Background: Blunt carotid artery injury (BCI) remains a rare but potentially lethal condition. Recent studies recommend that aggressive screening based on broad criteria (hyperextension-hyperflexion mechanism of injury, basilar skull fracture, cervical spine injury, midface fracture, mandibular fracture, diffuse axonal brain injury, and neck seat-belt sign) increases the rate of diagnosis of BCI by 9-fold. If this recommendation becomes a standard of care, it will require a major consumption of resources and may give rise to liability claims. The benefits of aggressive screening are unclear because the natural history of asymptomatic BCI is unknown and the existing treatments are controversial.

Hypothesis: The lack of an aggressive angiographic screening protocol does not result in delayed BCI diagnosis or BCI-related neurologic deficits.

Methods: A 10-year medical record review of patients with BCI was undertaken in 2 level I academic trauma centers. In both centers, urgent screening for BCI was performed in patients with focal neurologic signs or neurologic symptoms unexplainable by results of computed tomography of the brain as well as in selected patients undergoing angiography for another reason.

Results: Of 35212 blunt trauma admissions, 17 patients (0.05%) were diagnosed as having BCI. Six showed no evidence of BCI-related neurologic symptoms during hospitalization or prior to death as a result of associated injuries. Eleven sustained a BCI-related stroke, 9 of whom had it within 2 hours of injury. The remaining 2 had a delayed diagnosis (9 and 12 hours after injury) and received only anticoagulation because the lesions were surgically inaccessible. Just 1 of these 2 patients met the criteria for BCI screening and could have been offered earlier treatment, of uncertain benefit, if we had adopted an aggressive screening policy.

Conclusions: Of the few patients with BCI, most remain asymptomatic or develop neurologic deficits shortly after injury. Although a widely applied, resource-consuming screening program may increase the rate of early diagnosis of BCI, an improvement in outcome is uncertain. A cost-effectiveness analysis should be done before trauma surgeons accept an aggressive screening protocol as the standard of care.

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by because they developed early neurologic symptoms. In addition, the preferred option among the existing treatment strategies for BCI (anticoagulation, operation, stenting, or observation) is unclear. In the absence of level I evidence and because angiographic screening has risks and also consumes substantial human and financial resources, we have not instituted an aggressive angiographic screening protocol to detect BCI at either of our centers. This article reports our experience with BCI and stroke due to BCI. We specifically sought to examine the utility of selected risk factors that have been recommended as triggers for screening. The primary objective of this study was to identify whether the lack of an aggressive screening protocol has resulted in delayed diagnosis of BCI and its associated adverse outcomes.

**METHODS**

This study combined data from the trauma registries of 2 level I trauma centers: Oregon Health & Science University (OHSU), Portland, and Los Angeles County/University of Southern California (LAC/USC), Los Angeles. Each center performed a retrospective review of its trauma registry of all patients with *International Classification of Diseases, Ninth Revision* (ICD-9) codes at discharge beginning with 900 (injury to blood vessels of the head and neck) during a 10-year period (January 1, 1990, to January 31, 2000, at OHSU and January 1, 1992, to January 31, 2002, at LAC/USC). The medical records of these patients were reviewed for evidence of BCI and stroke secondary to BCI. We recorded patient age, sex, Glasgow Coma Scale (GCS) score at admission, Injury Severity Score, indication for cerebrovascular angiography, risk factors for BCI, interval between time of injury and time of stroke (in hours), and treatment of BCI.

The published risk factors for BCI that are recommended as indications for aggressive angiographic screening are cervical hyperextension or hyperflexion, a direct blow to the head and neck, cervical seat-belt sign, a GCS score of 6 or lower, diffuse axonal brain injury, cervical spine fracture, skull fracture, basilar skull fracture, midface fracture, and mandibular fracture. However, during the periods of this study, neither trauma center routinely screened asymptomatic patients for BCI based on these criteria. Urgent angiographic screening for BCI was performed in patients with focal neurologic signs or neurologic symptoms unexplainable by results of computed tomography of the brain. Selected patients with cervical seat-belt sign or major head and neck injuries had cerebrovascular angiography in addition to the angiography required for injuries in other areas (such as those of the aorta, liver, or pelvis). In the absence of a structured protocol for asymptomatic patients, occasional screenings with duplex ultrasonography (LAC/USC) or angiography (OHSU) were performed (based on the attending surgeon’s discretion) in patients with major neck seat-belt marks, following hanging, in patients with displaced mandibular fractures, or in those with major hyperextension-hyperflexion cervical spine injuries.

