THE NOVA SCOTIA MEDICAL JOURNAL

On Being Blamed

"If you can keep your head when all about you are losing theirs and blaming it on you."

Rudyard Kipling

For many reasons and from many directions blame is being laid on the medical profession for the current problems and state of affairs. Physicians are blamed by politicians as being one of the reasons the costs of health care are now unaffordable. Critics say that there are too many admissions to hospitals, too much surgery, too many lab tests and x-rays, and too many needless office visits.

Lately, journalists have also been laying blame on physicians' shoulders. For example, in a recent article in *Macleans,* Diane Francis initially shows insight when she states, "Doctors are not the culprits." Ironically, she concludes her article with the statement, "I would also suggest that doctors be forced to end featherbedding practices by permitting the licensing of nurse practitioners, midwives and other paramedics to save the system money." Her accusations relate to the blame we have also been receiving from other health professionals. It has been suggested that we are always acting in our own self interest and as well will not share "control of the team."

At the recent Canadian Medical Association Leadership Conference, the Honourable David Nantes, Nova Scotia's Minister of Health and Fitness, told his delegates that he feels poor communication between doctors and nurses was a factor in the nurses' discontent. He indicated this impression is the result of presentations made to Nova Scotia's Royal Commission on Health Care.

Patients also like to blame at times, sometimes holding physicians responsible for their illness or the lack of a cure. Fortunately, patients, when confronted with the dynamics involved in this type of blame usually become more understanding. However, increasing medical/legal costs certainly reflect the increased readiness to blame physicians, both appropriately and inappropriately.

We are also blamed for not preventing illness, but merely treating it, as if this was not an honourable occupation.

We are blamed for billing practices that utilize higher fee items. The fee for service system is based on high fee items, that helps to subsidize lower paid more time consuming service. Unfortunately, unreasonable disparities between specialities have developed and this leaves us open for more accusations.

Mr. David Nantes, again at the recent CMA Leadership Conference, accuses us of acting like trade unions in our negotiations over fees, attempting to protect the "status quo."

We have all been upset when the Post Office and other unions defend what seems like unreasonable positions and understand the problems this can cause. Physicians could probably discuss their own positions more reasonably, however, if they were feeling less like a "scapegoat."

Being blamed, however, engenders anger, frustration and reactions which creates a climate that is not always rational. Under such circumstances, it is often difficult to take a conciliatory approach to what seems like unfair criticism.

Blaming is one of the four patterns of communication identified in family therapy by Virginia Satir. Her efforts at clarifying communication are generally applicable to a more general range of problems, psychiatric and otherwise. She states the "blamer" is much more interested in throwing his or her weight around than really finding out about anything. Often blamers are domineering and attempt to gain power over the other party. Certainly blaming is dysfunc-tional communication and will not allow us to ever deal with the content of the problem. We cannot allow it to anger us, or allow ourselves to become "scape-goats" and isolated from the process of solving problems.

Although the Royal Commission, some columnists, our Minister of Health and fellow professionals seem to be particularly severe in blaming lately, we would be well advised to keep our heads "when all about are losing theirs."

The criticism we receive as a Medical Society probably has much basis. Twenty years ago we did things much the same as we do today and we were praised; now we are often criticized. One suspects society has changed around us and our conservative and insular attitude have left us isolated. Whether right or wrong, somehow we should continue our attempts to explain ourselves and our positions. We must remember that blaming, however negative, is still just an effort to communicate and we must continue and indeed, actively facilitate the process despite the many remaining misunderstandings. ☐

J.F.O'C.

Appreciations

DR. THOMAS J. McKEOUGH

With the passing of Dr. Thomas James McKeough on January 2, 1990, Medicine and Politics have lost a strong supporter and diligent worker.

After graduating from University of Ottawa Medical School in 1951, Dr. Tom began practice with his father in Sydney Mines. Eventually, he took over the practice and had worked very actively up until the Autumn of 1988 when he retired due to illness.

He was on the staff of Harbour View and Northside General Hospitals, serving on Committees with enthusiasm. He was very instrumental in Hospital Development in the Northside area. He is a Past President of the Nova Scotia Medical Society and was also elected to Senior Membership in the Society in 1987.

His concern for the well being of his patients and indeed all the people of his area, naturally led him into the Political Arena. He was elected as M.L.A. initially in 1960 and, after being re-elected for subsequent terms, he had served the second longest term in the Legislative Assembly. Retiring from Politics undefeated in 1978, he had held Cabinet posts in Municipal Affairs, Labour, Trade and Industry and Finance. He chaired a Provincial Task Force on Occupational Health and Safety in the workplace after his retirement from Politics.

Though he dedicated his life to the people, he managed to spend quality time with his family. He is survived by his wife, Marie-Paul and six children, Dr. Michelle, Sydney Mines, Louise (Mrs. Edward Carey)

DR. KENNETH V. GASS

With the passing of Dr. Kenneth V. Gass, age 67, who died on Monday, January 8, 1990 in Pugwash, the medical community of the province and especially the community of Pugwash lost a very loyal supporter.

Dr. Gass was born in Sackville, N.B. He was the son of the late William and Doris (Snowdon) Gass. He was a veteran of the second World War and served in England and Continental Europe. He attended Mount Allison University and received his medical degree from Dalhousie University in 1953. He practised as a family physician in Pugwash for 36 years and he was the only doctor in the area for 22 years.

He had been the past president of the Cumberland Branch of the Medical Society of Nova Scotia and also he was a member of Phi Rho Medical Fraternity. He was the past president and life member of the Royal Canadian Legion in Pugwash.

He is survived by his wife, the former Martha Horne; a son, Dr. K. Greg, Pugwash; a daughter, Julie, Halifax; and a brother, Cedric, Sussex, N.B.

J. Annson B. DeCroos, MD

Sydney Mines, Danielle, North Sydney, Marcel, Halifax, Brian-Paul, Edmonton and Denis, Calgary.

D.P. Hickey, MD ☐

Epidemiology of Child Motor Vehicle-Related Accidents in Nova Scotia, 1983-1987

Andrew S. Worster,* MSc and Lynn McIntyre,** MD, MHSc, FRCPC

Halifax, N.S.

We used the Nova Scotia Department of Transportation's motor accident report form to study the epidemiology of motor vehicle-related accidents among Nova Scotia children. Between 1983 and 1987, there were 12,495 motor vehicle-related accidents in Nova Scotia children under 15 years of age; 2212 (17.7%) accidents resulted in injury, of which 58 (2.6%) were fatal.

The child motor vehicle-related accident rate was 13.4 per 1000 and the motor vehicle-related injury and fatality rates were 23.6/10,000 and 6.6/100,000 respectively. The latter rate is the same as reported for US children. Peak rates occurred in 1985 with a marked decline in fatality rates thereafter. This decrease followed mandatory seatbelt legislation by one year.

Two counties bisected by the Trans-Canada highway each had significantly higher 5 year accident rates ($p < .001$) than the rest of the province. This finding is consistent with widespread publicity about the accident risk on that highway segment.

Accidental death is the most common cause of death in persons aged 1 to 35 years. For the past several decades, accidental injuries have also been the leading cause of childhood deaths in North America.[1-4] Motor vehicle-related accidents (MVRA) also result in severe, non-fatal injuries. Their associated disabilities incur enormous human, financial, and emotional costs in terms of the provision of medical care and rehabilitative services and in loss of future income.[5]

Geographic analyses of mortality from motor vehicle crashes have found rates to be higher in counties of low population density.[6,7] Less frequent seatbelt use, longer distances travelled, higher vehicle speeds and poorer roads have been proposed as possible explanations for rural-urban differences.[8] Children from low income areas have been found to have higher motor vehicle injury rates.[8-10] Between 1979 and 1983, the Province of New Brunswick and rural communities in Quebec and Alberta recorded the highest national rates of motor vehicle traffic fatalities in Canada.[3]

Studies of childhood traffic accident morbidity have gathered data through health interview surveys,[4] injury surveillance programs[11] and hospital monitoring systems.[9,12] Police reports have been found to be useful[9,10,12] in the identification of traffic accident victims but are less valuable in the classification of accident severity.[9]

Since 1983 in Nova Scotia, all police forces have used the Nova Scotia Department of Transportation's MV58A accident report form to record any MVRA resulting in injury to any person involved in the accident or in property damage in excess of $500.00. The MV58A form consists of 56 accident description categories (eg, weather conditions, type of injury) with more than 250 multiple choice responses.

We used the Department of Transportation's data derived from its accident report form to study the epidemiology of motor vehicle-related accidents among Nova Scotia children for the years 1983 to 1987.

METHODS

From data accumulated from MV58A reports for the time period January 1, 1983 to November 22, 1987, the Nova Scotia Department of Transportation provided us with the annual number of motor vehicle-related accidents reported in children under 15 years of age by county and by type of injury. For 1986, we also received the breakdown of accidents by victim class (eg, motor vehicle passenger) and sex.

