Original article

Influence of preoperative factors on the gain in flexion after total knee arthroplasty

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ABSTRACT

Final flexion mobility after a total knee arthroplasty is an important factor in patient comfort. Some patients gain in flexion mobility, others do not. Is it possible to identify the clinical factors related to the patient that predicted the final gain in flexion?

Materials and methods: A multicenter retrospective study directed by the Société française de la hanche et du genou (SFHG) was conducted on 1601 cases of total knee arthroplasty that had presented no complications and a minimal follow-up of 2 years. The gain in flexion was assessed by the difference between the preoperative and the final range of flexion. The range of the gain in flexion was tested based on eight factors: age, gender, etiology, body mass index, frontal deformed, preoperative flexum deformity and four levels of preoperative mobility: < 90°, 90°–109°, 110°–129°, and ≥ 130°.

Results: A mean gain in flexion of 8.4°±14° was found for the overall series. In 66% of cases, we found an increase of flexion and in 19% a loss of flexion. In cases with BMI higher than 35, varus deformity with an HKA angle < 166°, or flexum greater than 5°, the gain in flexion was significantly higher. A significantly different gain in flexion (P < 0.0001) was found in the four levels of preoperative flexum: the greatest gain in flexion was found in the “< 90°” group, then this gain was less in the next two groups, to become a significant decrease in the “≥ 130°” group. A decrease in flexion was noted in 51% of the cases in the latter group. Other factors such as age, sex, and etiology had no influence on the gain in flexion.

Discussion: After TKA, a gain in flexion was often noted. The amount of gain depended on the preoperative range of flexion: the lower this level was, the more flexion increased. The presence of a varus deformity, morbid obesity, or flexum was associated with greater gain in flexion, even if the final flexion was lower than the mean flexion in the overall population. The search for these factors made it possible to predict a gain in flexion and discuss this with the patient.

Level of evidence: Level IV. Multicenter retrospective study.

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1. Introduction

For a knee having undergone total knee arthroplasty (TKA) to function normally, it should provide 130° flexion and have complete extension [1–3]. These two criteria are necessary for ease in daily activities and to execute certain more demanding activities [4–8].
Postoperative mobility influences the degree of final patient satisfaction [5] and is a frequent question during preoperative consultations [9–11]. Knee flexion [12] is frequently restricted before surgery [13,14]. For several authors, preoperative flexion is the most important factor [15–17]. A high body mass index (BMI) may also decrease final flexion [18,19].

For Lizaru et al. [20], the severity of osteoarthritis may have a negative influence on the final range of flexion, and Matsuda et al. [21] reported that varius or valgus deformity may have an effect on final flexion.

For many authors, final flexion was found to be improved compared to preoperative flexion [1,3,9,10,13,14,17], even though this gain in flexion may not be consistent. The value of preoperative flexion also varies and may be influenced by clinical factors [4,7,8,11,12,18–21].

We therefore retained as the main criterion in our analysis the difference in range of postoperative and preoperative flexion, which we have called “gain in flexion”: this value was considered either a gain in flexion if positive or a loss of flexion if negative. The main hypothesis was that this gain in flexion was influenced by patient factors, and our secondary hypothesis was that total knee arthroplasty (TKA) increases range of movement in flexion.

2. Material and methods

Within the Société française de la hanche et du genou (SFHG [French Hip and Knee Society]), a multicenter retrospective study grouped 1601 knees that had undergone TKA in five centers. The TKAs were performed between 2000 and 2010 on cases of first-line treatment with a minimum follow-up of 2 years (mean follow-up, 51 months; range, 24–221 months).

The exclusion criteria were constrained or hinged prostheses as well as cases having presented complications that could have an effect on range of movement: infection, fracture, instability, loosening, or malposition.

The range of movement in flexion was assessed in degrees preoperatively and at the last follow-up. The presence of flessum was evaluated in the same way. The preoperative HKA angle was measured in all patients on long-leg films done to assess any frontal deformity.

We sought any influence of preoperative clinical factors on the variation in the gain in flexion: age, gender, preexisting pathology, BMI, frontal deformity, the presence of preoperative flessum, and the preoperative value of range of movement in flexion.

The patient’s osteoarthritis was categorized as idiopathic, rheumatoid, post-traumatic, postnecrotic, or other cause.

The preoperative BMI was categorized into five groups: thin (< 20), normal (20–24), overweight (25–29), obese (30–34), and morbidly obese (> 34).

