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Introduction.

It is interesting to review 1997 and wonder has trauma care advanced in Australia during that year? This question is difficult to answer because currently we may not be in a position to have an objective overview of the outcomes of the care we provide. Some outcome analyses are certainly being reported but these are often research based and relate to single institutions or a region. We have to ask ourselves is it not time for state and national outcome analysis? The advancement of Australian Trauma care requires us to be able to answer questions in relation to demographics, treatment and outcome of our trauma population.

In this our tenth edition of the Grapevine we look at a mother’s personal perspective of the effects of head injury. This insight to the aftermath of trauma is important and was shared with us during our recent Public Forum on head injury care. It reminds us that trauma reaches out to effect not only the patient but the family and friends of the patient.

There are some key challenges in trauma care and management of the elderly patients is one of them. Trauma in the elderly is frequent and associated with significant mortality. Professor Rick Bell highlights some key issues and we need to remember these the next time we might think of disregarding the "minor" injury in the elderly. Over the next year with hope to work with many of the different disciplines dealing with the care of elderly patients to develop guidelines for optimal care in certain specific injuries. Julie Fiendship has just joined the Department as case manager.

SWAN 6 in conjunction with the Australian Defence Force Health Services and the Ambulance Service of NSW will present 6 international speakers and a host of Australian experts for our 2 day meeting on May 1st and 2nd. The Public Forum on April 30th will focus on disasters and we are honoured to have Stewart Diver join us as guest speaker. Education is so important to us at Liverpool that we are always keen to have visitors do an attachment with us. If you would like to spend some time please ley us know. This month is the end of one era and the beginning of another with the transfer and modification of our web site, originally the work of Dr Jon Ryan. We hope we can build on Jon’s foundation.

Michael Sugrue
Director Trauma Services

The value of bronchoscopy in the management of burns

Severe inhalational injuries can remain completely asymptomatic for between 24 and 72 hours and lead to Adult Respiratory Distress Syndrome by the fifth day after the injury. The poor prognosis can probably improve with the use of early respiratory support at the
time of admission - especially with high-frequency ventilation (1).

According to Masanas et al (1994), bronchoscopy is sensitive (0.79) and highly specific (0.94) for the diagnosis of inhalation injury with high predictive value for ARDS. It is considered to be more reliable than the history of the injury & clinical findings (eg facial skin burns, singed nasal hairs, dysphonia) and is complementary to tests such as arterial blood gas analysis and platelet count. Therefore, bronchoscopy is recommended for all patients admitted for burn injuries, or inhalation of smoke or toxic gases within the first 24 hours (2). In cases when only discrete lesions are detected and the bronchoscopist is in doubt the diagnosis can be supported by histologic examination of staged biopsy or brush cytology (3).

References:


3 - Khoo AK, Lee ST, Poh. WT: Tracheobronchial cytology in inhalation injury: J Trauma 1997; 42:81-86

Comment

Inhalation injury is important to diagnose. Its presence doubles mortality from burns. Bronchoscopy is very useful diagnostic test to determine if inhalation injury has occurred, particularly looking for soot and erythema. I think the place of high frequency ventilation is still undecided. The study referred to by Martin is retrospective and uses historical controls. Masanes and colleagues suggests that prophylactic conventional ventilation improves outcome. Unfortunately there is a lack of prospective evaluation of these issues

Gill Bishop Director ICU Liverpool

Trauma in the Elderly: MORE THAN JUST AGE!

Richard M. Bell, M.D., F.A.C.S.

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INTRODUCTION

The population of people in the United States over the age of 65 constitutes about 11%. It is projected that this segment will increase by 6% over the next ten years and by the year 2025 these Seniors will represent 25% of all Americans. In addition, the percentage of people under the age of 44 is expected to decrease over this same time frame. Since our senior citizens already consume 33% of all money spent for trauma care, injury will
soon become a disease more closely associated with the elderly, not just a problem that affects the "young, potentially most productive members of our society." If by the year 2025 the average life span does indeed reach 85-115 years, the potential for injury will likely increase. Considering the fact that over 80% of the injured elderly patients can return to their pre-existing, independent level of activity following trauma, aggressive resuscitation and follow-up care is warranted. Increasing age correlates with increasing mortality in the low to moderately severe injury categories, however mortality remains relatively constant in the more severely injured groups, (ISS 25 or greater). The tendency to approach the seriously injured or ill elderly patient with an attitude of "avoidance or postponement" is myopic. Outcome can be maximized by a more aggressive approach, rather than one which is expectant.

