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Rheumatology is a little-known field, but one that is expanding fast. With the ageing of the population, individuals are suffering more and more from rheumatism – or at least they say they are – whether inflammatory or degenerative.

With this new publication, we wish to share our experience of the rheumatological pathologies that we encounter most often in our day-to-day rating work, and that we examined in some detail at the SCOR Medical Seminar. These seminars are an opportunity for us to share with you our reflections on the latest medical advances and their impact on the life insurance profession.

This Focus is intended to provide a rapid overview of rheumatology – a medical speciality in its own right in France, which is not the case in all European countries.

It is important to know the differences between the different pathologies, as this will help you to assess the risk and offer the most suitable conditions of acceptance.

It is for this reason we are going to look in depth at the pathologies most often encountered in the applications submitted to insurers:

- Osteoarthritis
- Rheumatoid arthritis
- Ankylosing spondylitis
- Osteoporosis
Rheumatology is the medical speciality that deals with bone diseases, but not exclusively. It includes:
- bone and joint pathologies
- peri- and abarticular pathologies, which affect the joint and the surrounding area: tendons, muscles, etc.
- musculoskeletal diseases
- systemic diseases (diseases close to Internal Medicine).

**Reminder**

A joint is a point where bones come together. The end surfaces of those bones are covered with articular cartilage and located inside a cavity known as the synovial cavity. The synovial membrane is the tissue that lines the inside of the joint and secretes the synovial fluid, a sort of “engine oil” for the joint. There is also an articular capsule, which provides the congruence of the joint, which is moved by muscles and tendons. The muscle is the part which contracts to move the bone and the tendon is the end part of the muscle which attaches the muscle to the bone.

1. **Main types of rheumatism**

The classification given below is a general and schematic one and may fail to take account of certain interactive and interlinked phenomena.

Generally, we use the term osteoarthritis when the cartilage is affected.

When the synovial membrane (the tissue that lines the inside of the joint) is affected or inflamed, we speak of arthritis. The synovial tissue secretes fluid, causing the joint to swell. Proliferation occurs, with tissue hypertrophy, and the inflammation “eats” into the cartilage and the bone, destroying the joint. The phenomenon is therefore different from that of osteoarthritis.

Tendinitis is the term used when the tendons are affected.

If the bone is affected, it can lead to osteoporosis. In fact, bone is a living organism, consisting of protein trabeculae which form a framework. When these trabeculae are damaged, the bone becomes fragile. Osteoporosis remains the most well known, but there are several other types of bone lesions.

When the muscle is involved, we talk of myopathy, myositis, etc.

When several organs are affected at the same time, we refer to systemic disease. This could be, for example, rheumatoid arthritis complicated by vasculitis with skin involvement, which can lead to serious cutaneous necrosis.

Traditionally, rheumatism is classified in two categories: “mechanical” pathologies and inflammatory pathologies.

2. **“Mechanical” pathologies**

These are pathologies without systemic inflammation. The most important of these pathologies is osteoarthritis. In this category we also find different bone lesions. Thus it will include osteoporosis, algodystrophies (particular forms of bone demineralisation), osteonecroses (holes in the bone), tendinopathies, abarticular rheumatism, etc.

3. **Inflammatory pathologies**

Inflammatory pathologies fall into four main categories:

- **Systemic diseases and connective tissue diseases**

It is in this first category that we find all the important rheumatological pathologies, and first of all rheumatoid arthritis. This is a very common disease, characterised by its importance in terms of disability, prognosis and care. Connective tissue disease covers disorders involving the conjunctive tissue.

Spondylarthopathies represent the second large type of pathologies. This is a general term which encompasses ankyllosing spondylitis or rheumatic pelvispondylitis, enterocolitopaties, etc.

We will also add to the list lupus, a complex pathology which is very difficult to assess in terms of impaired risk.

We also find other pathologies such as fibromyalgia. Although it has not been proved that this illness is one of the inflammatory pathologies, SCOR Global Life is consulted more and more often for cases involving
fibromyalgia, which are always delicate to assess. It is a condition that can lead to invalidity and major disability without necessarily threatening survival.

- **Metabolic or microcrystalline rheumatism**
  
  These are forms of rheumatism where there is crystal deposition in the joints. The classic example would be gout, which is most commonly known to the general public in its form affecting the big toe.

  Articular chondrocalcinosis consists of calcium pyrophosphate crystal deposition in the joints, which can lead to articular pain and arthritis.

  Hemochromatosis, or iron overload, consists of an accumulation of iron in the joints.

- **Infectious pathologies**
  
  Microbes can reach all the parts of the body, including the bones and joints. When a microbe or a germ reaches a joint by iatrogenic means, following an inopportune medical gesture or due to a particular terrain, this can lead to septic arthritis.

  In the vertebral column, the germs can reach the intervertebral disks and the super- and sub-jacent vertebrae. In these cases, we talk of spondylodiscitis.

  The germ may reach the bones themselves, causing infectious osteitis.

  Osler’s disease is a particular disease with which cardiologists are regularly confronted, where the germs are located in the heart valves. This disease can cause rheumatological manifestations, by septic embolism or other phenomena.

- **Tumoral pathologies**
  
  The bones can also be affected by tumours and cancers. Myeloma is a particular blood disease in which an abnormal secretion of an immunoglobulin (blood protein) attacks the bone. Leukaemia is another blood disease that can affect the bone. Primitive bone tumours start in the bone cells. They may be benign or malignant. Bone metastases are secondary tumours.

  Paraneoplastic syndromes are cancers which can cause manifestations in the bones or joints without there being any cancerous cells in the bones or joints. These are cancer-related manifestations.
Osteoarthritis today: some new concepts

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The typical arthritis sufferer is an elderly person who complains of pain to his doctor. The doctor will generally explain that “it’s old age”, that his joints are worn out and will prescribe anti-inflammatory drugs. Imagine yourself in the place of that patient: you are being told that it is the end, that you are a victim of old age, that medicine cannot do much for you and you resign yourself to taking the anti-inflammatory drugs. None of this is likely to cheer you up much. And yet it was still the way that we saw things about ten years ago. But now that traditional view is being overturned. This illness, which used to be neglected in medical schools because there was not much to say about it compared to others such as rheumatoid arthritis or connectivitis, is today being reconsidered following numerous medical advances.

Reminder of what osteoarthritis is

Osteoarthritis is a narrowing of the joint space. On the X-ray below, we can see that the joint space has disappeared [1]. Moreover, the bone is condensed and we can see bony constructions appearing, known as osteophytes. If we open up a joint, a knee for example, we may find some remaining healthy cartilage, but osteoarthritis is essentially characterised by the destruction of the cartilage. Osteoarthritis affects particularly the lower limbs, the knee and the hip, and, in the upper limbs, mainly the hands. A particular form of osteoarthritis is that of the distal interphalangeal joints, which leads to nodular formations that deform the joints, with consequences that are unattractive and painful. If the deformation is too pronounced, it can even become a disability, with the sufferer having difficulties holding objects. In this case, it is no longer just a case of the joint space narrowing: the cartilage is destroyed, and we see osteophytes forming or subchondral cysts, “holes” in the bone.

The other common location of osteoarthritis is the vertebral column: from the age of thirty-five, 100% of patients are affected by osteoarthritis in the vertebral column. The cervical rachis is affected in all cases, whereas, for some, the lumbar segment is unaffected. Osteoarthritis is characterised by osteophytes, lipping of vertebrae and condensation of the bone.

Osteoarthritis in the XXIst century

Two hitherto incontrovertible truths or “dogmas” about osteoarthritis are in the process of collapsing.

First dogma: osteoarthritis is a “disease of the old”

In 1933, we could say that osteoarthritis was to the joints what wrinkles are to the skin, or white hair to the scalp. This is no longer true, because the population is ageing [2] and osteoarthritis still starts at the same age. However, the notion of old age is a sociological concept. Being sixty-five in 1930 may have meant being old, but this is no longer the case today. Now, at sixty, we are enjoying our grandchildren, travelling, etc. Osteoarthritis generally starts at about sixty and peaks at the age of seventy. So
it is no longer possible to say that osteoarthritis is a “disease of the old” and to explain to these patients that it is because they are old that they are suffering. Of course, it is an age-related disease, but we can no longer content ourselves with this fatalist view, either from the point of view of the patient’s feelings or in terms of the doctor’s of addressing the problem.

The peak prevalence of osteoarthritis is situated at about sixty-five. The Americans and British, who observe in a much more pragmatic way that joints can sometimes be inflamed and painful and that the bone can be affected, have coined the expression “osteoarthritis” whereas the French speak of “arthrosis”. If we project ahead into the 2030s in the United States, individuals aged sixty-five to seventy-four years will represent 11% of the population in 2030, compared to 6.6% today. We know that we are moving towards an increasingly elderly population. We do not have such precise figures for other developed countries, but we can expect the phenomenon to be the same.

Second dogma: osteoarthritis is due to wear and tear on the cartilage

When we talk of wear and tear, we mean an alteration that appears to be extremely passive. Wear and tear occurs naturally as a result of repeated friction on any material, living or otherwise. Presented in this way, it is inevitable – a degenerative phenomenon that we cannot do anything about. This idea has now been completely called into question.

In 1920, it was believed that osteoarthritis was a disease of worn cartilage. Francis Broussais said that “any tissue that does not respond by inflammation to the causes of irritation or destruction that act on it may be considered as not being alive”. We say of a tissue that it is inflammatory when it is capable of expressing a certain degree of inflammation. Cartilage is a tissue that is not vascularised and therefore cannot manifest its inflammation. However, cartilage is in fact capable of expressing a certain degree of inflammation as it is defined today.
Osteoarthritis today; some new concepts

To refer to osteoarthritis, the terms “chronic senile rheumatism” or “chronic degenerative rheumatism” have also been used.

A definition of osteoarthritis

Osteoarthritis is a loss of cartilage, but not only that. Researchers are now taking an interest in synovial tissue, for osteoarthritis is also characterised by a certain degree of synovial inflammation, which has nothing to do with rheumatoid arthritis. They are also studying the bone more closely.

Cartilage is normally a smooth surface, whose superficial layer may crack. The tissue consists of cells, chondrocytes, which give it the character of living tissue. Chondrocytes, which are set in the cartilage matrix, tend to cluster at the edge of cracks; we even have the impression that they multiply, in a sort of reaction. Unfortunately, this reaction does not appear to be sufficient: the cartilage becomes detached and fragments fall into the articular cavity. The inflammation can be seen when we examine the synovial tissue under the microscope, in the form of blue focal points corresponding to the inflammatory cells. These are focal lesions with a certain degree of inflammation.

Another characteristic feature of osteoarthritis is bone modification[3]. Osteophytes (bone proliferation) are created as well as subchondral cysts (holes) underneath the cartilage in the subchondral bone. These are cracks that affect first the cartilage, then the bone and which act like valves: the synovial fluid enters, fills a small cavity, but cannot get out again, leading to the formation of a hole. This is how the subchondral cysts that we observe are now explained.

Cartilage is a tissue with a high content of sugar, due to the numerous glycoproteins it contains and which increase its capacity to retain water molecules. Cartilage that contains plenty of water has good mechanical properties. Indeed, it has to be extremely effective from a biomechanical point of view to be able to sustain several thousand movements a day. With osteoarthritis, the cartilage loses its sugar-rich proteins, and the type II collagen which makes up the framework of the cartilage is modified to become a collagen of a different type and lesser quality. These phenomena generate a poor quality matrix, rendering the cartilage more fragile and less able to cope with mechanical stress.

The second important point is the deterioration of the matrix, which is subjected to a very active process of destruction: enzyme synthesis by the chondrocytes. These cells start to synthesise enzymes that weaken the matrix. Thus we have gone from an extremely passive to a very active system; the degradation of the matrix is not the consequence of wear and tear, but of increased chondrocyte activity. We can therefore talk of inflammation, if we define the latter as an increase in the activity of a tissue.

It has been discovered that osteoarthritis is caused by a synthesis imbalance. In healthy cartilage, there is a bal-
An imbalance between anabolic factors, which produce the matrix, and catabolic factors, which destroy it. Osteoarthritis causes an imbalance in favour of the catabolic factors. There are then more matrix-destroying enzymes than anabolic factors capable of producing new matrix material.

Researchers wondered why chondrocytes start to synthesise more of these enzymes, called metalloproteases (MLP). If the chondrocyte starts to produce more MLPs, it is because it receives certain information in its cell membrane. Then, by “pressing” on a “button” – the receptor – a whole signalling cascade starts up from the membrane to the nucleus, where the library of all our genes is situated. For protein synthesis to take place, the book has to be taken from the library, then read. Pressing on the button engages a process that culminates in the book being removed from the library to be read. It is this book that allows the synthesis. This intercellular signalling leads to increased MLP synthesis. Today, we know what presses the button: it involves, in particular, pro-inflammatory cytokines such as IL1 (interleukin 1) or TNF (tumour necrosis factor). Other elements are also involved, which constitute different types of push-buttons. These aspects are important from the therapeutic point of view, for if we can prevent the molecules from communicating with each other, we can avoid MLPs synthesising; if we can inhibit the enzymes, we can prevent the degradation of the cartilage; if we prevent the button being pressed, the intercellular signalling will not take place, etc. We are beginning to see the different therapeutic targets of the future.

### Initiation of the osteoarthritis process

There are two hypotheses on this subject. A first approach concerns mechanical stress, an overload on normal cartilage resulting in it becoming degraded. Obesity – a risk factor in osteoarthritis of the knee – is clearly an example of mechanical stress, with excess weight constantly pressing on the knee cartilage. Genu varum or genu valgum, knee deformities, are other cases of mechanical stress.
exerted on the inner or outer compartment of the knee, depending on the case.

Although we are talking of mechanical stress and overload, this does not mean wear and tear for all that. In fact, mechanical stress plays a role in activating the chondrocytes. Certain receptors situated in the chondrocyte membrane are capable of responding to mechanical stress. In the case of overload, these receptors, the integrins, trigger the signalling cascade in the cell, which then starts to synthesise more enzymes. It is the overload that gives the cell a signal so that it starts to produce enzymes that degrade the matrix. This phenomenon can be studied in a laboratory by taking small pieces of cartilage and compressing them to see how the cells are activated.

The second hypothesis concerning the initiation of the osteoarthritis process concerns abnormal cartilage. The idea is that the cartilage is not of good quality from the outset. In this case, a genetic cause is suggested. In the case of osteoarthritis of the hands, overloading is rare and concerns only a few professions (seamstresses) or certain peoples (the Chinese, who eat with chopsticks). To explain osteoarthritis of the hands, the genetic factor is unknown, but a genetic predisposition exists. It has also been noted that patients affected by osteoarthritis of the hands also suffer from osteoarthritis of the hip or knee.

To simplify\(^{[5]}\), the activation of the chondrocytes results in a matrix altered by MLP synthesis. We may consider this a mechanical pathology, and yet this mechanical factor does not lead to a degenerative disease, but to a truly active disease. Certain molecules are capable of activating the chondrocyte: cytokines, in particular IL1 for cartilage. The genetic factor is also involved in weakening the cartilage.

The genetics of osteoarthritis are still a little-known field, but there are many teams working on this subject around the world.

To go from asymptomatic osteoarthritis to symptomatic osteoarthritis, there must necessarily be a trigger. 50% of patients suffering from osteoarthritis of the knee, for example, will feel pain and only 10 or 15% will end up going to see a doctor.

A study by Michel Lequesne, renowned for his work on osteoarthritis of the hip, observed that out of 100% of the population, 60% will suffer from anatomical osteoarthritis, 30% from radiological osteoarthritis, 15% from symptomatic osteoarthritis and only 5% will consult a doctor.

Symptomatic osteoarthritis is the combination of anatomical osteoarthritis, pain and possibly a disability linked to joint destruction. This notion of disability is important. In the United States, arthritis or rheumatism represent 17% of the causes of disability and 13.5% of cases of lumbago, which is considerably higher than the other causes of disability. Locomotor disability is a major disability compared to other possible disabilities. Also, the older the patients get, the more types of disability accumulate. If we separate out the different causes of disability, we find that osteoarthritis is a serious public health issue.

The consequences of osteoarthritis are considerable in terms of the number of days confined to bed and sick leave generated.