The *accident rate* was calculated as the number of accidents reported per 1000 population under age 15. The majority of accidents reported involved no personal injury. An injury was recorded if physical harm occurred to a child involved in the traffic accident. It was classified by the reporting police officer as minor (no hospital assessment required), moderate (treated and released), major (hospitalized) or fatal. The *injury rate* was recorded as the number of children injured in a motor vehicle-related accident per 10,000 children under age 15. The *fatality rate* was recorded as deaths per 100,000 children under age 15 years. Fatalities included all victims dying within 30 days of the accident.

The *accident-fatality* rate was calculated as the number of fatalities divided by the total number of accidents. It represents the proportion of accidents resulting in fatality. The *injury-fatality rate* was calculated as the number of fatalities recorded for all accidents which resulted in physical injury. This calculation is used to represent the proportion of reported injuries resulting in death.

We used census information from Statistics Canada to calculate provincial and county accident and injury rates for children under 15 years of age by year (with

*Third-Year Medical Student, Dalhousie University.

**Hospital Epidemiologist, IWK Children's Hospital, Assistant Professor, Dept. of Community Health & Epidemiology, Dalhousie University, Halifax, N.S.

Correspondence: Dr. Lynn McIntyre, Hospital Epidemiologist, IWK Children's Hospital, Box 3070, Halifax, NS, B3J 3G9 (902) 428-8517

adjustments for incomplete reporting in 1987). Statistics Canada also provided us with population density figures (expressed as population per kilometre squared) for Nova Scotia counties.

Statistical analyses were performed using SYSTAT on an IBM-compatible personal computer. The chi-square test was used to determine differences between health region accidents, injuries and fatalities compared to provincial counts. If significant differences were found, county differences were examined. Correlation was analyzed using Pearson's correlation coefficient. The level of significance chosen was $p < .05$.

RESULTS

Over the approximate five year period, there were 12,495 motor vehicle-related accidents in which Nova Scotia children under 15 years of age were involved; 2212 (17.7%) accidents resulted in injury to a child, of which 58 (2.6%) were fatal. For the years 1983-1987, the child motor vehicle-related accident rate was 13.4 per 1000 and the motor vehicle-related injury and fatality rates were 23.6/10,000 and 6.6/100,000 respectively. Figure 1 shows the yearly provincial motor vehicle-related accident, injury and fatality rates for children. Injury rates declined significantly over the period ($p < .001$, chi-square for trend) as did fatality rates after 1985 ($p < .04$, chi-square for trend).
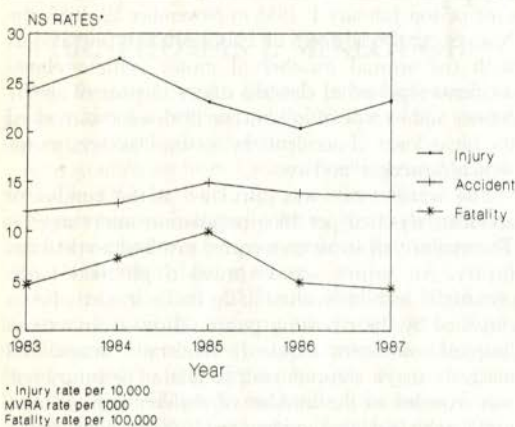
Fig. 1 Nova Scotia Child Motor Vehicle-related Accident, Injury and Fatality Rates, 1983-1987

Table I shows child MVRA rates per 1000 population in Nova Scotia and in the province's six health regions. Cobequid Health Region (Colchester and Cumberland counties) had a significantly higher rate of motor vehicle-related accidents in children than the rest of the province. Fundy Health Region (Hants, Kings and Annapolis counties) had significantly elevated fatality, accident-fatality and injury-fatality rates.

County by county analysis indicated that both Colchester ($p < .0001$) and Cumberland ($p < .002$)

counties independently had higher accident rates than the rest of the province. Cumberland's injury rate of 30.4 per 10,000 children was also significantly elevated ($p < .01$). Year by year analysis showed that the accident rate for both Colchester and Cumberland counties was highest in 1985 but had not changed significantly over the period.

REGION (Counties)	Accident Rate per 1000	Injury Rate per 10,000	Fatality Rate per 100,000	Injury-Fatality Rate (%)	Accident-Fatality Rate per 1000
WESTERN Digby, Yarmouth, Shelbourne	12.5	23.2	8.6	3.7	6.9
FUNDY Annapolis, Kings, Hants	13.0	23.0	13.0*	5.6+	10.0+
COBEQUID Cumberland, Colchester	15.6#	23.3	9.6	4.1	6.2
LUNENBURG-QUEENS-ATLANTIC Lunenburg, Queens, Halifax	13.4	23.3	3.3	1.4	2.4
NORTHUMBERLAND Antigonish, Guysborough, Pictou	13.2	24.3	8.1	3.3	6.1
CAPE BRETON Cape Breton, Richmond, Victoria, Inverness	12.9	24.2	4.1	1.7	3.2
NOVA SCOTIA	13.4	23.6	6.6	2.6	4.6

*$p < .01$, +$p = .009$, #$p < .0001$

Hants county alone accounted for Fundy's elevated rates. Its fatality rate was 21.4/100,000 ($p < .0007$), its accident-fatality rate was 17.9/1000 ($p < .001$) and injury-fatality rate was 10.8% ($p < .01$). All 9 deaths in Hants county were confined to 1984 and 1985.

The male:female ratio of accidental injuries in 1986 was 1.4 and the sex ratio of motor vehicle-related fatalities was 2.0. Figure 2 presents the 1986 distribution of injuries by victim class. Motor vehicle passengers and pedestrians represented 81.4% of known injuries. Nine children were killed in motor vehicle-related accidents in 1986; four motor vehicle passengers, two bicyclists, two pedestrians and one motorcycle driver.

There was no significant correlation between population density or child population size and accident rate, fatality rate, accident-fatality rate, injury-fatality rate or percent of accidents resulting in injury.
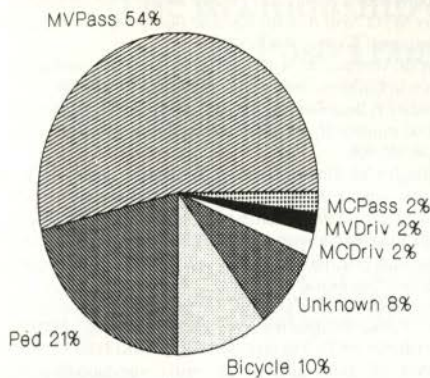
Fig. 2 Distribution of Child Motor Vehicle-related Injuries in Nova Scotia, 1986

DISCUSSION

In 1984, the motor vehicle-related fatality rate in U.S. children less than 15 years of age was 6.6/100,000, the same as Nova Scotia's five year rate.[6] Seventy-five percent of these deaths in U.S. children occurred when they were pedestrians or occupants of a motor vehicle. For 1986, 67% of Nova Scotia deaths occurred when the child was known to be either a pedestrian or motor vehicle passenger.

The 1981 incidence of traffic injuries in children under 15 years of age in Montreal was reported as 33.4/10,000.[9] In our study, the child MVRA injury rate was 41% lower at 23.6/10,000. In Montreal, 57% of injuries were pedestrian whereas in Nova Scotia, 23% of injuries occurred in child pedestrians. The use of police data alone in this study may have underreported child pedestrian injuries. The more rural nature of Nova Scotia with lower pedestrian risk may also be responsible for the difference in injury rate observed.

Mandatory seatbelt legislation, including child restraint provisions, was proclaimed in Nova Scotia on January 1, 1985. Seatbelts have been estimated to be responsible for a 40% to 50% reduction in MVA mortality in the United States.[13] The marked decline in child motor vehicle-related fatalities noted in Nova Scotia, commencing in 1986, may be a delayed result of seatbelt legislation. Information on seatbelt use, available in the Department of Transportation database, may be helpful in addressing the role of seatbelts in changing child MVRA mortality.

The calculation of county child MVRA rates assumes that these accidents occur in the child's resident area. Child pedestrian and bicycling accidents likely occur within a child's resident county. Many investigators have derived MVRA rates based on local population data and it seems reasonable to assume that most travel involving children is within easy driving distance of their homes.[9,12,14,15]

The high accident rates observed in the adjacent counties of Colchester and Cumberland counties may be

related to the fact that these counties connect New Brunswick and Nova Scotia by way of the two-lane Trans-Canada Highway. The public perceives this stretch of highway to be high-risk for accidents (Donna McCready, Information Officer, Nova Scotia Department of Transportation, September 1988; CBC 1st Edition Television News: *Death Alley*, January 25, 1989). It would be interesting to calculate accident rates in adjacent New Brunswick counties.

