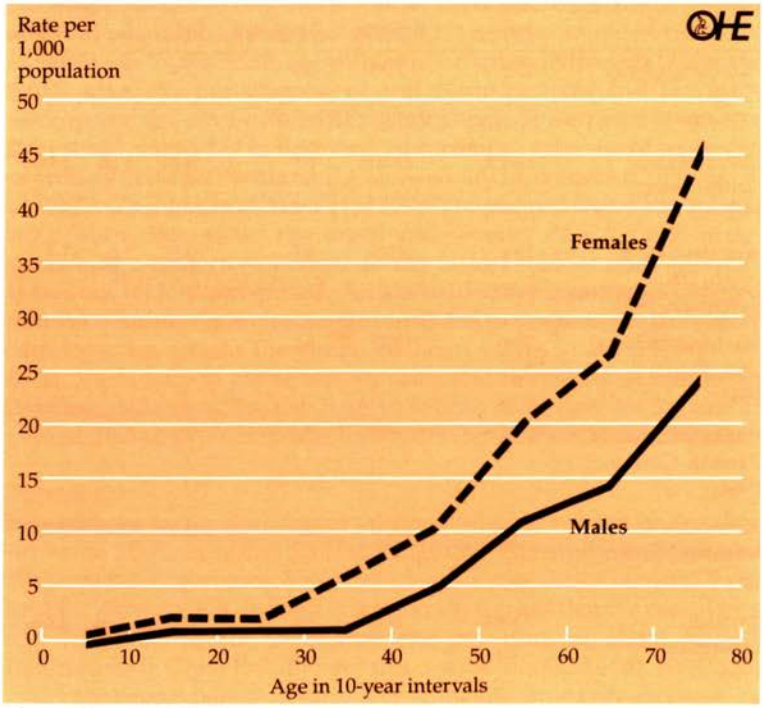
Study	*Patient age*	*No. of patients*	*% examined*	*Prevalence rate (%)* *M*	*F*	*Total*
Pittsburgh, USA Cobb. 1953-54	15+	798	60	0.4	1.0	0.7
USA National Health Exam. 1960-62	18-79	7710	87	0.51	1.38	1.08
Wensleydale & Leigh, UK. Lawrence, 1961	15+	1025	87	0.5	1.6	1.1
Blackfeet Indians USA. Burch. 1963.	30+	1281	86	–	–	1.2
Haida Indians Canada. Goften. 1964.	15+	492	88	0.42	1.01	0.69
Hiroshima & Nagasaki. Japan Wood. 1966	15+	18559	87	0.19	0.45	0.35
Jamaica. Lawrence. 1966.	35-64	600	89	0.90	2.24	2.08
Rochester, USA.* Linos et al. 1980	15+	400	–	0.66	1.31	1.05

Source: Mitchell, 1985; Hazes et al, 1990.
*Age-adjusted to US white population

in females at all ages. Lawrence found the prevalence rates for 'definite' RA increased from 1.6 and 0.5 per cent in females and males to five per cent and two per cent respectively over the age of 55 years (Hochberg, 1981).

Diverse prevalence rates have been reported in different ethnic populations. A rate of only 0.1 per cent has been found in black rural Africans (Beighton et al, 1975), whilst rates for urban African Negro population were more similar to the Western world (Solomon et al, 1975ab). However, in the USA no differences emerged between the black and white populations (Masi et al, 1989). However, these studies may not be comparable since the methodologies may have been different.

Figure 3 **Prevalence rates of rheumatoid arthritis in Rochester, Minnesota, by sex, on 1 January 1975**



Source: Linos et al, 1980

Mortality

RA mortality rates and life expectancy influence specific decisions such as the selection of drug treatment by the doctor. Yet the evidence on which such decisions are based is unclear.

The majority of studies show increased mortality and reduced life expectancy in RA patients. In 1989 in England and Wales 1423 people (OPCS, 1991) were recorded in the mortality statistics under rheumatoid arthritis. In addition, it has been suggested that the data underestimate mortality since RA is unlikely to be recorded on the death certificates, particularly as an underlying cause of death (Lindahl, 1985).

Care is needed when interpreting the data as different data bases (including hospital and other autopsy register and national statistics on the cause of death) could influence the conclusions of a study rather than the disease. Indeed, studies often involved patients who had severe symptoms and/or complications of either the disease or

the treatment, and even when a random sample of the general public was studied, Allebeck et al (1981) found an increase in mortality due to a subgroup of RA patients who had been hospitalised before the case selection.

Case acquisition bias was avoided in the Rochester study and no significant difference was found in the mortality of RA patients and the general population (Linos et al, 1980), however, the study may have been biased towards mild or even uncertain RA as it included 'probable' cases of RA.

Clearly some debate remains as to whether RA increases mortality. Recent studies have indicated about a three fold increase in mortality (Prior et al, 1984; Mutro et al, 1985), but it is generally agreed that the increased mortality occurs in a subgroup of RA patients who have a more severe disease course (Abruzzo, 1982; Vandenbroucke, 1984). Studies have found that this subgroup is susceptible to cardiovascular, respiratory and infectious diseases, perhaps as a consequence of the disease or even its therapy (Abruzzo, 1982; Prior et al, 1984). However, since many different treatments and dosages are used in RA it is not possible to determine the effect of the disease itself and/or the treatment on mortality.

Juvenile Chronic Arthritis

Different diagnostic criteria and methodologies make comparisons between juvenile chronic arthritis (JCA) studies difficult. In spite of this and the time span between the studies on Table 4, the incidence and prevalence rates show little variation.

Incidence rates vary from 0.12 to 0.19 per 1000, whilst prevalence rates range from 0.16 to 1.13 per 1000. These rates correspond to an estimate by the Arthritis and Rheumatism Council (ARC) in 1979 (Benjamin, 1990) of 12,000 children in Great Britain with JCA.

Table 4 **Prevalence and incidence studies of juvenile chronic arthritis**

Country (reference)	*Prevalence per 1000 children age 0-16 years*	*Annual incidence per 1000 children age 0-16 years*	*Year of study*
Taplow, UK (1968)	0.65	–	1959
Paris, France (1987)	0.77	0.19	1981-82
Brittany, France (1987)	1.00	0.13	1981-82
USA (1983)	0.16-0.43	–	1978, 1979
Rochester, USA (1983)	1.13	0.139	1960-80
Goteburg, Sweden (1987)	0.56	0.12	1983

Source: Benjamin (1990)

Table 5 **Juvenile chronic arthritis: age and sex characteristics according to disease subgroup**

Disease subgroup	*Sex ratio (M:F)*	*Age of onset*
Pauciarticular	1:2.5	Mean age 4 years
Seronegative polyarticular	1:2.5	2 peak ages of onset; 1-3 years and 8-10 years (mean age 5.5 years)
Systemic onset	1:1	Two-thirds younger than 5 years
Seropositive polyarticular	Mostly girls	From 5 years upwards (70 per cent of cases present over 10 years)
Juvenile ankylosing spondylitis*	5:1	From 3 to 15 years (mean age 12 years)

*comparative prevalence unknown
Source: Benjamin, 1990.

The various diseases which are included in the category of JCA each have there own epidemiology; the age and sex ratio give some indication of the variability of each subgroup (see Table 5), with each subgroup in descending order of prevalence.

Mortality in Juvenile Chronic Arthritis

The actual number of deaths from JCA in England and Wales is difficult to establish since it does not have its own International Classification of Disease code but is included in the category of Rheumatoid Arthritis and Other Inflammatory Polyarthropathies. Mortality statistics produced by the Office of Population Censuses and Surveys (1991) indicates that no individuals died in the age groups 0-19 years in this category in 1989, hence it can be inferred that no children died from JCA, although some cases may not have been recorded in the correct category.

Conversely, studies indicate that long term mortality ranges from one to three per cent in the United States to ten per cent in England and Europe. The differences may possibly be due to disease definition, increased frequency of secondary amyloidosis or selection bias. Certainly, more research is needed in this area.