<table>
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<tr>
<th>Patient</th>
<th>Timing, h</th>
<th>Risk Factors</th>
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<tr>
<td>1</td>
<td>0</td>
<td>None</td>
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<tr>
<td>2</td>
<td>0</td>
<td>Basilar skull and midface fractures</td>
</tr>
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<td>3</td>
<td>0</td>
<td>Severe blow to head and neck; basilar skull fracture</td>
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<tr>
<td>4</td>
<td>0</td>
<td>Broken helmet</td>
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<tr>
<td>5</td>
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<td>Baseball to head</td>
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<td>6</td>
<td>0</td>
<td>Hanging</td>
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<tr>
<td>7</td>
<td>0</td>
<td>Severe blow to head and neck; Glasgow Coma Scale score ≤7</td>
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<tr>
<td>8</td>
<td>2</td>
<td>Neck seat-belt contusion</td>
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<tr>
<td>9</td>
<td>2</td>
<td>Cervical spine fractures</td>
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<tr>
<td>10</td>
<td>9</td>
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<td>11</td>
<td>12</td>
<td>Cervical spine and midface fractures</td>
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**RESULTS**

Seventeen (0.03%) of 35212 patients who had experienced blunt trauma, with a mean±SD age of 28±12 years (range, 13-51 years) and a mean±SD Injury Severity Score of 34.2±13 (range, 9-99), had a diagnosis of BCI. The annual incidence was 1 BCI per 2083 patients with blunt trauma. Injury mechanisms included motor vehicle crash (n=8), motorcycle crash (n=2), industry mishap (n=2), fall from height (n=1), hanging (n=1), assault (n=1), bicyclist struck by automobile (n=1), and baseball to head (n=1). Six patients (35%) were either discharged without having developed BCI-related neurologic symptoms (n=4) or died of associated injuries (n=2) following mean±SD hospitalization periods of 21±20 days and 3.5±3.5 days, respectively. Eleven patients had strokes due to BCI at a mean±SD period of 2.3±4.2 hours (median, 0 hours; range, 0-12 hours) from the time of injury. The Table lists the timing of stroke and risk factors for BCI. Seven patients had strokes at the scene or en route, 2 patients had strokes in the hospital within 2 hours of injury, and 2 had delayed strokes 9 and 12 hours after the injury, respectively. The 2 patients who had strokes in the hospital within 2 hours of injury had risk factors for BCI: one had a neck seat-belt contusion, and the other had 2 cervical spine fractures (stable C2 and C7 vertebral body fractures treated only with a collar). Just 1 of the 2 patients who experienced delayed stroke had risk factors for BCI: this patient had both cervical spine and midface fractures. The other patient had no risk factors for BCI; he had been in a motorcycle crash that resulted in rib fractures and a splenic contusion. Both patients were treated with intravenous heparin sodium anticoagulation. The patient with rib fractures and splenic contusion had a bleeding complication and was ultimately treated only with aspirin.

