U·P·O·J

Open Fractures and Timing to Closure: A Review

T. Ty Fowler, MD[1]
Benjamin C. Taylor, MD[1]
Brian R. Williams[2]

[1] Department of Orthopaedic Surgery, Mount Carmel Medical Center, Columbus, OH

[2] University of Toledo, College of Medicine, Toledo, OH

Controversy exists between primary and delayed closure of open fracture wounds and complication rates. Traditional teaching has been to close open fractures in a delayed fashion. However, many investigators have shown low infection rates with primary closure of open fractures. Much of the literature available indicates primary closure can be performed on Gustilo and Anderson type I and II open fractures and even type IIIA with specific inclusion and exclusion criteria. Primary closure is contraindicated when wound debridement is inadequate, wound edges cannot be approximated without tension, or the wound remains contaminated. Timing of the closure of an open fracture wound can have implications on patient care, ranging from morbidity and mortality to economic considerations. We present a review of the published literature concerning the timing of closure of open fracture wounds.

Infection is a common sequela of open fracture injuries. In 1976, Gustilo and Anderson[1] (GA) classified open fracture injuries into three categories: type I, II, or III. A type I fracture is categorized as a wound that is less than one cm long and clean. Type II injuries are classified as a fracture with a laceration more than one cm, without extensive soft-tissue damage, flaps, or avulsions. Type III injuries were originally categorized as either an open segmental fracture, an open fracture with extensive soft-tissue damage, or a traumatic amputation. Special categories in type III were gunshot injuries, any open fracture caused by a farm injury, and any open fracture with accompanying vascular injury requiring repair. Type III injuries were further modified in 1984 by Gustilo et al[2] to include three subtypes. Type IIIA is described as adequate soft-tissue coverage of a fractured bone despite extensive soft-tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound. Type IIIB refers to inadequate soft-tissue coverage with periosteal stripping requiring flap reconstruction, often associated with massive contamination. Type IIIC describes any open fracture associated with arterial injury that requires vascular repair for limb salvage.

Delayed closure of open fractures, defined as wound closure occurring at any time after the initial debridement, was established as standard of care prior to the advent of current antibiotics, modern debridement methods, and improved fracture stabilization procedures[3]. In 1939, Trueta discussed the advantages and necessity of thorough debridement of war injuries to reduce infection[3]. However, in 1943, the use of penicillin resulted in further reduction of infection; therefore, the importance of performing a thorough debridement was no longer emphasized. The resulting recommendations were delayed closure, noting a lower infection rate when compared with primary closure complicated by inadequate debridements[3]. As stated by Hohmann et al[4], the standard of treating open wound fractures has become a combination of urgent aggressive debridement with excision of all dead and devitalized tissue, early stabilization of the fracture, and initiation of broad-spectrum antibiotics. Subsequently, the wound is debrided every 48 to 72 hours, with closure of the wound after final adequate debridement[3-5]. It has been shown that the optimal timing of wound closure or coverage is within seven days of injury, as delays greater than seven days are associated with higher infection rates[3-5].

According to the literature, the most important aspect of surgical open fracture treatment is thorough debridement and irrigation[1-20]. Multiple reports have shown that infections of open wounds likely are not caused by the initial contamination, but instead by organisms acquired secondarily through nosocomial routes[1-4]. In accordance, Patzakis et al[17] reported only 18% of infections related to open fractures are due to the initial organism. This has led to many authors suggesting that primary wound closure is safe and may actually reduce the rate of infection[3,4,7,9,17,19]. The present article reviews the controversy concerning the standard of care for open fracture closure and addresses the following two questions: Is there evidence in the literature to support primary closure at the time of initial debridement of open fractures? What are the contraindications to performing primary closure of open fractures?

Review and Discussion of the Pertinent Literature

Many authors consider a tension free closure in an open fracture wound a pre-requisite for primary closure[4,7,8,18-20]. As a result of significant edema and damage to the soft-tissue envelope after high-energy trauma, increased tension in the skin flaps is a concern when attempting primary closure. This increased tension theoretically has a negative effect on the cutaneous blood flow leading to necrosis of the skin edges and

Corresponding Author:
T. Ty Fowler, MD
793 West State Street
Columbus, Ohio 43222
ty_fowler@hotmail.com

eventual wound breakdown. In a pig model, Sagi et al[21] found that the Allgower-Donati suture-pattern had a significantly smaller effect on cutaneous blood flow with increasing tension when compared to a simple, vertical mattress, and horizontal mattress suture pattern.