Osteoarthritis

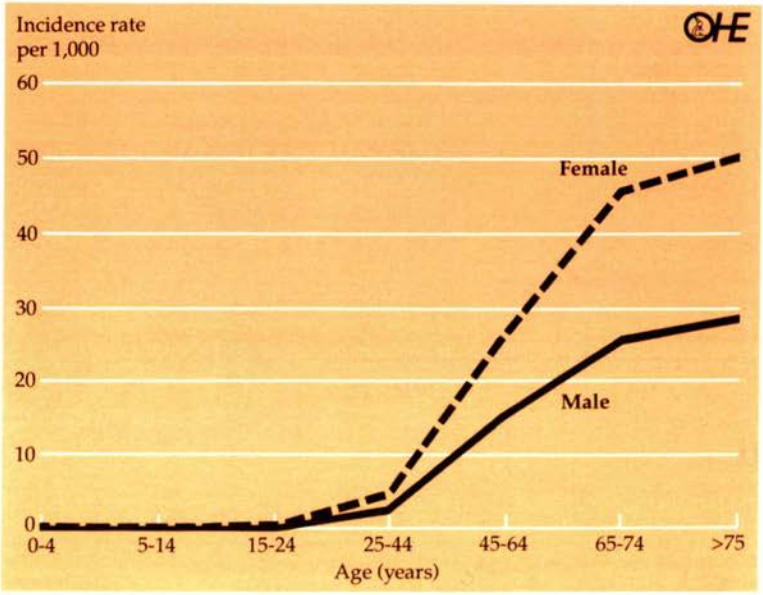
Osteoarthritis (OA) is the commonest rheumatic disease, second only to cardiovascular disease in producing severe disability (Wright, 1989). It is not surprising that OA has been described as 'an almost inevitable consequence of aging' (Kelsey, 1982) since it affects about ten per cent of the population aged 60 and over (Peyron, 1986); that is over one million individuals in Great Britain in this age group alone.

The epidemiology of OA is difficult as the symptoms lack specificity. Therefore, statistics in this area should be viewed with some caution since those based on X-rays may yield higher estimates because 40 per cent of individuals show evidence of OA on the X-rays but do not experience any symptoms, in addition difficulties may arise from different interpretations and definitions of early and mild OA.

Incidence

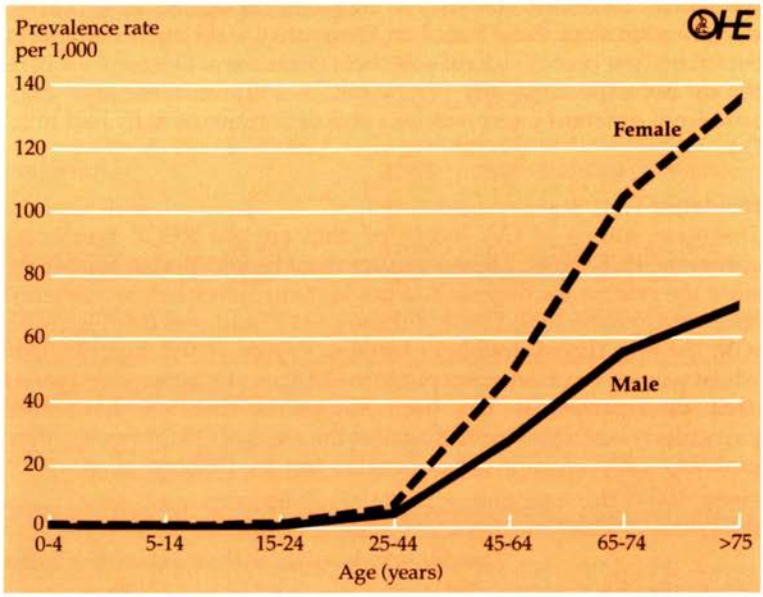
The main source of OA incidence data are the RCGP morbidity surveys (OPCS, 1986). These statistics must be interpreted cautiously since the criteria for diagnosis is not known. Nevertheless incidence rises with age reaching a peak at those aged 75 and over (see Figure 4), with the rate almost double in females. Peyron (1979) suggests that whilst women are more susceptible to OA they are 'somewhat spared until menopause'. It has been suggested that sex hormones, particularly oestrogen protect against the onset of OA. However, they probably only modify other genetic factors (Silberg et al, 1971; Sokoloff, 1969).

Figure 4 **Incidence rate of OA per 1,000 in England and Wales**



Source: OPCS, 1986

Figure 5 **Prevalence rate of OA per 1,000 in England and Wales**



Source: OPCS, 1986

Table 6 **Prevalence rates (%) of radiographic knee OA in different populations**

	Age Group (years)						
	25-34	*35-44*	*45-54*	*55-64*	*65-74*	*75+*	*35-74**
United States							
Males	0.0	1.7	2.3	4.1	8.3	–	3.8
Females	0.1	1.5	3.6	7.3	18.0	–	7.6
Northern England							
Males	–	7.0	12.1	28.7	42.3	–	19.0
Females	–	6.0	17.4	48.6	56.3	–	29.0
Holland							
Males	–	–	9.3	16.8	20.9	22.1	12.2**
Females	–	–	13.9	18.5	35.2	44.1	19.7
Sweden							
Males	–	0.0	3.0	4.5	4.5	4.5	2.8
Females	–	7.0	4.0	11.0	26.5	36.0	12.0

* Age-standardized rate using 1970 US population as standard
** Prevalence of 0 per cent assumed for ages 35-44 years.
Source: Adapted from Felson, 1988

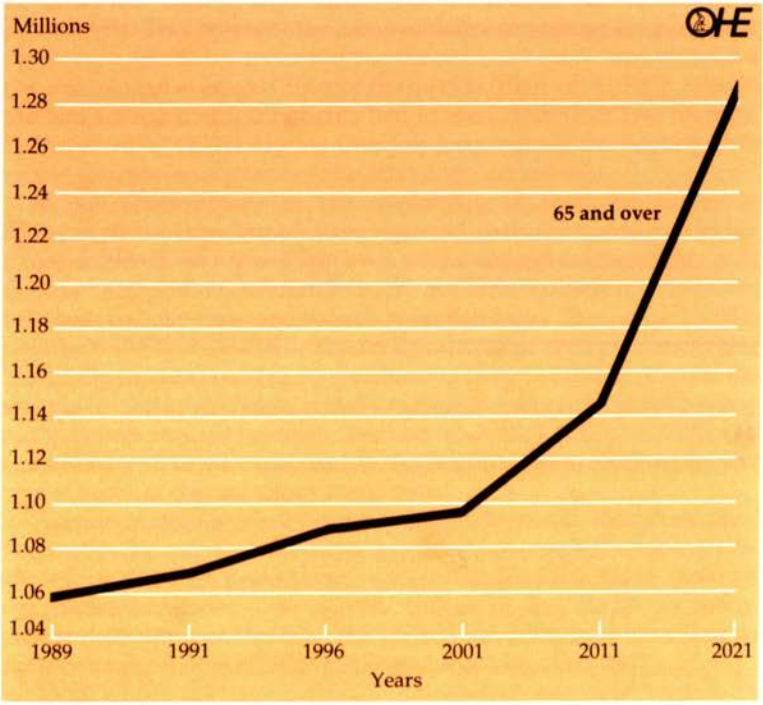
Prevalence

Age appears to have the greatest influence on the occurrence of OA. A study in the United States of 6672 adults in 1960-62 (National Center for Health Statistics, 1966) found that OA increased steadily with age from four per cent in persons aged 18 to 24 years to 85 per cent among individuals 75 to 79 years. OPCS data for England and Wales indicate the same trend (see Figure 5), although according to Peyron (1979) 'the mechanism by which aging influences the onset of OA is poorly understood at present'.

Figure 5 shows that the prevalence of OA is fairly evenly distributed between the sexes up to the age of 45 to 55 years, with a marked predilection for women thereafter. However, Lawrence et al (1980) argues that the sexes differed little from the overall prevalence of OA after reassessing 17 studies.