It is possible to measure the scale of the disability by the frequency of total joint replacements. Fitting an artificial joint supposes in fact that the patient is suffering from a
major disability. Now, an increase in total knee replacement operations in people over sixty-five has been observed. This is good news and bad news: surgeons no longer hesitate to carry out such joint replacements, but the frequency is increasing due to the ageing of the population. Projections for 2030 on the American population confirm this trend.

The same applies to total hip replacements. The number of hip replacements in the United States will practically double by 2030. We can suppose that other developed countries will follow more or less the same pattern.

How to correlate what is anatomical and what is symptomatic?

If an X-ray shows a case of asymptomatic osteoarthritis, does this mean that the patient has a greater risk of developing serious osteoarthritis culminating in a joint replacement or dependency than a person presenting symptomatic osteoarthritis with the same anatomical degree of destruction? Certain studies can provide the beginnings of an answer.

A 1999 study on total joint replacement at three years revealed that being in pain (over fifty on an analogical visual pain evaluation scale of one hundred) multiplies by almost two the risk of needing a total hip or knee replacement. A recent study goes in exactly the same direction for osteoarthritis of the hip, and it has also been confirmed for osteoarthritis of the knee. A patient who has pain in the knee and whose X-rays reveal osteoarthritis effectively has a greater risk of having a knee replacement within three years. Radiological osteoarthritis discovered during a health check-up may develop into more serious osteoarthritis, but the risk is less than in cases when the osteoarthritis is discovered following the appearance of pain.

This phenomenon is difficult to explain. A first method consists of comparing the patients suffering from symptomatic osteoarthritis with the others by using MRI
Treatments today are different from what it will be 2020. A genuine revolution, similar to the one we saw with rheumatoid arthritis between 1990 and 2000, is currently in progress.

Today, there are two types of treatments: drug treatments and non-drug treatments.

- Non-drug treatment
This consists of ensuring that patients maintain a decent level of physical activity. Regular activity helps combat other, related disabilities to which attention must be paid. Physical exercise plays a major role in maintaining a certain quality of the joint. This role has been demonstrated essentially in terms of quality of life, more than...
in terms of pain. As for rheumatoid arthritis, there are disease-modifying treatments and symptomatic treatments. There is no disease-modifying treatment for osteoarthritis other than physical exercise.

**Drug treatment**

There are several types of symptomatic drug treatments: analgesics, anti-inflammatories, symptomatic slow-acting drugs in osteoarthritis (Chondrosulf®, glucosamine sulfate, etc.), which have practically as little effect on osteoarthritis as paracetamol. Today, their prescription is linked to the presence of pain. We cannot say that they act as an anti-osteoarthritis treatment.

Drug treatment is slow acting and takes a few weeks to take effect. It also has a remanent effect after several weeks, if it is stopped. Whatever modern marketing says, this is a symptomatic treatment. Some very interesting studies are being conducted which may show a slight effect on the degradation of the cartilage, but their results are controversial for the moment.

There are other symptomatic treatments:

- local treatments, in the form of intra-articular injections of corticoids, effective in inflammatory osteoarthritis with joint effusion, nocturnal awakening or morning stiffness, or in the form of injections of hyaluronic acid, a symptomatic treatment considered as effective by rheumatologists and which constitutes an alternative that can sometimes be interesting if previous treatments have not been enough;

- physiotherapy (massages) can also help, with, for example, the rehabilitation of the vastus medialis in the thigh for femoropatellar osteoarthritis;

- the last of the various means used is total joint replacement.

And treatments of the cause, obesity in particular, should not be forgotten. A very good study has shown that weight loss is effective, but that certain sports must be avoided. Thus, a patient with osteoarthritis of the hip or osteoarthritis of the knee must avoid practising court and track sports (basketball, volleyball, athletics) at too high a level, whereas cycling and swimming pose no problems. There is, moreover, a list of occupational illnesses associated with osteoarthritis: tilers suffer from femoropatellar osteoarthritis; vibrating tools cause osteoarthritis of the elbow.

We have already moved on from the era of the degenerative disease due to wear and tear and treated with analgesics to multidisciplinary treatments of osteoarthritis, as exist for rheumatoid arthritis for example. The treatment of osteoarthritis must be approached in different ways.

Pharmaceutical companies are today trying to discover molecules capable of stopping the vicious cascade that leads to the degradation of the cartilage, by different means such as anti-cytokines. We are beginning to come closer to the drugs used to treat rheumatoid arthritis. Anti-TNF therapy is all the rage in rheumatoid arthritis at the moment, and rightly so. For osteoarthritis, a trial is underway which consists of injecting anti-IL1 into the joint, which no-one would have believed possible twenty years ago.

In 1973, Stanislas de Sèze, one of the great names in French rheumatology, expressed his wonder about the subject in these terms: "Will all these still fragile hypotheses one day lead to some progress in osteoarthritis therapy, which, let’s admit, is in serious need of some? We have to admit that, for the last half century, medical therapies for osteoarthritis have remained pitiful. Why should the dream of regenerating osteoarthritis cartilage not become a reality one day, at a time when we can see, on both sides of the Atlantic, that enzyme biochemistry is occupying the shrewdest researchers? The hope that the brains and test tubes of the modern alchemist will produce a therapy capable of regenerating osteoarthritis cartilage now seems no more irrational than it was twenty years ago to imagine a man walking on the moon. “ Stanislas de Sèze was very clear sighted. It has certainly taken longer than he hoped, but the research is advancing.
What is happening with the research on cartilage transplants as a treatment for osteoarthritis?

The idea is a simple one, the idea being to “plug the holes” that affect the cartilage. A Swedish team has conducted a study on this topic. After taking samples of cartilage from a non-weight bearing area, they extracted the chondrocytes and then cultivated them. They then recovered a pellet of chondrocyte cells capable of producing the cartilaginous matrix and positioned them in the hole in the cartilage. Then they used some bone to plug the hole over the cartilage to prevent the cells escaping. Eventually, the bone disappears and the cells have had the time to produce some matrix to fill up the hole. The idea is interesting, intelligent and has led to a very good publication showing the plugging of the hole. But the patients chosen were not suffering from osteoarthritis at all. They were young patients who had suffered trauma and whose cartilage was healthy, except for the place where the hole was, because of the trauma. For several patients, it gave positive results, but there was no control group. We do not know what would have happened if the hole had not been plugged. Furthermore, the study dates from several years ago and it is difficult to know what has become of these patients or what becomes of new patients treated in this way. A biotechnology company has gone into business in this market and the data has become difficult to obtain. It is possible today to send fragments of cartilage to this company, which multiplies the chondrocytes in the samples and then sends them back for use, although this technique is still very “acrobatic”.

In osteoarthritis, the rest of the cartilage is not healthy. There is a great deal of research going on into transplants. But today, it is difficult to get round the problem that resides in the fact that the peripheral cartilage is not healthy. Attempts are being made to understand the chondrocyte better to see if it is possible to modify it when it is diseased and can no longer produce enough matrix. Cell therapy enables us to better understand how this cell is different. We have some leads, but we cannot yet talk of effective cartilage transplants in osteoarthritis.

Do diabetics suffer less from osteoarthritis than other people? Is it possible to imagine injecting sugar into cartilage?

This is a question that is beginning to be asked, but for other reasons. Diabetics, like osteoarthritis cartilage, produce glycation end products, which accumulate in certain places and which have the ability to activate the chondrocyte. An epidemiological study on the frequency of osteoarthritis in diabetics compared to non-diabetics is ongoing.
We are going to examine the assessment of the risk involved in osteoarthritis in insurance, with first of all the case of a young applicant suffering from single site osteoarthritis, located only in the metatarsophalangeal joints. The insurer and the re-insurer had to deal with a claim for several million Euros. What sort of claim was it? It was a professional footballer belonging to a famous team, who had certainly over-stimulated his chondrocytes by kicking the ball too much. The consequences of this repeated mechanical stress were difficulties in running and therefore in playing football. This example shows that osteoarthritis can lead to disability.

We will look at the general rating rules. However, there are special cases for which it is necessary to produce custom solutions, drawing on our experience, in particular in cases of osteoarthritis that are unusual in their location and intensity, and which are connected to the person’s activity and/or profession.

It should be noted that dorsolumbar pain (pain in the vertebral column which may be osteoarthritic in origin) is the second main cause of people taking time off work.

### Death/TPD risk assessment

We asked ourselves a simple question: does suffering from osteoarthritis constitute an excess risk in terms of mortality? Can having worn cartilage, or having undergone a great deal of mechanical stress which has stimulated the chondrocytes in the cartilage, be correlated with a more general wear and tear on the body and therefore with higher excess mortality? Apparently, no one study has shown that osteoarthritis sufferers, at equal age, have a higher mortality rate than non-osteoarthritis sufferers. That being the case, osteoarthritis increases with age, as does mortality.

In principle, in the case of an applicant suffering from osteoarthritis (osteoarthritis of the hip or the knee), the risk is normal in most cases. Sometimes we may see an excess mortality, in particular if the applicant declares osteoarthritis covered 100% by the Social Security, as this pathology is not on the list of illnesses covered 100%*. If the doctor or the applicant indicates that the osteoarthritis is covered 100%, this means that it is a serious or incapacitating case. If the person is classed as disabled for osteoarthritis, this osteoarthritis is probably already severe, as it must be if it involves the continuous and significant taking of analgesics or NSAIDs, or if a joint replacement has already been done or is planned. In these different cases, we will apply a slight excess risk of 25%.

Two examples to better understand the reason for this excess risk:

The first concerns an individual who declared osteoarthritis covered 100%. It was osteoarthritis of the vertebral column, with a narrow lumbar canal, which was slowly compressing the spinal cord, leading to progressive paraplegia.

Regarding TPD, the risk was certain and his osteoarthritis was covered 100%.

The second example concerns an individual who declared that he suffered from osteoarthritis of the hip, treated by a total hip replacement. His operation resulted in MRSA and required a further operation.

In conclusion, in most cases we can qualify the risk as normal.

### Inability to work and disability

Dorsolumbar pain is the cause of a large amount of sick leave in France. In most cases, an exclusion is necessary. This is not satisfactory, but we have not found a better solution for the moment. We do everything we can to accept applicants classed as having an “inability” at reasonable rates. We are aware of the limits of exclusion, but it must be accepted that it must be applied quite often in the case of osteoarthritis.

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* The French health insurance system covers all the expenses relating to certain illnesses requiring prolonged and expensive treatment. These illnesses are known as “long-term illnesses” and feature on a precise list, but patients may obtain 100% cover for illnesses not on the list if they are particularly serious.
If the osteoarthritis is covered 100% by the French Social Security, if the person is already classed as disabled for osteoarthritis, and if he is continuously taking major analgesics or NSAIDs for osteoarthritis, prudence requires that we refuse such applicants for inability and disability.

The exclusion (the most common case) must be clear, unambiguous, comprehensible by the applicant, limited and formal. It must really be “obvious” to the applicant, who must be able to understand it. When claims are made, it is necessary to avoid disputes and conflicts between experts, which is not always easy.

By way of example, if an applicant were to declare osteoarthritis of the vertebral column, we would propose the exclusion of “any degenerative disco-vertebral, lumbosacral, cervical or dorsal impairment according to its location, repercussions and consequences”. This exclusion has the advantage of covering the intervertebral discs and vertebrae. However, the term “degenerative” is liable not to be understood by an applicant, as is “disco-vertebral” and so we prefer the following exclusion: “With the exclusion of any impairment of the vertebral column or any paravertebral impairment which is not due to an infection, a tumour or a fracture, its repercussions and consequences.” In this case, we explain what we will cover and we exclude all the rest. This exclusion has the advantage of being simple and comprehensible, but it is not applicable in all cases and cannot avoid all disputes.

In the case of osteoarthritis of the knee, the exclusion may concern “any degenerative impairment of the knee” or “any impairment of the knee which is not due to an infection, a tumour or a fracture”. This has the advantage of being simple, limited and precise even if no exclusion is perfect. The future and case law will tell us if these exclusions are correct.

Most often therefore we use an exclusion and from time to time we resort to a refusal.

Before we look at rating, let’s take another case: a thirty-five year old woman has declared generalised osteoarthritis and is on the point of being declared disabled although her X-rays are normal. What condition is this? She is suffering from fibromyalgia. Fibromyalgia has nothing to do with osteoarthritis, but sometimes it appears under the terms polyalgesia or generalised osteoarthritis, etc. It generally concerns young women with an environment of fibromyalgia (numerous treatments, hospitalisation, multiple check-ups and pains...). We should be wary of fibromyalgia, which can be hidden behind generalised osteoarthritis.

Rating remains possible in a case of more or less systematic, not very severe, non-disabling osteoarthritis which does not require any particular therapy. The excess risk may be 25, 50, 75%, etc. depending on the type of osteoarthritis and the person’s profession for certain particular types of osteoarthritis. Assessment will be on a case-by-case basis.

### Dependency

In France, osteoarthritis is present in very many dependency contracts. This seems normal insofar as these cases concern people who tend to be quite old. For the selection of the risk, everything begins with a simplified medical questionnaire completed by the applicant. If the latter declares a treatment, a disability or 100% Social Security cover, it will then be necessary to obtain a medical questionnaire completed by the doctor. Treated osteoarthritis is also concerned. For the dependency rating of osteoarthritis, the doctor will be asked in this questionnaire if the patient is capable of carrying out the tasks of daily life (walking, shopping, etc.). Whenever motor dysfunction is reported, especially concerning the lower limbs, the application is refused. An individual who declares osteoarthritis and is already using a walking stick will not therefore be able to be insured for dependency.

Depending on the location of the osteoarthritis, the number of joints affected (mono-, oligo-, poly-articular), the existence of a disability, the total coverage of health expenses by the public health insurance system, a planned hip or knee replacement, the risk will vary from a normal risk to a refusal, even a deferment if certain information is missing. Total or partial dependency cover may be granted for the least serious cases. In the most serious cases, partial dependency cover will not be granted. Only total dependency cover will.
In the case of osteoarthritis of the knee which has led to an exclusion, if the individual dislocates his knee, is he covered or not, bearing in mind that you only mention fractures, tumour or infection in the exclusion?

Dr Dominique Lannes

The claim should in fact be accepted as it is not connected to the osteoarthritis, but given that a dislocation is trauma and not a fracture, it will not be. When writing the exclusion, we hesitated between the terms “fracture” and “trauma”. This is a sensitive subject, since in plenty of cases we find that an insured person who suffers such trauma is not covered, whereas he deserves to be as this is not connected to the osteoarthritis. This is the limit of the exclusion, which can sometimes turn out to be unfair. We are continuing to consider the possibility of including trauma.

Michel Dufour

The aim was to be more restrictive in the wording of the exclusion and to give more leeway in examining the claim.

Dr Dominique Lannes

Indeed, but if we take the exclusion literally, in the absence of a fracture, the claim cannot be accepted.

Dr John Evans

We insure numerous professional footballers. We tend to exclude the joint when the anterior cruciate ligament, for example, has been operated on. And yet, often, the player starts playing professionally again and is able to continue his career without taking any time off. Do you think that the exclusion could be lifted during a footballer’s professional career? Or, once there has been an operation on a knee, do you consider that a reservation should be kept regarding that joint?

Prof Francis Berenbaum

This is a very difficult question. It all depends on the quality of the operation and whether the joint can be stable after the operation. If the knee remains unstable after the operation, the risk of osteoarthritis persists. In the case of a meniscectomy, even a partial one, the risk of osteoarthritis increases. In the case of an isolated tear in the cruciate ligament, when the operation repairs the ligaments and the joint is stable, with no related meniscal trauma, we can very well imagine that there will be no development of osteoarthritis. It all depends on the possibility of keeping a very stable joint or not. Whenever a sportsman is professional, there is hyper-use of the joint compared to a non-professional sportsman, which complicates things. From the outset, the professional already presents a risk. After knee trauma, everything depends on the sequelae and the performance of the operation. It is difficult to generalise, we have to work on a case-by-case basis.

Is the reasoning the same for a footballer who has suffered a torn cruciate ligament, but has not had an operation? Have any comparative studies been carried out on knees operated on and not operated on?

Prof Francis Berenbaum

I have no knowledge of any studies of this type. On the other hand, I have read a lot of studies on meniscectomies. It really is necessary to rely on the expertise of the orthopaedic surgeon, who is the...
only person able to assess whether the joint is stable or not from a clinical point of view. For a professional, it may be necessary to go further in assessing the stability. The key to osteoarthritis of the knee in sportsmen is stability.