The comparison of accident rates per miles travelled would be helpful in determining whether or not the increased accident rate is related to higher relative risk or to increased traffic density. Unfortunately, miles travelled are not calculated by the Department of Transportation (Ken O'Brien, personal communication, February 1988).

The higher accident-fatality rate in Hants county is likely related to multiple-victim crashes (eg, schoolbus accident) in two subsequent years. This could be easily verified by search of the MV58A database. It is reassuring that this predominantly rural county had no child motor vehicle-related fatalities and a decrease in its injuries over the last two years of the study.

Curiously, however, Chipman's analysis of 1970 Nova Scotian motor vehicle accident-related hospitalizations identified Fundy and Cumberland regions as having much higher rates of hospital admission of motor vehicle accident victims than was predicted by their accident rates.[12]

The higher male to female ratio of both injuries and fatalities has been observed elsewhere[10,16-18] and has been partially explained by more risk-taking activities by boys[18] and greater exposure of boys to risky environments.[2,8]

The child MVRA rates of Nova Scotia's only two urban counties, Cape Breton and Halifax, were remarkably similar to provincial rates. This study could not demonstrate a rural-urban difference in accident rates perhaps because of the highly rural nature of the province.

The MV58A report injury classification scheme provides insufficient information about the nature of the injury to identify risk factors for various outcomes. Pless combined police reports and emergency room and hospital records to study the epidemiology of medically attended childhood MVRAs.[9] In order to obtain complete and reliable medical data on injuries, a medical counterpart to the MV58A should be developed and its completion by the attending physician in hospital should be required by Department of Transportation regulation.

CONCLUSION

Continuing collaboration with the Nova Scotia Department of Transportation is necessary to answer some of the questions raised by this study and to identify modifiable risk factors for accidents in children which may lead to the implementation of effective preventive measures. The database generated by the MV58A report

form appears to have enormous potential for the study of child MVRAs. Similar forms are used in Saskatchewan and New Brunswick and may be equally useful in these provinces. ☐

ACKNOWLEDGEMENT

We thank Dr. Barry Pless for his review of an earlier version of this manuscript.

References

1. Millar WJ, Last JM. Motor vehicle traffic accident mortality in Canada, 1921-1984. *Am J Prev Med* 1988; **4**: 220-30.
2. Baker SP. Injuries: the number one threat to our children. *Dimensions Health Serv* 1985; **62**: 30-32.
3. MacWilliam L, Mao Y, Nicholls E, Wigle DT. Fatal accidental childhood injuries in Canada. *Can J Public Hlth* 1987; **78**: 129-135.
4. Klauber MR, Barrett-Connor E, Hofstetter Cr, Mick SH. A population-based study of nonfatal childhood injuries. *Prev Med* 1986; **15**: 139-149.
5. World Health Organization. *The epidemiology of accident traumas and resulting disabilities*. Report on a WHO symposium. Euro Reports and Studies 57, Regional Office for Europe. Copenhagen: World Health Organization, 1982: 20-26.
6. Centers for Disease Control. *Deaths from motor vehicle-related injuries, 1978-1984*. In: CDC Surveillance Summaries, February 1988. *MMWR* 1988; **37** (No. SS-1): 5-12.
7. Waller AE, Baker SP, Szocka A. Childhood injury deaths: National and geographic variations. *Am J Public Health* 1989; **79**: 310-315.
8. Baker SP, O'Neill B, Kapf RS. *The Injury Fact Book*. Lexington, MA, Lexington Books, 1984: 195-218.
9. Pless IB, Verreault R, Arsenault L et al. The Epidemiology of Road Accidents in Children. *Am J Public Hlth* 1987; **77**: 358-360.
10. Rivara FP, Bergman AB, LoGerfo JP, Weiss NS. Epidemiology of childhood injuries: II, sex differences in injury rates. *Am J Dis Child* 1982; **136**: 502-506.
11. Gallagher SS, Finison K, Guyer B, Goodenough S. The incidence of injuries among 87,000 Massachusetts children and adolescents: Results of the 1980-81 Statewide Childhood Injury Prevention Program Surveillance System. *Am J Public Health* 1984; **74**: 1340-47.
12. Chipman ML. Hospitalization after motor vehicle accidents in Nova Scotia. *Can J Public Health* 1973; **64**: 133-140.
13. Centers for Disease Control. *Occupant restraint usage in fatal crashes — Fatal accident reporting system, 1975-1986*. MMWR 1987; **36**: 636, 641-3.
14. Baker SP. Determinants of injury and opportunities for intervention. *Am J Epidemiol* 1975; **101**: 1.
15. Copeland AR. Accidental childhood deaths: the metropolitan Dade County experience, 1978-82. *Am J Forens Med Path* 1986; **7**: 101-103.
16. Fatal accidents among school-age children, *Statistical Bull* 1981; (July-Sept): 10-12.
17. Fatal accidents at the preschool ages. *Statistical Bull* 1982; (Oct-Dec): 12-15.
18. Gofin R, Palti H, Adler B, Edet E. Childhood injuries: a population-based study of emergency room visits in Jerusalem. *Paed Perinat Epidemiol* 1989; **3**: 174-88.

The Relationship of Motor Vehicle Accidents to Post-Traumatic Stress Disorder

Brian D. Dufton,* PhD

Kentville, N.S.

This article briefly describes the syndrome of PTSD in order to facilitate its recognition and appropriate referral. At present, the prevalence of PTSD associated with motor vehicle accidents is unknown. However, it seems likely that family physicians are in an ideal role to detect this disorder.

An individual has been in a severe car accident. He or she has recovered physically but now presents to the family doctor with a bewildering array of distressing psychological sequelae. For example, he or she is frequently bothered by thoughts and nightmares about the accident. As well, he or she avoids reminders of the latter and seems withdrawn. He or she also reports angry outbursts, trouble sleeping, and marked distractibility. In summary, this person has experienced an extremely distressing incident, and is subsequently suffering from cognitive "re-experiences" of the event, numbing of emotional and social responsiveness, and symptoms of over-arousal.[1]

He or she thus likely fulfills the diagnostic criteria for post-traumatic stress disorder (PTSD), first described in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) in 1980. This disorder has been the subject of both media attention and research, particularly with regard to the effects of "spectacular" tragic events. Research subjects, for example, have included school children after their playground was the scene of a sniper attack,[2] firefighters following intense exposure to a disastrous brushfire,[3] Lebanese University students following the Israeli invasion,[4] and, most notably, Vietnam combat veterans.[5,6,7] Yet more commonplace events can also give rise to PTSD and this is explicitly acknowledged in DSM-III. Participants in motor vehicle accidents, then, as described earlier, may exhibit symptoms associated with PTSD. It is not unlikely, therefore, that family physicians deal with cases of PTSD on a somewhat regular basis.

Patients with motor-vehicle-accident-related PTSD are often difficult to treat. A major hindrance is misdiagnosis. For example, patients may be seen as simply being "oversensitive". Alternatively, where litigation or insurance claims are involved, patient's complaints may be seen as exaggeration to amplify compensation. As well, somatic symptoms may be confused with the sequelae of neurological or physiological problems potentially resulting from the accident. A second hindrance to treatment is the ingrained nature of PTSD; it rarely responds to reassurance or sedative medication. This article first describes the psychological assessment and treatment of an individual with accident-related PTSD, and, second, suggests some guidelines when confronted with such a case.

CASE EXAMPLE

This forty year old man was referred to a local Mental Health Clinic by his family physician because of difficulties coping with the outcome of a motor vehicle accident a year before in which, of his two passengers, one died and the other lost the use of his right arm. The patient himself received a slight concussion but was otherwise unhurt. The accident occurred as a result of another car pulling out into an intersection and no blame was affixed to the patient. An assessment was conducted via an interview and standardized psychological questionnaires.

RESULTS OF ASSESSMENT

The psychometric evaluation was reflective of moderate depression and marked anxiety. He reported significant guilt about the accident despite being told by the RCMP officer that there had been nothing he could have done to have avoided the other vehicle. He described highly frequent and intrusive thoughts and feelings about the accident, which he attempted to block out from his awareness. He found that he was quite irritable with his children, had trouble sleeping, suffered from severe headaches, and had little energy. He had a very strong sense of the fragility of life and, as a consequence, wanted to finish a number of woodworking projects as a legacy before "something" might happen.

His distress was greatly increased by reminders of the accident such as driving near the site (he generally avoided doing so if he could help it), seeing the family of the man who had died, and seeing the injured man. The subsequent legal proceedings were extremely upsetting to him, particularly as opposing lawyers attempted to have him take partial responsibility for the accident. Thinking about future court involvement, therefore, was also quite upsetting for him. In general he felt little able to cope with even minor life stressors. This was in contrast to his life prior to the accident. He described himself as having been a cheerful man who got along

*Psychologist, Cornwallis House, Psychiatric Day Centre, Valley Health Services Association.

