

DISCLAIMER: These guidelines were prepared by the Department of Surgical Education, Orlando Regional Medical Center. They are intended to serve as a general statement regarding appropriate patient care practices based upon the available medical literature and clinical expertise at the time of development. They should not be considered to be accepted protocol or policy, nor are intended to replace clinical judgment or dictate care of individual patients.

# ANTICOAGULATION FOR BLUNT CAROTID ARTERY INJURY

## SUMMARY

The optimal management of blunt carotid artery injury (BCI) is not well established. Surgical intervention, anticoagulation and antiplatelet therapy have been proposed. The goal of anticoagulation therapy is to prevent cerebral embolization and avoid permanent occlusion. Anticoagulation therapy has been associated with improvement in neurological outcomes when compared to no anticoagulation therapy. In the absence of any contraindications, the use of systemic anticoagulation with heparin is safe and should be considered.

## RECOMMENDATIONS

- **Level 1**
  - **None**
- **Level 2**
  - **None**
- **Level 3**
  - **Heparin therapy is safe and should be considered if:**
    - **No contraindications are present AND the anticipated benefit outweighs the risk of bleeding in patients at high risk.**
    - **Warfarin therapy should be considered following treatment with heparin.**
  - **Antiplatelet therapy may be considered in patients without contraindications.**
  - **Therapy should be continued for 3 to 6 months.**

## INTRODUCTION

Blunt carotid injury (BCI) is a rare but potentially devastating injury with a reported incidence of 0.08 to 0.27% (1). BCI results in severe disability or death if undiagnosed. Rates of mortality and neurologic morbidity ranging from 5-40% and 40-80%, respectively, have been reported among patients presenting with BCI (1). The mechanism of injury is usually due to direct trauma mainly from motor vehicle crashes followed by assaults and falls (2). Various types of vascular injury may occur, including the development of intimal flap/dissection, occlusion/thrombosis, pseudoaneurysm, carotid cavernous fistula, complete transection or combination of these lesions (1). Complications of BCI may result from either damage to the arterial wall or intima. Dissection due to arterial wall damage results in hemodynamic instability and damage to the arterial intima exposes subendothelial collagen which is a thrombogenic surface and potent platelet aggregator (1).

The optimal management of BCI is not well-defined. Surgical intervention, anticoagulation and antiplatelet therapy have been described. The goal of anticoagulation therapy is to prevent cerebral embolization and avoid permanent occlusion (1). Several studies have evaluated the use of systemic heparin as the anticoagulant of choice for BCI.

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## EVIDENCE DEFINITIONS

- **Class I:** Prospective randomized controlled trial.
- **Class II:** Prospective clinical study or retrospective analysis of reliable data. Includes observational, cohort, prevalence, or case control studies.
- **Class III:** Retrospective study. Includes database or registry reviews, large series of case reports, expert opinion.
- **Technology assessment:** A technology study which does not lend itself to classification in the above-mentioned format. Devices are evaluated in terms of their accuracy, reliability, therapeutic potential, or cost effectiveness.

## LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

## LITERATURE REVIEW

Cothorn and colleagues conducted a prospective observational study evaluating the efficacy of continuous infusion heparin in reducing stroke rate in patients with BCI (2). The patient population included those with blunt trauma who were admitted to the Level I trauma center. Of the 13,280 patients admitted to the center, 643 patients underwent diagnostic four-vessel cerebrovascular angiography and 114 patients were identified as having carotid artery injuries. Of those with blunt carotid artery injury, 73 asymptomatic patients received one of four different anticoagulation regimens: 1) continuous infusion heparin, 2) subcutaneous low-molecular weight heparin, 3) anti-platelet agents, or 4) no therapy. Fifty-four patients (74%) received continuous infusion heparin at 15 Units/kg/hour without a loading dose, titrated to achieve aPTT between 40 to 50 seconds. Two patients (3%) received dalteparin sodium 5000 units once daily. Seventeen patients (23%) received antiplatelet agents (aspirin or clopidogrel). Forty-one patients did not receive anticoagulation. Ischemic neurological events did not occur in any patient who received anticoagulation while they developed in 46% (19/42) of those who did not receive any type of anticoagulation therapy. The authors did not comment on the comparative efficacy of the different pharmacological therapies in BCI (Class II).

Fabian and colleagues conducted a retrospective chart review of patients with BCI admitted to a Level I trauma center from December 1984 to September 1995 (3). Sixty-seven patients with 87 BCIs were treated. There were 54 intimal dissections, 11 pseudoaneurysms, 17 thromboses, 4 carotid cavernous fistulas, and 1 transected internal carotid artery. Anticoagulation with heparin was initiated at the time of diagnosis in 76% of patients while others received no treatment. The goal of heparin therapy was a partial thromboplastin time (aPTT) of 40-50 seconds. Heparin therapy was maintained for 1-3 weeks. Following heparin therapy, warfarin was initiated and continued for 3-6 months with a target prothrombin time of 15-18 seconds and an international normalized ratio (INR) of 1.8. Heparin therapy was independently associated with improvement in neurologic outcomes and survival. Heparin therapy resulted in decreased mortality when compared with those not on heparin treatment (20% vs. 100%,  $p < 0.01$ ) (Class III).

Anti-platelet therapy was compared with anticoagulation therapy in a study by Wahl and colleagues (4). A retrospective chart review were performed for all adult patients admitted to a trauma center from January 1992 to December 1999. Twenty-two patients were identified to have BCI injuries. Of the 22 patients, eight were observed, but not treated with heparin or antiplatelet agents. Of these, two patients died of head injuries and the remaining six survived with fair to good neurological exams at discharge. Seven of 22 patients were treated with heparin with a aPTT goal of 40-60 seconds. Heparin therapy was started within the first 24 hours after admission in three of seven patients. There were four major bleeding complications which resulted in discontinuation of heparin. None of these patients were over anticoagulated. All survivors had fair to good neurological exams at discharge and two patients were discharged on warfarin. Of the remaining seven patients who received antiplatelet therapy, all had fair to good neurological exams at discharge. There were no differences in neurologic outcomes between those patients receiving anticoagulation and those receiving antiplatelet therapy. Heparin therapy resulted in a significantly higher rate of bleeding complications ( $p = 0.05$ ) (Class II)

**Table 1 : Contraindications/Precautions to Heparin Therapy**

## REFERENCES

1. Singh RR, Barry MC, Irland A, et al. Current diagnosis and management of blunt carotid artery injury. *Eur Vas Endovasc Surg* 2004; 27:577-84.
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3. Fabien TC, Patton JH, Croce MA, et al. Blunt carotid injury, Importance of early diagnosis and anticoagulant therapy. *Ann Surg* 1996; 223(5):513-22.
4. Wahl WL, Brandt M, Thompson G, et al. Antiplatelet therapy: An alternative to heparin for blunt carotid injury. *J Trauma* 2002;52:896-901.