ANATOMY AND PHYSIOLOGY OF AGING

General surgeons frequently view children as "small adults", much to the dismay of pediatricians and pediatric surgeons. Elderly patients have been described as "stiff grown-ups". Many of the physiologic changes associated with the aging process render organs less compliant ("stiffer"), with a reduction in organ reserve function and ability to respond to the stress of injury.

Cardiovascular System

The stress imposed by injury can result in mortality and serious morbidity from a cardiac perspective often not attributable to direct cardiac injury per se. Anatomic changes in the cardiovascular system begin early in life with plaque formation present in 30% of individuals in their late teens and rising to involve 85% of people by age 45. By age 65, nearly half the population has coronary artery stenosis of greater than 50% of the vessel diameter. Cardiac output falls on the average of 1% per year after the fourth decade. Total blood volume decreases and circulation time increases. The total number of pacemaker cells in the sinus node decrease with advancing age and only 10% of the original number may remain at age 75. In addition, a reduction of 50% of the conducting fibers in the bundle of His is common after the 6th decade. The reduction of conduction fibers is accelerated by any cause of left ventricular hypertrophy. The aging heart also demonstrates an increase in collagen and lipofuscin, with valvular thickening and stiffening.

Elderly patients respond differently to the stress of injury than do younger patients. Youthful people are able to increase cardiac performance by increasing heart rate and myocardial contractility. Cardiac output is further augmented by a reduction in ventricular end-systolic volume. The ability of the elderly individual to increase heart rate is limited during stress or exercise, but these patients can increase cardiac output by increasing preload (end-diastolic volume). With increasing age there is a steady increase in left sided dysfunction and abnormal ventricular wall motion with exercise in 40% of patients at age 60 and present in 90% of patients at age 80 or greater. The response to endogenous catecholamine release with stress is also different. It is presumed that the failure to demonstrate a significant response to hormonal milieu of the stress response following injury is not due to a decrease in production of these mediators, but more likely related to a reduction in the responsiveness of the cellular membrane receptor.

For the elderly patients undergoing non-cardiac surgery, Goldman has developed a mathematical model for predicting the risk of a peri-operative cardiac event. Most surgical procedures necessitated by injury are emergent or urgent and the luxury to weigh the risk:benefit ratio is not always available. Further, the opportunity to formally evaluate cardiac function in detail, or to improve cardiac performance may be limited in the setting post-injury. Even injuries which do not require surgical intervention no doubt
generate the same, or greater, degree of a stress response that many operative procedures impose.

A common pitfall in the evaluation of the older patient following injury is the mistaken impression that "normal" blood pressure and heart rate indicate normovolaemia. Blood pressure generally increases with age due to stiffening of the vessels, ventricular hypertrophy and other factors. Blood pressure of 120mmHg may represent hypotension for a patient whose normal pre-injury blood pressure was 170-180 mmHg. Coupled with the limited ability to increase heart rate, significant blood volume loss may be masked by the absence of tachycardia early in the course, or even hypotension, whose onset may be delayed. The chronic high afterload state induced by elevated peripheral vascular resistance may limit cardiac output and ultimately peripheral and coronary oxygen delivery. Further, it must be remembered that blood pressure should not be equated with cardiac output.

Scalea and co-investigators carefully examined outcome in a group of geriatric patients. Early in their series, one third of the patients who appeared "stable" in the resuscitation area with a "normal" blood pressure and heart rates died within 24 hours of cardiac arrest. Survival was improved in their experience by early (first 1-2 hours) aggressive, invasive monitoring. With this approach, previously unsuspected states of hypoperfusion were identified and reversed much more quickly! Failure to recognize inadequate oxygen delivery creates an "oxygen debt that the geriatric patient is unable to pay." Haemodynamic resuscitation may require the use of inotropes after volume restoration in these patients.

Renal

Ageing of the kidney and decreasing renal function occur even in the absence of concomitant diseases which are known to have deleterious effects on renal parenchyma (eg hypertension, diabetes, etc.). Renal blood flow decreases by 1 % per year after age 40. This reduction in renal blood flow appears to be related to intra-renal morphologic changes. Blood flow may be further reduced by atheromatous plaque in the renal artery and arterioles. Glomerular senescence is manifested by a 20% reduction in renal weight between ages 40 and 80. As much as 50% of the total population of nephrons can be lost with the ageing process. Most of this weight loss is from the cortex. Histologically there is hyaline deposition in the afferent arterioles and anastomotic continuity between the afferent and efferent arterioles can be demonstrated. As basement membranes thicken, glomerular and tubular sclerosis ensues. Convoluted tubules can collapse due to tubular wall atrophy. The majority of these changes occur in the renal cortex, but interstitial fibrosis occurs primarily in the medulla. A reduction in length of the medullary tubule is also common.

