Tourniquet-induced wound hypoxia after total knee replacement

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We have investigated whether the thigh tourniquet used during total knee replacement (TKR) influenced the development of postoperative wound hypoxia and was a cause of delayed wound healing.

We allocated randomly 31 patients (31 TKRs) to one of three groups: 1) no tourniquet; 2) tourniquet inflated at low pressure (about 225 mmHg); and 3) tourniquet inflated to high pressure of about 350 mmHg. Wound oxygenation was measured using transcutaneous oxygen electrodes.

In the first week after surgery, patients with a tourniquet inflated to a high pressure had greater wound hypoxia than those with a low pressure. Those without a tourniquet also had wound hypoxia, but the degree and duration were less pronounced than in either of the groups with a tourniquet.

Use of a tourniquet during TKR can increase postoperative wound hypoxia, especially when inflated to high pressures. Our findings may be relevant to wound healing and the development of wound infection.

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More than 30 000 total knee replacements (TKRs) are performed in the UK each year, most of which have a successful outcome. Delay in wound healing has been reported to occur in up to 22% of cases and presents as a contusion, a haematoma, skin necrosis or a superficial infection.¹⁻³ While the incidence of wound problems with current surgical techniques and prostheses is low, these complications contribute significantly to deep infection which occurs in up to 5% of cases.⁴

Wounds need oxygen for healing and to prevent infection.⁵⁻⁸ While there are many causes of delayed wound healing, delivery of oxygen is the limiting factor, and measures to minimise hypoxia should reduce the incidence of delayed healing.⁹ We postulated that the thigh tourniquet, which is used to provide a bloodless operative field for surgery and is an obvious cause of perioperative hypoxia, was a significant risk factor for the development of postoperative hypoxia. This was based on two observations. Firstly, when a tourniquet is inflated and blood flow ceases, the distal microvasculature becomes blocked with cellular debris and by vasoconstriction.¹⁰⁻¹⁴ This block is not resolved on initial reperfusion and thus tissue reoxygenation is prevented, an effect that is likely to be enhanced in damaged tissue as found in the wound flaps of a TKR. Secondly, a tourniquet can only completely stop blood flow when inflated to a high pressure. Experimental investigations have determined that this should be between 300 and 400 mmHg.¹⁵⁻¹⁹ Lower pressures stop macroscopic blood flow, but allow some microcirculation which is beneficial to the tissues by minimising capillary blockage.^{20,21} For TKR this would mean inflating the tourniquet to the lowest pressure practicable. There are no agreed figures for the level of the optimal pressure of a tourniquet. We have therefore investigated whether the thigh tourniquet used during TKR influenced the development of postoperative wound hypoxia and therefore be a cause of delayed wound healing.

Patients and Methods

We recruited 31 patients who had received TKR. Approval of the Ethical Committee and informed consent had been obtained. We excluded patients who had non-osteoarthritic disease, previous open knee surgery, systemic or local hypoxia, were receiving anticoagulant or antiplatelet agents or steroids, had a significant varus or valgus deformity, a perioperative lateral release.

Patients were allocated randomly, using sealed envelopes opened at the induction of anaesthesia, to three groups: (i) no tourniquet (NT); (ii) a tourniquet applied at a pressure of 125 mmHg (LT); or (iii) at 250 mmHg (HT) above the patient's mean anaesthetic arterial blood pressure. A standard protocol was followed utilising a tourniquet 11.5 cm

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wide, with an effective pressurising width of 9 cm (DePuy UK Ltd, Leeds, UK), with exsanguination in extension using a Rhys-Davies device where appropriate. All patients had general (non-halothane) anaesthesia, a midline skin incision, a medial parapatellar approach, insertion of a cemented Insall-Burnstein II TKR (Zimmer, Warsaw, Indiana) and skin closure using continuous vicryl (Ethicon Ltd, Somerville, New Jersey) over a single drain. They were all mobilised on the second postoperative day. No patient received thromboembolic prophylaxis or used a continuous passive motion machine. Measurement of tissue oxygenation was performed with transcutaneous oxygen-pressure electrodes (Radiometer Ltd, Crawley, UK) placed on the skin. This is a simple, non-invasive, reliable, repeatable and well-established technique for measuring the cutaneous oxygen delivery.^{22,23} The electrodes were placed on each wound flap with a reference electrode sited in the infraclavicular region to determine intra- and interpatient differences in systemic oxygen delivery both before and after operation. The electrodes were attached to two Radiometer TCM3 TINA units (Radiometer Ltd). Measurements were taken before operation and on each day after surgery for one week or until discharge from hospital.