The prevalence of radiographic knee OA in different populations

Figure 6 **Total number of elderly with RA, OA and gout in England and Wales***



Source: OHE

*Prevalence rates applied to projected population

worldwide (see Table 6) concurs with OPCS data, with a general trend toward an increase with age and especially a rise in prevalence among women of advancing age. However, the rates in the various populations differ considerably in spite of essentially the same methodologies. However, these discrepancies may exist because of different radiographic interpretations, therefore more comparative population studies are needed to identify any geographical differences.

Health resources will be put under increasing pressure as the population of the United Kingdom is aging and the incidence of RA, OA and gout increase with age. It can be anticipated that there will be an increasing number of elderly and very elderly patients with these diseases (see Figure 6) since projections indicate that the over 65 age group will grow by 2.5 per cent, with the over 75's rising by 37.5 per cent between 1988 to 1996 (OHE,1989). This will increase demand for GP time and prescriptions dispensed and put health care resources under pressure.

TREATMENT

The effective care of people with arthritis and allied conditions involves treating both the disease and the person. Treatment combines the use of a team of health care professionals with medication and, in some cases, surgery. Indeed, 'of all the afflictions of man, it is arthritis that is seen as most clearly being the responsibility of the primary health care team' (Hynes, 1991).

The team

The general practitioner (GP) manages the day-to-day care of patients with arthritis and allied conditions. These diseases account for over four million GP consultations each year (OPCS, 1986)[9]. However, GP's may not have 'a full knowledge of the actual therapeutic and rehabilitative possibilities and their results' (Zinn, 1982) and may refer patients to a rheumatologist, who will confirm the diagnosis, recommend and initiate treatment.

The medicaments prescribed for RA can be monitored by the GP using blood tests whilst the progression of the disease in the hands can be observed with graded rings, and treatment can be adjusted accordingly. This prevents the inconvenience of waiting for a hospital opinion. In a British survey waiting list times for consultations with a rheumatologist averaged 3.4 months in 1986 (Blair et al, 1987), even in the best served districts patients had to wait more than two months. Helliwell et al (1991) argues that the long outpatient waiting lists reflect the continuing lack of rheumatology manpower.

Serious shortcomings in the availability of specialist advice in different parts of the country were reported by the Manpower Working Panel in 1986, who argued that even where consultants were in a post there were important deficiencies in the available back up. A ratio of one full-time (whole time equivalent) rheumatologist for every 150,000 population has been endorsed by the Royal College of Physicians, World Health Organisation and the Department of Health. Table 7 indicates the shortfall of rheumatologists in many regions; the situation may be worse as the figures include rehabilitation and other staff. The new NHS and Community Care Act may aid in highlighting and challenging the unmet needs of rheumatology manpower.

The rheumatology manpower situation is better in other countries. In Sweden the number of rheumatologists far exceeds the ratio of one for every 150,000 population, whilst the United States has an approximate rate of 1 to 85,000, indeed it has twice as many rheumatologists per capita as the United Kingdom and is aiming to achieve a ratio of 1 to 67,000 by the year 2000 (Meenan, 1991).

9 The figure is likely to be an underestimate as the OPCS data only classify 'serious' cases of arthritis and the definition is unknown.

Table 7 **Number of rheumatology consultants by Regional Health Authority (RHA) in England, 1990.**

RHA	*Population (millions)*	*Actual number*	*Required number**	*Shortfall***
Northern	3.08	16	21	5
Yorkshire	3.66	8	24	16
Trent	4.70	18	31	13
E Anglia	2.06	10	14	4
NW Thames	3.50	17	23	6
NE Thames	3.80	30	25	(5)
SE Thames	3.66	29	24	(5)
SW Thames	2.98	16	20	4
Wessex	2.94	18	20	2
Oxford	2.56	14	17	3
S Western	3.26	8	22	14
W Midlands	5.22	13	35	22
Mersey	2.40	5	16	11
N Western	4.02	18	27	9

* One rheumatologist for every 150,000
** Figures in brackets indicate surplus
† Whole time equivalent (rounded up)
Source: OHE

Whilst it is not possible to prevent the onset of most rheumatic disorders, physical disability can be minimised by regular exercise, physiotherapy and with help from health professionals.

Physiotherapists have an important role in informing the patient of exercises, movement and handling techniques which can help maintain and restore muscle power and joint movements as well as preventing and correcting deformity. Bed rest is usually only recommended during phases of excessive inflammatory activity.

Other members of the team include the occupational therapist, who assesses the needs of an individual in relation to daily living and can provide helpful equipment. The specialist rheumatology nurse can monitor and advise the patient on a regular basis and is becoming an increasingly valuable and cost effective part of hospital practice, in addition to the chiropody and orthotist departments. Splints and appliances are made to help reduce pain and improve function, also a variety of walking aids and special shoes are available.

The goal of a multidisciplinary approach is to help patients attain their maximum potential for daily living by making full use of the varied and considerable skills of therapists and others.

Management of pain

In spite of one sixth of the population believing that medicine can do

little or nothing to relieve the symptoms of the various forms of arthritis (Dunnell et al, 1972), it is possible to minimise the symptoms. Moreover if the symptoms are left untreated or treated half-hearted they will invariably get worse and the damage may be irreparable.

Treatment for rheumatoid arthritis is usually aimed at reducing pain and inflammation to allow normal function. Patients with mild and intermittent pain may be prescribed analgesics (see Table 8) to be taken when needed, unlike non steroidal anti-inflammatory medications (NSAIDs) which need to be regularly maintained in adequate dosages to obtain their full therapeutic effect. NSAIDs

Table 8 **Medication used in the treatment of arthritis and allied conditions**

Chemical name	*Effective for treating*
1 Analgesics	
Aspirin	Pain and inflammation in rheumatic disease and other musculoskeletal disorders.
Benorylate; Salicylates	Pain and inflammation in rheumatic disease and other musculoskeletal disorders.
Co-proxamol; Co-dydromol	Pain in musculoskeletal conditions.
2 NSAIDs	
Azapropazone	Pain and inflammation in rheumatic disease and other musculoskeletal disorders; gout.
Ibuprofen; Tolmetin	Pain and inflammation in rheumatic disease (including juvenile arthritis) and other musculoskeletal disorders.
Diclofenac Sodium; Naproxen; Piroxicam	Pain and inflammation in rheumatic disease (including juvenile arthritis) and other musculoskeletal disorders; acute gout.
Diflunisal; Fenbufen; Fenoprofen; Tiaprofenic Acid	Pain and inflammation in rheumatic disease and other musculoskeletal disorders.
Flurbiprofen	Pain and inflammation in rheumatoid arthritis, osteoarthrosis and ankylosing spondylitis.
Indomethacin	Pain and moderate to severe inflammation in rheumatic disease and other acute musculoskeletal disorders; acute gout.
Ketoprofen; Sulindac	Pain and inflammation in rheumatic disease and other musculoskeletal disorders; acute gout.

Chemical name	*Effective for treating*
Mefenamic Acid	Mild to moderate pain in rheumatoid arthritis (including juvenile arthritis), osteoarthritis and related conditions.
Nabumetone; Tenoxicam; Etodolac	Pain and inflammation in osteoarthritis and rheumatoid arthritis.
Phenylbutazone	Ankylosing spondylitis (in hospital).
3 Corticosteroids	
Dexamethasone Sodium Phosphate; Hydrocortisone Acetate; Methylprednisolone Acetate; Prednisolone Acetate; Triamcinolone Acetonide; Triamcinolone Hexacetonide	Local inflammation of joints and soft tissue (by local injection).
Prednisolone	RA and SLE which is unresponsive to other medication (by mouth).
4 Disease modifying medication	
Sodium Aurothiomalate	Active progressive rheumatoid arthritis, juvenile arthritis.
Auranofin	Active progressive arthritis when NSAIDs inadequate alone.
Penicillamine	Severe active or progressive rheumatoid arthritis, juvenile arthritis.
Sulphasalazine	Active rheumatoid arthritis
5 Antimalarials	
Chloroquine; Hydroxychloroquine Sulphate	Active rheumatoid arthritis (including juvenile arthritis), systemic lupus erythematosus.
6 Anti-gout medication	
Colchicine	Acute gout
Allopurinol; Probenecid; Sulphinpyrazone	Hyperuricaemia
7 Others	
Methotrexate; Chlorambucil; Azathioprine; Cyclophosphamide	Immunosuppression

Source: British National Formulary, 1991

reduce inflammation, but probably do not alter the course of the disease. Analgesics are the mainstay of treatment for OA with NSAIDS used only if analgesics prove to be ineffective.