The morphologic changes described above lead to glomerular hyperfiltration and intraglomerular hypertension. The glomerular filtration rate (GFR) declines by 6.5 mL/min/1.73m2/decade after age 40. After age 60 the rate of reduction in GFR declines by 17 mL/min/1.73m2/decade. Serum creatinine usually remains within the normal limits presumably due to a reduction in muscle creatinine production from the decrease in lean body mass which accompanies ageing. For patients over the age of 60 it may require over 30 hours to decrease sodium excretion in response to salt deprivation. Maximum concentrating ability of the kidney of an octogenarian is 850 mosm/kg. This is only 70% of the ability of the 30 year old kidney. Decreased production of renin and angiotensin are also observed, as is the responsiveness of the kidney to these mediators.

The changes associated with ageing in the geriatric kidney place this organ at increased
risk following injury. "Relative" hypo-perfusion may predispose the kidney to increased risks of ischaemic injury. The therapeutic window is significantly narrowed for many potentially nephrotoxic agents, especially those administered during the early evaluation and management of these patients before homeostasis has been totally restored, ie, radiographic contrast agents, antibiotics. Pitfalls in the resuscitation of the injured senior citizen include either over or under-fluid resuscitation. Interstitial solute is essential to preserve the counter current multiplier necessary for volume and sodium preservation, but the sluggish renal response to solute loading in the elderly, makes volume and solute over-loading quite easy. Early invasive monitoring is clearly helpful to assure adequate volume restoration, renal perfusion and oxygen delivery. Frequent monitoring of serum antibiotic levels and the indices of renal function is prudent.

Respiratory

Many of the changes which occur in the airways and lungs with ageing are difficult to ascribe precisely to the process of senescence per se and may be the result of chronic exposure to toxic gases throughout life. Regardless of the cause, the respiratory function of the elderly patient is significantly reduced. Calcification of tracheal cartilage retards the normal shortening and lengthening of the conducting airways with changes in lung volume. The loss of elastic tissue around non cartilaginous airways promotes distal airway collapse, air trapping, "auto-PEEP" and alveolar dilatation. The net effect is a chronic increase in the functional residual volume. This increased volume progressively flattens the diaphragms and ultimately reduces diaphragmatic excursion. Maximum inspiratory capacity is compromised. Capillary and alveolar basement membranes thicken reducing diffusion capacity. The sensitivity of the central nervous system receptors to changes in partial pressures of oxygen and carbon dioxide, as well as pH, is reduced. The limited ability to increase minute ventilation when necessary, renders the elderly patient at increased risk for respiratory failure, even with minor injuries, with the increased possibility that early intubation and mechanical ventilation will be required. With intubation, the risk for gram-negative pneumonitis becomes significant. In some series, the mortality in elderly patients who develop pneumonia exceeds 90%.

The mortality in patients who develop pneumonia exceeds 90%.

Chest injuries occur in elderly and non-elderly patients with essentially equal frequency. Mortality rates, however, for the elderly patient are higher. This difference in mortality cannot be attributed to an increase in non-thoracic injury, but may be related to the fact that more geriatric patients with serious thoracic injury arrive without vital signs, and that the ability to maintain haemodynamic normality is significantly less than their more youthful counterparts. Haemo- and even simple pneumothorax, are not well tolerated by the elderly patient. Pulmonary complications of serious injury occur in the elderly with equal frequency. Atelectasis, pneumonia and non-cardiogenic pulmonary oedema predominate. The latter two problems contribute significantly to the late deaths in the older group, but may not be the primary cause of mortality. As with injury to other organ systems, the male predominance in the population is lost with nearly equal numbers of men and women sustaining chest injury. Marginal respiratory reserve coupled with over zealous crystalloid fluid infusion increases the potential for pulmonary oedema or worsening of intraparenchymal pulmonary contusions.

Musculoskeletal

Disorders of the musculoskeletal system are the most common complaints of the middle and elderly population. These disorders are the most likely to cause restrictions in an individuals daily life and are the key components to loss of independence. Changes due to the ageing process in other organ systems (heart, central nervous system etc.) contribute to reductions in mobility and independence. Changes in soft tissue with age are most likely to account for these limitations of motion. Proliferation is further reduced by changes in DNA-transcription factors, proteins which control DNA replication. The
cellular products change substantially as the cell ages. The collagen produced, for example, is less soluble, more heat stable and contains increased cross-linking. This results in "stiffening" of ligaments, cartilage, intervertebral discs, joint capsules, etc. In addition to a general decline in responsiveness to many anabolic hormones, there is an absolute reduction in levels of growth hormones.