Why use the expression “vertebral column” in the exclusion rather than “disco-vertebral column”?

Dr Dominique Lannes

The advantage of using the term “vertebral column” is that the applicant knows what it means. Professor Berenbaum also spoke of the vertebral column and he also knows what it means. The discs are a part of the vertebral column… We thought, from a legal (case law) point of view, that we needed a concept comprehensible to the applicant and which would lead to as few disputes as possible. We arrived at the conclusion that we must use very simple terms. The vertebral column encompasses, in our opinion, the whole of the rachis and the discs. We also talk of the “paravertebral region”. We have seen a large number of claims where lumbar pain has transformed into paralumbar or paravertebral pain. The term “paravertebral region” allows us to neatly cover this point.

Why not envisage a higher premium rather than an exclusion?

Dr Dominique Lannes

We do sometimes offer higher premiums, mostly in cases of non-serious, generalised osteoarthritis. In the case of osteoarthritis mainly located in one site, the knee or the hip, the more “ideal” solution is the exclusion.

Michel Dufour

It is true that this type of application exists. We may, however, ask ourselves why the insuree absolutely prefers to pay a higher premium rather than accept an exclusion. This is a good reason to examine the application closely and reconcile all the parameters (professional context, age, environment…). This raises questions. We can accept such an application in a few cases of very slight osteoarthritis, but from our point of view as re-insurers, we are undeniably suspicious of this type of request.

Dr John Evans

It is possible to cover oneself by playing on the waiting period. The longer the waiting period before the payment of professional disablement benefits, the more willing we are to examine the application. When the waiting period is very short, especially for manual or exposed occupations, we prefer the exclusion.
In arthritis, the highly vascularised synovial membrane becomes very inflamed and secretes an extremely inflammatory synovial fluid. It is a potentially very aggressive inflammatory disease \[9\].

The different form of arthritis

Numerous other diseases can be concealed by arthritis.

A Infectious or septic arthritis

First of all we have what rheumatologists dread – infectious arthritis. It is always necessary to try to eliminate this cause before starting to talk of any other type of arthritis. When a germ affects a joint, it will destroy it. When a joint swells, it is systematically necessary to suspect infectious arthritis and have a biopsy done by a rheumatologist in order to eliminate the possibility of an infection. Infectious arthritis includes tuberculosis, staphylococcal or streptococcal sepsis, etc.

B Microcrystalline arthritis

The second cause of arthritis is found mainly in very old people. Microcrystalline arthritis takes root when a for-
An overview of rheumatoid arthritis

eign body such as crystal of calcium or a uric acid crystal (gout) introduces itself into a joint.

Among the non-infectious and non-microcrystalline forms of arthritis, we find other diseases such as rheumatoid arthritis.

**Rheumatoid arthritis**

Rheumatoid arthritis is not only synovitis. Generally, it is associated with a thickening and a multiplication of the synovial membrane, which invades the articular cavity: synovial pannus. Arthritis is a polymorphic disease, which can be manifested by the impairment of a single joint (rheumatoid monoarthritis), less than five joints (rheumatoid oligoarthritis), or more than five joints (rheumatoid polyarthritis).

Rheumatoid arthritis is a multi-factor disease, that is to say, it is triggered by several factors. It occurs in a particular genetic terrain, but for it to manifest itself, there must also be environmental factors which probably play a much greater role than the genetic factors. Very little is known about the genetic terrain. We are making progress every day in this field thanks to the genetic studies that are being conducted. It seems that the HLA (Human Leukocyte Antigen) system carried by the white blood cells, which make it possible to carry out transplants whilst guaranteeing compatibility between donor and recipient, is very specific in cases of rheumatoid arthritis. Most of the time, we can find an HLA system specific to rheumatoid arthritis. We find DR4, or DRB104 by its new name, in 40 to 60% of cases, DRB101, or DR1, in 20 to 30% of cases. So there really is a particular genetic terrain. Numerous environmental factors must be taken into account and not all of them are known.

Smoking is one of the most interesting factors: we can explain to the patient that one of the first treatments
Evolution of rheumatoid arthritis

Rheumatoid arthritis is a very fickle and sly disease. Attacks are totally unpredictable and periods of remission vary in their length and extent. Now, the first question a patient suffering from rheumatoid arthritis asks concerns its evolution. The problem is that we do not know exactly how this pathology evolves. This unknown quantity in terms of evolution, specific to chronic illnesses such as diabetes or multiple sclerosis, places the patient in a situation of uncertainty regarding his future and can seriously interfere with the doctor/patient or doctor/carer relationship, with, into the bargain, considerable therapeutic problems.

With rheumatoid arthritis, the patient’s life is in total upheaval due to the mental and socio-professional repercussions. We do not always realise the suffering caused by rheumatoid arthritis. The situation must be explained to patients to ensure they do not neglect their illness or turn exclusively to alternative medicine.

The physical effects are obvious: the patient is in pain, constantly. He cannot sleep at night, he is stiff when he wakes up – this is known as morning stiffness – and he is tired. He cannot go to work, as he has difficulties getting out of bed, opening a pot of jam, or even taking a shower. His life is totally disrupted. When the joints swell, the disease becomes visible. But pain is eminently subjective and the patient is often seen as a “skiver” if he does not go to work or get out of bed. In the discussion groups we run, patients tell us that they feel very isolated and misunderstood, both by doctors and by those around them. Their illness is a silent and often invisible one. Driven by tiredness and pain, the patient risks letting himself go and becoming sedentary, finally losing his autonomy and a certain quality of life (social and daily) with nothing but disability to look forward to. It is therefore a major problem.

We use the HAQ, a functional capacity scale[^1^], to measure patients’ ability to dress, get ready, get up, walk...
or reach an object. It is not entirely satisfactory, but it is the only index recognised in France and in Europe.

The mental impact is also very important. Patients are stressed and anxious faced with an unpredictable disease that obliges them to resign themselves to no longer having the life they had before. They risk withdrawing into themselves and often find themselves isolated. They find themselves obliged to change their life habits, they have to have frequent blood tests, follow special diets and take drugs that are often toxic. Some will have to wear splints at night. This is a disease which has consequences on people’s relationships, their love and sex lives. These aspects are very worrying for patients, and it is as important to deal with these worries as it is to prescribe medicines for them.

From a socio-economic point of view, rheumatoid arthritis leads to repeated absences from work, long-term sick leave, people working part time for health reasons, professional redeployment. Many patients are obliged to give up work, although they would prefer to continue. Working often enables them to forget about their illness. Stopping work results in a loss of income, leading to economic insecurity. Added to this are repeated visits to doctors and hospital stays, operations and expensive treatments. Talking about this disease more might well contribute to improving its care.

### Demographic data

Rheumatoid arthritis affects 0.5 to 1% of the population according to the country. It is three times more common in women. The average age of onset is about forty-five, an age at which the professional activity is still important. According to a survey done by the French Society of Rheumatologie, the prevalence (number of patients at a given moment in a population) in France is 0.31%, which is equivalent to the number of patients suffering from spondylarthritide. The annual incidence (number of new cases in France each year) is about 8.8/100,000. About 300,000 people are affected every year in France. Associations give higher figures, but these are difficult to verify. According to data emanating from the French Social Security, the annual number of declarations of rheumatoid arthritis was stable between 1989 and 1996. The annual average is said to be 8,500 cases reported, with an approximate incidence of 20/100,000 for the population in question and overall prevalence of 0.4% for the population as a whole. This estimation is equivalent to 150,000 cases for France.

### Diagnosis and initial care

#### Early diagnosis

Rheumatoid arthritis must be diagnosed very early. This is why we need to change the current situation and talk about this disease more. Often patients consult their doctor only after the disease has been developing for two or three years, when it is already rather late. And yet, it is very easy to make an early diagnosis in middle-aged women suffering from polyarticular pains, affecting the hands in particular (proximal interphalangeal joints and metacarpophalangeal joints), especially in cases where both sides are symmetrically affected with swelling of the joints. However, there are misleading modes of presentation for which the early phase is more difficult.
to identify: this could be, for example, a man suffering from monoarthritis in the knee. In this case, it is necessary to think not of osteoarthritis, but of rheumatoid arthritis. Signs in the feet may also be specific to rheumatoid arthritis, but often we do not think of this condition. Morning stiffness leading to difficulties in getting out of bed can also be a symptom. Doctors should not hesitate to seek the opinion of a rheumatologist when at least three joints are swollen, where there are symptoms in the feet or hands, and if morning stiffness lasts more than thirty minutes.

The diagnosis can be confirmed by a few extra tests. The sedimentation rate and C-reactive-protein (CRP), which are markers of inflammation, are not very interesting, for simple sinusitis would be enough to make them go up. If we combine them with other more specific markers and X-rays, they become much more interesting, although X-rays will often be normal at the beginning.

Often, patients come with a very positive Waaler-Rose latex serology test. But it must be borne in mind that 5% of elderly subjects are healthy carriers and do not suffer from rheumatoid polyarthritis. This marker is therefore not at all specific, but is interesting if it shows a high rate from the outset. In that case it is an unfavourable prognostic factor. 30% of rheumatoid arthritis is seropositive for rheumatoid factors within the first six months. 70% of rheumatoid arthritis becomes seropositive after three years of evolution: so we must not wait for seropositivity (and this term must be used with care in front of patients who may well associate it with other pathologies (Aids)).

Anti-CCPs (anti-citrullines) are interesting antibodies. They are a new marker, not yet very common, but specific and sensitive. This is a good marker for diagnosis: if it is positive, we can suppose that we have an early case of rheumatoid arthritis, although the diagnosis is never 100% certain.

**B Measurement of disease activity**

There are several ways of measuring disease activity.

The DAS 28 is a European index with four variables which measures the number of tender and swollen joints, the activity of the disease assessed by the patient on a scale of 0 to 10 and the sedimentation rate. The disease is active if the DAS 28 is higher than 3.2\[^{13}\].

Another index, the SDAI, is the numerical sum of five parameters. It measures in particular the number of tender and swollen joints and the global assessment of the disease by the patient and doctor. This index can be used instead of the DAS.

**C Analysis of X-rays**

Once we have measured the disease activity, we must look for radiological lesions. However, it can happen that the X-rays are normal and therefore of no help. An ultrasound scan (and even an MRI scan) may be requested to confirm the pannus. X-rays show lesions when there are “cysts” in the bone. The radiological lesions often progress rapidly within the first two or three years of the disease. A radiological lesion means the destruction of the bone and the cartilage; it is final and irreversible. 10 to 26% of patients suffer erosion in the first three months, 60% the first year and 75% within two years. This reinforces the need to begin disease-modifying drug treatment immediately. Rheumatoid arthritis is a true diagnostic and therapeutic emergency.

**D Bad prognostic factors**

We can determine the bad prognostic factors by seeing patients from the very onset of the disease. Rheumatoid arthritis risks being potentially erosive:

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### DAS 28 index

**Composite index with 4 variables:**

- number of tender joints
- number of swollen joints (synovitis)
- patient general health assessment
- sedimentation rate (ESR)

**Active disease:** DAS 28 > 3,2

**Remission:** DAS 28 < 2,6
An overview of rheumatoid arthritis

- if the evolution of the disease is long
- if it takes more than an hour to loosen up from morning stiffness
- if more than three joints are affected
- if there is pain when we press on the metacarpophalangeal joints
- if there are positive rheumatoid factors
- and if erosion has already started to appear.

The prognosis can also be bad with an acute start and a high number of joints affected, high initial functional disability, an unfavourable socio-economic situation and low educational level. The appearance of subcutaneous nodules is also, it seems, a bad prognostic factor, as are a high sedimentation rate and C-reactive protein.

There is often a dissociation between the inflammatory lesions and the destructive lesions. Certain patients are very satisfied with their treatment and general condition. Their general check-up is satisfactory, but on their X-rays we see that the lesions are progressing rapidly with considerable bone destruction. It is very difficult to explain to the patient that he must move on to much more aggressive treatments because the rheumatoid arthritis is causing radiological damage [14].

The consequences of the disease

Rheumatoid arthritis is a public health issue which has consequences on the patients' daily lives. In half of cases, the patients give up work altogether within five years. It is possible that the situation will improve thanks to new treatments, but the figures remain worrying. Currently, it is estimated that 25% of patients will have at least one joint replacement. Once again, we can imagine that surgery would be less frequently proposed if more incisive treatments were given earlier. Furthermore, 10% of patients will present a serious disability in less than two years, which is considerable. Life expectancy is reduced by five to ten years and mortality is multiplied by 2.26 compared to the control population of the same age. This mortality rate is linked to the disease itself or may be of iatrogenic origin. This must be kept in mind as rheumatoid arthritis is a potentially serious illness. It seems, however, that with disease-modifying drug treatments, in particular Methotrexate®, the situation is improving, particularly on the cardiovascular front, which was a source of high mortality.

The complications of rheumatoid arthritis are above all infectious. With the new treatments, we have also seen cases of tuberculosis, all the more so as this disease is seeing an upsurge in France. Osteoporosis can also appear because it is a consequence of rheumatoid arthritis, but also because the treatments cause it, or because we are dealing with pre-menopausal women. We also see cardiovascular complications and a higher number of lymphomas in patients suffering from rheumatoid arthritis.

Therapeutic strategy

Faced with rheumatoid arthritis, energetic action must be taken. Ideally we seek to induce a remission. We must
is a veritable revolution. It is necessary to treat quickly, ideally within the first three months, as the lesions progress rapidly over the course of the first two or three years. It is absolutely essential to see a specialist to start a disease-modifying drug treatment, because choosing the right one is not easy. The choice is made on a case-by-case basis, taking into consideration the level of disease activity, other treatments and any related illnesses.

Rheumatoid arthritis is an aggressive disease that requires aggressive treatment with dangerous drugs. It is very important to explain this to the patient so that he does not neglect to take the drugs prescribed. There is no precise consensus on this subject. We often choose as the first-line treatment drugs such as Methotrexate® or Leflunomide®, which are anchoring treatments. We can also use other, older drugs such as Plaquenil®, Salazopyrine® or Allochrysine®. All these treatments are potentially dangerous and require regular monitoring with blood tests to detect any side effects.

If the patient fails to respond to the treatment, we can either increase the doses or combine other therapies. If the disease is really aggressive or if there are bad prognosis markers, more recent treatments are available, biotherapies or drugs containing therapeutic agents of the anti-TNF Alpha type: Remicade®, Humira® or Enbrel®.

As well as the drugs that relieve the pain and inflammation (analgesics, anti-inflammatory), they must be given disease-modifying drugs as these are very important in fighting the pannus and avoiding osteocartilaginous destruction. It is possible to add local treatments. If a person is responding well to the symptomatic treatment and the disease-modifying drug treatment, but still has knee synovitis, we should not hesitate to offer a local injection or “infiltration” of a cortisone derivative. We can also offer surgery if a patient has a swollen wrist, to avoid tendon tears.

Multidisciplinary care is required. Thus, it is useful to offer patients some dietary guidance, as patients must pay some attention to what they eat when suffering from rheumatoid arthritis. It must also be explained to them that they need to take some exercise, wear splints, do some occupational therapy and adapt their home environment. If necessary, suggesting that they see a psychologist or contact patients’ associations is useful if the patients believe that the people around them will not cope well.

In the past, the disease-modifying drugs were administered progressively, in successive stages, from the least dangerous to the most aggressive. Today, the attitude is the opposite. We treat early and strike hard now, even if it means reducing the treatment later if necessary. This
Enbrel® is an anti-TNF, but not a monoclonal antibody. It is a soluble receptor. It is administered in the form of two subcutaneous injections every two weeks. The patient must return to the hospital every six months for a check-up and to ensure that there are no complications.

These drugs are interesting because they act very quickly. With Plaquenil®, it took four to six months for the drug to act. With Methotrexate® or Leflunomide®, it takes one to two months. With the anti-TNF agents, we often see a response from the first injection or intravenous infusion: this is what makes them so revolutionary. The effectiveness of each of these drugs is equivalent, and it is possible to move from one to the other if they do not work.