The presence of a preoperative frontal deformity was quantified using the hip and knee angle (HKA angle) calculated on a long-leg film. Five groups were distinguished according to the HKA angle: < 166°, 166°–176°, 177°–183°, 183°–193°, and > 193°. The presence of preoperative flessum was classified according to its severity: ≤ 5°, 6–10°, 11–15°, 16–20°, > 20°. Preoperative flexion was divided into four groups: stiffness (≤ 90°), limited flexion (91°–110°), normal flexion (111°–130°), and high flexion (> 130°).

Different TKA models have been used and we sought a difference in gain in flexion between the posterior stabilized prosthesis with a fixed-bearing or mobile tibial plateau, a prosthesis with great femoral flexion, a concave-convex geometry prosthesis, and a prosthesis preserving the posterior cruciate ligament.

The statistical analysis was done by the Lille centre de bio-statistiques (Lille Biostatistics Center) using SAS 9.3 (SAS, Cary, NC, USA). Normality of the quantitative items was verified using the Shapiro–Wilk Test. For the analyses comparing qualitative data, the McNemar Test was applied. To compare the numeric data, the Student t-test or the Wilcoxon test was used. With multiple comparisons, a Bonferroni adjustment was carried out. The first–species risk was set at 5%.

3. Results

The mean age of the patients was 71 ± 8 years (range, 22–96 years); the mean BMI was 29.1 ± 5.36 (range, 17–51); 71% of the subjects were female and 29% male.

The TKA resulted in increased range of movement in flexion. The gain in flexion of the overall series was 8.4 ± 14° (range, −40° to 95°) (P = 0.0001) with final flexion at 123° ± 12° (range, 75–155°) for preoperative flexion at 114.6 ± 15° (range, 30–150°). The gain in flexion was positive in 66% of the 1058 cases with a mean gain in flexion of 16° ± 14° (range, 5°–95°), it was null in 15% of the cases (237), and negative in 19% of the cases (306): −10° ± 7.1° (range, −5° to −40°).

Age, gender, and etiology had no significant influence on the gain in flexion (Table 1). The BMI significantly influenced the gain in flexion: the gain in flexion increased as BMI increased; the BMI > 35 group had a statistically different gain in flexion. However, the greater the preoperative BMI, the lower the preoperative flexion was, and despite this higher gain in flexion, the flexion at revision remained below the mean in the other groups (Table 2).

The existence of a substantial preoperative varus deformity (HKA ≤ 165°) was related to a greater gain in flexion compared to the “HKA 166–176°” group (P < 0.0026) or the “HKA 177–183°” group (P < 0.01) (Table 3). The patients who had a substantial varus deformity had lower preoperative flexion.

Preoperative flessum (> 5°) (29%; 467 cases) improved the gain in flexion: the greater the flessum the greater the gain (Table 4). There was a significant difference between the group with no flessum (< 5°) and each of the other groups. There was a significant difference between the (> 20°) group and the (6–10°), (11–15°), and (16–20°) groups. At follow-up, flessum (> 5°) persisted in 5% of the cases (83 cases).

A different behavior was noted for the four levels of preoperative flexion (°), with a statistically different gain in flexion between each group (P < 0.0001).

The “stiff knee” group (68 cases) had a greater gain in flexion: 35 ± 17° (range, 0°–95°). This group had the lowest preoperative flexion: 75 ± 16° (range, 30–80°). The gain in flexion was always positive except in one case. There was never a loss in flexion at the final follow-up. Despite this higher gain in flexion, the mean range of movement in flexion at revision (110° ± 13°; range, 85–135°) was not as high as that obtained in the other groups. The knees in this group increased one or two ranges-of-movement groups.

The limited flexion group (368 cases) had a gain in flexion of 17° ± 12° (range, −15° to 45°) with a mean preoperative flexion of 99 ± 5°. In 87% of the cases (319), a gain in range of movement was noted, in 9% (35) stability was observed, and in 4% (14) a loss of flexion. The mean flexion at revision was 117° ± 11° (range, 75°–145°). In more than eight cases out of ten, the patients in this group had a positive gain in flexion and found satisfactory range of movement in flexion.