After 25 years of age, muscle mass decreases by 4% every ten years and after age 50 the rate is 10% per decade. If levels of growth factors are low the rate may approach 35%.

This is manifested by a reduction in size and total number of muscle cells. The decrease in strength with ageing correlates directly with the decrease in muscle mass. Skeletal muscle is frequently injured at the time contraction. Initial injury as measured by the reduction in ability to contract is the result of the mechanical damage to the sarcomere. At three days following damage contraction is at its lowest and is thought to be the result of damage to the myocyte caused by toxic oxygen radicals.

The first changes due to ageing can be demonstrated in articular cartilage shortly after skeletal maturation. Clefts and fissures develop in the surface of the cartilage, there is increased density of subchondral bone and osteophyte formation begins. Although the chondrocyte population declines as the skeleton matures, the population remains relatively constant in adult life. Cellular production of more variable proteoglycans is responsible for the changes in the articular matrix which is less effective in repair of damaged surfaces.

Deterioration of tendons, ligaments and joint capsules follows similar pathways, leading to facility of injury, spontaneous rupture and providing less joint stability. This increases the risk of injury, not only to the musculoskeletal system, but adjacent soft tissue. The most dramatic changes of all musculoskeletal tissues occurs in the intervertebral disc. The loss of water and proteoglycans with aging affect the shape and compressibility of the disc. These changes shift the loads on the vertebral column to the facets, ligaments, and paraspinal muscles. This contributes to degeneration of facet joints and the development of spinal stenosis. Progressively, these alterations place the spine and spinal cord at increased risk for injury.

Immune

In theory, it may be the immune system which determines longevity by being the first organ system, especially the thymus, to reach the end of its genetic information. Thymic tissue is less than 15% of its maximum by age 50. Hepatic and splenic size also decrease. Changes occur in the number and function of T-cells and B-cells, but a substantial amount of contradictory information concerning specifics is notable in the current literature. Cell mediated immunity decreases with age as measured by skin test reactivity and can be directly correlated with survival. Whether its response is related to a decrease in numbers of T-cells or a reduction in their ability to respond to activation is speculative. B-cell senescence is manifested by a decline in antibody production. This phenomena may be, at least in part, related to alteration in T-Helper/T-Suppressor cell ratio or function. To confuse the issue further, the production of antibody against "self" appears to increase with age, ie an Increase in anti-native DNA, rheumatoid factor, antithyroglobulin, etc. There does not appear to be an increase in autoimmune diseases which parallels the aging process, however. Clinically, the elderly are less able to tolerate infection and are more prone to develop multiple organ system failure. The absence of fever, leukocytosis and other manifestation of the inflammatory response may be explained by changes in inflammatory cell numbers or response, or the change in cellular products (a reduction in interleukin-1 production or an increase in
prostaglandin-E2 as examples).

Central Nervous System

Brain weight falls about 10% by 70 years of age and is associated with progressive loss of neurons leading to cerebral atrophy. This loss is replaced by cerebral spinal fluid. In this setting, the opportunity for increased intracranial bleeding from injury exists. With advancing age, the dura becomes tightly adherent to the skull accounting for the reduction in acute epidural haematomas seen in the elderly patient. Subdural haematomas are the most common and are the result of increased tension on the bridging veins caused by separation of the atrophic brain from the dura. Tears in these veins result from even minor injuries producing subarachnoid as well as subdural haemorrhage. Abnormal proteins tangle inside the neuron while plaque from dendritic degeneration accumulates. Cerebral blood flow is reduced by 20% by age 70, and even further if atheromatous debris occludes conducting arteries. Peripherally, conduction velocity slows due to demyelinization. Clinically, subtle changes in mental status may be demonstrated by reduced acquisition or retention of information. Visual and auditory acuity decline, vibratory and position sensation is impaired and reaction time increased.

Medications

Concomitant disease may require the use of medications and many elderly patients are on multiple pharmacologic agents. Drug interactions are frequently encountered in this population. Side effects are much more common due to the very narrow therapeutic window in the elderly. Adverse reaction to some medications may even contribute to the injury producing event.

Beta-adrenergic blocking agents may limit chronotropic activity. Calcium channel blockers may prevent peripheral vasoconstriction and contribute to hypotension. Non-steroidal anti-inflammatory agents may contribute to blood loss due to the adverse effects on platelet function. Steroids and other drugs may further reduce the inflammatory response. Chronic anticoagulant use may increase blood loss. Chronic diuretic ingestion may render the elderly patient chronically dehydrated and with total body deficits of potassium and sodium. Hypoglycaemic agents not only may have contributed to the injury event itself, but may make control of serum glucose difficult if their use is unrecognized. Psychotropic medications, commonly prescribed for elderly patients, may mask injuries or become problematic if withdrawn abruptly. Changes in central nervous function as a result of these medications may have also contributed to the injury. In addition, elderly individuals usually neglect to keep their tetanus immunizations current.