In 1976, Gustilo and Anderson[1] completed a retrospective and prospective study of 1025 open long bone fractures before the current sub-classification of type III injuries. Their study demonstrated a decrease from a 20% infection rate to 6% in type I and II injuries with primary closure at initial debridement. In a subanalysis of type III injuries, sixteen of the injuries were closed primarily leading to a 44% infection rate, whereas five underwent delayed closure with a resulting infection rate of 20%. The authors concluded that type I and II injuries should be closed primarily, whereas type III fracture closure should be delayed[1]. It is necessary to reiterate the importance of debridement when treating open fractures, as quoted in their landmark article: "If there is the slightest doubt in the surgeon's mind as to whether there has been adequate debridement of the wound after open fracture…the safe rule is not to close the wound"[1].

A comprehensive literature review article prepared by Crowley et al[19] reviewed studies addressing primary and delayed closures of open fractures. The investigators recommended primary closure of open GA type I, II, and IIIA fractures, with the exception of wounds that are grossly contaminated with feces or stagnant water, or fractures resulting from farmyard-related injuries. However, type IIIB and IIIC injuries were recommended to be managed by specialized teams and the wounds should be closed at the earliest possible time[19].

A randomized, prospective study of eighty-two open fractures of the lower and upper extremities was completed by Benson and colleagues[18] that yielded no significant differences in infection rates between primary and delayed closure. The groups were comparable statistically with respect to time from injury to debridement, immediate internal fixation, subjective classification of gross contamination, and associated injuries. Forty-four injuries were closed primarily, three (6.8%) of which resulted in superficial infection. The other thirty-eight cases were left open for an average of four to six days before closure of the wound, resulting in two (5.2%) deep infections. Hohmann et al[4] had similar results in their retrospective study of the timing of open tibia fracture closure with deep infection being the end point. They included GA type I, II, and IIIA open fractures, and therefore excluded type IIIB and IIIC injuries, polytraumas, associated injuries, unrelated significant comorbidities, presentation greater than twenty-four hours post-injury, or surgeries within the previous six months. The authors reported no statistically significant difference in infection rates between forty-nine patients who were treated with primary closure at initial debridement with a 2 % infection rate, and forty-six patients treated with delayed closure who had an infection rate of 4.3%. However, the authors found almost twice the length of stay for patients who underwent delayed closure, and they concluded that primary closure is potentially more cost-effective in appropriately selected patients[4].

Furthermore, DeLong and colleagues[7] retrospectively studied 119 open fracture injuries and closures. These consisted of fractures involving the tibia (52), femur (27), radius and ulna (12), ankle (11), patella (9), and humerus (4). Seventy-six of the injuries were closed primarily and seventeen of the closures were delayed. Three of the primary closure cases resulted in infection and two of the delayed closures resulted in infection. The investigators concluded that there were no statistically significant differences in infection, delayed union, or nonunion between procedures. Inherent in retrospective studies, this study was not without limitations. These authors noted that the wounds in the delayed closure group were unable to be closed without tension on the skin edges, which is an accepted indication for delayed closure. Arguably these two groups are not equivalent and, therefore, should not be compared with respect to infection rate. To decrease variation between groups, the authors performed a subanalysis within the three types of open fracture grades and found no significant differences between the two closure methods. Similar to Hohmann et al's conclusion[4], DeLong et al[7] stated that primary closures provide a lower number of operations, and decreased health care costs and hospital stays with no increase in infection.

Gleuck et al[20] retrospectively examined forty-two open fractures of the distal radius and closure methods. Three of the twenty-seven cases that underwent primary closure resulted in infections. Investigators analyzed the correlations between infection and the following: Gustilo and Anderson type I-III open fracture, time from injury to irrigation and debridement, primary versus delayed closures, method of stabilization, and degrees of contamination. The degree of contamination was the only variable found to be statistically significant in its correlation to infection. The investigators suggested that primary closure is safe in relatively clean wounds. However, there is no evidence to support primary closure of contaminated wounds regardless of classification type[20].

Rajasekaran and colleagues[9] prospectively studied upper and lower extremity open fractures to determine safe criteria for primary closure of GA type III injuries. They excluded type I, II, and IIIC fractures immediately, as well as patients suffering from shock, peripheral vascular disease, drug-dependent diabetes mellitus, peripheral vasculitis, connective-tissue disorders, or wound contamination by sewage or farm substances. Specific inclusion criteria were reported as wound debridement within twelve hours of injury, no skin loss, stable skeletal fixation, and bleeding skin margins closed by direct apposition under no tension. The authors' findings showed that only five of the 173 (2.9%) wounds had deep infections. Therefore, they concluded that primary closure in open fractures is appropriate in properly selected patients[9]. It is worthy to note that strict criteria for primary closure, as stated above, led to only 185 fractures of 557 (33%) type III open fractures that were able to be primarily closed at the initial debridement.