The “normal knee flexion” group (110–129°) accounted for half of the patients in the study (832 cases). The gain in flexion was 7° ± 11° (range, −35° to 45°) for a mean preoperative flexion of 117° ± 6°. In 71% of the cases (594 cases), a gain in range of motion was noted, in 14% of the cases (114 cases), it remained identical, and in 15% of the cases (124 cases), the final flexion decreased. At revision, the mean flexion was 124° ± 10° (range, 80–155°). In this
Table 1
Gain in flexion of the series, influence of gender and etiology on gain in flexion.

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean gain in flexion ± SD</th>
<th>Preoperative flexion</th>
<th>Flexion at revision</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall series</td>
<td>1601</td>
<td>8.5 ± 14° (−40° to 95°)</td>
<td>114 ± 15° (30°–150°)</td>
<td>123 ± 12° (75°–155°)</td>
<td>&lt;0.0001*</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1141</td>
<td>9 ± 14° (−40° to 85°)</td>
<td>113 ± 15° (35°–95°)</td>
<td>122 ± 12°</td>
<td>=0.08b</td>
</tr>
<tr>
<td>Male</td>
<td>460</td>
<td>8 ± 13° (−35° to 95°)</td>
<td>116 ± 14°</td>
<td>124 ± 12°</td>
<td></td>
</tr>
<tr>
<td>Etiology</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary osteoarthritis</td>
<td>1451</td>
<td>8 ± 14° (−40° to 95°)</td>
<td>115 ± 13°</td>
<td>123 ± 11°</td>
<td>=0.55a</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>56</td>
<td>10 ± 16° (−25° to 45°)</td>
<td>110 ± 12°</td>
<td>120 ± 10°</td>
<td></td>
</tr>
<tr>
<td>Post-traumatic</td>
<td>20</td>
<td>12 ± 17° (−15° to 60°)</td>
<td>103 ± 17°</td>
<td>115 ± 15°</td>
<td></td>
</tr>
<tr>
<td>Secondary to necrosis</td>
<td>54</td>
<td>9 ± 15° (−30° to 55°)</td>
<td>116 ± 15°</td>
<td>125 ± 12°</td>
<td></td>
</tr>
</tbody>
</table>

* Preoperative flexion and flexion at revision are significantly different (P<0.0001).
b There was no significantly different gain in flexion between males and females.
a There was no significantly different gain in flexion between the different etiologies.

Table 2
Influence of body mass index (BMI) on gain in flexion.

<table>
<thead>
<tr>
<th>BMI</th>
<th>n</th>
<th>Mean gain in flexion ± SD</th>
<th>Preoperative flexion</th>
<th>Flexion at revision</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Thin” group (BMI &lt; 20)</td>
<td>26</td>
<td>6 ± 18° (−35° to 45°)</td>
<td>114 ± 21°</td>
<td>120 ± 17°</td>
<td></td>
</tr>
<tr>
<td>“Normal” group (BMI ≥ 20 to &lt; 25)</td>
<td>271</td>
<td>6 ± 14° (−30° to 50°)</td>
<td>118 ± 15°</td>
<td>124 ± 13°</td>
<td></td>
</tr>
<tr>
<td>“Overweight” group (BMI ≥ 25, &lt; 30)</td>
<td>652</td>
<td>8 ± 14° (−40° to 95°)</td>
<td>116 ± 15°</td>
<td>124 ± 11°</td>
<td></td>
</tr>
<tr>
<td>“Obese” group (BMI ≥ 30 to &lt; 35)</td>
<td>426</td>
<td>9 ± 14° (−35° to 80°)</td>
<td>113 ± 14°</td>
<td>122 ± 11°</td>
<td></td>
</tr>
<tr>
<td>“Morbidly obese” group (BMI ≥ 35)</td>
<td>224</td>
<td>12 ± 14° (−30° to 65°)</td>
<td>107 ± 15°</td>
<td>119 ± 12°</td>
<td>&lt;0.001a</td>
</tr>
</tbody>
</table>

There was a significant difference in gain in flexion between the five BMI groups (P < 0.001).
a The “morbidly obese” group had a significantly higher gain in flexion than the other groups (P < 0.001).

Table 3
Influence of preoperative frontal deformations on gain in flexion.

