Long Term Bone Changes in Vascularized Fibula Grafts

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INTRODUCTION

Bone grafts have been used since early twentieth century to repair bony defects and fuse joints. Early investigators recognized the benefits of a living bone graft, but only after the 1960s when Jacobson and Suarez pioneered the techniques and instruments for microvascular anastomosis did the use of free living bone transfer become prevalent[1]. In the following decades, the use of free vascularized fibula grafts (VFGs) has evolved into a successful solution in the treatment of large skeletal defects[2-6]. Numerous indications for free VFGs include: large segmental bone loss (due to trauma, tumor, and osteomyelitis), complex tissue loss (requiring composite flaps), and congenital bony defects.

Jupiter, in 1987, originally reported on the repair of large femoral defects with free VFG with his series of 7 patients[7]; he went on to publish successful results repairing defects of the humerus[8] and radius[9] with vascularized grafts. Most recently, a long term retrospective study was carried out reviewing 29 consecutive patients who underwent either femur or humerus repair with free VFGs[10]. Fourteen of 15 patients with femur defects went onto union at an average time of 19 weeks, and 13 of 14 patients with humerus defects went on to union at 12.4 weeks. Majority of grafts showed hypertrophy, and very few patients experienced complications of stiffness and infection. One illustrative case is shown in the Figure.

These studies span several decades. Even with improvement in surgical techniques and rehabilitation methods, there are numerous common themes. Here we review the long term bone changes in vascularized fibula grafts.

BIOLOGY OF BONE GRAFTS

For most bone defects less than 6cm, a conventional cancellous or corticocanellous bone graft is appropriate[11]. This is provided that the defect has a well-vascularized bed, adequate soft tissue coverage, and no infection. The process of bone graft incorporation is by "creeping substitution," which was first described by Barth in 1895[12]. It involves gradual vascular ingrowth, resorption, and replacement of necrotic bone. Creeping substitution results in rapid revascularization in small cancellous grafts, which has greater inductive capacity than cortical bone. However, as much as 40% to 50% of lamellar bone remains necrotic, and as a result, structural nonvascularized grafts have substantial problem with fatigue fracture, even years after the surgical procedure[11,13,14].

A vascularized bone graft, on the other hand, avoids the process of creeping substitution because of its preserved circulation and improved cell survival[11-13,15,16]. In a canine model, osteocyte viability in vascularized grafts was shown to be 78% of normal controls, compared to 46% of normal controls in nonvascularized grafts[15]. This increased osteocyte survival results in less remodeling, improved strength, better healing, and decreased incidence of stress fracture, when compared to nonvascularized autografts or allografts[15,17-21].

BONY UNION

Primary bone union in patients treated with VFGs to long bones occurs in upwards of 70% of cases in review of series[18,20]. Secondary bone grafting can be carried out in most nonunion cases. The mean time to vascularized fibular graft healing is 6 months, and the secondary graft can be attempted at around the 12 months mark. A largely cancellous autograft at the ununited junction is usually sufficient[18,20,22,23].

In a large Mayo cohort, in vascularized fibular grafts performed for non-osteomyelitis indications, the primary and secondary union rates were 69% and 84% respectively, but in series of defects with infections, the union rates fall to 49% and 77%[20]. In cases involving tumor, the highest union rates were reported: primary union 76%, overall 92%[20].

STRESS FRACTURE

As mentioned earlier, vascularized bone grafts have decreased incidence of stress fractures, but they do occur. A Mayo clinic series reports the rate to be 9%, with higher incidence in the lower extremity and in tumor patients who receive postoperative chemotherapy and radiotherapy[20,24]. Most fracture occurs within the first year of surgery, when the bone has insufficient time to hypertrophy[20,25]. Therefore, de Boer *et al* recommend that a vascularized graft should be protected against stress fracture during the first year, allowing a gradual increase in mechanical loading that enhances remodeling and hypertrophy[26]. If the graft has adequate vascularity, nondisplaced upper extremity fractures will heal in a cast alone, and displaced and lower extremity fractures require fixation with or without bone grafting[20,27].

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HYPERTROPHY

One of the benefits of VFGs as a reconstructive option is its ability to hypertrophy. In an earlier animal study, Fujimaki and Suda used a canine model to show that VFGs hypertrophy in absence of weight bearing and never exceed the width of the recipient bone[28]. These results were not observed in subsequent retrospective studies of human patients. Falder *et al* have shown that grafts hypertrophy in all cases where there was mechanical stress through the recipient bone and graft, but not in cases with stress shielding of the graft; furthermore, the grafts do hypertrophy beyond the width of the recipient bone[29].