Biomechanics of Injury in the Elderly

After the age of 50, injury from falls steadily comprises a greater percentage of the cause of injury, reaching over 60% of the injury causing events by age 80. The elderly are especially prone to injury from falling because of the physiologic changes described above. The ability to maintain balance, especially after tripping, is decreased in the elderly patient. Visual acuity is decreased compromising the ability to avoid hazardous objects. In addition, there are many disease states which may cause elderly patients to fall. Postural hypotension, cardiovascular changes, vertebrobasilar insufficiency, and hypoglycaemia are common in the older patients. Falls from the same level generally result in femoral neck fractures or fractures of the distal radius as the outstretched hand attempts to break the fall. Humeral neck fractures commonly occur after falling on the elbow. If the head is struck, subdural haematoma is likely for the reasons cited in the discussion of CNS changes with ageing above. Elderly patients who have fallen deserve
careful evaluation.

Motor vehicle crashes are the second highest category of injury mechanisms in the elderly population. These injuries generally occur close to the patients’ homes, as the elderly driver may voluntarily restrict their driving to familiar territory. The majority of vehicular crashes occur in daylight hours, presumably due to a reduction in night vision in this population which may further restrict their opportunity to drive. Interestingly, the majority of vehicle crashes are side impacts. This may reflect a decrease in peripheral vision in the elderly, or restrictions on the ability of the older individual to turn the head and neck to the side due to degenerative changes in the cervical spine. Side impacts result in energy transfer to the side of the body which is struck suggesting the possibility of ipsilateral extremity fracture, rib fracture, pulmonary contusion, spleen or liver injury.

Injuries from firearms decreases steadily with age, but of those that do occur a larger percentage of them are intentional efforts of the elderly to take their own life. Injuries to pedestrians remains relatively constant after age 50. The majority of these occur in cross walks as opposed to the younger pedestrian who is usually struck by a moving vehicle away from an intersection. Changes in visual acuity, presbycusis, slowed reaction times and inability to move quickly may contribute to this fact. The percentage of injuries due to burns is also consistent throughout life. Despite advances in medical technology, the estimation of mortality as age plus percent of body surface area burned remains valid.

Aggressiveness, Ethics and Outcome

Most elderly patients return to their pre-injury level of function and independence following recovery from traumatic events. Age increases mortality from injury significantly for the lower injury severity bins, while mortality is relatively constant in the more severely injured categories. More aggressive care, especially early in the evaluation and resuscitation of the elderly trauma patient, has been shown to improve survival. Attempts to identify which elderly trauma patients are at greatest risk for mortality have not found much utility in clinical application. Age greater than 75, Revised Trauma Scores of less than 7, hypotension, hypoventilation and Coma Scores of 3 pose the highest relative risks of death in one series. While these factors may be useful for explaining the mortality in a group of patients, they offer little in determining the clinical course of any individual patient or the appropriateness of providing care. Certainly there are circumstances in which the physician and patient, or family member(s), may choose to forgo heroic measures and provide only supportive care. The ethical issue of appropriateness of care in an environment of declining hospital resources and restrictions on finances is more than challenging.

REFERENCES:


McCoy GF, Johnstone RA, Duthie RB. "Injury to the elderly in road traffic accidents." J.Trauma 29: 494-7, 1989
Dear Sir,

RE: PAIN RELIEF FOR PATIENTS WITH SIGNIFICANT HEAD INJURIES

As an Accident and Emergency doctor, I have had several unresolved disagreements with various general surgeons on providing pain relief in both adult and paediatric patients who attended the Accident Department with the above condition. We are in agreement with regards to the role of analgesia in patients with mild and severe head injuries but the problem arises with patients whose GCS (Glasgow Coma Scale) lies between 6-10. I have enclosed for you three cases which summarise the great gulf in our opinions on these matters.

CASE 1

A young man, 26 years, was involved in a RTA (Road Traffic Accident) and he arrived in the Department after receiving pre-hospital care by our advance paramedical crew. He was resuscitated along ATLS guidelines with >90% oxygen and in-line spinal immobilisation. His main injuries involved the head and neck and fracture to the distal humerus. His airway was intact, hypoxia, hypovolaemia, and hypoglycaemia excluded, catheterised, and his fracture splinted. His GCS fluctuated between 6-8 and whenever he was restless, he pulled at anything which his hands could reach. A decision was made to give this gentleman a titrated dose of Dimorphine (total given, 2mg) intravenously. No further problems were encountered thereafter and his coma scale was not altered nor was his oxygen saturation. The general surgeon had wanted intramuscular Codeine Phosphate to which I disagreed.