<table>
<thead>
<tr>
<th>Preoperative deformation</th>
<th>n</th>
<th>Mean gain in flexion ± SD</th>
<th>Preoperative flexion</th>
<th>Flexion at revision</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HKA &lt; 166°</td>
<td>121</td>
<td>13 ± 14° (−25° to 55°)</td>
<td>109 ± 14°</td>
<td>122 ± 15°</td>
<td>&lt;0.0026a</td>
</tr>
<tr>
<td>HKA 166°–176°</td>
<td>736</td>
<td>8 ± 14° (−40° to 65°)</td>
<td>115 ± 14°</td>
<td>122 ± 14°</td>
<td>&lt;0.0026a</td>
</tr>
<tr>
<td>HKA 177°–183°</td>
<td>337</td>
<td>8 ± 15° (−35° to 95°)</td>
<td>116 ± 17°</td>
<td>125 ± 15°</td>
<td>&lt;0.01b</td>
</tr>
<tr>
<td>HKA 184°–194°</td>
<td>309</td>
<td>9 ± 13° (−30° to 45°)</td>
<td>116 ± 14°</td>
<td>125 ± 13°</td>
<td>NS</td>
</tr>
<tr>
<td>HKA &gt; 194°</td>
<td>98</td>
<td>10 ± 14° (−25° to 45°)</td>
<td>111 ± 14°</td>
<td>121 ± 14°</td>
<td>NS</td>
</tr>
</tbody>
</table>

There was a significant difference between the groups: P=0.0024.
a There was a significant difference between the “HKA < 166°” group and the “HKA 166°–176°” group (P = 0.0026).
b There was a significant difference between the “HKA < 166°” group and the “HKA 177°–183°” group (P = 0.01).

Table 4
Influence of preoperative flexion value and gain in flexion.

<table>
<thead>
<tr>
<th>Preoperative flexion</th>
<th>n</th>
<th>Mean gain in flexion ± SD</th>
<th>Preoperative flexion</th>
<th>Flexion at revision</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 5°</td>
<td>1133</td>
<td>7 ± 14° (−40° to 95°)</td>
<td>117 ± 14°</td>
<td>124 ± 11°</td>
<td></td>
</tr>
<tr>
<td>6–10°</td>
<td>211</td>
<td>11 ± 13° (−25° to 45°)</td>
<td>111 ± 13°</td>
<td>122 ± 11°</td>
<td>=0.002a</td>
</tr>
<tr>
<td>11–15°</td>
<td>149</td>
<td>11 ± 13° (−25° to 45°)</td>
<td>109 ± 12°</td>
<td>120 ± 11°</td>
<td>=0.03a</td>
</tr>
<tr>
<td>16–20°</td>
<td>54</td>
<td>14 ± 14° (−10° to 50°)</td>
<td>104 ± 14°</td>
<td>118 ± 11°</td>
<td>=0.005a</td>
</tr>
<tr>
<td>&gt; 20°</td>
<td>53</td>
<td>20 ± 16° (−20° to 65°)</td>
<td>98 ± 16°</td>
<td>118 ± 12°</td>
<td>&lt;0.0001a</td>
</tr>
</tbody>
</table>

There was a significant difference between the groups: P<0.0001.
a There was a significant difference between the (≤ 5°) group and the four other groups.
Table 5 Influence of the level of preoperative flexion on gain in flexion.

<table>
<thead>
<tr>
<th>Level of preoperative flexion</th>
<th>n</th>
<th>Mean gain in flexion ± SD</th>
<th>Preoperative flexion</th>
<th>Flexion at revision</th>
<th>Progression of flexion</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stiffness (&lt; 90°)</td>
<td>68</td>
<td>35 ± 17°</td>
<td>76 ± 11°</td>
<td>110 ± 13°</td>
<td>Gain: 67 cases (98%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(48%)</td>
<td>(30–80°)</td>
<td>(85–135°)</td>
<td>Identical: 1 case (2%)</td>
<td></td>
</tr>
<tr>
<td>Limited flexion (90°–109°)</td>
<td>368</td>
<td>17 ± 12°</td>
<td>99 ± 5°</td>
<td>117 ± 11°</td>
<td>Gain: 319 cases (87%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(23%)</td>
<td>(75–145°)</td>
<td></td>
<td>Identical: 35 cases (9%)</td>
<td></td>
</tr>
<tr>
<td>Normal flexion (110°–129°)</td>
<td>832</td>
<td>7° ± 11°</td>
<td>117 ± 6°</td>
<td>124 ± 10°</td>
<td>Decreased: 14 cases (4%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(52%)</td>
<td>(80–155°)</td>
<td></td>
<td>Gain: 594 cases (71%)</td>
<td></td>
</tr>
<tr>
<td>High flexion (≥ 130°)</td>
<td>333</td>
<td>−3° ± 9°</td>
<td>133 ± 5°</td>
<td>130 ± 8°</td>
<td>Decreased: 124 cases (15%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(21%)</td>
<td>(100°–150°)</td>
<td></td>
<td>Gain: 78 cases (23%)</td>
<td></td>
</tr>
<tr>
<td>Overall data</td>
<td>1601</td>
<td>8.5° ± 14°</td>
<td>114 ± 15°</td>
<td>123 ± 12°</td>
<td>Decreased: 168 cases (51%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(−40–95°)</td>
<td>(30–150°)</td>
<td></td>
<td>Gain: 1058 cases (66%)</td>
<td></td>
</tr>
</tbody>
</table>