Wound healing was not assessed as an outcome measure but it was examined by two observers for haematoma, infection, skin necrosis, dehiscence and contusion.

Statistical analysis. The results were analysed by two-way ANOVA for comparison within and between groups. Student's *t*-test was used for comparison when ANOVA detected significant differences. Fisher's exact test was used for categorical data relating to numbers of wounds in which the oxygen had reached the preoperative levels. Spearman's rank correlation was used for assessing the relationship between postoperative hypoxia and the length of time of inflation of the tourniquet. Confidence intervals (CI) are reported as 95%.

Results

No patient had cardiovascular complications or received supplementary oxygen after the first postoperative night.

Ten patients required blood transfusions of two units of packed cells which were given within 72 hours of surgery. Five were in the NT group, two in the LT group and three in the HT group.

The preoperative infraclavicular reference values, taken as an index of systemic oxygen delivery, did not differ significantly from the postoperative values either within or between groups (ANOVA, $p > 0.649$). Overall, the mean preoperative truncal transcutaneous oxygen pressure (ptcO₂) was 8.7 kPa (CI ± 0.52). The mean value on the first postoperative day was 8.1 kPa (CI ± 0.57) and was consistently the lowest recorded value on any day. On average, levels were 0.6 kPa less than the preoperative levels, but did not reach statistical significance ($p = 0.091$).

Preoperative levels of oxygenation of the medial and lateral flaps of the wound did not differ between any of the randomised groups ($p = 0.883$) or between flaps ($p = 0.56$). The mean preoperative transcutaneous oxygen pressure for the medial flap was 8.1 kPa (CI ± 0.41) and for the lateral 8.2 kPa (CI ± 0.36). The preoperative levels of both medial and lateral flaps were lower than the preoperative truncal reference values: 0.6 kPa ($p = 0.038$) and 0.5 kPa ($p = 0.093$), respectively.

Medial wound flap (Fig. 1). All three groups showed a similar pattern of postoperative wound hypoxia, although the level of hypoxia and degree of recovery differed.

In the NT group a clear reduction in oxygenation was seen during the first three days after surgery: with a maximum mean reduction of 1.38 kPa (17.4%) ptcO₂ (CI ± 1.56); this level was not statistically significant ($p > 0.17$). The recordings stabilised between the fourth and seventh postoperative day at normal or nearly normal levels.

In the LT group, in which the mean inflation pressure was 223 mmHg, the pattern of hypoxia was similar but more pronounced. Although there was no statistical difference between the results in this group and those from the NT group ($p > 0.086$), the levels did drop significantly from preoperative levels for the first four days after surgery ($p < 0.022$, with a maximum mean reduction of 2.29 kPa (27.5%) ptcO₂ (CI ± 1.39)).

In the HT group in which the overall mean inflation pressure was 352 mmHg, there were significant reductions in oxygenation compared with the preoperative level on all days after surgery ($p < 0.0021$); the maximum mean reduction was 3.20 kPa (40.6%) ptcO₂ (CI ± 1.27). The levels were significantly lower than those of the NT group after day two ($p < 0.042$), and were significantly lower than those of the LT group only on the sixth postoperative day ($p = 0.038$).

A pattern of hypoxia was thus evident in all groups. The medial wound flaps suffered maximal hypoxia during the first three days after operation, followed by a variable degree of recovery.