This patient had CT scan and was transferred to the surgical ward for continuous neurological observation as his GCS improved a few hours later.

Comment

Analgesia in head injury care is important. Pain relief must be given. Even patients with significant decreased level of consciousness need analgesia. I would administer morphine IV in initial 5mg boluses and then 2.5 mg iv. I would not recommend the use of Codeine Phosphate in this type of patient. In relation to his management In general we tend to intubate our patients with GCS < 9, not only to protect their airway, prevent hypoxia but
also to facilitate CT scanning as it is likely that the patient will not lie still for the scan.

CASE 2

A middle-aged gentleman, 53 years, was admitted to our department after the car he was repairing fell onto his face when the car jack slipped. He was resuscitated by the pre-hospital emergency team with in-line spinal immobilisation. On arrival, he was resuscitated along ATLS guidelines, hypoxia, hypovolaemia and hypoglycaemia excluded and was breathing >90% oxygen. He was very restless, bleeding from the nose, ear and mouth had an intact airway despite his GCS of 13, and complaining of pain. The surgeons wanted analgesics, but I suggested intubation in view of his facial injuries.

He was given Codeine Phosphate and prior to CT scanning, he was intubated. He sustained extensive facial, and intracranial injuries and was transferred to a neurosurgical unit for emergency intracranial decompression and facio-maxillary care.

Comment

In a patient with significant facial fractures Codeine Phosphate is inadequate and IV Morphine should be given, preferably in the pre-hospital phase. Analgesia may help relax the patient prior to intubation. Patients with this type of facial injury have difficult lying flat as it can compromise their airway and particular care in this group should be taken not to over sedate.

Case 3

A teenager, 17 years, fell approximately twenty feet, sustaining head injuries and a fractured right femur. Treatment was along ATLS guidelines with hypoxia, hypovolaemia and hypoglycaemia being excluded. His GCS was 6-7, restless, and he was given intramuscular Codeine Phosphate prior to splinting his fractured femur. His GCS fell and he was intubated and Naloxone given. CT scan revealed no intracranial abnormalities and he was admitted to the surgical ward for neurological observation after fixation of his femoral fracture.

Comment

In multi system or major trauma we avoid intra-muscular injections as they are slow to be absorbed and have a variable response. I am not sure why your patient received naloxone.

I have searched the literature for recent opinions on this matter but the majority of the authors fail to deal with this aspect of patient care adequately or properly. Opinions varied from intramuscular Codeine, titrated intravenous opiates, non-steroids, such as per rectum Diclofenac Sodium, no treatment until neurosurgical evaluation, or intubation.

Our trauma unit sees six (6) to ten (10) major trauma cases per week involving a wide variety of both paediatric and adult trauma. We do not have an on-site neurosurgical team and henceforth, consultation is via the telephone to our referral centre nearly forty miles away and this could take up to an hour. The neurosurgeons are in favour of
Codeine but a few are questioning the widespread use of this analgesia.

I would be grateful for your organisation's opinion and guidelines in dealing with these cases.

Yours sincerely,

Robert Ritchie
Specialist Registrar
Medway Accident Centre
Gillingham Kent UK

Reply

That you for your frank questions in relation to analgesia. I would recommend the use of morphine intra-venously. In general you will be undertaking a CT of the head in nearly all patients with a GCS of less than 13 and administration of morphine will not mask neurological signs.. It is extremely rare to cause respiratory embarrassment with morphine. It can be reversed with Narcan. We tend to intubate patient with a GCS <9, and give morphine even to these patients. I would not use Codeine.

Michael Sugrue

A Mother's Perspective

My name is Jenelle Saliba and I am a parent of a brain injured child. My son David, in June 1995, sustained a closed brain injury; he was 15 years old. David was riding a trailbike at night, with no headlights, on a country road; he collided with a parked semi-trailer.

His injuries were a frontal-lobe injury with facial lacerations, broken teeth, severed lip, fractured second and third ribs, collapsed lung, shattered thumb and patella and two brachial plexus lesions at the base of C5,C6 and C7. David also sustained trauma to his throat and was in a coma for nine days.

Firstly the shock: as a mother to be informed that your child has been involved in an accident, especially a road accident, is overwhelming. The mixed thoughts and emotions, the "what if's and if onlys", come flooding in; what if he is unconscious, what if he is on life support, what if he dies.