Correspondence: Dr. Brian Dufton, Cornwallis House, Psychiatric Day Centre, Valley Health Services Association, 100 Exhibition St., Kentville, N.S. B4N 1C4

well with others. From his description he still had a supportive marriage and good relationships with his children. Based on this assessment he was given a diagnosis of PTSD.

COURSE OF TREATMENT

This patient was seen over twenty-one one-hour sessions as an individual and on one occasion with his wife. The couple session was conducted to provide information to his spouse as to the nature of PTSD, to gain her view as to his functioning, and to determine the impact of PTSD on the couple as a unit. Individual treatment consisted of:

a) empathic psychotherapy (a method of supportive listening) to establish a supportive and trusting therapeutic alliance in which he could feel accepted and understood;

b) relaxation training to reduce somatic over-arousal;

c) cognitive therapy (a method of psychotherapy which emphasizes the role of thinking in controlling emotions) to help him gain a more realistic and adaptive perspective on the accident;

d) coping skills training (systematic rehearsal of coping strategies such as relaxation, positive thoughts and images in response to stressors) to help him gain greater control of his distress and to begin to deal with avoided reminders; and

e) education as to the nature of PTSD to help normalize his behaviour and reduce his anxiety about his condition.

Progress was gradual with periodic set backs. For example, following the death of a family pet, he re-experienced much of his acute distress even though he had been doing quite well prior to the event. Over time, nonetheless, he learned to be less self-blaming and less fearful when thinking about the accident. Moreover, he learned to recognize stressors likely to produce increased distress. A standardized post-treatment assessment was reflective of greatly reduced symptomatology.

SOME GUIDELINES

Clearly, many physicians will have neither the time nor the training to implement psychological interventions to treat PTSD in their patients. There are, however, some guidelines which may prove useful.

Recognize PTSD. If a patient has experienced a trauma and subsequently reports the symptomatology described here, then it may help both the physician and the patient to recognize what is going on; that is, the patient is not going "crazy" but is experiencing a predictable psychological pattern.

Be supportive. These patients do not need judgement. They need to be able to talk about their feelings with family and friends as well as with professionals. Family physicians are extremely important in this regard as they are often the most accessible professionals.

Watch for resurgence. Symptoms of PTSD tend to be much greater around anniversary dates of the trauma. As well, other reminders can serve to produce a resurgence of the pattern. Thus, if a patient is presenting with renewed difficulty, there is a good chance that something has produced re-traumatization. The family physician is often in a position to place the symptoms in context.

Refer. Patients with PTSD can often benefit from psychological approaches as offered by mental health professionals. The latter do not replace the support of the general practitioner but rather they offer unique expertise in the treatment of trauma-based psychological problems. □

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, D.C.: Author, 1980.
2. Pynoos RS, Frederick C, Nader K, Arroyo W, Steinberg B, Spencer E, Nunez F, Fairbanks L. Life threat and post-traumatic stress in school-age children. *Arch Gen Psychiatry* 1989; 44: 1057-1063.
3. McFarlane AC. The aetiology of post-traumatic stress disorders following a natural disaster. *Brit Psychiat* 1988; 152: 116-121.
4. Saigh PA. Pre and postinvasion anxiety in Lebanon. *Behavior Therapy* 1984; 15: 185-190.
5. Pitman RK, Orr SP, Forgue DF, deJong JB, Claiborn JM. Psychophysiologic assessment of post-traumatic stress disorder imagery in Vietnam combat veterans. *Arch Gen Psychiatry* 1987; 44: 970-975.
6. Fairbank JA, Keane TM, Malloy PF. Some preliminary data on the psychological characteristics of Vietnam veterans with post-traumatic stress disorders. *Consult Clin Psychol* 1983; 51: 912-919.
7. Keane TM, Wolfe J, Taylor KL. Post-traumatic stress disorder: Evidence for diagnostic validity and methods of psychological asessment. *Clin Psychol* 1987; 43: 32-43.
8. Watson IPB, Hoffman L, Wilson GV. The neuropsychiatry of post-traumatic stress disorder. *Brit Psychiat* 1988; 152: 164-173.

Serious Adult Head Injury in Nova Scotia
AN EPIDEMIOLOGIC REVIEW

David S. Malloy,* MD,FRCSC

Halifax, N.S.

Traumatic Brain Injury is a leading cause of death and disability. To better define the nature of such injuries in Nova Scotia, an epidemiologic review of serious head injury has been undertaken. During a 37 month period 150 adult patients with serious head injuries were admitted to the neurosurgical service at the Victoria General Hospital. Numerous parameters were assessed which have allowed for the development of recommendations for the prevention of these potentially devastating injuries.

It has long been recognized that Traumatic Brain Injury is a frequent occurrence.[1] Such injuries are associated with significant mortality and morbidity.[2,3,4] In many cases the consequences of this form of trauma are devastating. The disabilities produced place tremendous emotional and psychological strains on the survivors and their families. The stress applied to the health care system, in economic terms, is also high.

Most statistics concerning head trauma are derived from large American or European studies.[5,6] To our knowledge there has not been a recent review of head injury in Nova Scotia. Over the past several years we have collected a large database for each of our head-injured patients. This serves as the basis for this epidemiologic review. It will also serve as a source of data for further reviews of patient care and outcome.

For the purposes of this epidemiologic review we chose to study "serious" head injuries which occurred in our referral or catchment area. We have defined "serious" head injury as any injury a) associated with a Glasgow Coma Scale score of 8 or less after non-surgical resuscitation; b) requiring neurosurgical intervention; or c) necessitating admission to the tertiary care neurosurgical service for more than 48 hours.

We have excluded three patients admitted to hospital solely for the purpose of organ donation (all self-inflicted gunshot wounds). The period under study covers 37 months beginning in June 1986 and terminating at the end of June 1989.

Patients in this review were admitted to the neurosurgical service at the Victoria General Hospital in Halifax. No patient under the age of 15 is included. The Victoria General Hospital serves as the tertiary adult neurosurgical centre for the province of Nova Scotia. It provides facilities for operative care and neurosurgical intensive care. The hospital also functions as the primary centre for the Metropolitan Halifax region.

RESULTS

Patients were analyzed with respect to a number of variables. These included age, sex, severity of injury, mechanism of injury, seasonal occurrence, presence of multisystem trauma and mortality.

Incidence

During the 37 months of the study 150 patients were entered. The number of patients was fairly constant for each of the one year periods. 42 patients were entered in the first 13 months followed by 53 and 55 respectively in the two subsequent 12 month intervals. Overal the annual incidence of "serious" head injury was 7.3 per 100,000 population. The annual incidence of "serious" head injury in the Metropolitan area was almost identical to that of the province as a whole (7.4/100,000).

During the period of our study, a total of 552 patients were admitted to the Victoria General Hospital with head injuries. Included in that number are the 150 patients in this review. Ninety nine were from outside the Metropolitan region leaving 453 local patients. The annual incidence of head injury requiring hospital admission in the Halifax area is calculated to be 64 per 100,000. If we extrapolate this figure to the province we estimate the annual incidence of head trauma necessitating admission to be about 440 cases. Assuming a pre-hospital mortality of 15% (based upon US statistics) there are approximately 520 head injuries each year in Nova Scotia. This translates to 75 cases per 100,000. Roughly 89% of patients reaching hospital will have "Mild" injuries while 5% and 6% respectively are "Moderate and Severe" (according to GCS).[7]

Age/Sex Distribution

Males dominate the head injury population in this review. 124 patients were male (82.3%). The male: female ratio was 4.8:1.

Head-injured patients in this review were young, the majority less than 34 years. Table I illustrates the age and sex data for our study group. Figure 1 illustrates the incidence rates per 100,000 population. Figure 2 shows the frequency of injury in each age/sex group. The mean age in the entire group was 30.9 years.

Mechanism of Injury

The activities leading to head injury are outlined in Table II. Motor vehicle accidents were the leading

*Assistant Professor, Department of Neurosurgery, Dalhousie University, Halifax, N.S.
From the Department of Neurosurgery, Victoria General Hospital, Halifax, N.S.

mechanism (46.7%). All vehicular trauma (including vehicle-pedestrian accidents) accounted for 68.7% of the patients. "Other" injuries included patients struck by objects (falling trees, marine equipment, hockey pucks). (See Figure 3).