The efficacy of NSAIDs has been clinically demonstrated. 'Differences in anti inflammatory activity between different NSAIDs are small but there is considerable variation in individual patient response' (British National Formulary, 1991) therefore patients may try a number of NSAIDs throughout the course of their disease. The withdrawal of NSAIDS is soon followed by the recurrence of inflammation, often described as a 'flare up'.

Over the past decade the recommended dosages of NSAIDs have risen (Baum et al, 1985) which increases the cost of prescribing. In 1990 the number of anti-rheumatic scripts (mainly NSAIDs) dispensed totalled 23.3 million at a cost of £219 million[10], that is one in every 20 NHS prescriptions. If rheumatoid arthritis symptoms persist despite the use of NSAIDs it becomes necessary to use other medication such as disease modifying medication or corticosteroids. The latter share some of the NSAIDs characteristics by rapidly reducing established inflammation but probably do not prevent the progression of joint damage.

Disease modifying medicaton used in the treatment of RA and psoriatic arthritis gradually reduces disease activity over a period of months, but lacks the immediate analgesic and anti-inflammatory properties of NSAIDs. All are potentially toxic, therefore are used when the disease is severe and progressive, although recently it has been argued that early and consistent use of these medicaments might alter the rate of an individual's functional loss and mortality. Certainly, rheumatologists have adopted a more aggressive and earlier treatment regime as the time from referral to the use of disease modifying drugs has fallen from seven years (1967-1971) to five months in 1982-1986 (Spector et al, 1988).

There is little doubt that these treatments can cause side effects. Whilst the incidence of adverse reactions varies between different medications, the overall profile of toxicity is similar. However, many of the adverse reactions could be avoided by careful and regular monitoring of a patient by the GP, including regular blood and urine tests, and any side effects being reported by the patient.

The management of juvenile chronic arthritis and gout differ significantly from other types arthritis. The aim of therapy in children is to prevent joint deformity and maintain joint and muscle function in the growing child, therefore the physiotherapist is a vital member of the management team. NSAIDs are normally the only medicaments required, with surgery only necessary in children with significant functional limitation.

10 Calculated at an average net ingredient cost of £9.40.

The symptoms of gout can be controlled by medication, therefore it is vital that the disease is accurately diagnosed. The typical impression that a gout patient is a man in middle age indulging in too much alcohol and rich food is no longer appropriate. Indeed, a substantial proportion (15-30 per cent) of newly diagnosed gout patients are women (Macfarlane et al, 1985; Lally et al, 1986) with its onset almost invariably at menopause.

The management of gout is fairly simple and straightforward. In some patients a reduction in weight may sufficiently reduce the amount of uric acid in the blood. However, many gout patients do need treatment to stop acute attacks and to prevent recurrent attacks and complications. Acute attacks require high dosages of NSAIDs, whilst long term treatment with anti-gout medication such as allopurinon alters uric acid metabolism.

Compliance[11]

Non-compliance is a problem since the benefit from treatment is largely dependant on whether the patient adheres to the prescribed therapy, whether it is medication, psychosocial intervention, physical or occupational therapy.

Adherence to arthritis therapies is a significant problem, yet has received little attention. The majority of reports suggest that only 40 to 60 per cent of patients follow the prescribed regime correctly (Belcon et al, 1984; Deyo et al, 1981; Hicks, 1985). Compliance among children is similar to adults with about 50 per cent adequately complying (Jay et al, 1984; Rapoff et al, 1982). The extent of non-compliance is remarkable given that medications are frequently given to provide relief from symptoms (Jay et al, 1984; Rapoff et al, 1982). The search for a typical non-compliant patient has met with little success, no consistent relation has been found for variables like age, sex, social class or personality traits.

According to Ehrlich (1980) compliance will not be achieved merely by educating the patient, but suggests that the best remedy is to involve the patient in the planning of the programme, rather than to dictate it to the patient.

Surgery

Severe rheumatoid, osteoarthritis and juvenile chronic arthritis may require surgery. The various types of surgery typically undertaken in arthritis and its allied conditions are explained in Table 9.

One of the most important advances in the surgical treatment of arthritis has been the introduction of prosthetic replacements. Before

11 Compliance is simply the extent an individual's behaviour coincides with medical or health advice (Haynes et al, 1979). Therefore non-compliance ranges from those individuals who fail to have their prescription dispensed to those who stop their course of treatment before completion.

Table 9 **Types of surgery for arthritis and allied conditions**

Minor	Includes repair of damaged tendons and ligaments and removal of large cysts and nodules.
Synovectomy*	Removal of inflamed lining (the synovium) of a joint or tendon sheath.
Osteotomy*	The cutting and resetting of a joint at slightly different position.
Debridement or Arthroscopy	The 'spring cleaning' of joint to remove unwanted bony spurs and loose pieces of bone and cartilage floating in cavity.
Arthrodesis	Bone permanently fixed to bone eliminating any joint.
Arthroplasty	Replacing a joint either in part or completely.

Source: Phillips, 1989
* Procedures now rarely performed

artificial joints were available the only option was to cut the joint out (excision arthroplasty); a procedure rarely used today.

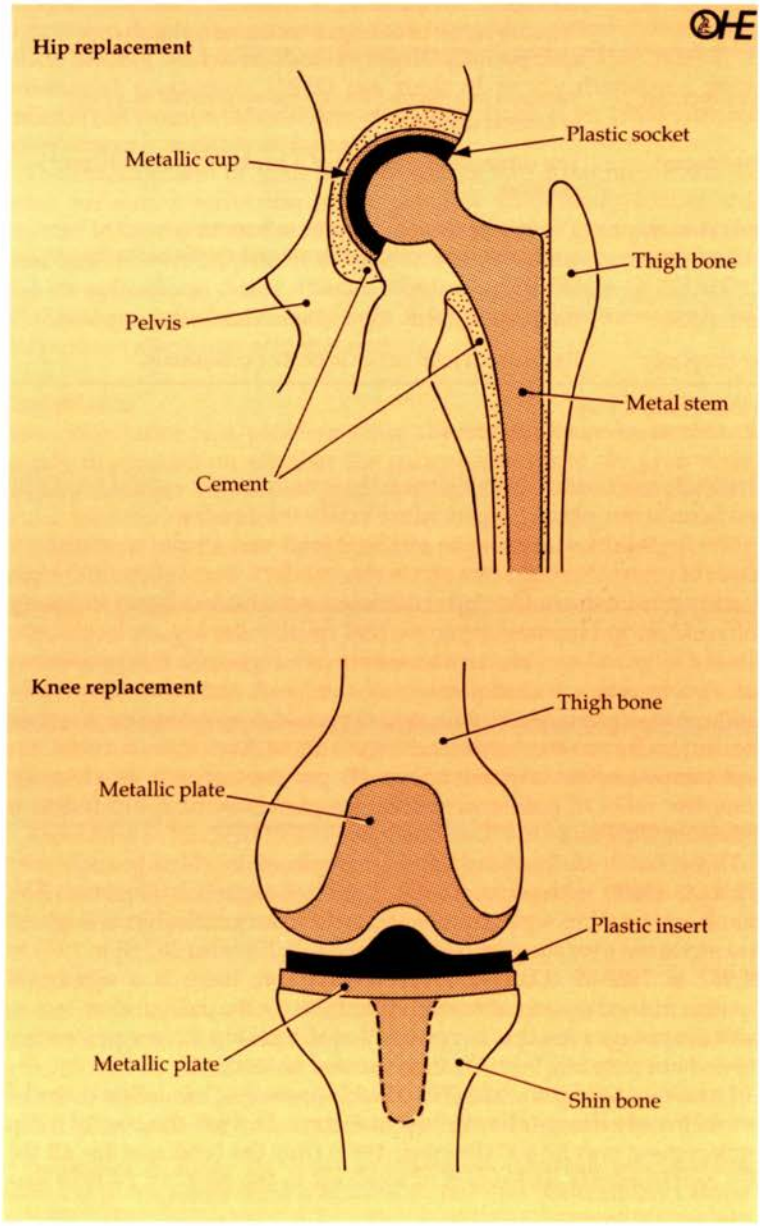
The first implantation of an artificial joint was a knee replacement made of ivory over 100 years ago, today modern metal alloys and high density plastics have allowed artificial joints to be developed for many different sites. The most common and reliable are hip replacements, closely followed by knee replacements (see Figure 7), but prostheses are also available for small joints of the hand, shoulder, elbow and ankle. A successful joint replacement provides an adequate range of movement for most everyday activity without pain, indeed a total hip replacement offers a greater than 90 per cent chance of virtually complete relief of pain and restoration of near normal hip function (Stauffer, 1989).