Lateral wound flap (Fig. 2). In the NT group, significant reductions in oxygenation were seen for the first four postoperative days ($p < 0.018$), with a maximum mean reduction of 2.39 kPa (29.1%) ptcO₂ (CI ± 1.19). A gradual return to normal levels was observed over the following three days.

In the LT group, significant reductions from preoperative levels were identified on all postoperative days ($p < 0.0021$), with a maximum mean reduction 3.37 kPa (41.6%) ptcO₂ (CI ± 1.56). Statistically significant lower levels compared with the NT group were seen between the fourth and seventh days ($p < 0.027$), implying that the gradual return to postoperative levels was not occurring.

In the HT group the levels of hypoxia were most marked and most prolonged. There were significant reductions in oxygenation from preoperative levels on all days after surgery ($p < 0.0097$); the maximum mean reduction was

Fig. 1

Graph showing mean values ($\pm$ SEM bars) of the oxygenation of the medial wound flap in each of the three randomised groups.

Fig. 2

Graph showing mean values ($\pm$ SEM bars) of the oxygenation of the lateral wound flap in each of the three randomised groups.

6.60 kPa (79.5%) ptcO₂ (CI $\pm$ 1.38). The levels were significantly lower than those for both the NT and the LT groups on all postoperative days ($p < 0.017$), indicating that the hypoxia in the HT group was prolonged.

Return to preoperative levels of oxygenation. As a measure of recovery from the initial postoperative hypoxia, a calculation was made of the number of wound flaps which had returned to within ± 0.5 kPa ptcO₂ of preoperative levels at one week.

In the NT group, 14 out of 20 wound flaps had returned to normal within one week of surgery. This compared with 10 out of 20 in the LT group and 3 out of 20 in the HT group ($p = 0.000567$).

Critical hypoxia (Table I). We recorded the development of hypoxia and the level of wound oxygenation below which normal cell function and therefore wound healing may be affected. We chose a level of 3.0 kPa ptcO₂ since this reflects data on the function of fibroblasts and leukocytes.²⁴

Table I. Number of wound flaps becoming critically hypoxic after TKR

	Lateral flap	Medial flap	p value
No tourniquet	2	1	0.16
Low-pressure tourniquet	3	1	
High-pressure tourniquet	6	2	

In 15 patients, levels of hypoxia in wound flaps which were 'critically' low were recorded on two consecutive days, usually in the first three days after surgery. Hypoxia of the medial wound flap was always associated with hypoxia of the lateral flap but not necessarily vice versa.

Length of time of application of the tourniquet (Fig. 3). This was examined, as a possible independent variable affecting wound oxygenation, by performing a Spearman rank-correlation analysis. The graph shows that in our study there was no relation between the period of inflation and the development of postoperative hypoxia in either tourniquet group.

Fig. 3

Scatter plot and correlation lines showing the relationship between postoperative hypoxia and length of time of tourniquet inflation. Correlation values are 0.127 and 0.097.

Subjective assessment of wound healing. Five skin flaps (three lateral and two medial) in three wounds were identified with delayed wound healing. Two lateral (one HT and one NT group) and one medial (HT group) flaps were noted to be contused. One wound (two flaps) was considered to be superficially infected (LT group). This was confirmed by culture and resolved by conservative treatment.

All the flaps which were identified as contused were critically hypoxic for at least 48 hours after surgery. The infected wound had marked hypoxia, but was not in the 'critical' range.

Complications. One patient was withdrawn because the chosen tourniquet pressure in the LT group was too low to provide adequate haemostasis. This occurred mid-way through the study and necessitated including an extra patient, thus increasing the study size to 31.

Discussion

The assessment of wound hypoxia after TKR provides an objective outcome measure for comparison of different groups and is an alternative method for the subjective assessment of wound healing and infection.

In a previous study Johnson⁹ studied wound hypoxia after TKR. All the procedures were performed with a tourniquet and the role of the incision, continuous passive motion and lateral release on wound hypoxia was assessed. Neither the role of the tourniquet nor its pressure was examined as separate risk factors for hypoxia.