Degree of hypertrophy depends on a number of variables, including patient age, weight-bearing location, type of graft used, method of fixation, as well as the vascular status of the graft. It occurs primarily in young individuals (age<20) and is more extensive in the lower extremity. Hypertrophy is less observed when the graft is extensively shielded by rigid fixation[20,22,26]. Hypertrophy, when it occurs, is seen by an average 13.5 months postoperatively[22].

The mechanism of hypertrophy is a different process from the reactive callus formation of fracture healing[22,28]. De Boer delineates three types of hypertrophy: 1) periosteal: increase in diameter due to irregular bone formation around the graft; 2) endosteal: cortex and medullary canal both increased in diameter; 3) combination of periosteal and endosteal hypertrophy[26]. His study, however, did not go on to examine these types of hypertrophy in his series of patients; in fact, de Boer measured graft hypertrophy based on the change of width of fibula over time, defined by outer cortices on radiographs. Indeed it is very difficult to measure the endosteal diameter of fibula grafts consistently through plain radiographs due to dense appearance of hypertrophied bone. In one long term follow up study of 10 patients, vascularized fibula grafts hypertrophied, while computed tomography (CT) scans showed that medullary canal of grafts stayed the same size, this being consistent with a periosteal mechanism[22].

Most studies, including de Boer's, have measured hypertrophy in one-dimension. A more appropriate examination of fibula graft hypertrophy would be studying the change in cross section area of cortical bone.

Fibula is triangular in cross section at time of harvest, but after hypertrophy, it becomes a square[22]. One can measure the width of the outer cortex of fibula on radiographs at surgery (or contralateral fibula) and at follow up.

One can introduce the size of adjacent recipient bone into the equations for control. The size of medullary canal, if it can be adequately measured on radiographs or if CTs were available, would provide a more accurate measurement of graft hypertrophy.

SUMMARY

The free vascularized fibula graft is a useful technique for complex reconstruction to salvage limbs with large bony defects. Because of its biology, vascularized grafts obviate the process of creeping substation and therefore the osteopenia that may be associated with conventional bone grafts. It produces rapid bone union in majority of cases with low rates of stress fractures if adequately protected initially after surgery. Graft hypertrophy is well recognized, but there remain numerous questions relating its extent and mechanism.

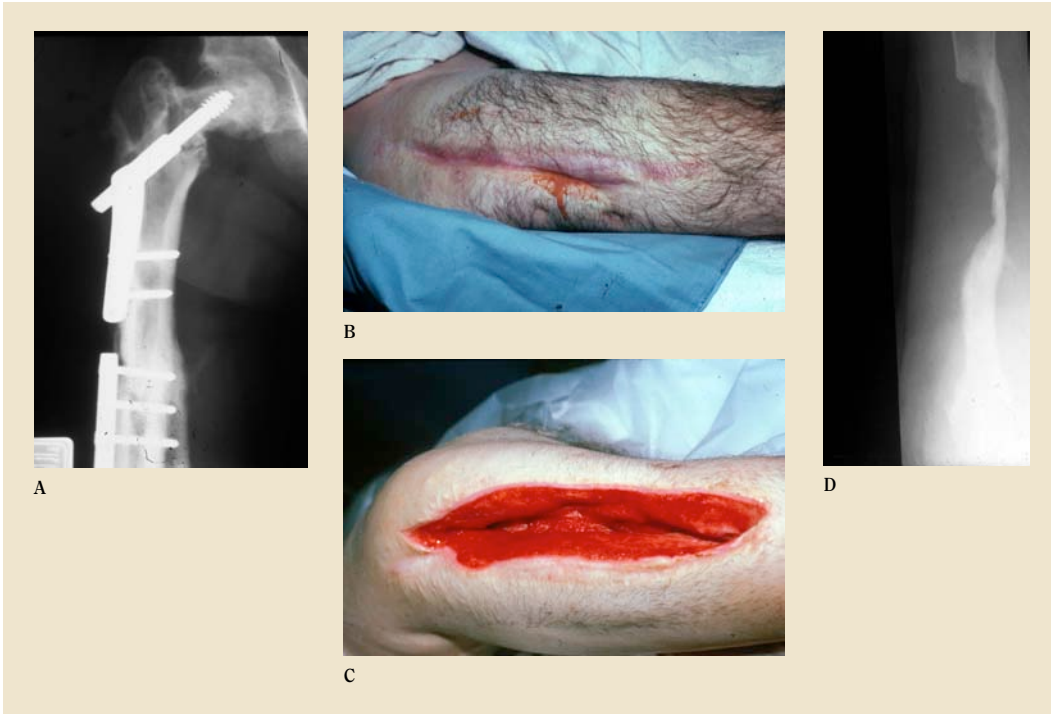
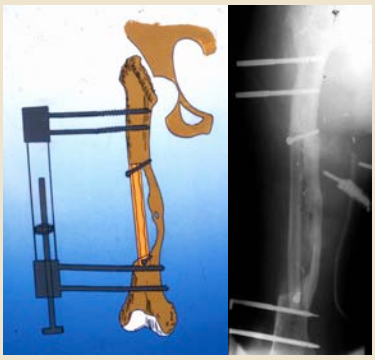
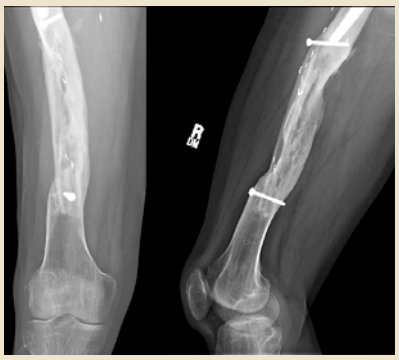