**COMMENT**

The results of our study lead us to question the benefits and thus the suitability of instituting an aggressive angiographic screening protocol that has been advocated by other investigators to detect asymptomatic BCI. This study is distinct from most studies of BCI because instead of focusing on the detection of BCI in a screened population, we focused on the incidence of delayed stroke from BCI in an unscreened population. Of the few patients with BCI detected during the 10-year periods in which there were no screening protocols at either trauma center, most either remained asymptomatic or developed neurologic symptoms immediately after injury, before screening could be performed. Only 2 of 35000 patients in 10 years developed stroke many hours after admission; of these, only 1 fit into a high-risk category. Therefore, a screening protocol would rarely have led us to offer potentially (but not certainly) effective stroke prevention therapy to an asymptomatic patient with BCI.
This study highlights several clinical pathophysiologic features of BCI that should be considered before a policy of aggressive screening is accepted as a desirable standard of care: (1) most BCI-related strokes occur before or shortly after arrival to the hospital, before any meaningful study or intervention can be instituted; (2) not all patients with BCI will benefit from therapy because they do not universally proceed to stroke; (3) not all patients with BCI can undergo anticoagulant therapy because of the risk of hemorrhage from associated injuries; (4) the institution of liberal screening to identify the rare patient who might benefit would pose a major burden on the human and financial resources of an institution, particularly of a busy trauma center with many patients at risk; and (5) even if such a protocol were instituted, delayed BCI-related stroke would still occur because the published risk factors are not all-inclusive.

Several investigators have proposed that stroke due to BCI is preventable because if the vascular injury can be detected early, intervention with anticoagulation or stenting may be efficacious.5,7,12,13 Miller et al5 showed that when they liberalized their angiographic screening protocol and aggressively treated BCI with anticoagulation, they obtained a decrease in stroke-related mortality from 24% to 13% (P = .03). Despite this impressive decrease in the mortality rate, the frequency does not represent a large number of patients with BCI or BCI-related stroke and mortality. According to the authors, the reduction in stroke-related mortality amounted to 8 patients across 5 years or fewer than 2 patients per year. The total number of angiograms performed to gain this benefit was not reported. It is unclear whether this frequency is related to regional population characteristics and therefore should be validated in many other trauma centers. In support of this argument, Eachempati et al8 questioned the necessity of BCI treatment because in their experience, no patient worsened neurologically even though only 52% of patients with BCI were treated with heparin. Wahl et al14 found no benefit of full anticoagulation compared with antiplatelet therapy for BCI. In addition, most series of BCI have reported significant and even fatal hemorrhagic complications from anticoagulation with unfractionated or low-molecular-weight heparin.6,7,11,13 None of the studies that have recommended treatment are randomized trials; these recommendations are based on level III evidence.3,5,7,11-13

Reports from 2 institutions have characterized the increased incidence of BCI, owing to intensive screening, as an “unrecognized epidemic.”5,12 However, this increased incidence may not necessarily translate into a higher rate of stroke. If the argument of these authors were true, many BCIs occurring throughout the world must have remained untreated. If the natural history of BCI were consistent with frequent strokes, one would wonder why unexpected morbidity from undiagnosed BCI is infrequently observed. One can compare advocacy for aggressive screening for BCI with aggressive screening for spinal injury, a problem that if missed is likely to lead to spinal cord deficit and is frequently linked to litigation. In contrast to an undiagnosed spinal injury, evidence indicates that most BCIs heal and remain asymptomatic without therapy. Biffi et al7 showed that 57% of grade 1 injuries (which represent most BCIs found by aggressive screening) healed within 10 days whether or not the patient underwent anticoagulation. This is not surprising; a significant percentage of asymptomatic arterial injuries throughout the body are known to heal without causing clinical abnormalities. Studies of the natural history of lower extremity arterial injuries indicate that the outcome of clinical occult lesions is overwhelmingly benign.15,16 This principle is also valid for penetrating injuries to the neck that can be managed expectantly based on the absence of clinical symptoms.17,20 The vast majority of occult penetrating cervical vascular injuries remain asymptomatic, and there is little evidence that this clinical tenet should be different for blunt cervical vascular injuries.