The use of a tourniquet during TKR is widespread but there are risks of the occasional associated local necrosis, neurapraxia, vascular injury and local postoperative pain. In our study we have shown that wound hypoxia is more likely to occur when a tourniquet is used. The tourniquet and the pressure employed affected not only the level of hypoxia, but also the duration.

It could be argued that wound hypoxia is not important unless it reaches levels at which breakdown is likely. Critical levels of hypoxia at which normal cell function would be affected¹⁹ were seen in all three groups, but most

often in the those with a high tourniquet pressure. The lateral flap was most at risk despite having similar pre-operative levels to the medial side. These findings are in agreement with those of Johnson.⁹ It is possible that the vascular supply is particularly prone to damage in the thin lateral flap which is often created during surgery.

In order to minimise the number of complicating factors which could have affected our results, no patient received thromboembolic prophylaxis or supplementary oxygen after the first postoperative night. With thromboembolic prophylaxis, it is possible that the agents used by many surgeons (e.g. heparin, warfarin, NSAIDs) may interfere with the intravascular activity which occurs on inflation and deflation of the tourniquet thus affecting tissue reoxygenation. Whether this would enhance or reduce oxygenation is unknown but will be the subject of a future trial. When supplementary oxygen therapy is administered by nasal cannula or mask, it would seem sensible to try to maintain this treatment for at least 48 hours in all patients after surgery to minimise hypoxia of the flap. Compliance with this could not easily be assessed and may be difficult to enforce if chosen.

Potential complicating factors in the assessment of hypoxia after TKR in which one group has been operated on with a tourniquet and the other without, relate to blood loss and to differences in the circulatory delivery of oxygen either from anaemia or the reduced oxygen-carrying capacity of transfused blood. We do not believe that this would have adversely affected our results since it has previously been shown that in the absence of circulatory failure, the haemoglobin level does not affect wound healing.^{25,26} While there was no significant difference in the frequency of postoperative blood transfusion, it was clear that the patients operated on without a tourniquet received their transfusions earlier than the other groups whether during or after operation. Our results have clearly shown that the NT group had the highest postoperative levels of wound oxygenation, suggesting that blood transfusion has little effect on this. Surgery without a tourniquet is usually somewhat more demanding, with the possibility of increased damage to the skin due to prolonged

or excessive retraction, but in our study the wound flaps of the NT group were well oxygenated.

The tourniquet failed in one out of 11 patients in the LT group and none of the HT group. The lack of haemostasis in the patient in the LT group was unexpected but was noted immediately on incising the wound. This patient had to be excluded from the study. In attempting to minimise tourniquet pressures, it is quite probable that some failures will occur. A larger study of 46 patients with tourniquet pressures of 100 mmHg above systolic blood pressure recorded three failures (6.5%).¹⁷ Usually, there appear to be no ill-effects if the problem is immediately recognised and the tourniquet pressure increased, as occurred in our single failure. No further per- or postoperative complications were noted.

If a tourniquet is to be used for TKR it should be inflated to the lowest possible pressure to minimise wound complications. In our study we used a value of 125 mmHg above the mean anaesthetic arterial blood pressure. Higher pressures offer no advantage. Performing the operation without a tourniquet minimises postoperative hypoxia, but does not eliminate it completely.