You enter the Emergency Department, announce you are David Saliba's mother and a nurse looks at you with a well rehearsed (not quite perfected smile) which says, stay calm, this is serious, he is in with the Trauma Team. She then escorts you to the "Green Room". The "Green Room" where a telephone is provided for you to ring and inform the family. You now really know this is serious.

Time flies by one minute and when you look at the clock it's actually been several hours. Your mind wafts, you flash back to happier times, you see naked baby images on rugs, then to blood red, carnaged images of this person on that bed, but you never quite see the face. Thoughts of organ donation are soon quashed by "he'll be O.K." An emergency doctor enters and explains David's condition, what has been done, CT scans, spinal x-rays etc. She then takes a deep breath, which you somehow mimic with her and prepare yourself for the worst.
"Mrs Saliba, David is on a respirator, there are many I.V. drips, he is on a heart monitor - he is in a critical condition - you can come in now". She verbally prepared me with facts and tried, I suppose, to soften the impact of what I was about to see.

The Trauma Team were brilliant, efficient and caring, they walked me through the Emergency Department.

I then saw David. I gasped, he didn't look like David, his eyes blackened, his face almost unrecognisable with swelling, he had tubes going into his mouth, blood in his nostrils and hair, and a bandage on his lip.

The medical equipment constantly flashing and beeping all around him. I stood and took everything in. I could smell petrol, I could see monitors and I could hear beeping and alarms, but the sound of the bellows of that respirator still haunt me. I thought I was going to pass out, I had to touch him! All my other senses were on overload. I walked closer and touched his forehead, he was quite warm; this somehow surprised me as everything else was cold and alien. I asked if he could hear me, the doctor looked and said "talk to him".

I thought that he had been sedated, that medication had rendered him unconscious. It wasn't until three days later in ICU that a word jumped out at me - COMA - a word I had not allowed to surface. How that word effected me! "But if his CT is clear, why is he in a coma" I kept asking. "He has hit his head, Mrs. Saliba, at high speed, Mrs Saliba; he has a head injury" Still not registering that anything could be wrong with his brain. No one mentioned the word brain'. "Head injury, OK run this by me again! If the scans and x-rays are clear why is he on life support, in a coma? Where is the physical proof of what is causing this?" I am sure someone explained it all to me again, but somehow it just didn't sink in.

ICU - the reality hits. The nursing staff were the most incredible people I had ever met, professional, courteous, angels with invisible wings. I would ask questions, and they would patiently explain everything, every time a procedure was carried out they would explain to us why and what it was. Unfortunately there was a breakdown in communication between the doctors and us. One doctor had presumed another had kept us up to date. The only information we extracted was an eight-hourly hand-overs or by eavesdropping to hear doctors exchanging opinions at the end of David's bed.

On day three my mother had a mild heart attack at David's bedside, she was admitted to CCU, I now had two patients to worry about. A hospital Social Worker was sent to see mum; my husband and I only saw her briefly. After nine days, David regained consciousness and was breathing on his own, he had a tracheostomy and was transferred to a ward.

This was it, his orthopaedic wounds would heal, he would be home in a couple of weeks. Not so! Waking up from a coma was slow; every time David's eyelids fluttered to open, we would hold his hand and speak to him. "It's O.K. David, it's mum, you are in hospital, we are all here" My husband and I cried with joy. He's back!

He opened his eyes, he was panic stricken, we reassured him, he was alright, and that he had a tube in his throat to help him breath, and that he couldn't talk now but would be able to when it came out. David lifted his hand, felt the trachy and gave a thumbs up signal, he smiled then went back to sleep.

Little information or support was given to us at that time, although we knew that he was being cared for with such expertise. It was only after he was transferred to a
neurological ward many weeks later that PTA was discussed and doctors from the Brain Injury Unit came to assess him for his suitability to undergo rehabilitation there.

The Brain Injury Unit. We had thought David would feel relaxed here as there were patients chatting on the verandah, having coffee and others involved in woodwork classes. There was a relaxed feel about the place. We were introduced to medical staff, physio and speechies. David was initially frightened by the other patients, some of whom were in a very bad state. Some were groaning and one patient screaming out obscenities constantly. Another female patient sat in a chair chanting in rhyme. What was this place? David spent everyday with a regime to help rehabilitate him physically, cognitively and many skill building exercises and activities were organized.

Although improving physically, he also appeared to be more confident in himself yet emotionally and behaviourally he seemed cold towards his family. He angered quickly and would use obscene language frequently.