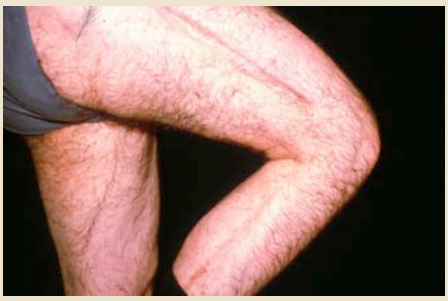
Figure: A case example of free vascularized fibula graft.
A. and B. Preoperative radiograph and picture of a patient who had traumatic femoral shaft and femoral head fractures, underwent initial fixation, and then experienced osteomyelitis with a draining sinus tract. **C. and D.** Clinical picture and radiograph of thigh and femur after serial debridement and Girdlestone resection of femoral head. There is a segmental defect.



E. Free vascularized fibula graft transfer to segmental defect of femur. Fixation with lag screws and an external fixator.



F. Graft hypertrophy of the femur 25 years after the free vascularized fibula graft transfer, and 20 years after ipsilateral total hip arthroplasty.



G. Clinical picture of thigh after free graft.

References

1. **Jacobson JH, Suarez EL.** Microvascular Surgery *Chest.* 1962;41:220-224
2. **Malizos KN, Zalavras CG, Soucacos PN, Beris AE, Urbaniak JR.** Free vascularized fibular grafts for reconstruction of skeletal defects. *J Am Acad Orthop Surg* 2004 Sep-Oct;12(5):360-9.
3. **Arai K, Toh S, Tsubo K, Nishikawa S, Narita S, Miura H.** Complications of vascularized fibula graft for reconstruction of long bones. *Plast Reconstr Surg* 2002 Jun;109(7):2301-6.
4. **Minami A, Kasashima T, Iwasaki N, Kato H, Kaneda K.** Vascularised fibular grafts. An experience of 102 patients. *J Bone Joint Surg Br* 2000 Sep;82(7):1022-5.
5. **Aronson J, Jownson E, Harp JH.** Local bone transportation for treatment of intercalary devects by ilizarov tecniqueL Biomechanical and clinical considerations. *Clin Orthop* 1989 Jun: 243:71-79.
6. **Lin C, Wei F, Chen H, Chuang DC.** Outcome Comparison in Traumatic Lower Extemity Reconstruction by Using Various Composite Vascularized Bone Transplantation. *Plast Reconstr Surg* 1999 Sep:104(4):984-992.
7. **Jupiter JB, Gerhard HJ, Guerrero J, Nunley JA, Levin LS.** Treatment of segmental defects of the radius with use of the vascularized osteoseptocutaneous fibular autogenous graft. *J Bone Joint Surg Am.* 1997 Apr;79(4):542-50.
8. **Jupiter JB.** Complex non-union of the humeral diaphysis. Treatment with a medial approach, an anterior plate, and a vascularized fibular graft. *J Bone Joint Surg Am.* 1990 Jun;72(5):701-7. Erratum in: J Bone Joint Surg Am 1990 Sep;72(8):1270.
9. **Jupiter JB, Gerhard HJ, Guerrero J, Nunley JA, Levin LS.** Treatment of segmental defects of the radius with use of the vascularized osteoseptocutaneous fibular autogenous graft. *J Bone Joint Surg Am.* 1997 Apr;79(4):542-50.
10. **Jawa A, Concannon MJ, Duffy FJ, Yaremchuk M, Jupiter JB.** Free vascularized fibular grafts for segmental defects of the femur and humerus. In Press.
11. **Bieber EJ, Wood MB.** Bone reconstruction. *Clin Plast Surg* 13:645-655, 1986.