A prospective study was completed by Shtarker et al[6] of thirty-two open tibia fractures, including eleven type I, ten type II and eleven type IIIA open fractures, with primary closure. Two of the closures resulted in skin necrosis leading to skin

grafts; however, there were no infections. Similar to Gustilo and Anderson[1], Shtarker et al[6] recommended primary closure if the surgeon is confident that the wounds are free of contamination.

Weitz-Marshall and Bosse[3] performed a literature review of the timing of wound closures. They concluded that it was the surgeon's judgment gained by experience that is required for primary closure protocol along with an adequate amount of debridement. Furthermore, contraindications for primary closure included grossly contaminated wounds with feces, stagnant water, dirt, or injuries occurring in the farm setting. Moreover, primary closure was not suggested if the antibiotic treatment was delayed for more than twelve hours, if a questionable amount of viable tissue at initial surgery remains, or if there was a concern regarding adequacy of debridement[3].

Reuss and Cole[8] retrospectively reviewed eighty-one open tibial shaft fractures. Thirty-two patients underwent delayed closure and the remaining forty-nine wounds were closed primarily at the time of initial debridement. Delayed closures resulted in 19% infection rate, whereas only 2% of the primary closures became infected. All the infections were GA type III injuries, one type IIIA (primary closure group), and six type IIIB (delayed closure group). However, the groups in their study are not equivalent, as the more severe injuries required more debridements and were closed on a delayed basis. There was a statistically significant association between multiple debridements and infection, and a higher fracture grade. The authors stated that primary closure is acceptable based on the surgeon's discretion regarding the adequacy of debridement and low contamination of the wound.

Lavelle et al[14] performed a web-based survey to determine the methods of treatment for open fractures by ACGME accredited residency programs. They found that the vast majority of programs were primarily closing GA type I (88%) and type II (86%) open fractures. Type IIIA open fractures however, were only primarily closed by 57% of programs.

Other investigators who have studied the current topic disagree with the recommendation of primary closures of appropriately selected wounds. In an instructional course lecture article Zalavras and colleagues[12] reviewed the treatment of open fractures and concluded that the duration of antibiotic therapy, the time between injury and surgery, or the type of wound closure are not significant variables in outcomes. The authors stated that, in their review of the literature, infection rates are equal between primary and delayed closure; however, they warn that there is an increased risk of *Clostridia perfringens* contamination causing gas gangrene with primary closure despite providing no evidence to support this claim[12]. In stark contrast to their own literature review and the previously mentioned studies, they recommend that open wounds should not be closed primarily due to the risk of gas gangrene and that no rationale exists for primary closure, since delayed closure is a time-tested standard of care[12]. However, there may be some bias due to the selective reporting of the sources the investigators reviewed, as well as their personal preference for treating open fractures.

Russell et al[5] retrospectively reviewed ninety cases of extra-articular open tibia fractures from 1981 through 1986. Despite the difference in groups (primary closure group had twice as many GA type III open fractures), results showed that primary closures resulted in a 50% infection rate versus a 33% infection rate for delayed closure. If type III fractures were removed and only type I and II fractures were analyzed, there was no statistically significant difference in the infection rate between primary and delayed procedures. Despite their results with respect to type I and II fractures, they concluded that there is no place for primary closure of any open tibia fracture[5].

Conclusion

This article reviews the controversy concerning the standard of care for timing of closure of open fractures and addresses the following two questions: Is there evidence in the literature to support primary closure at the time of initial debridement of open fractures? What are the contraindications to performing primary closure of open fractures?

It is the authors' opinion and that of our institution that primary closure is an acceptable procedure for treatment of appropriately selected open fractures. It is strongly agreed in the literature that adequate debridement is the most important factor in minimizing infection while treating open fractures[1-20]. Given the available evidence in the literature, which lacks an abundance of Level I studies, very low infection rates with primary closure of type I and II open fractures can be obtained when ensuring adequate debridement[1-4,6-9, 13, 16,18-20]. Moreover, Rajasekaran and colleagues[9], as well as Shtarker et al[6] have shown acceptable low infection rates of type IIIA fractures treated with primary closure. Crowley et al's systemic review[19] also advocated primary closure in type IIIA open fractures when gross contamination is absent. Rajasekaran et al[9] controlled for very specific parameters whereby primary closure of open fracture wounds was acceptable: debridement within twelve hours of injury, appropriate stabilization of the fracture, and closure of skin margins by direct apposition with no tension on wound edges. The authors advocate the use of Rajasekaran's criteria and further emphasize thorough wound debridement and the use of the Allgower-Donati suture for closure.