Our study is limited by its retrospective design. It is possible that we did not identify every patient with a BCI-related neurologic event during these periods. However, even if we were to double the abnormalities identified, BCI and BCI-related morbidity would still be very rare, and our conclusions would be unchanged. We did not conduct long-term follow-up on the patients who had BCI and were discharged without developing BCI-related symptoms. Nonetheless, these patients spent a mean duration of 3 weeks in the hospital. There is no evidence to suggest that stroke may occur at a later time. We also could not identify the total number of patients who were screened for BCI in our centers during the study period. Therefore, we do not have a precise idea of our cost savings related to the lack of aggressive screening and how these savings would have been offset by the additional medical costs of the 1 patient whose stroke might have been prevented. In addition, we did not include vertebral artery injuries in this study because only 1 injury was identified between the 2 centers.

Our study did not examine if duplex ultrasonography, computed tomographic angiography, or magnetic resonance angiography could replace conventional 4-vessel cerebrovascular angiography. Because cerebrovascular angiography is expensive, requires mobilization of an interventional radiology team including a highly specialized interventional radiologist, and is associated with a risk of stroke (albeit low), each of these less invasive and less expensive modalities has been evaluated as a diagnostic alternative. Duplex ultrasonography has been reported to detect cervical carotid injuries nearly as effectively as angiography but has not been seriously considered as a replacement because it cannot evaluate the intracranial vasculature.21,22 Computed tomography and magnetic resonance imaging are still being investigated, and controversial results have been published.6,23

What are appropriate triggers for cerebrovascular angiography at our trauma centers? Unfortunately, many risk factors and physical findings have been associated with BCI.3,11,24,25 Even with the optimized criteria for angiographic screening to include only patients with a mechanism of injury consistent with a cervical hyperextension or hyperrotation, hyperflexion, or direct blow to the head and neck who manifest a GCS score lower than 6, petrous bone fracture, diffuse axonal brain injury, Le Fort II or III facial fracture, or cervical spine fracture, the average trauma center would be casting a wide net.11 Perhaps the consideration of the most significant or a combination of significant risk factors might in-
increase the specificity of screening asymptomatic patients and therefore make it usable. Even so, the prevention of stroke-related mortality is expected to be infrequent at best according to our experience.

In summary, BCI and stroke due to BCI were rare occurrences at our trauma centers. Although it is likely that some BCIs went undetected during these periods, an aggressive angiographic screening protocol according to a wide range of risk factors would not have prevented a significant proportion of strokes. Most BCI-related strokes occurred too early for intervention or in the absence of identifiable risk factors. With such a low incidence of preventable strokes due to BCI, it would be hard to justify aggressive angiographic screening protocols at our trauma centers. We conclude that aggressive angiographic screening for BCI has a low potential for stroke prevention in our populations. Suggestions of aggressive screening policies are still premature and may carry unnecessary litigious potential for trauma centers. A cost-effectiveness analysis must be performed before the trauma community as a whole embraces one more costly and invasive standard of care.

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Corresponding author: John C. Mayberry, MD, Department of Surgery/L223A, Oregon Health & Science University, 3181 SW Sam Jackson Park Rd, Portland, OR 97201 (e-mail: mayberrj@ohsu.edu).

REFERENCES


DISCUSSION

Erwin R. Thal, MD, Dallas, Tex: Dr Mayberry and his group brought to us a very timely subject. Stimulated by the work of Dr Biffl and his group in Denver, Colo, and Dr Fabian and his colleagues in Memphis, Tenn, we have become aware of an increased incidence of blunt carotid artery injuries when selected screening has been utilized. In this retrospective review from 2 level I trauma centers, the authors have concluded, however, that these blunt carotid injuries and strokes related to these injuries were rare occurrences, and therefore aggressive angiographic screening would be hard to justify.

The group in Denver identified independent predictors of injury, and having 1 of these in a setting of a high-risk mechanism was associated with a 41% risk of injury. Miller and colleagues reported that the stroke rate for patients with injured vessels treated with heparin or antiplatelet therapy was 6.8% compared with 64% in those untreated, and this was significant at P<.001. Over the past 7 years, there have been more than 10 reports that support aggressive screening, and I think we are about to hear another one this morning.