The gain in flexion is statistically different between each group (P < 0.0001).

4. Discussion

This study confirmed the influence of certain preoperative clinical factors on the gain in flexion and did not confirm the classic notion of final range of movement in flexion equal to preoperative range of movement after TKA. This study reports a different behavior in terms of gain in flexion depending on preoperative flexion. The gain in flexion is influenced by certain preoperative patient factors. Like Harvey et al. [4], Kotani et al. [9], and Lizarz et al. [20], this series showed no influence of age. Contrary to Ritter et al. [1] and Harvey et al. [4], there was no difference in gain in flexion between the male and female subjects. The etiology did not influence gain in flexion, contrary to the series reported by Ritter et al. [14], Kotani et al. [9], and Harvey et al. [4], who observed greater gain in cases of rheumatoid arthritis. However, our series included a greater number of primary osteoarthritis cases than other series.

The existence of morbid obesity (BMI > 35) resulted in a significantly greater gain in flexion, but the preoperative flexion and therefore, the final flexion were lower than that in the other groups. These results are in agreement with Dennis et al. [10], who found that obesity was a factor of worse final flexion.

The presence of significant preoperative varus deformity (HKA < 166°) of the lower limb had an influence on the final range of movement [17], increasing the gain in flexion, but it did not increase the flexion at revision [9]. Compared to Ritter et al. [14], we observed an influence of preoperative varus on the gain in flexion, with greater gain in flexion when varus was more severe.

This study found a different behavior depending on preoperative flexion. The gain in flexion was greater for knees with low preoperative flexion, contrary to knees with preoperative flexion at 110° or more. The final flexion value was not predicted by preoperative flexion. A knee with preoperative flexion less than 90° was systematically improved, whereas half the knees with 130° preoperative flexion resulted in decreasing flexion. A knee with preoperative flexion between 110 and 129°, i.e., half the patients, improved flexion by less than 10° in seven cases out of ten.

We found no significant influence in gain in flexion based on the type of prosthesis despite the diversity of models used. The femoral components with high flexion that increased flexion for Kim et al. [22] may facilitate recuperation of natural flexion [23,24]. However, the present study did not demonstrate a significant difference in gain in flexion, contrary to the study reported by Massin et al. [25]. In addition, as underscored by Banks et al. [26], Mont [27], Matsuda et al. [28], Ishii et al. [29], and Russell et al. [30], tibial plateau mobility had no influence on final flexion.

The results of this multicenter retrospective study grouping a large number of subjects show the benefit on flexion of a total knee replacement on knees with less than 130° of preoperative flexion.

5. Conclusion

Our hypothesis was confirmed: the gain in flexion is influenced by preoperative factors. The gain in flexion does not reflect final flexion: a stiff knee undergoes a greater gain in flexion than a knee with high preoperative flexion. Knowledge of these factors can assist in providing the patient with more precise information.

Disclosure of interest

Gilles Pasquier declares that he is an educational and research consultant for Zimmer.

Bruno Tillie declares that he is an educational and research consultant for Symbios.

Sébastien Parratte declares that he is an educational and research consultant for Zimmer.

Jean Noel Argenson declares that he is an educational and research consultant for Zimmer and Symbios, and a designer for Zimmer.

Gérard Deschamps declares that he is an educational and research consultant for Tornier and a designer for Tornier.

Michel Berchov declares that he is an educational and research consultant for Biomet and a designer Biomet.

Julia Salleron declares that she has no conflicts of interest concerning this article.