12. **Barth H.** Histologishe Untersuchungen uber Knochen Transplantation. *Beitr Pathol Anat Allg Pathol* 17:65-142, 1895
13. **Burchardt H.** The biology of bone graft repair. *Clin Orthop* (174):28-42, 1983.
14. **Springfield DS.** Autogenous bone grafts: Nonvascular and vascular. *Orthopedics* 15(10):1237-1241, 1992.
15. **Arata MA, Wood MB, Cooney WP.** Revascularized segmental diaphyseal bone transfers in the canine: An analysis of viability. *J Reconstr Microsurg* 1:11-19, 1984.
16. **Berggren A, Weiland AJ, Dorfman H.** Free vascularized bone grafts: Factors affecting their survival and ability to heal to recipient bone defects. *Plast Reconstr Surg* 69:19-29, 1982.
17. **Doi K, Tominaga S, Shibuta T.** Bone grafts with microvascular anastomoses of vascular pedicles: An experimental study in dogs. *J Bone Joint Surg Am* 59:809-815, 1977.
18. **Weiland AJ, Moore JR, Daniel RK.** Vascularized bone autografts: Experience with 41 cases. *Clin Orthop Rel Res* (174):87-95, 1983
19. Pho RW, Levack B, Satku K, *et al.* Free vascularized fibula graft in the treatment of congenital pseudarthrosis of the tibia. *J Bone Joint Surg Br* 67:64-70, 1985.
20. **Han CS, Wood MB, Bishop AT, Cooney WP.** Vascularized bone transfer. *J Bone Joint Surg Am* 74:1441-1449, 1992
21. **Enneking WF, Gady JL, Burchardt H.** Autogenous cortical bone grafts in the reconstruction of segmental skeletal defects. *J Bone Joint Surg Am* 62:1039-1058, 1980.
22. **Ikeda K, Tomita K, Hashimoto F, Morikawa S.** Long-term follow-up of vascularized bone grafts for the reconstruction of tibial nonunion: Evaluation with computed tomographic scanning. *J Trauma* 32:693-697, 1992.
23. **Bishop AT, Wood MB, Sheetz KK.** Arthrodesis of the ankle with a free vascularized autogenous bone graft: Reconstruction of segmental loss of bone secondary to osteomyelitis, tumor, or trauma. *J Bone Joint Surg Am* 77:1867-1875, 1995.
24. **Aberg M, Rydholm A, Holmberg J, Wieslander JB.** Reconstruction with a free vascularized fibular graft for malignant bone tumor. *Acta Orthop Scand* 59:430-437, 1988.
25. **Minami A, Kaneda K, Itoga H, Usui M.** Free vascularized fibular grafts. *J Reconstr Microsurg* 5:37-43, 1989.
26. **de Boer HH, Wood MB.** Bone changes in the vascularized fibular graft. *J Bone Joint Surg Br* 71(3):374-378, 1989.
27. **Minami A, Kimura T, Matsumoto O, Kutsumi K.** Fracture through united vascularized bone grafts. *J Reconstr Microsurg* 9:227-232, 1993.
28. **Fujimaki A, Suda H.** Experimental study and clinical observations on hypertrophy of vascularized bone grafts. *Microsurgery* 15:726-732, 1994.
29. **Falder S, Sinclair JS Rogers CA, Townsend PLG.** Long-term behaviour of the free fascularised fibula following reconstruction of large bony defects. Br J Plast Surg. 2003 Sep;56(6):571-84