Three hundred thousand hips are replaced worldwide each year (Philips, 1989), with about 42,000 in NHS hospitals in England. The number of total hip replacements and arthroplasty of the hip in England has increased over the past decade (see Table 10) from 28,788 in 1979 to 41,947 in 1988-89 (Dorrell, 1991). In addition, there is a significant number of total hip replacements undertaken in the independent sector, with data suggesting that 28 per cent[12] of all total hip replacements were carried out privately (over 7000 operations) in 1986.

The average cost to the NHS of each operation has fallen over the years, mainly due to shorter hospital stays. In 1988 the cost of a hip replacement was £2,400 (Frankel, 1991) thus the total cost for all the hip replacements performed in England to the NHS in 1988-89 was

12 This figure only includes total hip replacements and not other arthroplasty of the hip.

Figure 7 **Joint replacement**



Source: Phillips, 1989

Table 10 **Estimated number of principal operations performed, ordinary admissions and day cases in NHS hospitals in England**

All patients total hip replacement and other arthroplasty of hip	
1979	28,788
1980	31,660
1981	32,775
1982	33,171
1983	36,926
1984	36,926
1985	36,539
1986	41,000
1988-1989	41,947
Total	319,038

* The figures for 1987-88 are not available
Source: Dorrell, 1991

£100 million. Another way to approach the cost is in terms of quality adjusted life year (QALY)[13] with hip replacements being a cost effective intervention with a cost per QALY gained of £1,180 (Maynard, 1990). Frankel (1991) believes that current operation rates may accommodate the incidence of the disease, and therefore does not advocate an increased mean level of total hip replacements. Nevertheless there are still long waiting list times with a mean time of 22.2 months in 1986 for a hip replacement surgery in Britain, and even in the most efficient districts there is a wait of over 16 months (Blair et al, 1987), although the governments initiative on reducing waiting list times may have some affect on those individuals waiting more than two years.

Arthritis of the knee is commoner than hip arthritis, but requirement for knee arthroplasty is less. Arthritis often affects both knees equally and the replacement of one knee may produce little benefit in terms of patient mobility or quality of life. The rate of knee replacements is still increasing with 10,000 knee replacements performed in 1989 in England (Bulstrode, 1989). In the United Stated knee replacements have overtaken hip replacement operations, with the number of operations doubling from 50,000 operations in 1980 to 110,000 in 1985 (Phillips, 1989), with estimates suggesting that there is a need for 143,000 operations a year (Quam et al, 1991). Studies indicate the success rate to be good. A large study of 9,000 knee replacements carried out between 1971 and 1987 indicates that 91 per

13 The most commonly used form of QALY in the United Kingdom has two dimensions of quality of life, that is of distress and disability. For a full explanation see Williams (1985).

cent of prostheses were still in situ at five years, 80 per cent at ten years, and 69 per cent at 15 years (Rand et al, 1991).

The success of an operation depends on patient selection, choice of prothesis and surgical skill. An artificial hip and knee is expected to last about 15 years, therefore a revision procedure may be required for those patients who had an artificial joint before the age of 60 years. Age should not preclude individuals from joint replacements, consideration is required if quality of life has significantly deteriorated.

As technology advances newer and better material for joint replacement will become available. At present, research is centred on bone grafting. There are two types of grafting, autografting where the bone is removed from one site and grafted on to another in the same person, and allografting where the bone from one person is grafted in another. Indeed, Britain's first bone bank at Glenfield Green hospital in Leicestershire has found that the main demand is for thigh bones for revision of hip joint replacement operations.

Other Treatments

Over the centuries individuals have turned to 'alternatives' in the search for an aid to rheumatic diseases – copper bangles have replaced the nutmegs and new potatoes of our grandparents. A report in the United Kingdom reveals that 91 per cent of patients with rheumatoid arthritis (RA) use an average of nine self-prescribed remedies (Higham et al, 1982). This survey concurs with results from the United States where an estimated 94 per cent of patients with rheumatic diseases tried as many as 13 unproven remedies each (Brown et al, 1980; Wasner et al, 1980) at a cost of $1 billion annually (Hecht, 1980). Social class or educational background seem irrelevant (Brown et al, 1980) in search of pain relief and control of the disease.

Dietary manipulation as a form of therapy has increased in popularity. The addition to the diet of fish oil and evening primrose oil is particularly common. However, a review by Buchanan et al (1991) on the effect of diet concluded that it is doubtful whether fish oils and/or evening primrose oils have any significant long term benefit for patients with RA; little research has been undertaken on their effect in OA. Nevertheless, the Arthritis Foundation of the USA states that 'The possible relationship between diet and arthritis has been thoroughly and scientifically studied. The simple proven fact is: no food has anything to do with causing arthritis and no food is effective in treating or 'curing' it' (Arthritis Foundation, 1976). Controlled double blind studies are needed to resolve the issue completely.

Naturally, weight reduction in the overweight, vitamin supplements in the vitamin deficient and the removal of any foodstuff to which an individual is allergic are all sensible and helpful, but at present many individuals are spending large sums of money on remedies of unproven benefit.

THE COST OF ARTHRITIS

It is necessary, although at times difficult, to attribute costs to all aspects of arthritis and its allied conditions in order for society to make the most efficient use of its limited resources. The costs of arthritis include all medical services consumed, the impact on the community in terms of lost production and personal costs to patients and their families.

Studies in the United States have attempted to measure the total cost of arthritis, estimates range from £12,000 million to £33,000 million a year at 1990 prices (Arthritis Foundation, 1982; McPherson Brown, 1988). If it is assumed that the United Kingdom has one-fifth the population of the USA and the cost of arthritis is one half the lower figure, it can be estimated that arthritis in the United Kingdom cost £1,200 million in 1990[14].

There are a number of medical costs for arthritis which can be calculated for the United Kingdom, including hospital and GP costs (see Table 11). In 1989 the hospital sector absorbed £231.3 million for individuals with arthritis, about 1.6 per cent of all hospital costs. This figure reflects the cost of both in-patient and outpatient services. The latter estimate must be treated with caution as it excludes those cases entering the outpatient system from sources other than general practice referral, such as in-patient discharge, and may inadequately reflect the true resource burden of managing the disease in this setting. Considerations such as these suggest that the outpatient cost may be an underestimate, however since it is only a small proportion of total hospital services it is unlikely to substantially alter the overall figure.

Arthritis accounted for 2.23 per cent of all general practice consultations in 1989, at a cost of some £44.78 million. In the pharmaceutical services, arthritis cost £219 million, a sum approaching one tenth of the total cost of pharmaceutical services, naturally 'over the counter' products (OTC) purchased to relieve the symptoms would increase the cost considerably. The pharmaceutical cost is relatively high because patients frequently need prolonged medication. Excluding the other parts of the service for which figures are not available, this gives a total cost of arthritis to the National Health Service of £495.08 million.

Other costs are difficult to quantify, particularly indirect costs such as lost productivity. Nevertheless, in 1989 over 41 million working days were lost, that is nearly 11 per cent of all days lost, resulting in an expenditure of approximately £308 million in annual benefit

14 The costs are based on the Arthritis Foundation estimates of $13,500 million in 1982, inflated to 1990 prices and converted in to sterling using the mean exchange rate for 1990 of $1.75:£1.