These drugs are being closely monitored by the Observatory set up by the French Society of Rheumatologie. It checks in particular for the absence of infectious complications, lymphoma or cancer. For the moment, the data concerning the occurrence of lymphoma after taking this drug is quite reassuring, but more time is needed.
The importance of care

The general practitioner must be involved in the treatment, and patients must be able to consult him immediately if any problems arise. Indeed, we are prescribing complicated, aggressive and difficult-to-monitor treatments. Without the support of his GP, the patient is left to his own devices. Furthermore, patients’ associations exist, which can pass on information and give advice. Global care must be physical, psychological, social and nutritional, centred on the patient and his family, with networking between the hospital and the GP and patients’ associations.

There are also collective patient education programmes on offer. At the Hôpital Cochin, we talk to patients about their disease, treatments, pain and stress management, their social life and nutrition. This programme is run by a multidisciplinary team and includes a nurse, a dietician and a social worker. The programme takes place over two days, during which we explain to the patients what is happening to them and we give them the chance to talk to each other so that they feel less isolated. Francis Berenbaum also practises patient education at the Hôpital Saint-Antoine, individually over a one-day hospital stay. In the provinces, other teams are also educating patients suffering from rheumatoid arthritis.

As far as physical education is concerned, a rehabilitation specialist explains what activities should be practised. He shows why it is a good idea to wear splints or special soles to provide relief for the feet and how to cope better with this disease. He explains to patients the benefits of relaxation: certain simple breathing exercises can help patients to avoid taking extra drugs against the pain.

Conclusion

Rheumatoid arthritis requires a rigorous strategy, a rapid diagnosis, early treatment, specialised care and a global approach. The patient must be given the chance to meet doctors, a psychologist, social worker, nurse, occupational therapist, etc. Without this global approach, the care provided will not be satisfactory. Rheumatoid arthritis also requires regular monitoring.
What sort of diet do you recommend?

Dr Janine-Sophie Giraudet-Le Quintrec

There is no standard diet. We explain to patients that certain dietary manipulations can act on certain factors of inflammation, such as giving arachidonic acid, which acts on certain fats, for example. We explain to them that in spite of the tiredness and sedentary lifestyle, they must try to have a varied, balanced diet. There is no point in taking food supplements, if they eat a small amount of everything. If they are taking cortisone, they should not eat too much salt, but more proteins. We also explain that rheumatoid arthritis and its treatments can lead to osteoporosis and that therefore they must try to eat a diet rich in calcium. We give patients explanations so that they can understand food labels better. We also advise them to take vitamin D to help fix the calcium. There is a lot that must be said and we devote a whole afternoon to it. Often the patients are very satisfied with this information on nutrition. There are no foods that must be avoided; we simply show what is recommended and what is not. We have prepared this information with a dietician, emphasising both the medical aspect and the practical dietary aspect.

With erosive rheumatoid arthritis that has been in remission for several years, what is the risk of a new acute flare-up occurring?

Dr Janine-Sophie Giraudet-Le Quintrec

That depends, for I believe that each case is different.

Prof Francis Berenbaum

The fact that there has been erosion is not a good sign. With erosive rheumatoid arthritis, the risk of a new flare-up is higher than with non-erosive rheumatoid arthritis. Nevertheless, it is very difficult to give an opinion on this case-by-case risk.

If the patient is under treatment and in remission, do you try to reduce the treatment? What is your strategy in this respect?

Prof Francis Berenbaum

To my knowledge, only one study has looked at this question. It included two hundred patients suffering from rheumatoid arthritis and who were in remission under disease-modifying drugs. The disease-modifying drug treatment was stopped in one hundred patients and continued for the hundred others. After one year, 40% of the patients who no longer had the disease-modifying drug treatment had had a flare-up, whereas only 20% had had a flare-up in the group that continued the disease-modifying drug treatment. If we are optimistic, we can consider that 60% of the patients who interrupted the treatment did not have a new flare-up. If we are pessimistic, we can consider that the patients who stopped the disease-modifying drug treatment have twice as many flare-ups as those who continued.

Dr Janine-Sophie Giraudet-Le Quintrec

We always try, as far as possible, to limit or reduce corticoids. But we explain to “corticoid-reluctant” patients who find it difficult to understand the prescription of corticoids, that studies have shown that they have an effect on bone erosion.
we can have an effect on mortality. The latest studies no longer really show any excess mortality for a group of patients monitored who are in fact taking an effective disease-modifying drug. This is a notable change.

Dr Janine-Sophie Giraudet-Le Quintrec

This is the hope that reinforces the idea that we need to act fast.

Dr John Evans

There are several markers of inflammation, including some that are present even before the impairment of the joint. CRP is an example that speaks volumes. We now freely hand out statins as they seem to reduce CRP and protect the patient against coronary complications. What is happening in rheumatology?

Dr Janine-Sophie Giraudet-Le Quintrec

Statins are riding high in rheumatology as they are anti-inflammatory factors. They are very controversial as far as osteoporosis is concerned. It is possible that in the future they will be part of the array of treatments offered by the rheumatologist.

Prof Francis Berenbaum

A study published in May 2007 demonstrated that statins reduce disease activity.

What are the main causes of death connected to ankylosing spondylitis? The same as for rheumatoid arthritis?

Dr Janine-Sophie Giraudet-Le Quintrec

This is a difficult question. A study is currently showing that spondylarthritis causes more lymphomas.

The terrain is not the same, as ankylosing spondylitis occurs in younger patients. Furthermore, the evolutive profile is not identical. There are certainly some common factors, but probably also some different factors. The sex ratio is also very different.
As far as the treatment of rheumatoid arthritis is concerned, you are taking the same route as oncologists by using more and more dangerous drugs. What iatrogenic effects have you observed, in particular in terms of the fatal effects of anti-TNFs?

We do not yet have a long enough view of this subject. It is too early to draw any conclusions. There are infectious risks that can be fatal. Certain opportunistic infections by exceptional germs can be extremely serious and we can have problems, but for the moment we are monitoring the situation and have not had enough time to determine whether there is a risk of cancer or lymphoma induced by this drug.

The comparison with tumoral affections is striking. Our obsession is risk assessment. Now, we see that the iatrogenic aspect is fundamental with the products you are using, as well as the impact of the disease and its consequences. The impact on the chance of survival and the complications that these molecules can cause are impressive.

Since we have been treating rheumatoid arthritis earlier and more incisively, we have had fewer problems. It is because there are risks that the patient must be followed by a specialist. We are familiar with these risks and we can control them by monitoring patients, even if we cannot avoid certain extremely serious and rare complications. If the transaminases increase, you can reduce the doses or stop the drug. We know well the adverse effects of the drugs in the short and medium term. We even know the long-term effects of molecules such as Methotrexate®, in use since the 1980s and which we have had sufficient time to judge. Regarding cardiovascular mortality, the results have been better since we have been using this drug. We can ask ourselves if there is an interaction with folic acid which reduces the risk of hyper-homocysteinemia.

It seems that there is less talk of using gold compounds…

There is less talk of them. Today it is no longer possible to wait six months for a treatment to give results. Arava® or Methotrexate® act on the same cell, with onset of action after four weeks, even a little more in some cases. Therefore we do not hesitate to give the patient a much quicker chance of improvement.

We begin with Methotrexate®, we wait three months and if the response is insufficient at the maximum effective dose, we add an anti-TNF. This is the best attitude to adopt in benefit-risk terms. Our therapeutic attitude is today simpler than before. If the Methotrexate®, whose doses we can increase progressively, is ineffective in the end, we add an anti-TNF. This attitude is today practically consensual, except for a few reservations. In the United States, doctors are even experimenting with anti-TNFs as a first-line treatment. A study is about to begin to try to validate a treatment based on Methotrexate® + anti-TNF for three months. The idea is to begin by striking hard and then to consider stopping the anti-TNFs if necessary. From the doctor’s point of view, it is the best attitude in benefit-risk terms. True, there are some infectious risks, but these are better and better controlled. We know how to control tuberculosis: we can look for it and treat it before administering the anti-TNFs. For other infections, if the patient is already taking immunosuppressants, the risk is higher. In the studies, the infections are most often ENT infections, although there are some examples of serious infections. The tumoral problem remains, and for this we have seven years’ hindsight covering tens of thousands of patients: the Americans have not managed to find any difference in the risk of lymphoma or cancer among patients who are on anti-TNFs compared to those who are not, but this is with the reserve that not enough time has passed to be able to assert that the risk is insignificant. The infection is known and...
the risk can easily be assessed. As far as cancer and lymphoma are concerned, in spite of the tens of thousands of patients treated for several years and the absence of any significant difference observed, we still do not have sufficient hindsight to assert that there is absolutely no risk. Over the short and medium term, we are sure of what we are doing and we are monitoring the patients on anti-TNFs, whereas even with Methotrexate®, we monitor every two months with a hepatic profile, a numeration, etc.

Dr Patrick Malamud

Has the idea that rheumatoid arthritis constitutes a supplementary risk of the lymphoma been verified?

Dr Janine-Sophie Giraudet-Le Quintrec

Absolutely, according to the latest studies, the risk is multiplied by two. We have twice the risk of having a lymphoma when we suffer from rheumatoid arthritis. This also applies to ankylosing spondylitis.

Dr Dominique Lannes

Whenever the patient suffers from an auto-immune condition, whether it is lupus or rheumatoid arthritis, the risk of lymphoma is necessarily higher. Lymphomatous disease occurs when there is an anomaly. If a very specific part of the lymphocyte genome is excited, with a poor terrain, the risk of lymphoma is increased. It is the same type of stimulation as that which causes Burkitt’s lymphoma with EBV. The phenomenon is less blatant for rheumatoid arthritis or lupus, but it justifies an extra risk.

Prof Francis Berenbaum

If special monitoring is recommended, it is because TNF is also a molecule that is involved in the defence against tumours. We can therefore fear that by prescribing an “anti-defender” we will alter the barrier. However, for the moment, statistically we cannot see any significant difference in patients treated with anti-TNFs. On the other hand, we have all seen examples of lymphoma or leukaemia appearing in a patient who is on anti-TNFs, as in patients who are not, of course.
To rate rheumatoid arthritis, we must first be certain of the diagnosis, which is not always easy in an insurance application file as we do not examine the patient. A good knowledge of the complications is also essential, since the disease is potentially fatal. A good appreciation of the prognostic factors will also enable us to assess the excess mortality related to a case of rheumatoid arthritis. Finally, we need to have a complete selection file.

The diagnosis

When a patient goes to see a doctor, the latter is able to examine him and afterwards consult all the radiological or biological examinations that the person has in their possession, and can ask for further examinations. The difficulty for the insurance company medical officer or underwriter is that he cannot have access to this information, but he nevertheless needs precise items if he is not to risk incorrectly assessing the file, either by under- or over-rating the rheumatoid arthritis.

Rheumatoid arthritis is a very polymorphous disease and can lead to diagnostic difficulties. The presentations of rheumatoid arthritis can be extremely deceptive and can mislead doctors. To be certain of the diagnosis, a certificate must be provided by a certified rheumatologist, who will be able to give all the information needed to allow a correct rating:

- the diagnosis
- the date when the diagnosis was made
- the case history (beginning of the disease, time of evolution of the rheumatoid arthritis before treatment)
- the treatment followed by the patient
- any complications.

The prognosis

Once the diagnosis is known, we must try to find out about all the prognostic factors. These are far from featuring in files labelled arthritis… First of all the arthritis is not necessarily rheumatoid arthritis. More details are necessary in order to find out about the prognostic factors. It is possible at least to demand a certificate specifying certain information:

- the number of joints affected, swollen and tender
- the duration of morning stiffness, if any
- the existence of nocturnal awakenings due to articular pain
- the scores on the functional disability scales, as long as these are reliable and reproducible.

Knowledge of the biological prognostic factors is very useful. The sedimentation rate is very important in rheumatology. It would even be a little more reliable in showing the existence of a flare-up of rheumatoid arthritis. The CRP rate is at least as important. The serous auto-immunity factors, if there are any, are essential biological factors (rheumatoid factors given by the Waaler-Rose or Latex tests). Anti-citrullines are also beginning to be common practice. We can also look at genetic factors HLA DR1 and DR4. But these are not important prognostic factors for us. It is preferable to content ourselves with the ESR, the CRP and the Latex and Waaler-Rose serous auto-immunity factors (rheumatoid factors).

Add to this the radiological prognostic factors – joint space narrowing, erosion and the number of artificial joints the rheumatoid arthritis has required. This last factor is really very important.

Is the treatment a prognostic factor? We are accustomed to considering that if the corticotherapy is less than 7.5 mg, the rheumatoid arthritis is “under control”. Everyone knows that Methotrexate® and anti-TNFs are not harmless drugs. But without these aggressive treatments, the survival of patients will not increase. We are careful not to take these treatments as a prognostic factor, except in the case of complications attributable to them, although for the moment we are not aware of any long-term iatrogenic effects.

The prognosis is essentially linked to the presence or not of visceral complications (vasculitis) or extra-articular locations in general. A file which mentions vasculitis must not be accepted due to the life risk which is not acceptable in an insurance file.
More details

Prof Francis Berenbaum

Does this concern purely cutaneous vasculitis? [17/18]

Dr Patrick Malamud

I was thinking more particularly of systemic vasculitis. In the event of cerebral or cardiac complications, it is clear that a file must be refused.

A question: is a nodule not a sign of systemic vasculitis?

Prof Francis Berenbaum

No. Purely cutaneous vasculitis and nodules are not factors that aggravate the life risk.

Dr Patrick Malamud

Therefore, apart from nodules and purely cutaneous vasculitis, all other visceral locations must be refused.

Classification

To facilitate the work of insurers, we propose to simplify matters by referring to four forms of rheumatoid arthritis.

A Light form

This form is characterised by occasional flare-ups in an oligo-articular mode, long periods of remission, so-called “light” disease-modifying treatments. In fact, it is not possible to talk of light treatments since the best prognoses concern patients with treatments that are aggressive from the outset. In this light form, there is no inflammatory syndrome or erosion.

B Moderate form

The signs are greater than in the light form, but this is still a form of rheumatoid arthritis that is not uncontrollable.

C Severe form

In this form we find signs such as synovitis, duration of morning stiffness, nocturnal awakening, resistance to

Cutaneous vasculitis
On the survival curves of the patients in the Minaur study, we can see a marked difference between the control populations and the populations suffering from rheumatoid arthritis\(^{[19]}\). Since the excess mortality is double that of the general population, we must expect an over-rating of the files.

**Rating the risk-of-death risk**

**Dr Patrick Malamud**

*For a light form, with few flare-ups and long periods of remission, an excess mortality of 50 to 75% seems quite suitable. The patient will therefore pay 1.5 or 1.75 times his premium.*

*It is obvious that if we had proof that anti-TNFs will cause an excess mortality of 1.2 after twenty years, we would immediately revise the ratings: the additional premium would then only be 20 to 25%. But the data is not yet available. So we are obliged to refer to studies that track patients over the long term.*

**Prof Francis Berenbaum**

*Do you take age into account?*

**Dr Patrick Malamud**

*The basic rate takes account of age by definition. An insured person aged sixty pays a higher premium than a person of twenty-five. In the case in point, we are talking about over-rating above the average rating.*

*For a more developed case of rheumatoid arthritis with signs suggesting that it is not totally under control, we propose a rating of 100 to 150%.*

*For the severe forms, with a treatment that is not giving good results, multiple erosion sites, a hip replacement and a knee replacement, multiple clinical signs, the ratings exceed 200% and we may even go as far as refusing in the case of a visceral, cardiac or other form.*

*For the serious forms, we refuse the applications.*

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**Rating rheumatoid arthritis**

Disease-modifying drugs, inflammatory syndrome and the rapid destruction of joints.

**Serious form**

The serious form is characterised above all by vascular complications, linked essentially to vasculitis (with the exception of purely cutaneous vasculitis and nodules).

**Mortality study**

Rheumatoid arthritis is a serious disease. A study by Minaur and Coll. which appeared in 2004 reports on the follow-up over forty years of a hundred patients suffering from rheumatoid arthritis. The aim of the study was to discover the functional future of the patients, but also to analyse the causes of death. These were cases of rheumatoid arthritis diagnosed over forty years ago. This must not be taken as a reflection of how the disease is treated today, but of what we find in patients treated for forty years.

