OBESITY AND ARTHROSIS

DEFINITION

The body mass index (BMI) for an individual is their weight in kilograms (kg) divided by their height in squared metres, and this correlates with their total body fat. (IMC = \( \frac{W}{H^2} \)) Obesity is defined as a BMI > 30 kg/m² and morbid obesity as a BMI > 40 kg/m².

MECANISM OF ACTION OF OBESITY LEADING TO ARTHROSIS?

Overweight leads to arthrosis through various factors:
1) the level, frequency, and type of constraints induced by overweight have a direct mechanical effect on the cartilage, meniscus, and ligaments
2) mechanical receptors of the cellular membrane of chondrocytes allow the chondrocyte to evaluate the articular pressure. This mechanical signal induces chemical liberation of mediators and enzymes that lead to the destruction of extracellular matrix of the cartilage, and to modification of the inflammation process.

DOES OBESITY INFLUENCE ARTHROSIS OUTCOME?

Obesity increases pain level and decreases function of the arthritic knee, motion being limited by the mass of soft tissues surrounding the knee.
The less the patient moves, the more he increases his weight, leading to an acceleration of arthrosis outcome (1-2-3-4).
Malalignment of the limb is a factor that increases arthrosis.

There is strong evidence to suggest that obesity may be a predisposing factor in the development of osteoarthritis of the knee, while the relationship is not obviously demonstrated in hip arthrosis outcome (5).
Increasing 1 point of BMI leads to 15% augmentation of arthrosis outcome (6).

A shift from normal body mass index (BMI 25 kg/m²) to overweight (BMI >25 kg/m²) is associated with a higher relative risk of knee OA requiring arthroplasty than persistent overweight from 20–50 years of age, compared with those with normal relative weight during the corresponding age period (7).

Obese joint arthroplasty patients in USA have increased significantly since 1990. The mean BMI increased significantly from 27.8 in 1990 to 31.3 in 2005. Percentage of obese patients (BMI >30) in 1990 was 30.4% and increased significantly to 52.1% in 2005. The percentage of obese patients (BMI, 30-39) in 1990 was 25% and increased significantly to 39% in 2005 (8).
In contrast, the percentage of normal patients in 1990 was 26% and decreased significantly reaching a low of 10% in 2005 (8).

DOES OBESITY INFLUENCE JOINT REPLACEMENT OUTCOME?

Patients with a BMI > 30 have a functional result lower than those with BMI < 30 (15-16).
The literature is divided over the influence of obesity on outcome inTKR. Some reports have found similar results for obese and nonobese patients. Several studies have shown no difference in outcome at one, five, and ten years between the two groups, and the rate of peri-operative complications was similar (9-10-11-12-13), whereas others describe obesity
as having a negative influence on outcome (14-15). An analysis of results in obese patients who are morbidly obese has consistently demonstrated worse results in the morbidly obese patients when compared with nonmorbidly obese and nonobese patients (16-17-18).

GENERAL COMPLICATIONS: COMORBIDITY

The BMI is a predictor of morbidity and mortality from several chronic diseases, including diabetes mellitus, coronary artery disease and stroke (13).

Obesity has been generally held to increase the risk of venous thromboembolism although with effective prophylaxis this should be negated (19-20-21).

There was a high rate of revision and of peri-operative complications; 22% had wound complications and 10% deep infection (22).

In a study of mainly morbidly obese patients, the intra-operative subcutaneous tissue oxygen tension was found to be significantly lower than in a nonobese control group. As the risk of infection is inversely related to tissue oxygen partial pressure, a lower peri-operative tissue oxygenation may explain the high rate of wound complications noted in morbidly obese patients. Infection may be significantly higher for patients with a BMI > 35 kg/m² (23).

SURGERY

The technical aspects of the procedure is more difficult needing a large approach, (more bleeding), Increased operative times, Increased risk of complications, and Increased hospital stays.

It has been noted an increase in peri-operative blood loss in patients with a BMI greater than 30 kg/m² compared with those in whom it was lower (24-25).

We could add the increased physical stress and injury to surgical team.

PROSTHESIS OUTCOME

Health risks increase as the BMI rises from normal (BMI < 25) to overweight (BMI = 25 to 30), to obese (BMI = 30 to 40) and to morbidly obese (BMI > 40) (26-27).

Bodyweight has not been shown to have a consistent effect on the rate of wear of polyethylene. Wear has been shown to be a function of use and since the obese tend to have a lower level of activity, their increased weight is unlikely to affect the wear characteristics of the acetabular component.

Increased body-weight results in increased loading across a TKR and the surrounding bone (5). There are more complications and revision for loosening. They are more frequent and earlier than in normal subjects (29-30).

AMIN and Al (31) demonstrated that there is a significantly higher rate of radiolucent lines around the TKA implants in the morbidly obese patients compared with the nonobese group (29% and 7%, respectively), with approximately half being progressive. Using revision as an end-point, the five-year survivorship was 74.2% (53.8 to 94.6) in the morbidly obese group, compared with 100% in the non-obese group.

Using revision and pain as the end-point, the five-year survivorship was 72.3% (52.1 to 92.5) in the morbidly obese group and 97.6% (92.9 to 100) in the nonobese group (31).

Murray (32), and Foran (30) showed worse results in midterm survival as shown in the following figures.
from: Murray & Al (32): Kaplan-Meier five-year survivorship curve using revision and pain as an end-point.

from Foran & al (30): Kaplan-Meier survivorship curves for failure of the prosthesis, with a reoperation, clinical failure (a KSS <80 points), and radiographic failure as the end points, revealing a 91.7% ± 11.8% chance of survival in the morbidly obese subgroup, an 83.6% ± 8.7% chance in the nonmorbidly obese group, and a 98.7% ± 1.9% chance in the nonobese group.

Wear has been shown to be a function of use and since the obese tend to have a lower level of activity, this may compensate for the higher load across the knee replacement, so that it could not affect the wear characteristics of the polyéthylène.

**LOOSING WEIGHT**

Loosing weight is important, but not anyhow.
First step is to do a medical checkup to evaluate:
- BMI evolution
- determine factors involved in overweight: malnutrition, way of life, starting events, genetic problems, etc.
- muscular mass and fat tissues evaluation

Weight is related to what the person ate a long time ago, and to the maladjustment between food and current physical activity.
How to loose weight? The practician will give you a specific treatment but the principles are to loose weight progressively, not to re-overweight, to pratice physical exercises, and to be well minded.
In case of BMI > 40 advices from a nutritionist will be necessary.

CONCLUSION

Health risks increase as the BMI rises from normal (BMI < 25) to overweight (BMI = 25 to 30), to obese (BMI = 30 to 40) and to morbidly obese (BMI > 40).

However, morbidly obese patients may expect improvement in pain and function following joint replacement, although the overall results are inferior when compared with non-obese patients. TKA will improve considerably their quality of life and, provided they have been made aware of the increased risks, operation should not be withheld. We consider there is no justification for denying a joint replacement to a patient with disabling arthritis solely because of obesity.

But of course, patients with a body mass index superior to the normal (specialy those with a BMI >30) should be advised to lose weight prior to TKA and to maintain weight reduction. They should also be counselled regarding the inferior results which may occur if they do not lose weight before surgery.
REFERENCES