I have several concerns about the design of this study and the conclusions made therefrom. This is a small retrospective, uncontrolled study of only 17 patients culled from 2 institutions during 2 different time periods. There was no structured protocol or surveillance of the asymptomatic patient. The follow-up period was less than 3 weeks. While there is good evidence to suggest that many minor vascular injuries will heal without sequelae, Biffl and colleagues reported a study of 76 patients in which 70% of the grade 2 injuries progressed after being treated with heparin for 7 to 10 days. The group in Miami, Fla, has reported on the safety of nonoperative therapy for extremity injuries. However, missed carotid injuries can have a far greater adverse outcome. In fact, this study supports that, as 11 (65%) of their 17 patients had a stroke. The authors only looked at patients with known lesions, and since they did not screen asymptomatic patients, it was not possible to know the true incidence of these injuries.

The authors refer to the burden on human and financial resources; however, no data are presented to support this hypothesis. All of us must be cost conscious, but let us not overemphasize cost concerns to the detriment of good patient care. Prevention is a healthy investment, and the elimination of 1 stroke may pay significant dividends.

This paper is important because it raises questions that are yet to be answered. What are the proper screening parameters? What is the best way to screen? Is there an increased burden, and can we keep the cost at a reasonable and acceptable level? I would like to ask the authors 3 questions: (1) How do you account for...
the fact that over a 10-year period and combining 2 large centers, you only came up with 17 injuries, which is far less than any other published series? (2) What was the grade of injury found in those patients who had strokes? (3) How do you plan to perform the cost-effective analysis that you recommend?

Dr Velmahos: First I would like to recognize the major contribution of the Denver and Memphis groups in this area. Our fear is that if their recommendations become the standard of care, we may be exposed to litigation in the absence of hard proof.

Thank you, Dr Thal, for these very important questions. Before I answer them, I want to address a few of the issues you mentioned in your discussion. The difference in our study is that we looked at blunt carotid injury–related stroke. We didn’t focus on the incidence of carotid injury if it didn’t relate to stroke, so we may have missed blunt carotid artery injuries but we didn’t miss strokes. As far as the heparin is concerned, I would like to remind the audience that the evidence on the value of heparin is unclear. As Dr Thal mentioned, 70% of the Denver group’s grade 2 carotid artery injuries progressed to higher grades despite heparin. So I am not sure that heparin really works or should be given.

Our stroke rate was only 0.05%, which is consistent with other studies. Before the Denver and Memphis data, the incidence was between 0.05% and 0.08%. With aggressive screening according to these groups, the incidence increases to 0.9%. This means that among 35,000 patients in our study, we missed 300 blunt carotid injuries. However, according to the Denver data, asymptomatic carotid artery injuries progress within 7 to 10 days. Our patients spent an average of 3 weeks in the hospital, and therefore it is unlikely that we missed so many injuries in 2 mature level I trauma centers with dedicated trauma teams. So we need to re-examine the issue.

With regard to your questions: We don’t have the grade of the injury, our incidence was consistent with many studies, and we have to think carefully about how we will consider our cost-effectiveness analysis, but this is a matter of detailed discussion.

M. Ashraf Mansour, MD, Grand Rapids, Mich: Have you used carotid duplex scans to screen these patients? I have been using noninvasive studies as a tool to help me decide whether to get an angiogram on a high-risk patient or not. In other words, if the duplex scan is completely normal, that would avoid an angiogram and that would speak to the cost savings that you discussed. And if you had an abnormal scan, then you would go ahead and get an angiogram.

Dr Velmahos: The duplex scan has 2 limitations. The first one relates to its reliability compared with the gold standard, which is angiography. A lot of people would say that it is not as reliable as angiography, particularly in the area that you really care about, the distal part of the internal carotid artery injury.

The second limitation relates to availability. For example, in my center, I cannot have a duplex scan on Friday evening. If the patient is injured on Friday evening, the patient has to wait until Monday morning to get a duplex scan, and I am sure that this is true in many centers. Duplex scan still needs to be explored as a potential noninvasive test for this area.