On David's first weekend leave, he seemed agitated. Some friends called around and they all went for a walk. They had to catch up. David didn't come home, it was late, my older son Damien went to search for him. David, when found, refused to co-operate, he became aggressive towards his brother, his friends were also concerned as David was behaving somewhat "out of character" to how he was before. Damien rang and said "come and get him, he's out of control". We drove him home, under much protest, he yelled abuse at us and was physically shaking. He had consumed alcohol (not a great quantity and this had obviously helped trigger him off.

My elder son and I sat mortified as to what we had just witnessed, was it the alcohol, was it frustration at being away from his peers, was it the brain injury?

On Monday morning back at the Unit, Dr. Hodgkinson explained the dangers of consuming alcohol; he stood a greater risk of epileptic fit.

David shrugged this off, he bragged about his weekend adventure and continued his rehabilitation.

Several months had passed and David was coming home. I was relieved, but I also dreaded it. A fear of inadequacy, how were we supposed to handle him, how was I to pacify his anger. I cried, I felt isolated. I'll dig, I thought, out came the mattock and spades, out came my frustration, with every sod of drought-hardened clay, with every anger-filled swing I dug away, crying, yelling at the ground, looking to the sky for help. What were we to expect, how would we handle him, how to pacify his anger. All I heard from the family and friends was, "he's a teenage boy, doing teenage boy things, it's normal, he'll come good"!

Prior to the accident, like any other adolescent, David was becoming aware of his sexuality, he was in transition, somewhere not quite a child but definitely not quite a man. He had typically adolescent problems and really knew nothing of life except home, school and friends. He was going through body and hormonal changes. He was a walking GONAD! This is often a difficult time for the child and parents alike, with door slamming, diet-fads, pimple squeezing, bathroom hogging, parading in front of any image - reflecting surface, how to be cool - gotta, gotta, gotta have those Nikes - and - "by the way mum and dad you know nothing".

David was a popular kid, the phone would never stop ringing, he played sports and participated in many activities. Never the academic he enjoyed the social aspects of school. If you take all the above and add brain injury you have every emotion
exaggerated, every frustration harder to cope with, aggression sharpened to violence.

It has been 2 years and 4 months, and yes he's I suppose come good. He is alive, he is now 17 and has a driver's licence, he has done part-time work, he parties and goes out with his brother and his BROTHER'S FRIENDS. He's a gypsy, a free agent who never feels comfortable in one place. He has no fears of walking the streets at night or train hopping. He explodes unpredictably, putting his fists through walls and doors, his behaviour quite unacceptable. David uses obscene language and says inappropriate things, like cluing in his elderly granny, quite explicitly about his sexual encounters, (granny nearly went back to C.C.U. after that little discussion).

The telephone doesn't ring for David anymore, the house is void of goofy teenage boys playing loud music and reading hot-car magazines. There are no friends, no girlfriends anymore. No-more school - David dropped out due to ongoing difficulties and lack of support of understanding by school officials. Before the accident David planned an apprenticeship in car mechanics. Now he finds it hard to accept that his brachial plexus lesions and brain injury make that impossible.

He has become more child like in some respects. He is totally consumed with whatever he is doing at that moment. If he is working on an airfix model, for example, he doesn't leave or deviate from his task; he doesn't allow for breaks, for meals and even toileting, he just sits and glues his model, totally entranced, fixated.

He paces the floor like a lion in a cage. He struggles with guilt, after all it was his fault, it's his head, his change of lifestyle and his loss of friends.David's grief, anger, sense of hopelessness and exclusion has never really been dealt with, his psychological problems are real, his fears genuine. He is crying out for help. He will at times self-mutilate himself by burning his arm with a cigarette, he now drinks and smokes.

Although David was expertly cared for by the Brain Injury Unit, somehow he didn't fit into either an adult or paediatric unit. At the time of the accident he was a 15 year old blob with attitude. Experts in adolescent problems and issues need to be consulted in such cases. I would like to see group therapy sessions for other adolescent "headies" as David calls them. They need to laugh at themselves, cry at themselves and share their fears, experiences and difficulties.

For the family, as carers, tomorrow is our time to go on and build networks. We need to improve our understanding that brain injury affects the entire family unit and its extended community supports. It effects peers and colleagues, friends and relatives, lifestyles and goals. Tomorrow we need hope and support and we deserve respect. As a mother, I often felt unheard, labelled "overprotective" or "David's biggest problem", but at the end of day I know that I knew David better than anyone else. I felt his feelings, shared his grief. At the end of the day when the professionals go home David's family hold things together, pick up the pieces. Then again tomorrow isn't good enough, we need help today.