TABLE I

AGE/SEX DISTRIBUTION

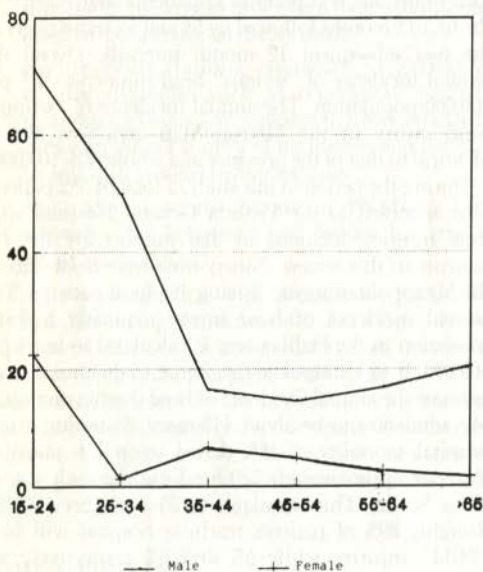| Age	Male	Female	Total
15-24	56	17	73
25-34	37	1	38
35-44	10	4	14
45-54	6	2	8
55-64	6	1	7
>65	9	1	10
	124	26	150

Age Distribution
/100,000

Figure 1

In the youngest age group (15-24) vehicle related trauma accounted for 83.6% of all injuries. Older patients were more likely to be victims of falls (41.2% of all patients >55). Assaults were most common in the 25-34 year old group. Assaults were an exclusively male injury. Of the two gunshot wounds, one was self-inflicted (see Figure 4).

Location of Injury

Patient records were analysed to determine the institution in which they received their primary resuscitation. We then classified the patients based upon usual travel time from each of these hospitals to our

Age/Sex Distribution
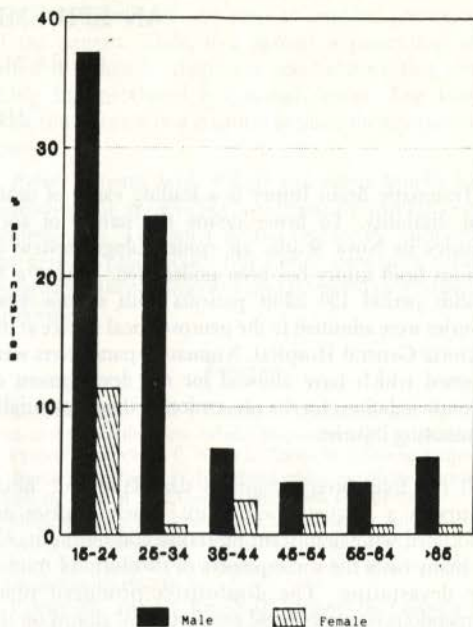
Figure 2

TABLE II

MECHANISM OF INJURY

Mechanism of Injury	Total	Male	Female	%
MVA	70	57	13	46.7
MCA	13	11	2	8.7
ATV	4	2	2	2.7
Bicycle	2	2	0	1.3
Pedestrian	14	8	6	9.3
ALL VEHICULAR	103	80	23	68.7
Fall	20	18	2	14.0
Assault	12	12	0	8.0
GSW	2	2	0	1.3
Other	13	12	1	8.7

TABLE III

LOCATION OF INJURY

Other System	Frequency
Metropolitan	52 (34.7%)
<1 hr. transport	1 (0.7%)
1-2 hr. transport	39 (26%)
2-3 hr. transport	21 (14%)
>3 hr. transport	29 (19.3%)

facility. Table III indicates the distances from the tertiary care centre where injuries occurred. Almost 2/3 of our patients suffered their injuries at least one hour away from the Neurosurgical centre.

Mechanism of Injury

Vehicular 68.7
Other 8.7
Assault/GSW 9.3
Fall 14

Figure 3

Mechanism of Injury

- ■ Vehicular
- ▨ Falls
- ▢ Assault/GSW
- ▢ Other

Figure 4

TABLE IV

MULTISYSTEM TRAUMA

Other System	Frequency
Facial	39 (49.4%)
Orthopedic	38 (48.1%)
Thoracic	23 (29.1%)
Spinal	12 (15.2%)
Abdominal	6 (7.6%)
Renal	6 (7.6%)
Cardiac	2 (2.5%)
Vascular	1 (1.3%)

Seasonal Distribution

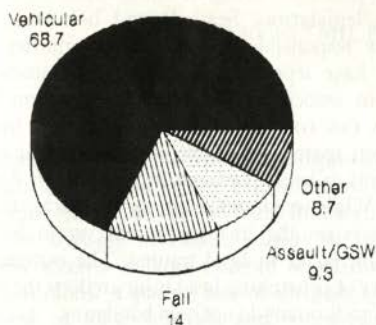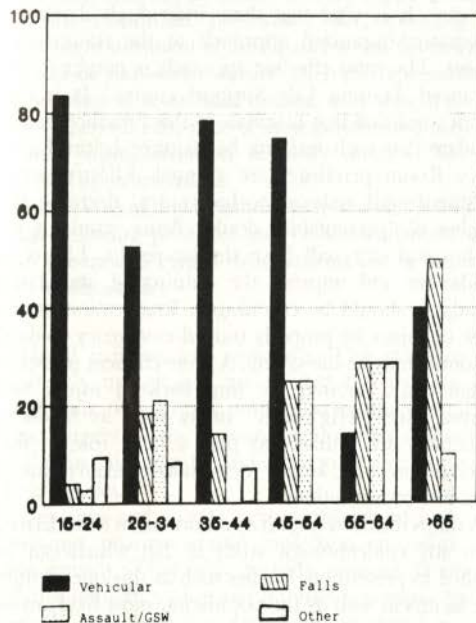As noted earlier, the annual incidence of serious head injury remained fairly constant. There was a marked increase in cases during the summer months 44.7% of our patients were injured in June, July and August. As Figure 5 illustrates, July and August were the busiest months with respect to head injury.

Seasonal Distribution

Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec
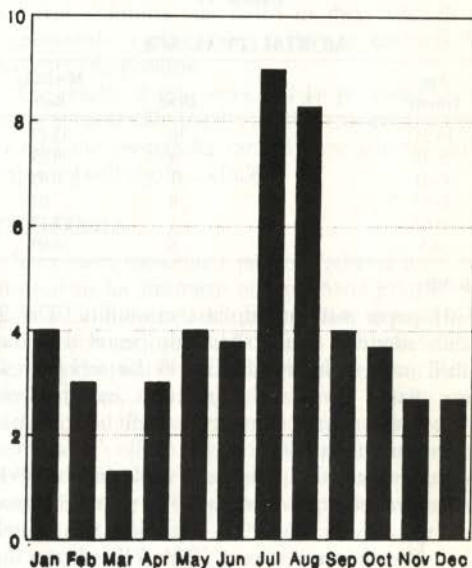
Figure 5

Severity of Injury

Injury severity was defined using the Glasgow Coma Scale.[7] GCS of 8 or less implies severe head injury, usually associated with the highest incidence of mortality and morbidity. Eighty-two patients had GCS scores of 8 or less after non-surgical resuscitation (54.7% of all patients). Fourty-four of these individuals were in the most severely injured group with GSC scores of 3-5. Thirty-four patients had "mild" injuries and an equal number were "moderately" injured. As noted earlier it is estimated that almost 90% of patients reaching hospital will have "mild" injuries. The larger proportion of moderate and severe injuries in this study reflects our definition of "serious" head trauma.

Other Injuries

Seventy-nine patients (52.7%) suffered injuries to other body regions in addition to the brain. Table IV illustrates the frequency with which other areas were traumatized. Facial and orthopedic trauma (long bone and pelvic fractures) were most common. Table V illustrates the relationship between mechanism of injury and the presence of multi-system trauma. Multiple injuries were observed in 2/3 of all patients with vehicular trauma.

TABLE V

MULTISYSTEM TRAUMA IN RELATION TO MECHANISM

Mechanism	Frequency
Vehicular	66.0%
Fall	19.1%
Assault/GSW	21.4%
Other	30.8%

TABLE VI

MORTALITY vs. AGE

Age Group	N	Dead	Mortality Rate
15-24	73	10	13.7%
25-34	38	4	10.5%
35-44	14	1	0.8%
45-54	8	0	0%
55-64	7	1	1.3%
>65	10	5	50.0%

Outcome

This paper will not discuss morbidity. The 21 patients admitted during the study period who died resulted in a mortality rate of 14% for serious head injury. Based upon our population and incidence estimates the overall mortality for all head injuries reaching hospital is 1.6%.

Mortality was noted to increase with age (Table VI). The mean age of those dying was 37.4 years. The mean age for survivors was 29.8. Mortality was closely associated with injury severity. Table VII indicates that mortality in the most severely injured group (GCS 3-5) was 34.1%. In the GCS-defined severe group death occured in 22%. The single death in the mild group (GCS 13-15) occurred in an elderly patient suffering cardiothoracic complications four weeks after multisystem injury.

TABLE VII

MORTALITY vs. SEVERITY

GCS	N	Dead	Mortality Rate
13-15	34	1	2.9%
9-12	34	2	5.9%
6-8	38	3	7.9%
3-5	44	15	34.1%

DISCUSSION

One of the striking results of this review is the relatively low annual incidence of head trauma in comparison with other reports. It has been widely noted that the annual incidence of traumatic brain injury approximates 200/100,000 population.[1] Our estimates suggest a yearly frequency of around 75 cases per 100,000. The lower incidence of head trauma in Nova Scotia is likely a reflection of several factors.