Table 11 **The cost of arthritis to the National Health Service, UK, 1989***

Health service sector	*Cost attributed to arthritis £ million*	*Total cost £ million*	*Per cent attributed to arthritis*
Hospital services	231.3	14,265	1.6
General practice	44.78	2,008	2.23
Pharmaceutical services	219.0	2,738	8.0
Others	N/A**	7,069	N/A**
Total	495.08	26,080	1.90

** N/A – not available
Source: OHE

* The estimates are calculated as follows:
Pharmaceutical Services – Number of prescriptions for rheumatic preparations multiplied by the net ingredient cost for 1989. The total cost of pharmaceutical services includes charge paid by patient.
Hospital Services – in-patient – number of in-patients for arthritis multiplied by mean length of stay, multiplied by average cost per day of £140 in 1988/89 (OHE estimate).
Outpatient – referral rate (OPCS, 1986) applied to number of people consulting for arthritis in 1989 (OHE estimate) multiplied by average cost of £42 (OHE).
General Practice – The proportion of consultations specifically for 'serious' osteoarthritis, rheumatoid arthritis and gout as defined by the OPCS (1986) was applied to the total expenditure on the General Medical Services in the UK in 1989. This figure may be an underestimate as it only accounts for 'serious' cases.

payments. This sum is not a cost but a transfer of wealth within the community, as the real price of sickness absence is lost productivity. There are also hidden costs for individuals with arthritis such as lack of employment opportunities and premature retirement from work, in addition to individuals changing jobs because of their arthritis thus wasting costly skills. Indeed, a survey found that, on average, workers with arthritis were earning only 50 per cent of their predicted income (Meenan et al, 1981). Moreover, people with arthritis may have additional expenditure due to lack of mobility and special equipment such as raised toilet seats and handrails to maintain their independence. The OPCS (1988) survey of disability found that 60 per cent of those with arthritis spend on average an addition £2.20 per week solely due to their disability and around a quarter of the people with arthritis spend a proportion of their income on at least one item of disability equipment at a cost of £78.00 (OPCS, 1988).

Personal costs such as pain, depression and low self esteem are largely intangible or incalculable. Nevertheless, a number of studies have attempted to measure pain. The studies show consistency in reported pain and that the relief from pain is the most important

component in explaining medication usage (McKenna et al, 1985; Kazis et al, 1983). Furthermore, in a survey of patients with chronic conditions[15] the impact of pain was greatest in patients with arthritis (Stewart et al, 1989). Pain and depression are interrelated with depression common among patients with arthritis and other types of chronic disease (Frank et al, 1985; Kashani et al, 1984). Newman et al (1989) found that a number of factors contributed to depression in people with rheumatoid arthritis, including demographic, disease related, disability and social variables. Arthritis also has costs in terms of a gradual reduction in the ability to carry out daily living activities since 67 per cent of people with arthritis have problems with dexterity, 64 per cent have reaching and stretching problems and 56 per cent have locomotor disabilities (OPCS, 1988).

Pain, depression and lack of employment opportunities can lower an individuals self esteem causing sexual and social problems (Ehrlich, 1983). At present studies have yielded insufficient information on the burden of arthritis to the individual and to the family to derive complete and accurate costings.

In the future costs may be expected to escalate as the number of elderly people with arthritis continues to grow (see p27). The total cost to the NHS for arthritis will increase from £495.08 million in 1989 to £564 million by 2001 at 1989 prices (OHE estimate) simply due to the increase in the number of individuals with arthritis – an increase in real terms of 14 per cent. Therefore the costs and benefits of the health care sectors will be subject to closer scrutiny in the search for greater economic efficiency.

15 The conditions considered included hypertension, diabetes, congestive heart failure, myocardial infarction, arthritis, chronic lung problems, gastrointestinal disorders, back problems and angina.

CONCLUSION

The Government recently set out its proposals for developing a health strategy in England in the consultative document *The Health of the Nation* (1991) in response to the World Health Organisation's (WHO) Health for All programme. The WHO set a number of targets; target 4 stated that the average years people live free from major disease or disability should be increased by at least ten per cent. It is surprising therefore, that the government failed to include arthritis in this document, since in the OPCS survey of disability (OPCS, 1988) arthritis was the most frequently mentioned cause of disability. It is even more remarkable as *The Health of the Nation* (p29) recognises that musculoskeletal problems are the largest contributor to self reported long standing illness. Laing and Hall (1991) suggest that '...it is perhaps indicative of a persisting low level of interest in the special health problems of older people', this is in spite of an aging population which will increase the total burden of arthritis since it is most common in middle and old age.

Arthritis was presumably excluded because it does not fulfil the three criteria set out in *The Health of the Nation* for the selection of key areas – perhaps the suitability of these criteria should be questioned. In any case arthritis does meet certain elements of the criteria and targets could be set in those areas. The government could aim to increase the expectation of active life by secondary preventative programmes and/or intervention, such as an improvement in the hip replacement programme.

Targets could also be set to ensure that the provision of rheumatologists met the standard endorsed by the World Health Organisation, Department of Health and Royal College of Physicians of one full time rheumatologist for every 150,000 people. By 1986 the average for the United Kingdom was 62 per cent of the recommended standards; whilst some areas are fully up to strength others are poorly served such as Yorkshire which had only 40 per cent of that recommended. The situation has improved a little since then although 18 districts still had no rheumatology sessions by 1990 (Symmons et al, 1991). The new NHS and Community Care Act may highlight this shortage of rheumatology manpower as 'purchasers' are now required to purchase one session of consultant rheumatology time for every 15,000 population – more if the population is mainly elderly (Symmons et al, 1991).

Rheumatologists are important as patients are frequently referred by their doctors for diagnosis, since many general practitioners (GP's) are not adequately trained to diagnose accurately these diseases. Indeed, as late as 1979 only 40 per cent of medical schools offered a formal course in rheumatology (Hull, 1988), consequently many GP's probably start practice relatively untrained in this discipline.

Therefore appropriate undergraduate and postgraduate training is essential to acquire specific skills such as joint and soft tissue injection techniques in addition to education initiatives for established GP's.

The government could also take an educational role. Patients require information concerning areas such as therapy, exercise and benefits available (for example, sickness benefit and disability allowance). Many of the myths need to be dispelled since one sixth of the population believe medicine can do little for them even though therapy minimises the symptoms. Also the public profile of arthritis and its related diseases needs to be raised, this may increase funding particularly of research projects. Research areas could include the risk factors which predispose the causes, methods of early detection and alternative forms of therapy which may reduce the need for surgery. It is disappointing that in *The Health of the Nation*, the government has so far failed to address the issue of arthritis and the many millions of individuals affected by this disease. It deserves a higher priority than it has received in the past.

REFERENCES

Abruzzo J L (1982). Rheumatoid arthritis and mortality. Arthritis Rheum; 25: 8: 1020-1023.

Adams I D (1979). Osteoarthritis and sport. J R Soc Med; 72: 185-187.

Agnew T (1991). Cost-cutting cripples arthritis patients. GP; September 20: 50.

Allebeck P, Ahlbom A, Allander E (1981). Increased mortality among persons with rheumatoid arthritis, but where RA does not appear on the death certificate. Scad J Rheumatol; 10: 301-306.

Ansell B M, Wood P H N (1976). Prognosis in Juvenile Chronic Polyarthritis. Clinics in Rheumatic Diseases; 2: 2: 397-412.

Ansell B M, Swann M (1983). The Management of Chronic Arthritis of Children. J of Joint & Bone Surgery; 65-B: 5: 536-543.

Arnett F C, Edworthy S M, Bloch D A et al (1988). The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum; 31: 315-324.

Arnett F C (1989). Immunogenetics and Rheumatic Diseases. In:McCarty D J (1989). Arthritis & Allied Conditions. Lea & Febiger; Philidelphia.

Arthritis Care (1989). Arthritis: The Painful Challenge. Arthritis Care.

Arthritis Foundation (1976). Arthritis: The basic facts. Atlanta. Arthritis Foundation.

Arthritis Foundation (1982). The Arthritis 1975 Annual Report. New York: Arthritis Foundation.