David H. Wisner, MD, Sacramento, Calif: How many workups did you do to find the 17 injuries that you found? Was there a 50% yield or a 1% yield from workups? The second question I have relates to your protocol for working up patients with neurologic findings that weren’t explainable by CT [computed tomographic] scan. How reliable is that approach? Is it possible that you had patients with blunt carotid injuries and strokes, but you didn’t know the stroke was secondary to carotid injury because you looked at their CT scans and thought the neurologic dysfunction was from their head injury?

Dr Velmahos: Thank you, Dr Wisner. The answer to your first question is that I do not know because we did not have this information to collect. My experience is that the yield even with our limited number of studies is low. Regarding the second question, I agree that the early CT is not reliable to identify strokes. Later, CT scans are. Once you have a patient with a low Glasgow Coma Scale score and inconsistent CT findings, essentially a negative CT scan, we often categorize this patient as having a diffuse external injury when the cause may be a stroke that does not appear in this very early CT scan. We routinely follow our patients with repeated head CT scans every 8 hours if that’s the case, so had it been a stroke, we would have identified that.

Edward T. Peter, MD, Red Bluff, Calif: I had 1 patient who had this injury secondary to a chiropractic manipulation. I wonder if you have seen this. And I wanted to know about the mechanism of injury. The ones that I have seen are in young people who have normal arteries. Some had intimal disruption with a flap dissection and ultimate clot or impairment of flow.

Dr Velmahos: For the most part, the mechanism of injury is a motor vehicle crash but can be really any mechanism. I do not have an opinion about the chiropractic manipulation.

Thomas V. Berne, MD, Los Angeles, Calif: George, do you think that CT angiography of the cerebral vessels is going to change this picture?

Dr Velmahos: Thank you, Dr Berne. I am not sure about that either. It’s a matter of technology evolution, and there is no doubt that this technology will reach the stage of being reliable. At this point and according to Drs Biffi and Morice, grade 1 and grade 2 carotid injuries cannot be reliably identified by CT scan or MRA [magnetic resonance angiography].

Gregory J. Jurkovich, MD, Seattle, Wash: Dr Velmahos, I appreciate your bringing this to our attention, and I share your concern about how to go about making these diagnoses. We struggle in our institution as well with the question of who should be screened and is it cost effective. I wanted to challenge 1 statement you made. If I have this correct, you said that you did not miss any strokes. Yet I do not believe your screening criteria were for stroke, but rather were for carotid injury. Did you really screen your entire 35,000 trauma admissions for a diagnosis of stroke, not just at discharge diagnosis but as part of their hospital admission? In a way this is a similar question to Dave Wisner’s. How do you really know that some of these injuries that you are calling a head injury aren’t really the result of a stroke due to a carotid injury? For that reason I’m reluctant to really accept the conclusions of this presentation.

Dr Velmahos: We did use as a criterion for inclusion in this study the presence of a carotid artery injury but also the presence of stroke.

Clayton H. Shatney, MD, San Jose, Calif: Interesting study, Dr Velmahos. I have a tough question for you. You implied that because of the 0.05% incidence of stroke, and I think that is a good end point, it wasn’t worth going after those few patients, with which I concur. But that brings up a big question in light of Dr Thal’s comment that if you could just save 1 of the 35,000 individuals, it might be worthwhile. My question is, is 0.05% something we should be going after from a cost-effective standpoint? If you are not willing to make that call, do you think perhaps the AAST [American Association for the Surgery of Trauma] ought to come up with a committee whereby we establish a certain percentage that is worth going after, in light of the fact that as human beings we cannot be perfect?

Dr Velmahos: This is certainly one issue that should be attacked by a major organization like the AAST or the COT [Committee on Trauma] in order to structure a multicenter trial. It would be a relatively simple observational study and would provide the massive number of patients that we need in order to define a stroke rate and a blunt carotid injury rate for that purpose across the nation. It seems like there is a lot of variation among trauma centers, certainly among ours and Denver. But regarding the first part of your question, what is a human life worth? I have no answer to that.