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References

1. Insall JN, Lachiewicz PF, Burstein AH. The posterior stabilized condylar prosthesis: a modification of the total condylar design: two-to-four-year clinical experience. *J Bone Joint Surg [Am]* 1982;64-A:1317-23.
2. Matthews LS, Kaufer H. The spherocentric knee: a perspective on seven years of clinical experience. *Orthop Clin North Am* 1982;13:173-86.
3. Simison AJM, Noble J, Hardinge K. Complications of the Attenborough knee replacement. *J Bone Joint Surg [Br]* 1986;68-B:100-5.
4. Bengtson S, Knutson K. The infected knee arthroplasty: a 6-year follow-up of 357 cases. *Acta Orthop Scand* 1991;62:301-11.
5. Niinikoski J, Grislis G, Hunt TK. Respiratory gas tensions and collagen in infected wounds. *Ann Surg* 1972;175:588-93.
6. Stephens FO, Hunt TK. Effect of changes in inspired oxygen and carbon dioxide tensions on wound tensile strength: an experimental study. *Ann Surg* 1971;173:515-9.
7. Silver JA. The physiology of wound healing. In: Hunt TK, ed. *Wound healing and wound infection: theory and surgical practise*. New York: Appleton Century Crofts, 1980:11-28.
8. Lundgren CE, Zederfeldt BH. The influence of low oxygen pressure on wound healing. *Acta Chir Scand* 1969;135:555-8.
9. Johnson DP. Infection after knee arthroplasty: clinical studies of skin hypoxia and wound healing. *Acta Orthop Scand Suppl* 1993;252:1-48.
10. Lee YH, Wei FC, Lee J, Su MS, Chang YC. Effect of post-ischaemic reperfusion on microcirculation and lipid metabolism of skeletal muscle. *Microsurgery* 1995;16:522-7.
11. Menger MD, Steiner D, Messmer K. Microvascular ischaemia-reperfusion injury in striated muscle: significance of 'no reflow'. *Am J Physiol* 1992;263:1892-900.
12. Sirsjo A, Lehr HA, Nolte D, et al. Hyperbaric oxygen treatment enhances the recovery of blood flow and functional capillary density in post-ischaemic striated muscle. *Circ Shock* 1993;40:9-13.
13. Concannon MJ, Kester CG, Welsh CF, Puckett CL. Patterns of free radical production after tourniquet ischaemia: implications for the hand surgeon. *Plast Reconstr Surg* 1992;89:846-52.
14. Naesh O, Haljamae H, Skielboe M, et al. Purine metabolite washout and platelet aggregation at reflow after tourniquet ischaemia: effect of intravenous regional lidocaine. *Acta Anaesthesiol Scand* 1995;39:1053-8.
15. Klenerman L, Crawley J. Limb blood flow in the presence of a tourniquet. *Acta Orthop Scand* 1977;48:291-5.
16. Pauers RS, Carocci MA. Low pressure pneumatic tourniquets: effectiveness at minimum recommended inflation pressures. *J Foot Ankle Surg* 1994;33:605-9.
17. Estersohn HS, Sourifman HA. The minimum effective mid thigh tourniquet pressure. *J Foot Surg* 1982;21:281-4.
18. Hargens AR, McClure AG, Skyhar MJ, et al. Local compression patterns beneath pneumatic tourniquets applied to arms and thighs of human cadavera. *J Orthop Res* 1987;5:247-52.
19. Shaw JA, Murray DG. The relationship between tourniquet pressure and the underlying soft-tissue pressure in the thigh. *J Bone Joint Surg [Am]* 1982;64-A:1148-52.
20. Mohler LR, Pedowitz RA, Lopez MA, Gershuni DH. Effects of tourniquet compression on neuromuscular function. *Clin Orthop* 1999;359:213-20.
21. Graham B, Breault MJ, McEwan JA, McGraw RW. Occlusion of arterial flow in the extremities at subsystolic pressures through the use of wide tourniquet cuffs. *Clin Orthop* 1993;286:257-61.
22. Gothgen J, Jacobsen E. Transcutaneous oxygen tension measurement I: age variation and reproducibility. *Acta Anaesthesiol Scand Suppl* 1978;67:66-70.
23. Versmold HT, Linderkamp O, Stuffer KH, Holzmann M, Riegel KP. In vivo versus in vitro response time of transcutaneous PO₂ electrodes: a comparison of four devices in new-born infants. *Acta Anaesthesiol Scand Suppl* 1978;68:40-8.
24. Niinikoski J. Oxygen and wound healing. *Clin Plast Surg* 1977;4:361-74.
25. Hunt TK. Current challenges for wound healing research. *J Trauma* 1970;10:1001-9.
26. Hunt TK, Zederfeldt BH, Goldstick TK, Conolly WB. Tissue oxygen tensions during controlled hemorrhage. *Surg Forum* 1967;18:3-4.