ARC Handbook. Rheumatoid Arthritis. ARC.

Arthritis & Rheumatism Council. Introducing Arthritis. ARC.

Barnard C, Evans P (1984). The Arthritis Handbook. Michael Joseph Ltd.

Baum C, Kennedy D L, Forbes M B (1985). Utilization of nonsteroidal antiinflammatory drugs. Arthritis Rheum; 28: 686-692.

Beighton P, Solomon L, Valkenburg H A (1975). Rheumatoid arthritis in rural South African Negro population. Ann Rheum Dis; 34: 136-141.

Belcon M C, Haynes R B, Tugwell P (1984). A critical review of compliance studies in rheumatoid arthritis. Arthritis Rheum; 27(11): 1227-1233.

Benjamin C M (1990). Juvenile Chronic Arthritis. Brit J Rheum; 29: 231-233.

Bird H (1991). Work-related rheumatic problems. Arthritis Research Today; 81: 5.

Blair P, Grahame R, Wood P H N (1987). The Practitioner rheumatology survey: an analysis. The Practitioner; 231: 969-972.

Brewerton D A (1988). Causes of arthritis. Lancet; 8619: 1063-1066.

Brewer E J, Bass J, Baum J et al (1977). Current proposed revision of JRA criteria. Arthritis Rheum; 20: 195-199.

British National Formulary (1991). No 21: British Medical Association & Royal Pharmaceutical Society of Great Britain.

Brown J H, Spitz P W, Fries J F (1980). Unorthodox treatments in rheumatoid arthritis (abst). Arthritis Rheum; 23: 657.

Buchanan H M, Preston S J, Brooks P M, Buchanan W W (1991). Is diet important in rheumatoid arthritis? Brit J of Rheum; 30: 125-134.

Bulstrode C J K (1989). Knee replacements. Update; 15 June: 1355-1360.

Bywaters E G L (1968). Diagnostic criteria for Still's disease (juvenile rheumatoid arthritis). In: Bennett P H, Wood P H N eds. Population studies of the rheumatic diseases. Proceedings of the third International Symposium. New York, June. Amsterdam: Excerpta Medica: 235-240.

Calabro J J (1985). Rheumatoid Arthritis Beginning in Childhood. In: Utsinger P D, Zvaifler N J, Ehrlich G E (1985). Rheumatoid Arthritis. Etiology, Diagnosis, Management. Lippencott.

Clayton J (1991). Confusion in the joints. New Scientist; 1767: 40-43.

Cobb S (1971). The frequency of rheumatic diseases. American Public Health Assoc Monograph. Cambridge. Harvard Uni Press.

Del Junco et al (1985). Do oral contraceptives prevent rheumatoid arthritis? JAMA; 254: 1938-1941.

Deyo R A, Inuie T S, Sullivan B (1981). Compliance with arthritis drugs: magnitude, correlates, and clinical implications. J Rheumtol; 8: 931-936.

Dieppe P A (1988). Arthritis. British Medical Association.

Dieppe P A (1991). Osteoarthritis: Clinical and Research Perspective. Brit J Rheum; 30 (suppl 1): 1-4.

Doherty M, Abawi J, Pattrick M (1990). Audit of medical inpatient examination: a cry from the joint. J R Coll Physicians London; 24: 115-118.

Dorrell S (1991). Parliamentary Debates (Hansard); 194; 142: 410.

Dugowson C E, Bley L, Koepsell T D et al (1989). Incidence of rheumatoid arthritis in women (abst). Arthritis Rheum; 32 (suppl 1): 25.

Dunnell K, Cartwright A (1972). Medicine Takers, Prescribers and Hoarders. Routledge & Kegan Paul.

Ebringer R W (1991). Rheumatoid Arthritis Bacteria. Arthritis Research Today; 81: 14.

Ehrlich G E (1980). Patient compliance in rheumatic disorders. In: De Blecourt J J (ed). Patient compliance. Bern: Hans Huber.

Ehrlich G E (1983). Social, Economic, Psychologic, and Sexual Outcomes in Rheumatoid Arthritis. American Journal of Medicine: 27-34.

Felson D T (1988). Epidemiology of Hip and Knee Osteoarthritis. Epidemiologic Reviews; 10: 1-28.

Felson D T et al (1989). New Research Findings in Arthritis. JAMA; 261: 5: 680.

Frank R G, Beck N C, Parker J C et al (1988). Depression in rheumatoid arthritis. J Rheumatol; 15: 920-925.

Frankel S (1991). Total hip and knee joint replacement: Assessing a district's needs. THS Health Summary. May; 6-8.

Golding D N (1989). A Synopsis of Rheumatic Diseases. 5th Ed. Wright.

Harris E D (1985). Rheumatoid Arthritis: The clinical spectrum. In: Kelle W M et al (eds). Textbook of Rheumatology (ed II). W B Saunders, Philadelphia.

Haynes R B, Taylor D W, Sackett D L (1979). Compliance in Health Care. John Hopkins Press.

Hazes J M W, Silman A J (1990). Rheumatoid Arthritis. Brit J of Rheumtol; 29: 310-312.

Hecht A (1980). Hocus-pocus as applied to arthritis. FDA Consumer; 14: 24.

Helliwell P S, Wright V (1991). Referrals to rheumatology. Brit Med J; 302: 304-305.

Hicks J E (1985). Compliance: a major factor in the successful treatment of rheumatic disease. Compr Ther; 11(4): 31-37.

Higham C, Jayson M I V (1982). Nonprescribed treatments in rheumatic patients (abst). Ann Rheum Dis; 41: 203.

Hochberg M C (1981). Adult and juvenile rheumatoid arthritis:current epidemiological concepts. Epidemiol Rev; 3: 27-44.

Hospital In-patient Enquiry (1985). London: HMSO.

Hull S (1988). Rheumatology and primary care today. The Practitioner; 232: 1203.

Hynes D (1991). Team work needed in arthritis. The Independent Community Pharmacist. September: 44-45.

Jacoby R K, Jayson M I V, Cosh J A (1973). Onset, Early Stages, and Prognosis of Rheumatoid Arthritis: A Clinical Study of 100 Patients with 11-year Follow-up. Brit Med J; 2: 96-100.

Jay S, Litt I F, Durant R H (1984). Compliance with therapeutic regimens. J Adolesc Health Care; 5: 124-136.

Kashani J H, Frank R G, Kashani S R et al (1984). Depression among amputees. J Clin Psychiatry; 44: 256-258.

Kazis L E, Meenan R F, Anderson J J (1983). Pain in rheumatic diseases. Arthritis Rheum; 26: 1017-1022.

Kellgren J H, Lawrence J S (1952). Rheumatism in miners. II. X-ray study. Br J Ind Med; 9: 197-207.

Kellgren J H, Lawrence J S (1958). Osteoarthrosis and disc degeneration in an urban population. Ann Rheum Dis; 17: 338-397.

Kellgren J H, Jeffrey M R, Ball J (1963). The epidemiology of chronic rheumatism. Vol 2. Oxford: Blackwell Scientific.

Kelsey J L (1982). Epidemiology of musculoskeletal disorders. New York: Oxford University Press; 78-86.

Khaligh N, Wood P N H (1986). Arthritis and rheumatism in the eighties. ARC.

Laing W, Hall M (1991). The Challenges of Ageing. ABPI. P42.

Lally E V, Ho G Jr, Kaplan S R (1986). The clinical spectrum of gouty arthritis in women. Arch Intern Med; 146: 2221-2225.

Lawrence J S (1961). Prevalence of rheumatoid arthritis. Ann Rheum Dis; 20: 11-17.

Lawrence J S (1977). Rheumatism in populations. London: William Heinemann Medical Books.

Lawrence J S, Sebo M (1980). The geography of osteoarthrosis. In: Nuki G (ed). The aetiopathogenesis of osteoarthritis. London: Ottoman Medical. Chap 16: 155-183.

Lindahl B J (1985). The casual sequence on death certificates: errors affecting the reliability of mortality statistics for rheumatoid arthritis. J Chron Dis; 38: 47-57.