After 39.7 years, sixteen patients were still alive. Thirteen deaths were directly linked to rheumatoid arthritis. The sixty remaining deaths were mainly linked to cardiovascular causes. However, we observe more tumours and lymphomas than in the general population. Eleven deaths were related to rheumatoid arthritis and/or its treatment. In these cases, it was not possible to prove the origin of the death. We know that corticotherapy can kill if it is badly monitored. It must be borne in mind that this study covers a forty-year period. In the 1950s, corticoids were being prescribed simply for general fitness. With the Minaur study, we see a progressive aggravation of mortality. The standardised mortality rate (excess mortality compared to a reference population) increased constantly: it was 1.15 after eleven years of the study, 1.42 after twenty years and 2.13 after forty years. Excess mortality is about twice as high as in the general population. Rheumatoid arthritis is therefore a serious illness that kills, at least indirectly.
Regarding dependency, complementary insurance, it is not possible to propose a rating for people with rheumatoid arthritis. We cannot grant inability to work or disability insurance to these patients for we would necessarily have to pay a claim. These people are sure to have time off work, whether or not they are well treated. Furthermore, they are tired and cannot always go to work.
Mrs Le Quintrec pointed out that rheumatoid arthritis was an unpredictable disease. It can pass from a benign form to a medium form. This is a situation that is very difficult for an insurer to assess. Furthermore, the applications that we must issue a decision on are often incomplete. When they join the insurance, the applicant and his GP tend to minimise the seriousness of the disease. It is necessary to understand that we are committing ourselves over twenty, thirty or forty years with no way back possible. What do you advise?

Dr Patrick Malamud

The more precise the medical certificate, the closer our rating will be to what is presented to us.

Prof Francis Berenbaum

It is very difficult to judge a prognosis for an early case of rheumatoid arthritis, even if prognostic criteria are currently coming into existence. But if it is a case of rheumatoid arthritis that has already been evolving for ten years and which is in our first category, you will not be running a great risk by insuring it. It is in the early years of the disease that the lesions appear. Once these first years have passed, if the rheumatoid arthritis is still in a benign form, with no articular destruction, with a simple and well-balanced treatment, the risk is low as these patients, who have come through those first few years, have a rather favourable prognosis.

As for the inability to work, I heard Mrs Le Quintrec indicate that 50% of patients suffering from rheumatoid arthritis are no longer working after five years. We know very well that certain patients suffering from rheumatoid arthritis continue to work and will not be declared unable to work. But if we reason en masse, on the basis of the statistics, we cannot do anything else but refuse these applicants.

Prof Francis Berenbaum

If you go on the figures, you are certainly right. However, it is absolutely necessary that you reassess very regularly because the therapeutic context is evolving rapidly and will be changing completely.

Dr Patrick Malamud

That is precisely the meaning of our mission! To do the rating we use special software and according to the data in this software, rheumatoid arthritis cases with a remission of more than five years are taken at the normal death insurance rate.

Dr John Evans

Do you not think that with rheumatoid arthritis, there is an excess cardiovascular risk?

Prof Francis Berenbaum

The excess cardiovascular risk is related to the fact that in the past we let inflammation persist whereas with the current disease-modifying drugs, we eliminate this inflammation. By doing this, we are also acting on the cardiovascular factor. Furthermore, disease-modifying drug treatments are more and more effective and enable us to reduce the corticotherapy, which is an extra risk factor, as well as sedentariness. Together these elements will contribute to the excess cardiovascular risk in patients whose illness is well controlled, regressing and even disappearing in the years to come.

Are you often confronted with complaints from patients against insurance companies?
Patients are desperate because they cannot find an insurance company that will accept them. At least that is what we hear from them. They already have a dreadful disease and they do not have the possibility of being able to take out a loan. It is catastrophic for them.

Often, patients enquire about a loan. Their bank, without any complementary guarantees, refuses the loan. Patients can only experience this as a refusal. It is a recurrent problem.

Dr Janine-Sophie Giraudet-Le Quintrec

This difficulty does not concern the majority of contracts. In a good many cases, borrower policies can be issued with death cover and TILA cover. The inability to work/disability guarantee does not represent the majority of borrower policies.

Certain banks refuse to grant loans without guarantees. You can take out individual insurance, but not as a collective borrower.

Michel Dufour

Effectively, for collective borrower policies, which are first level policies, the basic all causes death guarantee will not be granted. Action needs to be taken at this level.

We could not ask for more, as insurers and reinsurers, than to have markers, even if it means granting cover, even for inability to work, over very short periods. If you give us the relevant parameters enabling us to engage in cover for five years, even ten years, we will do that.

Dr Patrick Malamud

Regarding therapeutic progress, I could quote the example of Glivec. This is an extraordinary molecule which, at a dose of four capsules a day, allows complete cytogenetic and molecular remission from chronic myeloid leukaemia. It is easier to put up with than a bone marrow transplant. I have had to make a decision on several applications presenting a complete cytogenetic and molecular remission. But we have only four or five years’ experience of this drug and I had to commit myself for a twenty-year loan. I could not do so as I have no proof that this young man of twenty-seven in remission will still be alive in ten years’ time.

As long as we have no references, we cannot take such decisions. And the references regarding anti-TNF therapy are still perfunctory.

Why not use exclusions in certain circumstances?

Dr Patrick Malamud

I do not see how we can foresee exclusions on a general illness.

Dr Dominique Lannes

It seems difficult to use the exclusion in the case of the inability to work due to rheumatoid arthritis, all the more so as Mrs Le Quintrec told us that her patients suffered above all from asthenia and tiredness. We cannot include asthenia in the list of exclusions for joints. It is difficult to use the exclusion in this pathology, unless it is to cover inability to work due to an accident.

We give the impression of being very restrictive, but we have genuinely done the best we can to reduce the ratings. We have explained that certain forms of rheumatoid arthritis had a practically normal level of risk, and we take account of this. But as regards the inability to work, we are moving towards refusal given the criteria that we have studied.

With the benefit of experience, we are however willing to review our position once the aggressive first-line treatments have borne their fruit.

Dr Patrick Malamud

I do not see how we could make exclusions that are sound, formal and simple to understand on general illnesses without exposing ourselves to claims. How do we prove that a lymphoma is due to the rheumatoid arthritis? The experts would laugh us out of the room!
Michel Dufour

The exclusions must indeed be formal and limited. We are absolutely not in that frame.

Dr Patrick Malamud

For a clearly localised case of osteoarthritis, it is easy to exclude certain things. The same does not apply to rheumatoid arthritis.

Do you adopt the same attitude concerning ankylosing spondylitis for sick leave and disability cover?

Dr Patrick Malamud

It all depends on the form of ankylosing spondylitis. It is necessary to analyse the form (peripheral, axial) on a case-by-case basis. Peripheral forms of ankylosing spondylitis can be related to rheumatoid arthritis. The rating is nevertheless globally lower than for rheumatoid arthritis. We do not provide for any exclusions for the sick leave cover. On the other hand, we accept this cover for certain light cases of ankylosing spondylitis with a moderate additional premium. These are cases of spondylarthritides without acute attacks, with the occasional taking of anti-inflammatories. In this case we can apply a moderate additional premium for inability/disability.
This chapter deals with ankylosing spondylitis (ASA) and the spondylarthropathies.

Ankylosing spondylitis is a subgroup of the spondylarthropathies. This common inflammatory rheumatism, which affects young adults, is a polymorphous disease in its clinical presentation and severity. From this point of view, it is important to obtain a certain amount of information to be able to rate correctly the applications presented.

If anti-TNFs have constituted a major advance in the treatment of rheumatoid arthritis, it is even more true for ASA. The information below, which concerns the consequences of this type of rheumatism in terms of professional activity or mortality, must be qualified insofar as no study on mortality or the impact on professional activity includes data on patients treated with anti-TNFs.

1 A polymorphous disease

Spondylarthropathies are extremely polymorphous diseases.

They do not just consist of the rheumatism that occurs at the age of twenty on average and leads to ankylosis of the vertebral column. The term “ankylosing” is, moreover, inappropriate as it tends to worry the patient and because, thanks to the new therapies, this type of evolution will be able to be avoided.

Spondylarthropathies constitute quite a large set of conditions.

The form that affects the vertebral column is ankylosing spondylitis. A patient suffering from this pathology has back pain at night, feels the need to move about, to get up at about five o’clock in the morning to relieve his pain. Apart from this axial form of the disease, spondylarthropathies can also take the form of arthritis that affects the peripheral joints, often of the lower limbs (knee, ankle). Psoriatic rheumatism is also associated with the spondylarthropathies. Reactional arthritis, or rheumatism that occurs in the month following a genital or digestive infection, have practically disappeared. SAPHO is an inflammatory form of rheumatism characterised by bone inflammation. Enterocolopathy rheumatism follows on from a chronic inflammation of the digestive tract, more commonly known as Crohn’s disease. Finally, spondylarthropathies can take the form of undifferentiated ankylosing spondylitis. This is the association in the same patient (frequently a woman) of arthritis and inflammatory spinal pain, without the ankylosing form in the vertebral column.

Consequently, we cannot reduce the spondylarthropathies to ASA alone.

What are the characteristics common to these different forms of rheumatism?

1 These forms of rheumatism are associated with the HLA B27 antigen. This natural antigen is present in 7% to 8% of the population in general. On the other hand, it concerns between 50% and 90% of patients affected by this type of rheumatism and is therefore a factor of genetic predisposition, even if this predisposition remains unexplained to date. HLA B27 serves, in the immune system, to present antigens and to defend itself against microbes. Depending on the form of rheumatism, the association with HLA B27 is obvious. However, on the psoriatic forms of spondylarthropathy, HLA B27 is only present in 18% of the population.

2 These types of rheumatism all present an impairment of the enthesis, i.e. an inflammation of the area where the tendons are inserted into the bone. This inflammation often leads to pain at the back of the heel[20].
They can affect the vertebral column, the top, median or lower part of the rachis, as well as the sacroiliac joint (axial impairment). Inflammation occurs in the vertebral column as well as on the ligaments around the column. Over time, this inflammation leads to a “bamboo spine” with the development of bony bridges.

They can take the form of peripheral arthritis, on a toe for example.

These types of rheumatism are accompanied by extra-articular manifestations (psoriasis, uveitis (inflammation of the eye), inflammatory enterocolopathies). Psoriasis consists of a thickening of the skin, which becomes whitish, and can affect the scalp, the nails, the eye...

In short, this rheumatism can take very varied forms and cannot be reduced to its “lead” disease, ASA. Furthermore, the applications we have to judge do not necessarily contain the term “ankylosing spondylitis” but rather that of “spondylarthropathy”. A description of the manifestations in the patient must be enclosed with the application.

Epidemiological data

Spondylarthropathy is a form of inflammatory rheumatism affecting young adults. On average, it occurs at the age of about twenty-four. However, the diagnosis is often not made immediately. In fact, this type of rheumatism manifests itself quite slowly, sometimes in atypical fashion, which can delay the diagnosis.

The only epidemiological study concerning spondylarthropathies available in France was conducted in Brittany by Alain Saraux, by means of a telephone survey. It seems that the prevalence of spondylarthropathy (0.47%) is very close to that of rheumatoid arthritis (0.62%). These two types of inflammatory rheumatism are the most common in Europe.

In the past, it was said that ASA was “men’s rheumatism”, as it was estimated to be ten times more common in men than in women. In actual fact, it would seem to be three times more frequent in men than in women. On
The consequences, day to day, of spondylarthropathy are many, from both a physical and mental health point of view. The treatment allows an improvement of 40% in physical health, in particular the physical functions. The improvement is also appreciated on the rheumatological parameters (number of joints swollen, pain in the morning…).

A German study has shown that the use of one molecule, Remicade®, given in an intravenous infusion, allows a 50% improvement in the BASDAI (Bath Ankylosing Spondylitis Disease Activity Index). The improvement under a placebo was only 9%. It is moreover recommended that a BASDAI questionnaire be used, as it gives a good idea of the patient’s state of health. The BASDAI, which includes simple criteria, is now systematically used for patients consulting with this type of inflammatory rheumatism. The BASDAI is a visual analogical scale that the patient completes. It covers the degree of tiredness, the level of pain in the neck, hips, peripheral joints, the intensity of the stiffness… so many elements that must be taken into account to assess the activity of a rheumatism.

The improvement in patients’ health is also measured through their consumption of anti-inflammatories. In 72% of cases, the patients are able to reduce by more than half their consumption of anti-inflammatories and even stop them in 60% of cases. In this last eventuality, the functional consequences of the patient’s rheumatism are nil.

The consequences of spondylarthropathy

The consequences of spondylarthropathy on professional activity and mortality are rare. Furthermore, the data available does not take account of the effect of anti-TNFs.

### On professional activity

Most of the results in the field come from the work of the Boonen team.
Compared to the general population, spondylarthropathy increases mortality by 50%. Furthermore, the risk of losing one’s job is three times higher. This aspect, like mortality, is correlated with the disease activity and the functional disability.

One of the factors holding back the introduction of anti-TNFs on the market is their high cost. Treatment with anti-TNF costs, over a year, about 13,000 Euros. Consequently, in spite of its efficacy, it is not possible to administer this treatment to all people suffering from inflammatory rheumatism. The more frequent use of this treatment has therefore raised the question of the cost of treatment in general. A patient who takes anti-TNFs and whose state of health is considerably improved will reduce his consumption of anti-inflammatories, will not go into hospital, will not need any more X-rays or local injections, and will not take time off work... In the end, studies have shown that the cost of the anti-TNFs balances out with that of the other treatments. In the end, even if this drug is expensive, it allows significant savings to be made elsewhere.

To date, between 61% and 89% of patients suffering from a spondylarthropathy are exercising a normal professional activity. Inability to work is relatively marginal (from 3% to 9% of patients).

Work has been undertaken in several countries on the interruption of the professional activity due to a spondylarthropathy. Permanent disability is thought to be only 3% in Mexico, no doubt because of the costs of such an interruption. In Europe, the rate of interruption is of the order of 36% after twenty years’ evolution of the disease. In the United States, 5% of patients are forced to give up their professional activity entirely after five years’ illness.

These studies have also tried to highlight the predictive factors for the interruption of the professional activity. They show that when the diagnosis is made before the age of thirty-five, the person has double the risk of having to give up work. Manual work is another aggravating factor in giving up work. People who, because of their rheumatism, try to limit their activities (by changing their workstation) have 2.3 times the risk of permanently giving up their professional activity. For people forced to change their professional activity completely because of their rheumatism, the risk of total abandonment is 6.9 times higher.

An American study has identified some other predictive factors for the interruption of the professional activity:
- age at which the disease occurs
- years of education: people with a higher level of education are generally less disabled, no doubt due to better information
- physical activity
- females: women give up their professional activity more easily
- comorbidity: the presence of another disease at the same time favours the interruption of the activity.

A Dutch study also pointed out the following factors:
- difficulties in accessing the place of work
- mobility difficulties at the place of work
- colleagues’ negative attitude
- superiors’ negative attitude
- lack of support from colleagues/superiors.

Even if they do not give up their professional activity completely, certain patients suffer periods of unemployment. Different studies have in fact emphasised the non-negligible prevalence of unemployment in people affected by inflammatory rheumatism (between 14% and 50%).

An American study attempted to identify the predictive factors for unemployment in this population: sex, quality of life, pain... European studies, for their part, highlight the role of age and the BASDAI. When the latter registers high values, the risk of unemployment for the population concerned can increase by 25%.

On mortality

A study carried out in the United States and published in 1998 looked at the risk of mortality in the case of psoriatic rheumatism. The latter evolves in practically the same way as rheumatoid arthritis.

The SMR, the standardised mortality ratio, taking into account the age and sex of the patients, emphasises that the risk of mortality is 62% higher for people suffering from this type of rheumatism compared to the general
population. The predictive factors of mortality identified are the following:

- sedimentation rate (inflammation)
- the disease-modifying drug treatment on inclusion, emphasising the most severe form of rheumatism
- the presence of erosion, which means that the rheumatism is not under control.

Consequently, the patients who have a higher risk of mortality are those who present chronic inflammation and uncontrolled inflammatory rheumatism.

In the case of ankylosing spondylitis, in its axial form, the studies have provided results that are not always reliable insofar as, previously, one of the treatments of this condition consisted of irradiation of the vertebral column. Now, irradiating a rachis can lead to the development of a blood disease. Thus, the figures showing an excess risk of mortality were distorted by the risk of blood disease as well as by the risk of colon cancer.

In actual fact, half of the studies available include vertebral column radiotherapy data. According to the studies, these treatments lead to an increase in cancer of between 50% and 140%.