Nova Scotia is a sparsely populated "rural" province. Common sense would seem to dictate that more densely populated regions are more likely to experience higher rates of trauma. Nova Scotia has recently embarked upon a course of stringent enforcement of impaired driving legislation. Seatbelt and helmet laws are probably responsible for fewer head injuries. Other authors have reported a decrease in traumatic brain injury in association with such legislation.[8] Nova Scotians can consider themselves fortunate that they have been spared the degree of interpersonal violence that seems to be so prevalent in large urban American centres. While recent trends appear to indicate that such violence is on the increase, it has yet to become a significant factor in head trauma. The extremely low incidence of penetrating head injury reflects the fact that most Nova Scotians do not own handguns.

Since Nova Scotia is a relatively "rural" province it is not surprising that a large proportion of our patients were injured at locations quite remote from the tertiary care centre. The implications of this observations are obvious. Medical practitioners in the outlaying centres will most frequently be the providers of acute resuscitative care. It is vital that these individuals develop a systematic, organized approach to the traumatized patient. The most effective approach is taught in the Advanced Trauma Life Support course.[9] It is this author's opinion that hospitals in this province should mandate that such training be required before Emergency Room privileges are granted. Effective early treatment will reduce morbidity and decrease the number of "preventable" deaths. Better standards for pre-hospital care will have similar results. Efforts to standardize and improve the training of ambulance attendants should be encouraged. Intervention at the scene of injury by properly trained emergency medical personnel may be life-saving. A more efficient transport system could shorten the time between injury and trauma care. "High-tech" items such as Medevac helicopters are unlikely to play a large role in this province, however, largely (and understandably) due to economic considerations.

Perhaps the most important information to be derived from any epidemiologic study is that which can be applied to prevention. Studies such as this one identify risk factors as well as the population most likely to be affected by them. Clearly, those at highest risk for traumatic brain injury are young males aged 15-24. Equally clear is the fact that the most dangerous activities are those which involve motor vehicles. This reviewer suggests that formal driver education should be mandatory. Exposure to the consequences of head injury might prove to be a sobering part of such education. Strict enforcement of impaired driving laws, helmet laws and seatbelt legislation is essential. The argument that the latter infringes upon civil rights is ludicrous to those of us who deal with the carnage of highway trauma. Enforcement should be particularly stringent during the summer months when head injury is most frequent.

A Review of Pre-Transfer Care of Acute Hand Injuries in Nova Scotia

J.F. Watkins,* MD, FRCSC and W.S. Parkhill,** MD, FRCSC

Halifax, N.S.

Splinting is basic to the first aid, transportation and subsequent care of most injured extremities. As the only tertiary care referral service for hand injuries in Nova Scotia, the Division of Plastic Surgery at Dalhousie University receives a large number of referrals from other institutions. A general lack of adequate splinting was noted in these referrals and, consequently, a prospective study was undertaken to document the problem.

All outside referrals for acute hand injuries seen by the authors in the Victoria General and IWK Hospital emergency rooms between July 1, 1988 and January 1, 1989 were included in this review. The type of injury, referring institution, pre-transfer care, splinting and subsequent treatment of these patients was documented. A total of sixty-seven outside referrals, representing a broad range of acute hand trauma, were seen in the six-month period. Fifty-nine of these patients required pre-transfer immobilization yet only seven (12%) were splinted upon arrival.

The results of this series will be reviewed, the basics of hand splinting presented and recommendations as to how pre-transfer care of these patients can be improved will be discussed.

Hand injuries are common. The majority are relatively minor and are treated by primary care physicians; only a small proportion require referral to a hand surgery service. Dressings and splints are essential in the first-aid, transfer and subsequent care of most of these injuries.

Dressings and splints serve a number of functions. Dressings provide wound protection, antisepsis and debridement. They also provide pressure if needed, relative immobilization and improved cosmesis. Splints provide immobilization and facilitate elevation. As a consequence, the risk of further injury or contamination is reduced, a functional position is maintained and most importantly the patient's comfort in the acute phase of the injury is greatly enhanced.[1,2] The majority of hand injuries, therefore, require some form of immobilization as part of their initial management.

As the only tertiary care referral service for hand injuries in Nova Scotia, the Division of Plastic Surgery

at Dalhousie University receives a large number of referrals from other institutions. A general lack of adequate splinting was noted in these referrals and consequently a prospective study was undertaken to document the problem.

The results of this series will be presented and the basics of hand splinting discussed. Recommendations as to how the pre-transfer care of these patients can be improved will also be included.

THE SERIES

Sixty-seven consecutive patients, referred from other institutions for treatment of acute hand injuries, were included in the series. All patients were assessed by the authors between July and December 1988 at the Victoria General Hospital or IWK Hospital Emergency Rooms. Major burns and multiple trauma patients were excluded. Ambulatory clinic referrals and patients sent from doctor's offices or radiology departments were also excluded. Telephone advice on splints and dressings was not given for the period of the review unless requested by the referring physician.

METHOD

Standarized data sheets were completed upon initial evaluation of each patient. The following information was specifically documented: nature of injury, institution of referral, presence and type of dressings and splints, tetanus immunization status, administration of analgesics, and the transfer of X-rays and referral documents with the patient. The subsequent treatment of these patients was recorded. An assessment of the severity of hand injury and the need for pre-transfer immobilization was made by the authors and recorded.

RESULTS

Between July 1, 1988 and December 31, 1988, the authors received 67 outside referrals for acute hand injuries at the Victoria General and IWK Emergency Rooms. In the authors' opinions, 59 of these patients required immobilization yet only 7 (12%) were splinted prior to transfer. (Table I) The majority of those injuries splinted were fractures. There was a conspicuous lack of splinting of soft tissue injuries such as flexor and extensor tendons, digital nerve, and muscle lacerations. With respect to the Health Unit from which the patient was referred, there was a slightly higher incidence of splinting in the distant as opposed to local referrals; however, the overall percentage of appropriately splinted injuries was low in both groups.

*Clinical Fellow of Plastic and Reconstructive Surgery, Dalhousie University.
**Associate Professor, Department of Surgery, and Head, Plastic Surgery Dalhousie University.
From the Division of Plastic Surgery, Department of Surgery, Dalhousie University, Halifax, N.S.

TABLE I

Injury	Number	Splint Required	Splinted Upon Arrival
Flexor Tendon(s)			
Laceration(s)	8	8	0
Extensor Tendon(s)			
Laceration(s)	7	7	0
Metacarpal Fractures	6	6	2
Phalangeal Fractures	6	6	0
Finger Tip Injuries			
with fracture	4	4	1
without fracture	5	0	0
Amputations (no Finger Tip)			
complete	1	0	0
incomplete	3	3	1
Wrist Laceration(s)			
multiple structures	4	4	1
Finger Fracture — dislocations, ligament injuries	5	5	1
Infections	4	4	0
Nerve Lacerations (isolated)	4	4	0
Boxer's Fractures	4	4	1
Major Soft Tissue Injuries	2	2	0
Minor Soft Tissue Injuries	2	0	0
Forearm Muscles or Tendon Lacerations	2	2	0
Totals	**67**	**59**	**7**

Although there was a general lack of pre-transfer splinting of acute hand injuries, a number of positive aspects of the care of these patients was documented. All referring physicians discussed the patient with the accepting service prior to transfer. Each patient arrived with a referral letter, emergency room notes and X-rays when appropriate. Only one patient in the series was transferred with inadequate tetanus immunization. Dressings were universally applied to open wounds with only one dressing being constructed from non-sterile material. Antibiotics and analgesics were commonly administered when appropriate. All patients were transferred expeditiously to the accepting service.

The only widespread deficiency in the initial treatment of these acutely injured patients related to the general lack of appropriate immobilization.

DISCUSSION

The prospective series documents a major deficiency in the pretransfer care of acute hand injuries in Nova Scotia. In a six-month period, only 12% of these patients referred to the authors that required a splint were appropriately immobilized.

The benefits of immobilization were well accepted. Immobilization aids in elevation, protects the hand from further injury, maintains a functional position and greatly enhances patient comfort. The only acute hand injuries not requiring initial immobilization are minor lacerations not involving underlying structures, minor soft tissue injuries, complete amputations and stable finger tip injuries not associated with fractures.

The principles of hand dressings and splints are not complex and the materials for these are readily available in all emergency rooms. A simple hand dressing should consist of a thin sterile permeable non-adherent layer next to the wound. Commonly, a tulle gauze sheet coated with an ointment such as Vaseline serves the purpose. These wide mesh gauze sheets are available with or without impregnated antibiotics. The initial layer is followed by a sterile absorbent layer such as fluffed gauze or absorbent cotton. Web spaces are lightly padded with this material to prevent intertigo when adjacent digits are bandaged. A non-sterile wrap of "Kling" gauze or, rarely, an elastic bandage, secures the dressing.[3] Finger tips should be left exposed where possible to allow monitoring of circulation.