Based on the literature reviewed and the authors' opinion, contraindications for primary closure include inadequate debridement. If a surgeon is unable to approximate wound edges without tension to the wound, then closure should be delayed. Furthermore, if the wound is heavily contaminated with drug-resistant anaerobic or gram negative bacteria including those found in feces, stagnant water, or farm setting, primary closure should be avoided.

It should be emphasized that the studies reviewed, many of which were retrospective, also are plagued by low numbers and inadequate power. There is a strong need for well-designed prospective Level I studies to further support, potentially modify, or refute the above recommendations.

Acknowledgement

The authors acknowledge the editorial assistance of Janet L. Tremaine, ELS, Tremaine Medical Communications, Dublin, Ohio.

References

1. Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. *J Bone Joint Surg Am* 1976;58-A:453-458.

2. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of Type III (severe) open fractures. *J Trauma* 1984;24:742-746.

3. Weitz-Marshall AD, Bosse MJ. Timing of closure of open fractures**.** *J Am Assoc Orthop Surg* 2002;10:379-384.

4. Hohmann E, Tetsworth K, Radziejowski MJ, Wiesniewski TF. Comparison of delayed and primary wound closure in the treatment of open tibial fractures**.** *Arch Orthop Trauma Surg* 2007; 127:131-136.

5. Russell GG, Henderson R, Arnett G. Primary or delayed closure for open tibial fractures. *J Bone Joint Surg Br* 1990;72-B:125-128.

6. Shtarker H, David R, Stolero J, Grimberg B, Soudry M. Treatment of open tibial fractures with primary suture and Ilizarov fixation. *Clin Orthop Relat Res* 1997;335:268-274.

7. DeLong WG Jr, Born CT, Wei SY, Petrik ME, Ponzio R, Schwab CW. Aggressive treatment of 119 open fracture wounds. *J Trauma* 1999;46:1049-1054.

8. Reuss BL, Cole JD. Effect of delayed treatment on open tibial shaft fractures. *Am J Orthop* 2007;36:215-220.

9. Rajasekaran S, Dheenadhayalan J, Babu JN, Sundararajan SR, Venkatramani H, Sabapathy SR. Immediate primary skin closure in type-iii A and B open fractures: results after a minimum of five years. *J Bone Joint Surg Br* 2009;91-B:217-224.

10. Charalambous CP, Siddique I, Zenios M, Roberts S, Samarji R, Paul A, Hirst P. Early versus delayed surgical treatment of open tibial fractures: effect on the rates of infection and need of secondary surgical procedures to promote bone union. *Injury* 2005;36:656-661.

11. Olson SA, Schemitsch EH. Open fractures of the tibial shaft: an update. *Instr Course Lect* 2003;52:623-631.

12. Zalavras CG, Marcus RE, Levin LS, Patzakis MJ. Management of open fractures and subsequent complications. *Instr Course Lect.* 2008;57:51-63.

13. Gardner MJ, Mehta S, Barei DP, Nork SE. Treatment protocol for open AO/OTA type C3 pilon fractures with segmental bone loss. *J Orthop Trauma* 2008;22(7):451-457.

14. Lavelle WF, Uhl R, Krieves M, Drvaric DM. Management of open fractures in adult patients: current teaching in ACGME accredited residency programs. *J Surg Orthop Adv* 2007;16(3):111-117.

15. Al-Arabi YB, Nader M, Hamidian-Jahromi AR, Woods DA. The effect of the timing of antibiotics and surgical treatment on infection rates in open long-bone fractures: a 9-year prospective study from a district general hospital. *Injury* 2007;38(8):900-905

16. Levin LS. Early versus delayed closure of open fractures. *Injury* 2007;38:896-899.

17. Patzakis MJ, Bains Rs, Lee J, Shepherd L, Singer G, Ressler R, Harvey F, Holtom P. Prospective, randomized, double-blind study comparing single-agent antibiotic therapy, ciprofloxacin, to combination antibiotic therapy in open fracture wounds. *J Orthop Trauma* 2000; 14:529-533.

18. Benson DR, Riggins RS, Lawrence RM, Hoeprich PD, Huston AC, Harrison JA. Treatment of open fractures: a prospective study. *J Trauma* 1983;23:25-30.

19. Crowley DJ, Kanakaris NK, Giannoudis PV. Debridement and wound closure of open fractures: the impact of the time factor on infection rates. *Injury* 2007;38:879-889.

20. Glueck DA, Charoglu CP, Lawton JN. Factors associated with infection following open distal radius fractures. *Hand* 2009;4:330-334.

21. Sagi HC, Papp S, Dipasquale T. The effect of suture pattern and tension on cutaneous blood flow as assessed by laser Doppler flowmetry in a pig model. *J Orthop Trauma* 2008 Mar;22(3):171-175.