Linos A, Worthington J W, O'Fallon W M, Kurland L T (1980). The epidemiology of rheumatoid arthritis in Rochester, Minnesota: a study of incidence, prevalence and mortality. Am J Epidemiol; 111: 97-98.

Macfarlane D G, Dieppe P A (1985). Diuretic-induced gout in elderly women. Br J Rheumatol; 24: 155-157.

Manpower Working Panel (1986). A report on rheumatology manpower 1986. Br J Rheumatol; 25: 306-312.

Martin J, Meltzer H, Elliott D (1988). The financial circumstances of disabled adults living in private households. HMSO.

Masi A T, Medsger T A (1989). Epidemiology of the rheumatic diseases. In: McCarty D J (ed). Arthritis and Allied Conditions. Philadelphia: Lea & Febiger.

Maynard A (1990). Harsh choices for UK NHS. Scrip; 1568: 7-8.

McCarty D J (1989). Arthritis & Allied Conditions. Lea & Febiger; Philadelphia.

McKenna F, Wright T (1985). Pain and rheumatoid arthritis. Ann Rheumatol Dis; 44: 805.

Meenan R F (1991). Rheumatology Manpower – The US Perspective. Editorial. Brit J Rheum; 30: 81-85.

Meenan R F, Yelin E H, Nevitt M, Epstein W V (1981). The impact of chronic disease. Arthritis Rheum; 24: 544-549.

Melvin J L (1989). Rheumatic Disease: occupational therapy and rehabilitation. 3rd Ed. F A Davis Co.

Mitchell D (1985). Epidemiology. Chap 6. In: Ustinger P D, Zvaifler N J, Ehrlich G (1985). Rheumatoid Arthritis. Etiology, Diagnosis, Management. Lippencott.

McPherson Brown T, Scammell H (1988). The Road Back – Rheumatoid Arthritis: Its Cause and Its Treatments. Macmillian London.

Murray-Leslie C F, Lintott D J, Wright V (1977). The knees and ankles in sport and veteran military parachutists. Ann Rheum Dis; 36: 327.

Mutro D, Laakso M, Isomaki H, Koota K (1985). Ten year mortality and causes of death in patients with rheumatoid arthritis. Brit Med J; 290: 1797-1799.

National Center for Health Statistics (1966). Public Health Service, Publication No 1000, Series 11, No 15, Washington, US. Government Printing Office.

Newman S P, Fitzpatrick R, Lamb R, Shipley M (1989). The Origins of Depressed Mood in Rheumatoid Arthritis. J of Rheumtol; 16: 6: 740-744.

Novosti Press Agency (1990). The Search for a Rheumatoid Arthritis Treatment. Advances of Science & Technology; 18: 233.

OHE (1973). Rheumatism and Arthritis in Britain. OHE.

OHE Compendium of Health Statistics (1989). OHE.

OPCS, RCGP, DHSS (1986). Morbidity Statistics from General Practice. Third National Study. 1981-2. London: HMSO.

OPCS (1989). General Household Survey. London: HMSO.

OPCS (1988). Survey of disability in Great Britain. Vols 1-6. London: HMSO.

OPCS (1991). Mortality Statistics: cause 1989. London: HMSO.

Persellin R H (1989). The effect of pregnancy on rheumatoid arthritis. In: McCarty D J (ed). Arthritis and Allied Conditions. Philadelphia: Lea & Febiger.

Peyron J G (1979). Epidemiologic and Etiologic Approach of Osteoarthritis. Seminars in Arthritis and Rheumatism; 8: 4: 288-307.

Peyron J G (1986). Osteoarthritis: the epidemiologic viewpoint. Clin Orthop; 213: 113-119.

Phillips H (1989). A patient's guide to hip and knee replacements. Park Sutton Ltd.

Prior P, Symmons D P M, Scott D L, Brown R, Hawkins C F (1984). Cause of death in rheumatoid arthritis. Brit J Rheumatol; 23: 92-99.

Puranen J et al (1975). Running and primary arthritis of the hip. Brit Med J; 2: 424-425.

Quam J P, Michet C J, Wilson M G, et al (1991). Total Knee Arthroplasty: A population-based study. Mayo Clin Proc; 66: 589-595.

Rand J A, Ilstrup D M (1991). Survivorship analysis of total knee arthroplasty. J Bone Joint Surg; 73A: 397-409.

Rapoff M A, Christopherson E R (1982). Compliance of paediatric patients with medical regimens: a review and evaluation. In: Stuart R B (ed): Adherence, Compliance, and Generalization in Behavioral Medicine. New York, Brunner/Mazel.

Ropes M W, Bennett G A, Cobb S (1959). 1958 Revision of diagnostic criteria for rheumatoid arthritis. Arthritis Rheum; 2: 16-20.

Ropes M W et al (1957). Proposed diagnostic criteria for rheumatoid arthritis. Ann Rheum Dis; 16: 118-125.

Silberberg R, Silberberg M (1971). Skeletal growth and aging. Docum Geigy Acta Rheumatologica, Ciba-Geigy-Bale.

Silman A J (1983). Has the incidence of rheumatoid arthritis declined in the UK? Br J Rheumatol; 27: 77-78.

Sokoloff L (1969). The biology of degenerative joint disease. Chicago: The University Press of Chicago.

Solomon L, Robin G, Valkenburg H A (1975)a. Rheumatoid arthritis in urban South African Negro population. Ann Rheum Dis; 34: 128-135.

Solomon et al (1975)b. Rheumatic disorders in the South African Negro. Part 1. Rheumatoid arthritis and ankylosing spondylitis. S Afr Med J; 49: 1292-1296.

Spector T D, Thompson P W, Evans S J W et al (1988). Are slow-acting antirheumatic drugs being given earlier in rheumatoid arthritis? Br J Rheumatol; 17: 498-499.

Stauffer R N (1989). Correction of arthritic deformities of the hip. In: McCarty D J (ed). Arthritis and Allied Conditions. Philadelphia: Lea & Febiger.

Stewart A L, Greenfield S, Hays R D et al (1989). Functional Status and Well-being of Patients With Chronic Conditions. JAMA: 262: 7: 907-913.

Symmons D P M, Jones S, Hothersall T E (1991). Rheumatology Manpower in the 1990's. Brit J Rheum; 30: 119-122.

The Health of the Nation (1991). London: HMSO.

Utsinger P D, Zvaifler N J, Ehrlich G E (1985). Rheumatoid Arthritis. Etiology, Diagnosis, Management. Lippencott.

Vandenbroucke J P, Hazevoet H M, Cats A (1984). Survival and death in rheumatoid arthritis: a 25 year prospective follow-up. J Rheumatol; 11: 158-161.

Wasner C K, Cassady J, Kronenfield J (1980). The use of unproven remedies (abst). Arthritis Rheum; 23: 759.

Williams A (1985). Economics of coronary artery bypass grafting. Brit Med J; 291: 326-329.

Wingrave S J (1978). Reduction in incidence of rheumatoid arthritis associated with oral contraceptives. Lancet; 1: 569-571.

Wood P H N (1978). Special meeting on: nomenclature and classification of arthritis in children. In: Munthe E (ed). The care of rheumatic children. EULAR monograph Series No 3. Basle: EULAR Publishers.

Wright V (1989). Osteoarthritis: Diagnosis is not as simple as it seems. Brit Med J; 299: 1476-1477.

Wright V, Hopkins R, Burton K E (1979). What shall we teach undergraduates? Brit Med J; i: 805-807.

Zinn W M (1982). Rehabilitation of patients with chronic polyarthritis. International Rehabilitation Medicine; 4: 114.

Acknowledgements

The Office of Health Economics wishes to acknowledge the assistance and advice of the following:

Arthritis Care, 5 Grosvenor Crescent, London

Dr Deborah Symmons, Deputy Director
ARC Epidemiology Research Unit
Stopford Building, Oxford Road, Manchester

Dr R G Hull, Consultant Rheumatologist
Queen Alexandra Hospital
Cosham, Portsmouth

Useful Addresses

Arthritis Care
5 Grosvenor Crescent
London SW1X 7ER

Arthritis & Rheumatism Council
41 Eagle Street
London WC1R 4AR