Consequently, the results relating to mortality due to ASA are largely distorted by the mortality due to cancer, which is secondary to the irradiation.

The most realistic values on mortality due to ASA have been identified by English and Finnish studies. They show an SMR of between 1.66 and 1.50 inclusive.

One of the major factors in the excess mortality is due to cardiovascular problems. The same applies to rheumatoid arthritis. Chronic inflammation leads to a modification in the artery walls and thus an excess risk of cardiovascular mortality. This is why the diagnosis of rheumatism must be accompanied by a cardiovascular check-up. In the presence of other risk factors than chronic inflammation, such as high cholesterol or high blood pressure, patients are treated immediately. It is essential to take good account of the cardiovascular dimension.

An American study concerning a population of 50,000 patients identified an excess risk of mortality of 50% and emphasised that a large part of the excess risk of mortality can be attributed to the cardiovascular risk.

**Conclusion**

We should remember that ankylosing spondylitis only constitutes a small group of the spondyloarthropathies. This group is particularly polymorphous. The majority of the forms of this type of inflammatory rheumatism are benign.

Anti-TNFs have revolutionised the treatment of the spondyloarthropathies. The severe forms are candidates for treatment by anti-TNFs, but are also those which will have an impact on the professional activity and mortality. Consequently, they deserve to be paid particular attention. The studies including the efficacy of anti-TNFs will be particularly interesting from this point of view.
The arrival of anti-TNFs really constitutes a revolution in rheumatology. We know that the efficacy of this treatment, in particular in ankylosing spondylitis, is impressive.

There are three anti-TNF drugs: Enbrel®, Remicade®, Humira®. It is necessary to measure their effect on spondylarthritis as well as the cardiovascular condition of the patient. The side effects of the anti-TNFs are not yet clearly identified. We will find out more about them over the years to come.

Dr Corinne Miceli-Richard

There were some fears around these treatments concerning the possible occurrence of tumours or lymphomas. TNF means Tumour Necrosis Factor. It is a protein that serves in anti-tumoral defence.

The first studies on rheumatoid arthritis showed a slight excess risk of lymphoma. However, it was very difficult to confirm this link insofar as chronic inflammation itself leads to an excess risk of lymphoma of 100%. Was this excess risk under anti-TNFs really linked to the molecule or rather to the disease activity? The latest studies are completely reassuring on this point and establish a direct correlation with the disease activity: the molecule itself is not implicated in the lymphomas.

A second question concerns solid tumours. The most recent studies have concluded that there is a minimal risk of skin cancer. However, it is necessary to observe a much wider sample of patients to draw lessons.

What happens if the anti-TNFs are stopped?

Dr Corinne Miceli-Richard

Whereas it is very easy to determine when it is necessary to administer these products, it is much more complicated to work out the right moment to stop the treatment.

In rheumatoid arthritis, the treatment is suspensive. It is given by intravenous infusion every four or six weeks. Each time the treatment stops, the patients suffer. In spondylarthropathies, it is easier to space out the infusions, to reduce the doses and even, for some patients, interrupt the treatment. I have in fact seen patients for whom the interval between administrations by intravenous infusion was more than twelve months and for whom the treatment was stopped without these patients relapsing. However, this treatment remains suspensive; patients need it over the long term.

Rheumatism takes different forms. In some cases, it evolves by a succession of flare-ups and in this case, an interruption of the treatment can be envisaged. In other cases, it evolves continuously, and it is difficult to interrupt the treatment.

What about Fiessinger-Leroy-Reiter syndrome?

Dr Corinne Miceli-Richard

In France, we like to give rheumatisms the names of the doctors who described them. This is now known as “reactional arthritis”. It is a marginal syndrome. It affects fewer than 2% of patients.

What happens when you ask your patients to fill in a questionnaire on quality of life? What is your experience in this respect? When, as insurers, we ask our insurees to give this type of information, they tend to overestimate their capacities…

Dr Corinne Miceli-Richard

All these questionnaires, including the BASDAI, are essential elements in monitoring a patient. The latter will always reply in the same way. His answers
will be based on the same items, whether it is about his pain or his disability. Consequently, it is not the absolute but the relative value, that is the changes in the appreciations, that must be taken into account. It is thus possible to follow a given patient over the long term. On the other hand, comparing the results of one individual’s questionnaire with another’s is not very relevant in that each patient has his own experience and his own appreciation of pain. However, whether rheumatoid arthritis or APA is concerned, we know that patients’ tolerance of pain is excellent: in spite of their pain, patients behave almost normally. Their ability to “cope” with the disease is high. Conversely, patients suffering from fibromyalgia react badly to their pain although their disability is relatively low on a day-to-day basis.

There are therefore no studies measuring the quality of life and linking it to the inability to work…

Could you say something about the HLA B27 antigen and the inflammatory diseases presenting a cardiovascular risk?

There is no direct link between this antigen and the cardiac pathologies. This link, if it exists, is very loose. Certain conditions, such as aortic insufficiency, are not necessarily linked to HLA B27 insofar as these pathologies are observed in many forms of inflammatory rheumatism.

There is no direct link between this antigen and the cardiovascular pathologies either.

We have highlighted the direct link between HLA B27 and rheumatism thanks to transgenic rats. We made the rat express HLA B27 on its cells. Thus, the rat presented an inflammatory disease of the vertebral column, arthritis, and an inflammation of the eye. On the other hand, its heart was not affected nor was there any cardiovascular disease.

Consequently, on the basis of this somewhat artificial means, we have no reason to think that HLA B27 is involved in the cardiovascular manifestations, but rather in the inflammatory manifestations of the eye, joints or vertebral column and even in digestive inflammatory manifestations.

If the inflammation does favour cardiovascular pathologies, the link between the latter and HLA B27 is not direct.

Is gender involved in the evolution of the disease?

We have seen that being a woman has more repercussions on the professional activity. I am not aware of the content of the analyses carried out. The result is perhaps due to the fact that, in certain families, the woman is able to stop work more easily than her husband. In actual fact, the reasons for this excess risk have not been analysed.

On the other hand, we do know that the forms of this type of rheumatism in women are often more benign. The ankylosing impairment of the vertebral column affects men more. In the population in general, men are also more exposed to manual work. In this respect, the gender factor is more unfavourable to men.

I thought that the peripheral forms were more serious than the axial forms. Thus, the hip being affected was considered as a bad prognostic factor.

These bad prognostic factors were studied by Bernard Amor. The hip, curiously, is a peripheral joint. For spondylarthritis, we consider that the axial form is more severe.

Psoriatic rheumatism is common in people with spondylarthopathy. Is the opposite true?

Psoriatic rheumatism only affects 2% to 3% of the general population and 20% to 30% in the spondylarthopathies group. There is clearly an excess risk. However, this is only valid on very small general
What is the infectious risk of anti-TNF treatments?

Dr Corinne Miceli-Richard

This risk is a real one. In particular there is a risk of a resurgence of tuberculosis. Screening is now done for tuberculosis. Furthermore, we see a relative risk of 100% to 200% of developing a severe bacterial infection in the population that has received the treatment compared to the placebo population. These risks have been proven.

Furthermore, in the case of ASA, the studies were published in 2002. In actual fact, the studies were published on analyses begun in 2000. We do therefore have a certain degree of hindsight. The first applications in rheumatoid arthritis date from about ten years ago. On this subject, there is no reason to see any particular infections occurring. However, concerning the risk of tumours, to the extent that the sample of patients must be large, some questions remain.

Is the activity of anti-TNF Alphas maintained over time?

Dr Corinne Miceli-Richard

In rheumatoid arthritis, we see a loss of effectiveness to the point where we are forced to replace one anti-TNF by another. In rheumatoid arthritis, most of the patients who take Remicade® are on Methotrexate®. Antibodies develop that contribute to this loss of effectiveness. In ASA, the effect is less significant. A preliminary study carried out at the Hôpital Ambroise-Paré indicates that the combination of Methotrexate® and Remicade® is not necessary. The antibodies that develop against the molecule do not harm the efficacy of the treatment.

The maintenance of the treatment in ankylosing spondylitis is much better than in rheumatoid arthritis. Furthermore, the spacing out of the treatments cannot be envisaged with rheumatoid arthritis whereas it is with ankylosing spondylitis. In the same way, it is much less necessary to change anti-TNF in spondylarthrits than in rheumatoid arthritis, even if we do not know exactly for what reasons.
Rating ankylosing spondylitis

Dr Gabriela Mendoza Sassi • Associate Medical Director - SCOR Global Life

To rate spondylarthitis, it is necessary to be sure of the diagnosis, to know the complications and appreciate the prognostic factors.

To be certain of the diagnosis, we need a specialised rheumatological certificate, which must mention:
- the date of the diagnosis, so that we can calculate the disease evolution time
- the history of the disease, to determine the lesions (biological markers) presented by the patient
- the past or current treatments followed by the patient
- the complications.

The prognosis

When we examine the file of an applicant affected by spondylitis, we do not know how his disease will evolve. To envisage this evolution, we can use seven predictive factors described by Professor Amor. These factors, when they are observed during the early years of the evolution of the disease, will enable us to predict the evolution of the spondylitis for the next fifteen years.

Professor Amor’s prognostic criteria are the following:
- inflammation of the coxofemoral joint: 4 points
- sedimentation rate (>30): 3 points
- poor response to NSAIDs: 3 points
- stiffness of the vertebral column observed by the patient: 3 points
- the presence of a "sausage"-like swelling of a finger or toe: 2 points
- the presence of oligoarthritis: 1 point
- the beginning of the disease before the age of sixteen: 1 point.

If the total number of points is three or less, then the disease will be benign (sensitivity: 92.5%, specificity: 78%).

If the total is seven or less, the disease may be severe, but the sensitivity is only 50% (specificity: 97%).

Rating

A Epidemiological data

Epidemiological data in terms of mortality is used to establish the rating. A 1993 study by K. Lehtinen on a cohort of 398 patients suffering from ankylosing spondylitis, followed for twenty-five years, determined mortality 1.5 times higher than the general population. The rating must also be based on data relating to inability to work and disability. After fifteen years, 50% of patients suffering from severe spondylarthritis are no longer working.

B Clinical date, signs of evolution and complications

The rating is also based on signs of evolution: presence of pain, synovitis, morning stiffness, high sedimentation rate and CRP. These signs are negative factors for the evolution of the disease.

We count as complications the following items:
- visceral lesion
- hip affected
- claw toes
- artificial joint
- advanced kyphosis
- respiratory impairment.

C Presence and type of treatment

Rating is also based on whether or not the patient needs to follow a treatment and on the type of treatment (anti-inflammatory or disease-modifying drug treatment). In the case of disease-modifying drug treatments, we distinguish immunosuppressants from anti-TNFs.
Practical rating examples

A Example I
A man aged thirty-eight asks for cover for a real estate loan, for a capital of 85,000 Euros over twenty years. This applicant had a diagnosis of spondylarthritis in 2003. His medical report details a treatment by NSAIDs, a normal ESR and CRP, no pain or complications.

We can consider that this is a “light” case of spondylarthritis, presenting some positive points:
• absence currently of any clinical evolution
• ESR and CRP normal
• no disease-modifying drug treatment
• no complications.

For a “light” case of spondylarthritis, we apply the following principles:
• death: no excess mortality
• TPD: accepted
• inability/disability: additional premium of 50%.

However, depending on the radiological lesions presented by the applicant, the complementary cover may be refused.

B Example II
A man aged fifty-two asks for cover for a real estate loan, for a capital of 85,000 Euros over ten years. This applicant has been suffering from ankylosing spondylitis since 1982, confirmed by the medical report, treated by NSAIDs and sulfasalazine, and which is non-evolutive. A kyphosis and moderate respiratory impairment have been identified as well as the absence of any visceral lesion, hip lesions, claw toes or artificial joint. The sedimentation rate is 30 whilst the CRP stands at 15. The functional respiratory tests are not available.

Here we have a “moderate” case of spondylarthitis, which has been evolving for over twenty years, but which, at the present time, does not present any clinical evolution. This is a positive point for the rating. On the other hand, several negative points must be noted:
• the persistence of a disease-modifying drug treatment
• high ERS and CRP
• the presence of kyphosis
• moderate respiratory impairment.

In this case, the rating will be as follows:
• death: additional premium of 50-75%
• TPD: accepted
• inability/disability: refused.

C Example III
Example III concerns a man aged thirty-two who is asking for cover for a real estate loan for a capital of 145,000 Euros over twenty years.

In the medical questionnaire, the applicant reports ankylosing spondylitis, starting in 2000, which is being treated with Remicade® and has stabilised, as well as the persistence of morning stiffness lasting forty-five minutes, with hip lesions and claw toes, but no kyphosis or respiratory impairment, and no artificial joints. Furthermore, the sedimentation rate is 35 whilst the CRP stands at 12.

This corresponds to a case of “severe” spondylarthritis. The positive point is that the patient has been clinically stabilised by Remicade®. On the other hand, several negative points must be emphasised:
• morning stiffness
• high ESR and CRP
• hip affected
• claw toes.

In this case, the rating will be as follows:
• death: additional premium of 75% to 100%
• TPD: refused
• inability/disability: refused.
Conclusion

The number of applicants suffering from ankylosing spondylitis has been increasing since 2000.

The rating takes account of the prognostic factors, positive factors as well as negative factors.

Anti-TNFs are in the process of changing the natural history of the disease. However, we do not have enough hindsight to judge how they will influence mortality and inability/disability.
Dr Corinne Miceli-Richard

Exercising your profession is difficult. Making assessments based on medical files is not a simple task.

I am a little surprised that you do not integrate into your system the type of work the patient does or the length of the periods of sick leave caused by the rheumatism. This applies most particularly to the inability to work. As a doctor, considering the three examples presented, I am much more worried for patient II, whose disease-modifying treatment seems not to be very effective, than for patient III, who is on Remicade®. It is a delicate matter to assess these situations.

Among the Amor criteria, peripheral lesions do not have much weight. The important items are rather the axial disease and hip lesions. For doctors, a serious spondylarthropathy is one which gives rise to treatment by anti-TNFs. To administer the latter, we identify above all hip lesions, an inflammatory syndrome, a high sedimentation rate, a poor response to NSAIDs, stiffness of the vertebral column. You are right to make use of these criteria. And yet the applicant’s professional activity is missing.

Michel Dufour

Our reflections, in the past, were based on a biomedical model. Now, as has already been indicated, we have to take account of the “bio-psycho-social” aspect, that is to say, the person’s environment. This approach is not simple for the insurer in France, given that he has the greatest difficulties in obtaining any information on these aspects. Asking for such information is sometimes considered as an infringement of civil liberties. Indeed, we must be very careful to respect the individual.

We are considering how to deal with the inability to work risk. In the past, the response was simply to refuse the insurance or insert an exclusion. Today, we are trying to rate files in such a way as to insure our clients on as many points as possible. Unfortunately, we lack the data.

Dr Dominique Lannes

The drawback of exclusions, which can be quite practical when studying an application, is that they partly empty the policy of its substance. Assessment is difficult, to be sure, but it represents progress compared to systematic exclusion.

Michel Dufour

We take account of the individual’s profession, sometimes without really being aware of it. In any insurance application file, the applicant’s profession is mentioned. Any underwriter who is studying an application is aware of the pathology and the profession. These elements are taken into account when increasing or reducing the rating of the policy. When inability to work insurance is refused, it is not unusual that the underwriter be asked to justify his decision. Nevertheless, these approaches remain informal, they are based on experience. It is difficult to integrate these items into rating software.

Dr Corinne Miceli-Richard

It is also necessary to take account of the length of the sick leave.

Michel Dufour

We do take account of it. The underwriters are aware of the periods of sick leave and act accordingly.
Do certain applications give rise to an exclusion?

Dr Dominique Lannes

In general, inflammatory rheumatism does not give rise to an exclusion insofar as it is very difficult to exclude a general illness. However, refusals are possible.

The whole issue is that, in the event of claim, it is difficult to conclude that the sick leave is due to the rheumatoid arthritis and to invoke the exclusion.

When we do place an exclusion on an illness and the patient is off work due to that illness, the patient will not claim any benefits... The problem only arises in the case of disability. It has been proven that a certain number of applications for benefits are avoided when the exclusion is placed.