Splints are readily fabricated from casting material and, when appropriate, are incorporated into the outer wrap of the dressing. Aluminum splints and buddy-taping are occasionally used for isolated digital injuries. Splints are generally applied in the "safe" or protective position (Figure 1) and except for some isolated digital injuries, the wrist should be immobilized with the hand. The safe or protective position implies moderate wrist extension, thumb extension and abduction, metacarpal-phalangeal joint flexion at 60° to 90° and slight interphalangeal joint flexion at no more than 5° to 10°.[4,5] Usually, a volar plate slab or thumb spica are used to splint the hand in the safe position. Figures 2 and 3 illustrate these splints. It should be noted that some injuries such as flexor tendon lacerations are immobilized in a position other than the safe position after definitive repair; however, when first seen, these injuries are still most appropriately immobilized in the safe position.
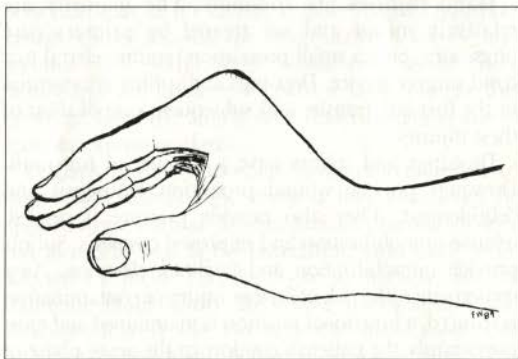

Fig. 1 "Safe" Position

Despite the clear benefits and relative simplicity of hand dressings and splints, the series documents a deficiency in their use in acute hand injuries prior to transfer in Nova Scotia. Several recommendations can be made in hopes of improving this situation. All accepting physicians should instruct the referring physician, to ensure that appropriate dressings and splints are applied prior to transfer.
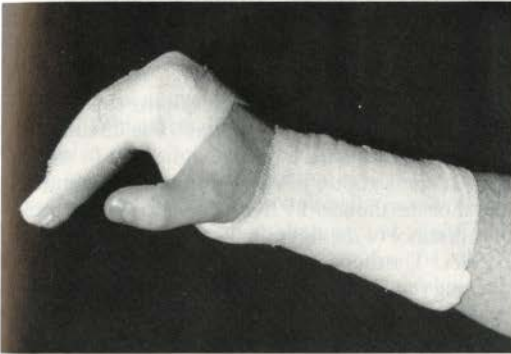
Fig. 2 Thumb Spica

Fig. 3 Volar Slab with hand in Safe Position

Referring physicians should check all these patients prior to transfer, especially if the application of dressings and splints were delegated to other emergency room staff. Medical education at all levels should emphasize the first aid, assessment and pre-transfer care of these patients, as well as their definitive treatment. Finally, all physicians involved in the care of these injuries should adopt the attitude that short-term splinting in the safe position is never detrimental in the acute stage of a hand injury.

If these recommendations result in a more widespread application of appropriate dressings and splints to acute hand injuries; patient comfort, subsequent treatment and ultimate rehabilitation of these injuries will be significantly enhanced. □

References

1. Beasley, RW. *Hand Injuries*, Philadelphia: W.B. Saunders Co., 1981, pages 66-94.
2. Noe, JM. In *Acute Hand Injuries*, Walfort, F.G., Boston: Little Brown and Co., 1980, pages 241-248.
3. Flatt, AE. *The Care of Minor Hand Injuries*, 3rd ed., Saint Louis: C.V. Mosby Co., 1972, pages 37-42.
4. Tajima, T. Treatment of Post Traumatic Contracture of the Hand, *J Hand* Surg 1988; **13-B:** 118-129.
5. O'Brien, ET. *Fractures of the Metacarpals and Phalanges, in Operative Hand Surgery*, 2nd ed., Green, DP, ed., New York: Churchill Livingstone, 1988, page 745.

## SERIOUS ADULT HEAD INJURY IN NOVA SCOTIA
An Epidemiologic Review

The medical profession and the health care system need to take leading roles in the prevention of head injury. We must continually strive to get our message out to those at the greatest risk. Prevention is the key to reducing the mortality and morbidity associated with traumatic brain injury. □

References

1. Kraus JF. Epidemiology of Head Injury. In: Cooper PR, ed. *Head Injury*. Baltimore: Williams and Wilkins, 1987: pg 1-19.
2. Becker DP, Miller JD, Ward JD, Greenberg RP, Young HF, Sakalas R. The outcome from severe head injury with early diagnosis and intensive management. *J Neurosurg* 1977; 47:491-502.
3. Bowers SA, Marshall LF. Outcome in 200 consecutive cases of severe head injury treated in San Diego County. *Neurosurgery* 1980; 6:237-242.
4. Jennett B, Teasdale G, Braakman R, Minderhoud J, Heiden J, Kurze T. Prognosis of patients with severe head injury. *Neurosurg* 1979; 4:283-289.
5. Jennett B, Teasdale G, Galbraith S, et al. Severe head injury in three countries. *J Neurol Neurosurg Psychiatry* 1977; 40:291-298.
6. Klauber MR, Marshall LF, Barnett-Conner E. Prospective study of patients hospitalized with head injury in San Diego County, 1978. *Neurosurg* 1981; 9:236-241.
7. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: A practical scale. *Lancet* 1974; 2:81-84.
8. McSwain NE, Lummes M. Impact of repeal of motorcycle helmet law. *Surg Gynecol Obstet* 1980; 151:215-224.
9. American College of Surgeons. Advanced Trauma Life Support Course.

COMMON COURTESY IS JUST NOT COMMON ENOUGH
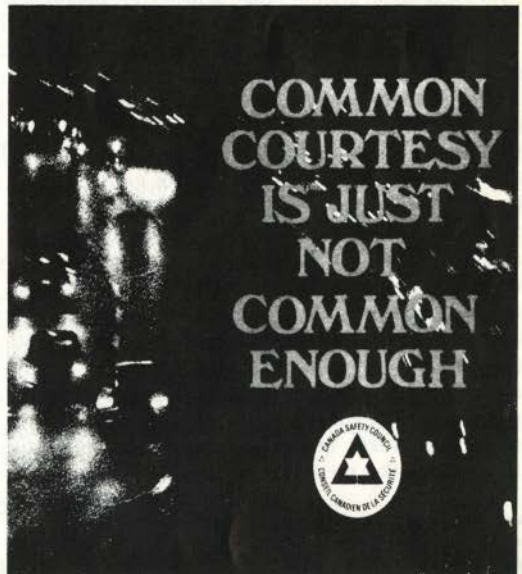
Lightning Injuries

Ted Bachynsky, *BSc (Hon) and Ian R. Morris, **BEng, MD, FRCPC (Anaes & EM)

Halifax, N.S.

It is difficult to determine the incidence, morbidity and mortality associated with lightning strike as the reporting of such incidents is not required[1] and there is no standardized nomenclature.[2] However, it is estimated that in the United States, 150-250 deaths occur annually, more than from any other natural disaster.[1] Most individuals who are struck by lightning (70-80%) survive[2]; usually with some permanent sequelae.[3] Most deaths and injuries due to lightning strike occur among outdoor sports enthusiasts, notably golfers, campers, and joggers.[14] As expected, almost all of the injuries (90%) occur during afternoons between May and September when people tend to be found outdoors.[4]

PHYSICAL PROPERTIES

Lightning is usually, but not always, associated with cumulonimbus (thunder) clouds[2], and is a result of the electrical difference between a cloud and the earth.[5] Lightning is produced by the collision of particles in thunderclouds associated with the continuous updrafts and downdrafts of storm formation.[2,6] These collisions result in a buildup and layering of static electricity within a thundercloud, the lower layers of the cloud becoming negatively charged relative to the earth.[5] When the potential difference existing between earth and cloud exceeds the insulating strength of air, a flash of lightning occurs.[6] There are four modes of lightning discharge: intracloud, cloud to cloud, cloud to ground, and ground to cloud.[2] This paper is concerned mainly with the cloud to ground variety which represents about 20% of all lightning flashes.[2]

The lightning stroke begins as a downward directed, stepped "leader" stroke, ionizing the air in its pathway.[5] As this branching leader stroke nears the surface of the earth, an upward discharge ("pilot" stroke) rises from the ground or an object such as an isolated person in an open area, to complete the pathway of ionization at a height of about 50 metres above the earth.[6,7]

Although the main electrical discharge occurs in the return (pilot) stroke, the lightning is perceived as having travelled from cloud to ground because the leader stroke is slow relative to the pilot stroke.[5] The leader stroke is also more luminous than the pilot because of the large amount of energy required initially to ionize the air channel.[5] As many as 40 secondary and return strokes may occur in rapid succession, the average number of secondary strokes being four to seven.[5] Lightning frequently strikes the same place during a single storm, as structural conditions making an object susceptible to the original strike, usually remain in effect following the initial strike.[1]