Dr Gabriela Mendoza

The exclusion often concerns the vertebral column. It happens, in cases of ASA, that any disease that is not tumoral or infectious be excluded. Thus, if the person is suffering from a vertebral tumour or infection, we take account of that element.
Dealing with osteoporosis: 
a choice for thirty or forty years

Dr Mickaël Rousière • CHU Saint-Antoine, Paris

1

Physiopathological reminders

The bone tissue is a tissue in the body that plays different essential roles:
• support frame for the body overall
• protection (the ribs for the lungs and heart, the skull for the brain)
• movement
• storage of minerals
• formation of red blood cells (by the bone marrow).

We distinguish compact bone (80% of the mass of the skeleton, 20% for bone remodelling), trabecular bone (20% of the mass of the skeleton, 20% for bone remodelling).

The bones are made up of:
• a framework, the matrix
• cells with a role in the destruction and recomposition of the bone
• crystals loaded with calcium, which give the bone its solidity.

The bone is a living tissue which is permanently renewing itself. First of all, the bone is destroyed by osteoclasts. Then other cells called osteoblasts will rebuild the bone in order to repair any lesions. This process of destruction/reconstruction of the bone is balanced quantitatively and is called bone remodelling. The integrity of the skeleton is preserved.

In the event of osteoporosis, the cells that rebuild the bone are not very effective whereas the cells that destroy it are hyperactive, which leads to bone loss, causing bone fragility. The bone loss will get worse over time.

2

The definitions of osteoporosis

The definition of osteoporosis has changed a great deal over time. Etymologically, osteoporosis means porous bone, containing small holes. At the beginning of the XXth century, it was thought that the gravity of osteoporosis was measured by fractures, in particular the femoral neck fracture. Then, histological studies were conducted with bone biopsies. The true definition of osteoporosis dates from 1993, following a consensus conference. However, this very literary definition is not easy to use in practice: “A systemic skeletal disease characterised by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture.”

An osteoporotic bone presents bone trabeculae that are thinner, perforated and poorly connected to each other.

The “practical” definition was given by the WHO in 1994 and concerns bone densitometry. It is based on the distinction between “osteopenia”, “osteoporosis” and “confirmed osteoporosis”.

3

Recognising osteoporosis

Osteoporosis is therefore a systematic disease of the skeleton characterised by a reduction in bone mass and disruptions in the microarchitecture with the following consequences:
• an increase in bone fragility
• an increase in fracture risk.

The recognition of osteoporosis cannot be limited to a medical examination consisting of measuring patients’ height. Indeed, this criterion is one that comes far too late, since it is accepted that a fracture of the vertebrae leads to a loss of height of two to three centimetres. Other causes, such as scoliosis, can also lead to loss of height, so this sign is also non-specific.

An X-ray of the vertebral column is not satisfactory either insofar as it will only show up a fracture.

As for biological examinations, to date there is no blood test to diagnose osteoporosis.
It is therefore necessary to use the bone mineral density test, which is the reference examination for the diagnosis of osteoporosis today. This examination is reliable and harmless, as the irradiation is very low. Bone densitometry allows the bone mineral density of the lumbar spine and femoral neck to be measured. X-rays pass through the human body. The denser the bone, the more the X-rays are stopped and the less they are picked up by the detector. This totally painless examination takes ten minutes to perform.

The examination measures the quantity of calcium present in the bone, which is then compared to a bone surface to determine the bone mineral density. This bone mineral density provides figures that are quite difficult to understand on their own. However, bearing in mind that the bone mass is at its maximum between the ages of twenty and thirty, it is possible to compare a patient’s bone mineral density to a reference population considered as normal. We therefore study the patient’s bone density value in relation to the value for an individual of the same age (Z score) as well as the variation in this value compared to a young subject (T score). For example, the bone densitometry of a woman of sixty is compared to that of a young woman in order to determine the quantity of bone lost over the years.

The measurement of the bone mineral density will reflect the risk of possible future fractures. Each time the T score is reduced by one unit, the risk of fracturing the femoral neck is multiplied by two. The lower the bone density, the higher the fracture risk. This examination therefore allows us to predict a possible fracture risk for a given person.

The consensus conference fixed a threshold value from which osteoporosis is defined. Thus, when the standard deviation from the T score is between – 1 and + 1 inclusive, the density is normal. If it is between – 2.5 and – 1 inclusive, we talk of osteopenia. If it is below – 2.5, we talk of osteoporosis. It is from this value that we consider in fact that the fracture risk is very high (+ 400%). Osteopenia is not an illness but a transitional state. An American study involving several tens of thousands of patients showed that a reduction of one point of the standard deviation doubled the fracture risk.

### Epidemiology of osteoporosis

The number of women in France with osteoporosis is estimated at four million. Most of them are unaware of it. More precise estimates refer to 3.7 million osteoporotic women. Almost half of them already present fractures. However, only 802,000 patients have been diagnosed and treated.

The prevalence of osteoporosis is as follows:

- Women over 50 years old: 30%
- Women over 65 years old: 50%
- Women over 80 years old: 70%

A study by Ribot conducted in 1995 shows that, out of a sample of women aged over forty-five who go to a menopause consultation, 40% are suffering from osteoporosis, at least of the lumbar spine. This condition is therefore particularly common.

Osteoporosis is a silent epidemic, given the lack of pain and symptoms... until the fracture occurs. This characteristic poses problems in terms of diagnosis and treatment.

The epidemiology of osteoporosis is becoming better and better known. That of the fractures has already been known for a long time. A fracture is a painful event which necessarily entails a medical consultation, which gives us more complete databases. This is particularly true for fractures of the femoral neck insofar as the individuals are systematically taken into hospital.

Apart from the pain, these fractures (wrist, vertebrae, femoral neck) have major consequences in terms of morbidity, mortality and socio-economic costs. They are all the more frequent as the population ages. From the age of seventy, the increase in the prevalence of femoral neck fractures is exponential. It is expected that by 2040 taking care of these fractures will constitute the major part of the professional practice of orthopaedic surgeons. However, a wrist fracture is often the first fracture suf-
Vertebral fractures are very frequent. They affect one woman in three aged over sixty-five. These fractures often herald other fractures or spinal compression.

Each fracture announces the next one. A patient who suffers a wrist fracture risks a vertebral fracture and, if nothing is done, a femoral neck fracture. Vertebral fractures are a common pathology. But in one case in two, they are not recognised as a real fracture. Indeed, unlike the wrist fracture or femoral neck fracture, the vertebral fracture is not always apparent. It may only give rise to a small amount of pain. A woman of sixty-five who has some back pain immediately thinks she is suffering from osteoarthritis. She therefore considers that there is no point in seeing a doctor, whereas it is possible that she is actually suffering from a vertebral fracture. The problem is that a vertebral fracture has the same functional impact as a femoral neck fracture in terms, for example, of the risk of a further fracture. Thus, even if the vertebral fracture does not cause great pain, it still means there is a high risk of a femoral neck fracture.

The risk of a vertebral fracture increases exponentially with age. A French study conducted with women in the Picardy region also shows that one in two women aged over eighty-five has suffered from at least one vertebral fracture.

When we compare a normal bone with a bone suffering from a vertebral fracture, we see that the top of the bone has totally collapsed. The framework is no longer supporting the edges of the bone.

Vertebral fractures cause acute chronic pain. They also cause a loss of height, which, moreover, can have a serious impact on patients’ mental state. Furthermore, women who have multiple vertebral fractures can suffer from breathing difficulties, even digestive problems and...
in very rare cases, problems of incontinence. Nevertheless, the essential risks are loss of height and chronic pain. A woman suffering from vertebral fractures may also suffer from depression linked to the symptoms mentioned previously (loss of height, chronic pain).

Vertebral fractures herald other fractures. An American study on this subject was conducted with 9,600 women over eight years. It shows that a woman who has had a vertebral fracture sees her risk of having another vertebral fracture multiplied by four and the risk of a femoral neck fracture multiplied by two. All the studies agree that vertebral fractures are predictive of fractures of the femoral neck. Furthermore, the more a woman who has had a vertebral fracture advances in age, the higher her risk of having a femoral neck fracture.

Among women who have had a first vertebral fracture, one in five will have a second vertebral fracture within the next year. Furthermore, a woman presents a higher fracture risk if she has already had fractures in the past. Thus, on average, 20% of women have subsequent vertebral fractures. This rate reaches 25% when the patient has already had two previous vertebral fractures. In the final analysis, the more fractures one has suffered, the greater the risk of having more. A patient who has suffered a fracture of the vertebrae runs a greater risk of a further vertebral or non-vertebral fracture, a femoral neck fracture, for example[23]. In medical jargon, we use the term “fracture cascade” to characterise this succession of fractures. We thus see women who are hospitalised every year or every six months for another fracture. This explains the importance of providing care from the first fracture.

Among the osteoporotic fractures, vertebral fractures are the most well-known consequence of post-menopausal bone loss. However, non-vertebral fractures – and in particular those of the femoral neck – are also frequent, and also serious, costly and responsible for a large part of the mortality, morbidity and expenses related to osteoporosis.

French rheumatologists are taking more and more of an interest in osteoporosis, and French studies are now available[24]. Half of osteoporotic fractures are not vertebral, but about 40,000 vertebral fractures are recorded every year in France. However, if all the vertebral fractures found in radiological data are counted, the number of vertebral fractures reaches 120,000 a year. Finally, we record each year in France 50,000 fractures of the femoral neck, 40,000 wrist fractures and 40,000 other types of fracture (pelvis, humerus, etc.). Thus in total, 200,000 fractures can be linked to osteoporosis every year.
Dealing with osteoporosis: a choice for thirty or forty years

The incidence of hip fractures increases exponentially from the age of seventy. It is multiplied by three for women who have had a previous fracture. Moreover, a hip fracture can have serious consequences. Thus, out of four people having suffered a hip fracture, one risks dying in the following year, the risk of mortality after a fracture therefore being in the order of 25%. Of these same four people, only one will be able to return to her normal activities, and two will see a reduction in their functional capacities. Of these two people, one will enter an institution (retirement home) and the other will need help at home. Globally, after such a fracture 5% of people die within the following year, 25% of people get better, 50% of people will need total (in an institution) or partial (help at home) assistance.

A hip fracture is therefore serious as it can lead to death. It is also costly as the patient will be forced to finance the home help or care in an institution.

Medico-socio-economic consequences of fractures

Osteoporotic fractures are frequent and increase with age. From the ages of fifty-five to fifty-nine, the fracture risk (in particular of the wrist) already increases significantly.

Osteoprotic fractures are expensive. An American study has estimated this cost but it only takes account of the cost of the hospitalisation due to the fracture. We can therefore see that a femoral neck fracture costs 19,000 Euros. A vertebral fracture only costs 500 Euros. However, it must be borne in mind that most people do not go into hospital, but are treated at home, and the cost of this service is significant. An English study gave similar figures to those of the American study. This study emphasises that the direct cost of a femoral neck fracture is very high. Indeed, the person needs hospital treatment, which represents a cost of more than 1,000 Euros. Moreover, this type of fracture also requires rehabilitation, an

Cost of a femoral neck fracture
- High and relatively constant
- Estimated average cost (EU) per patient = €19,100

Cost of a vertebral fracture
- Lower and variable.
- Estimated average cost (EU) per patient = €500

Kanis J et al, Health Technology Assessments 2002
operation and a joint replacement (costing 1,500 Euros). Finally, a Swedish study estimates that the cost of a femoral neck fracture, if we count the direct cost in the following week and all the costs that will be generated in the year after the fracture (consultations, rehabilitation, drugs, physiotherapy sessions, home help), amounts to 14,000 Euros per patient. A vertebral fracture represents a lower direct cost, but if we count all the expenses—bearing in mind that this type of fracture causes chronic pain—we arrive at a total cost of 12,000 Euros per year per patient.

In the end, these femoral fractures are therefore costly, both for society and for the patients. An assessment of the quality of life of women suffering from osteoporosis and having had fractures has shown that after a hip fracture, the patient’s quality of life is reduced by 80% after one year. In the case of a vertebral fracture, the reduction in the quality of life is 70%.

One vertebral fracture in ten gives rise to a hospital stay. In all the other cases, the fractures are treated at home. Sometimes these fractures do not give rise to a hospital stay because they are not recognised as such: the patients think they simply are having a flare-up of osteoarthritis. A person who suffers a vertebral fracture sees the risk of going into hospital in the following year increased by 18%. The vertebral fracture, and fractures in general, are probably evidence of the poor health of the patients. Whatever the reason for a person going into hospital, the fact that she has already had a vertebral fracture increases the hospital stay by five days on average, which represents an extra cost.

Vertebral fractures also cause other pathologies. We say that these are silent fractures, but it is not true. 41% of women who have suffered a vertebral fracture suffer from chronic pain. Similarly, 41% of women who have had a vertebral fracture suffer from functional disability afterwards. Moreover, whether they are radiological or clinical, fractures cause on average almost a year of moderate pain. Often, patients are told that fractures of the vertebrae heal in six to eight weeks—this point needs to be relativised since they actually suffer for almost a year. Similarly, a clinical fracture requires on average fifty days’ complete bed rest and almost a hundred days of limited activity.

Osteoporotic fractures and mortality

All osteoporotic fractures increase mortality. Mortality increases for femoral fractures, but also for vertebral fractures. American companies have even managed to evaluate the number of years of life lost after a fracture. In the case in point, if a person suffers a fracture of the vertebrae at the age of sixty, she loses globally two years compared to normal life expectancy. If a person suffers a femoral neck fracture at sixty, her life expectancy is reduced by eleven years. But in fact, the older a person gets, the closer she gets to her normal life expectancy. The calculation concerns the number of years lost compared to the theoretical life expectancy. Consequently, a person aged eighty has certainly reached her theoretical life expectancy, but in the event of a femoral neck fracture, the probability that she will die within the following year is 25%.

We have therefore been able to calculate mortality curves. Other figures, taken from other studies allow certain trends to be identified. Thus the following graph shows that fractures of the forearm do not increase mortality much. Indeed, the green curve is almost the same as the mortality curve for the general population. On the other hand, when a person suffers a fracture of the femur or a vertebra, her mortality increases by 50% after five years. These figures are severe, but they show clearly that after an osteoporotic fracture, mortality increases by a high proportion. In fact, in the field of osteoporosis, we often use this saying: “The more serious it is, the more serious it is.” If we take the case of a first vertebral fracture, the risk of mortality is multiplied by 2.3 within ten years.

To conclude, we can say that osteoporotic fractures are painful, frequent and costly both in terms of functional disability, illness and mortality. These fractures are serious and cause a high rate of mortality. Even if the figures are a little at variance with each other, we can see that 20 to 25% of patients die in the following year, and that one patient in two will see a reduction in their life capacities. The graph below shows the relative survival rate after a femoral neck fracture and a vertebral fracture. You can see that between the expected mortality and what we can observe, there is a clear difference. We certainly have a multiplication of the rate of mortality by a factor of at least three after these fractures.
Treating osteoporosis

How to prevent and improve the treatment of osteoporosis.

First it must be detected. This is not always easy, faced with people aged sixty who are beginning to have osteoarthritis, high blood pressure, cholesterol etc., and who must therefore deal with a large number of drugs. In these cases, we need to look for the risk factors and systematically offer patients an examination of their bone mineral density. It is also necessary to give them preventive treatments. Certain drugs have proved their efficacy in treating osteoporosis. Calcium and vitamin D represent the minimum treatment. But we must also envisage other more effective treatments.

Treatment involves both drugs and other treatments. The first element is "life hygiene". Thus we encourage patients to practise sport and eat foods rich in calcium. The cardiologist often advises elderly patients to watch their cholesterol. It should be remembered that "light" products contain less fat, but not less calcium. Thus in a normal yoghurt and a 0% fat yoghurt, the quantity of calcium is the same. The same applies to milk. We also advise women to take plenty of physical exercise (at least forty-five minutes’ to one hour’s walking a day) and smokers to give up.

There are various drug treatments available. The pharmaceutical industry is well aware that osteoporosis is a more and more important subject and it has launched a whole array of molecules onto the market. It is a little difficult to know in which order they will be used. We have been using hormone replacement treatment less and less during the menopause, even if we are seeing a return to this method at the moment.

The Drug Agency published a prescription guide in 2004, updated in 2006. We therefore have treatment models for patients according to their fracture backgrounds, their age and their bone mineral density. We can thus determine when treatment for osteoporosis can be started. The drugs all operate in more or less the same way. Globally, all the drugs prevent about 50% of the risk of a vertebral fracture. In other words, when we propose a treatment to an osteoporotic woman, we only prevent one fracture in two. As far as non-vertebral fractures are concerned, in particular femoral neck fractures, the risk is reduced by 30 to 50% by some of these drugs. Globally, none the molecules have proven their efficacy for the femoral neck, except three: Actonel TM, Fosamax TM and Proteos TM.

The doctor must think to offer drug treatment. But it is also necessary to educate people in order to increase the effectiveness of these drugs, which represents a real problem. Often we do not know how to go about this. We know that when we reduce the number of times the drug needs to be taken, observance is better. However, with modern osteoporosis treatments which require the taking of one tablet a week, we have observed that after one year, observance is only 60%. In other words, only 60% of people are still taking the drug prescribed after one year, which is regrettable.

Osteoporosis is not a fatality. We can screen for it and prevent it; and diagnose and treat it.
Mortality after osteoporotic fractures

Johneil, Kanis et al. Osteoporosis Int 2004

Femoral neck fractures are serious

Relative survival rate after osteoporotic fractures

Adapted from Cooper C. Am J Epidemiol. 1993
Dr Gabriella Mendoza

Tobacco may also be a common factor.

Dr Mickaël Rousière

The women we are seeing today are those who smoked little in the past. Nowadays, they all smoke, whereas before they did not.

We talk in our circles of the effect of the statins used against cholesterol as a protective factor in osteoporosis. Have you also observed this phenomenon?

Dr Mickaël Rousière

We have seen a sort of boom in the use of statins. And for a while, we believed in it. But finally, if the effect exists, it is probably marginal. We have been able to show, through the inflammation, that they increase bone mineral density by very little and reduce bone hyper-modelling somewhat. But in the final analysis, if we look again at the major cardiological studies that have included information on fractures, we see that there is no reduction in fracture risk. We had the same hopes for beta-blockers. Hypotheses had been put forward on the role of betablockers. Finally, the physiopathological model is an attractive one. But in practice, we do not have the impression that it really reduces the fracture risk. That said, it is not a bad thing in itself. Betablockers and statins are not deleterious for the bone.

Dr Gabriella Mendoza

Even with developments in medicine, if we compare the studies done twenty years ago and those done very recently, this mortality remains the same. Today, patients are still dying as much from a femoral neck fracture as they were twenty or thirty years ago.

One woman in four dies in the year after a femoral neck fracture. That is an impressive figure.

Dr Mickaël Rousière

Because we do not screen enough for osteoporosis?

Dr Gabriella Mendoza

The problem is to identify the real cause of death. It could be post-operative complications. A surgical operation can cause pulmonary infections in the elderly. There is also very high cardiovascular mortality. There is probably a combination of the risk factors of osteoporosis and the cardiovascular risk factors. An osteoporotic woman should systematically be sent to a cardiologist to see if there are any cardiovascular risk factors. Conversely, women suffering from cardiovascular problems should be sent to the rheumatologist to screen for osteoporosis.

Dr Mickaël Rousière

Why does osteoporosis mainly affect women? Following on from that question, are the prognosis and complications the same?

To answer your second question, osteoporosis in men is serious. Men die just as much from a femoral neck fracture as women do. Why do we talk less about male osteoporosis? It is probably a little less frequent than female osteoporosis, for one major reason, which is the absence of the menopause in men. Men do not suffer at the age of fifty from this hormone deficit, which is nevertheless a genuine factor in triggering osteoporosis in women.
We have been interested in female osteoporosis for a very long time. Some data is now beginning to appear concerning men. The pharmaceutical industry has started to take an interest in this condition, which is developing more and more. Finally we are realising that this condition is not just anecdotal in men. It is, moreover, likely that the majority of cases are not diagnosed. I think this is possible, all the more so because men have poor life habits: they drink and smoke. If we were to carry out densitometry studies on the population, we would probably find cases of osteoporosis in men. The definition of osteoporosis must also be considered. We can say that men may suffer from osteoporosis, but it is true that they suffer fewer fractures than women because the architecture of the bone is a little different. The cortical bone is thicker. The mesh is a little tighter and better interconnected. At equal bone mineral density between men and women, even if it is low, there are still more fractures in women than in men, at least until the age of eighty. After eighty, it is relatively common for a man to break the neck of his femur. But in fact, we are short of epidemiological data. Currently, major studies are underway in the United States. They consist of densitometric monitoring in men over fifty. The objective is to obtain epidemiological data on osteoporosis in men. Very little is said about this subject. And yet, we will need to take an interest in it since men’s life expectancy is also rising.

Among women with an early menopause, do you see earlier onset, i.e. does osteoporosis occur correspondingly early? Is the risk the same or aggravated by earlier occurrence?

Dr Mickaël Rousière

This is a difficult question because there are in fact very few studies on women with an early menopause. Osteoporosis is nevertheless rare in women before the menopause. Generally, it is related to a notable pathological event. This can be for example a woman suffering from cancer, a blood disease or a disease that requires heavy treatment.

Before the theoretical age of the menopause, these women do not have a low bone mineral density.

They do not suffer many fractures. Generally, we do not offer them treatment for osteoporosis as such. Often, we ask the gynaecologist for hormone treatment, which moreover does not pose any breast or cardiovascular problems until the theoretical age of the menopause. Consequently, until the age of fifty, osteoporosis does not pose a problem. On the other hand, after fifty the evolution is the same as for other women. I would point out, furthermore, that no pharmaceutical company offers a drug for women with an early menopause.

In the absence of a deficiency, is vitamin-calcium supplementation useful? Do they not have a deleterious effect, in particular from a renal point of view?

Dr Mickaël Rousière

Yes and no. This is something to look at, for sure. And yet, should we propose systematic supplementation? Probably not. The major American studies, which have come out recently, show that supplementation with vitamin D and calcium does not reduce the risk of fracture. From a statistical point of view, these studies were well done. However, from a practical point of view, they are useless because they concern people who already have a satisfactory intake of calcium and vitamin D. It is probably necessary to give supplements to people who need them. As far as calcium is concerned, a very simple approach can be taken. This involves a dietary questionnaire that takes the patient five minutes to complete and the doctor two minutes to analyse. It gives an idea of the daily intake. In post-menopausal women, the theoretical intake of calcium is situated at about 1.2 grammes a day. It is very easy to know if a woman has an intake of more or less than 1.2 g. Personally, I systematically use this questionnaire. I only supplemen t women whose daily intake is less than 1 gramme. As far as vitamin D is concerned, a simple method consists of dosing vitamin D. It should be noted that, on average, vitamin D deficiency affects between two thirds and three quarters of post-menopausal French women. Moreover, recently, the lower normal dose was increased. Before, the normal dose was between 20 and 50. Today, we consider that it is between 30 and 50 nanogrammes inclusive per
millilitre. The lower limit has therefore been increased and, consequently, the number of people under this norm will have increased. With this norm, we arrive at three quarters of post-menopausal women with vitamin D deficiency. We could almost propose systematic supplementation. I would also point out that this does not only concern post-menopausal women: an American study has thus shown that one in two twenty-five-year-old medical students has vitamin D deficiency.

Dr Gabriela Mendoza

The threshold has been raised, but on a scientific basis. This new threshold was considered on the secondary hyperparathyroidism reaction. Until now, the threshold was too low. Today, we have a higher threshold which corresponds to the physiological reality. So 80% of patients now need vitamin D supplements.

Dr Mickaël Rouillé

In the zone between 20 and 30 nanogrammes inclusive, there was still bone hyper-remodelling. We were not managing to reach normal bone remodelling.

I see this in my practice. Dosing of vitamin D is relevant. Five times out of six, it leads to a therapeutic approach. Moreover, the drugs used to treat patients, which are very expensive, work all the better on people who are not deficient in calcium and vitamin D. The drugs used to treat osteoporosis work less well on people with a vitamin D deficiency. There is even a theoretical risk that a serious vitamin D deficiency facilitates fractures.

For calcium, there is a French questionnaire that is very simple to fill in. It estimates the dietary calcium intake very accurately. As far as vitamin D is concerned, I personally choose to supplement almost 95% of my patients.
How to rate osteoporosis?

Dr Dominique Lannes • Associate Medical Director - SCOR Global Life

1. Osteoporosis in an insurance context

Today, osteoporosis affects women over fifty, after the menopause. Let’s imagine an insurer’s portfolio, whose target population it is. The risk feared by the insurer is the fracture, and that increases with age.

Insurance policies and bank loans are getting longer and longer. Many women take out insurance at the age of thirty, forty or fifty. Eventually they will join this population at risk of osteoporosis. Moreover, in France new “seniors” policies have been introduced. The age limit for them is seventy-five. A person can insure themselves up to the age of eighty-five. We are there precisely in the major osteoporosis risk zone. Finally, “dependency” policies are appearing more and more. The average age for taking out these policies is sixty-two years, precisely in this population threatened by osteoporosis. For these policies, which are getting longer, a new approach to risk is appearing. A new segmentation in insurance no doubt needs to be invented.

2. How to identify osteoporosis

A. Context

Osteoporosis therefore concerns women over fifty. It can happen that osteoporosis is not detected in an insurance declaration. But some women are osteoporotic without knowing it. Other women are not, yet receive preventive treatment. In theory, they are at no risk. There are also women who have established osteoporosis, whether it is treated or not, and who present a fracture risk or have already suffered fractures. Finally, it is this population in principle that interests us in our assessment of the risk, bearing in mind that there is still the problem of women who declare nothing and yet are osteoporotic.

What we have to do therefore is try to identify women with established osteoporosis and presenting a high risk of fractures or who have already had fractures. We can imagine that the highest risk relating to a fracture concerns dependency. Temporary inability to work and disability are also important risks, but to a lesser degree. In this section of the population, we can ask ourselves if we should not reconsider our approach to these people who take out death insurance a very late age, in view of the statistics mentioned by Dr Rousière.

B. How to spot osteoporosis on a medical questionnaire

By examining the applications, we can identify the people and their treatments by the way they answer. Quite often, women know the word “osteoporosis” but they may also use terms like “bone demineralisation”, “décalcification” or “bone fragility”. These are the four terms most commonly used.

Of course, they may declare fractures. There is no question of saying that a multiple trauma in a woman of thirty-five after a car accident is a case of osteoporosis. We are clearly talking about the case of a woman over fifty who declares fractures resulting from falling over. These will mainly be fractures of the femoral neck, the wrist and the famous vertebral fractures. In this respect, the term “spinal compression” is still in common use. Spinal compression is still referred to very often in our medical questionnaires. We must be attentive to these fractures that affect women over fifty.

The question of treatment is more complicated. It is difficult to always be able to work out what is meant. By consulting the list of declared treatments in a medical dictionary, we find references to additional treatments, adjuvant treatments, first intention preventive treatments, second intention preventive treatments, fracture prevention treatments, etc. The simplest thing is to look up the name of the drug in the reference dictionary and to find out to what class of osteoporosis it corresponds.
How to spot osteoporosis on a medical report

In medical certificates, doctors talk about osteoporosis with more or less (great) risk of fracture. Some doctors also mention osteopenia.

How to spot osteoporosis in the complementary examinations

Osteodensitometry is the most obvious solution. The problem is that this examination is only prescribed occasionally, and furthermore we do not ask for it. In France, the Social Security pays for this examination, which is the most reliable and the most objective. We believe that eventually every menopausal woman will undergo this examination. We will then be able to ask the person for her last bone densitometry results. We will be asking for this test more and more often, in particular for dependency insurance.

Selection and rating

Medical selection

When we identify osteoporosis, a medical certificate must be completed (causes, treatments, fractures, spinal compression).

As far as the last bone densitometry test done is concerned, as we have just seen, the subject is still being debated. Indeed, this examination can predict fractures, but also possibly mortality or dependency, in a non-negligible way. At the moment we do not systematically ask for it, but in years to come, we will certainly require it for certain types of insurance.

Death/TPD rating

Today, we consider that osteoporosis does not have much impact in terms of death rating. This is the case in particular because osteoporosis never appears in medical certificates as a cause of death. For the moment, treated osteoporosis without any fractures represents a normal risk for death cover. Untreated osteoporosis represents a risk rated at “normal to 25%”. Osteoporosis with fractures also represents a risk of 25%. Perhaps we need to re-examine the data provided by Dr Roušière on the risk of mortality with this approach. We now have osteoporosis within our sights and we will be monitoring this subject attentively.

Dependency rating

Apparantly osteoporosis, through the fracture, is a gateway to dependency. For the moment, osteoporosis without any fractures represents a 25% rating. A person who declares that she is receiving preventive treatment for osteoporosis and who is in good health will be given a normal rating. One day this will almost certainly require a bone densitometry test, which will be the objective reflection of the person’s situation regarding the risk of fractures.

In the case of osteoporosis with a fracture, we systematically defer for three months for dependency. After this three-month period, the sequelae of the fracture are assessed. For example, if it was a femoral neck fracture and the person can no longer walk, the decision will be
a refusal. If there is proof of the efficacy of a treatment, the application will be accepted with a rating of 25%. If the fracture is not treated and we have the impression that it is an osteoporotic fracture, the rating will be 50% and partial dependency will be refused. We are a little more severe in these cases.

In the case of osteoporosis with several fractures, we apply the same approach. We may then go as far as a refusal or a 50% rating if there is an effective treatment.
Dr John Evans

What was said about bone densitometry is more or less what was being said about pulmonary function tests (PFT) fifteen years ago. It was said that we would never obtain the PFT, that it was too expensive. Today, it is virtually systematic. Any bronchial disease almost always gives rise to a PFT. This no longer shocks any insurer. It will be the same thing for bone densitometry in ten or so years’ time. It is an objective instrument; it will be difficult to do without it.

Do you not think that a menopausal woman, who reads women’s magazines which are full of articles on the subject, will ask for this examination when she goes to see her gynaecologist or possibly her rheumatologist, especially knowing that it painless?

Dr Patrick Malamud

This is effectively a very favourable point. This examination does not require any kind of injection. It is relatively quick to do. It only lasts ten minutes or a quarter of an hour. It is certain that this is a lead for the near future. I am thinking of the parallel with cardiac ultrasound scans. When we started to work on rating, we did not always have scan results in the files. Today, it has become commonplace.

Dr Mickaël Rousière

The examination is reliable as long as it is well done technically. Sometimes it is poorly done and the results are of no use. In this case, I am obliged to ask for another examination, and the results are sometimes completely different. It is therefore necessary to have the right apparatus, the right reference curves. Above all, it is an examination that is very sensitive to the position of the patient. In particular, if you do two examinations, you must respect the same conditions of measurement. This is an easy examination as long as a little time is taken to get the patient in the right position.

How do we recognise the quality of a bone densitometry test?

Dr Mickaël Rousière

This is the problem of specialisation in rheumatology. For example, for the femoral neck, the spine must be straight during the examination. Sometimes the patients are installed the wrong way across the table... The bone must be in the centre of the frame of a measuring window and relatively straight. Moreover, certain apparatus has an American measurement curve adaptable to the French population. Now the old 91 and 92 curves are wrong. If the apparatus has a 91 or 92 reference, we can suppose that the examination might not be reliable. It can also happen that the technician does not remember to change the reference curve. Furthermore, we must also take account of the skin colour. There are in fact genetic differences between white, black and Asian skin. It is necessary for the radiographer to be used to carrying out this examination, but I note that for at least one bone densitometry test in two, significant criticisms can be made. There remains much progress to be made in this domain.
In this Focus we have presented the rheumatological pathologies most frequently encountered in our impaired risk activity. Of course there are others, but we have chosen to deal with those most representative of our activity so that you may benefit from our experience.

We can conclude that over the last few years, medical progress and its consequences for rheumatology have been considerable concerning:
• the appearance of new treatments
• their place in therapeutic strategies
• the improvement of care and patients’ quality of life
• the increase in survival rates in certain rheumatismal pathologies.

These advances, in particular the improvement in quality of life and survival, have important repercussions for the way we analyse and assess the applications of people affected by these diseases in insurance medicine.

At SCOR Global Life we are constantly trying to adapt our rating practices to the advances made in the fields of diagnosis and treatment. We must take into account these medical advances and integrate them into our risk selection and rating policies so that we are able to offer insurability solutions to the great majority of sick people. Thus, whenever it is possible, and depending on the information collected allowing an objective assessment of the risk, we will try to offer the most appropriate conditions of insurance.