The average duration of a lightning flash is 0.2 seconds[2], the discharge current during this time period being between 10,000 and 20,000 amperes with a voltage of between 20 million and 1 billion volts and a temperature of 300,000°C.[7] Lightning has been described visually in many forms: "streak" lightning described above generally accounts for human injuries while "sheet" lightning travels within clouds, and nearer ground-directed discharges include ribbon, bead, and ball lightning.[6]

Thunder occurs because of the explosive expansion of air ionized by the lightning stroke.[5] The distance of a flash can be calculated by dividing the time in seconds between the sighting of the lightning stroke and the arrival of the thunder by five.[2] This gives the approximate distance of the lightning stroke from the observer in miles.[2] The thunder begins with a short clap followed by a long rumble at distances of a few hundred metres; at greater distances, it begins with a rumbling sound, and beyond 15 miles, thunder is rarely heard.[2]

The clinical severity of an electrical injury is determined by the type of circuit (AC or DC), duration of contact, voltage, amperage, tissue resistance and pathway of current flow.[2] Lightning injuries differ from other high voltage injuries in several ways. Since lightning is instantaneous DC shock, it causes a single and often forceful contraction of muscle followed by relaxation.[2] High voltage AC shocks (industrial/household electrical accidents) on the other hand, cause tetanic muscle contraction which may link the victim to the conductor for an extended period of time.[2,5] This prolonged contact may result in severe internal burns and charring of tissue.[5] With lightning, an explosive shock wave occurs and contusions of internal organs are more the rule.[5] Lightning bolts may impart energies of 100 million volts and over 200,000 amperes peak current, magnitudes rarely seen in industrial/household electrical accidents.[5]

MECHANISM OF INJURY

In lightning injury, current is delivered to the victim by:

1. Direct strike;
2. Side flash;
3. Stride potential; and
4. Flashover.[2]

A *direct strike* is the most serious mode of delivery since the primary pathway of current flow is directly

* First Year Medical Student, Dalhousie University.
** Emergency Room Physician and Staff Anaesthetist, Victoria General Hospital, Halifax, N.S.

through the victim[2] and, as direct strikes are often to the head, they incur a high morbidity.[6] They are, however, not uniformly fatal.[2] The chance of a direct strike increases when an individual is wearing metal objects, such as hairpins, or is carrying metal objects such as an umbrella or golfclubs outside during a thunderstorm.[2,6]

Side flashes, a more common mode of lightning energy delivery, result when the direct pathway of current flow (such as through a tree) has a higher resistance than the air between the tree and the victim.[3] The lightning, therefore, jumps from the tree to the individual.[6]

Stride potential occurs when a person standing near the point of a lightning strike has a potential difference between the legs, and the resistance to flow of energy through the ground is greater than through the body tissues.[2] The current enters through one leg, and exits the other.[2] The side flash and stride potential mechanisms of lightning strike probably account for reports of mass casualties in open areas from a single stroke.[2,6]

Flashover is a phenomenon peculiar to lightning strikes. With metal conductors, electric current travels on the outside of the conductor.[2,5,6] Flashover exhibits a similar mechanism. It is believed that most of the energy in some lightning strikes flows around the outside of the victim's body, blasting apart clothing and vaporizing sweat.[2,5,6] Flashover significantly diminishes the energy dissipation within the body consequently resulting in less injury and greater survival.[8]

Different body tissues show a graded resistance to flow of electricity: nerve < blood vessels < muscle < skin < tendon < fat < bone.[2] Skin resistance is affected by moisture, cleanliness, thickness, and vascularity; wet skin (sweat, wet garments) may facilitate flashover conduction.[2]

CLINICAL EFFECTS BY ORGAN SYSTEM

Cardiovascular Complications

Characteristically, lightning injury effects multiple systems with variable severity.[6] Lightning can act as a massive, single DC countershock depolarizing the entire myocardium at once.[3,6] This causes a single systolic contraction followed by a period of asystole which varies in duration.[6] In most cases, cardiac activity returns spontaneously, initially at a bradycardiac rate which then gradually increases.[6] However, if apnea also occurs due to lightning induced paralysis of the respiratory centre, the cardiac rhythm may deteriorate and hypoxia can lead to ventricular fibrillation — a secondary arrest.[6] The duration of apnea rather than of asystole seems to be a prime determinant of morbidity and mortality.[2] Transient tachycardia and hypertension can also occur.[2]

Electrocardiographic changes observed following lightning injury may be due to a primary electrical injury or burn of the myocardium without coronary artery occlusion and usually resolve without complications.[6] Nonspecific ST-T wave abnormalities (especially T wave inversion[2]) are typical following lightning strike.[4] Prolongation of the QT interval, axis shifts, P wave changes, and diminished voltages are also seen.[3]

Neuropsychiatric Complications

Initially following a lightning strike, there is an altered level of consciousness which can vary from disorientation with retrograde amnesia, to a loss of consciousness.[6] Paralysis of the respiratory centre may occur, resulting in sudden death or a rapid onset of cerebral oedema can lead to brainstem herniation.[6] Epidural hematoma and chronic subdural hematoma have also been reported.[2] For most patients, however, a return to consciousness occurs.[3]

In many cases, the return of consciousness is followed by a transient motor paralysis known as keraunoparalysis.[9] This is the most common of the immediate neurologic sequelae and is associated with extreme vasoconstriction and loss of sensation.[9] It can affect one or more extremities[9] usually lasting less than 24 hours.[3] Following lightning injury extremities may be blue, cold, mottled, and pulseless.[1,2] This results from sympathetic instability causing vasospasm and usually clears spontaneously within a few hours.[1,2]

Alteration of deep tendon reflexes occur with lightning strikes[4] as do seizures and coma.[2] Psychiatric disorders such as severe thunder and lightning phobias and hysterical blindness, deafness, and loss of speech have been described.[3]

Eye Injuries

Effects of lightning strike on the eye include corneal ulcers[2] and more commonly cataracts (early or late developing[6]) which may totally resorb in time.[3] Cataracts also are known to result from man-made source high voltage accidents but are generally later in onset than the lightning induced form.[6] Retinal detachment or macular holes[10] have been described following lightning injury, and optic nerve injury and abnormalities of accommodation or pupillary light reflexes can occur.[2] An initial blindness or pupillary reflex abnormalities can be permanent or transient.[2]

Ear Injuries

Otic sequelae of lightning strike can be caused by the direct passage of electric current through the ear, or by the blast effect.[6] The shock wave, expanding from the lightning channel, causes tympanic membrane rupture in 50% of lightning strike victims.[4] The direct passage of lightning current can cause external ear burns[11], and trauma to the middle[12] and inner ear.[13] Neurosensory deafness or dizziness of central or vestibular origin may occur, however, the mechanism of their causation is uncertain.[2] Persistent tinnitus may follow lightning strike.[11]

Skin Injuries

Initially, the skin may show no signs of injury. However ultimately, over 90% of the victims of lightning

strike show some form of cutaneous burns.[5] Lightning burns are generally more superficial than those resulting from man-made high voltage current.[3] Deeper thermal burns, resulting directly from a lightning strike, are rare and occur secondarily, from contact with hot or ignited substances. "Feathering" burns are superficial dermal complexes quite characteristic of lightning strike.[6] They are not true burns and do not actually damage the skin.[1] These fern shaped "burns" are probably related to flashover and disappear in a few days.[6] Recognition of this burn pattern may be lifesaving for an unaccompanied comatose victim who is breathless and pulseless, by signalling the need for immediate resuscitative efforts.[6]

Flashover may also cause linear burns via steam production in areas where sweat or water accumulate.[1] Punctate burns resemble multiple small "cigarette" burns.[1] Entrance and exit burns may also be evident.[4]

Abdominal and Musculoskeletal Injuries

Internal damage (muscle, abdominal viscera), directly due to the passage of lightning energy, is uncommon when compared with injury from man-made high voltage currents. However, intestinal perforation due to expansion of bowel gasses has been reported[14], and myoglobinuria from severe muscle damage has occurred following lightning strike but is much more commonly seen with man-made high voltage electrical accidents.[3] The release of myoglobin can lead to acute renal failure secondary to myoglobinuria.[6]

Musculoskeletal injury following lightning strike is more commonly the result of secondary mechanical trauma.[6] Triggering of violent muscular contractions, as well as the shock wave from a lightning strike, can throw a victim to the ground or against some object with great force, resulting in significant blunt trauma.[2,4]

Pregnancy and Lightning Strike

A special situation is lightning injury involving a pregnant woman. In a number of such cases reported, maternal survival did not seem to be affected by the pregnancy. However, fetal survival was only 50%.[15] The fetus, it might seem, is at greater risk of injury because of immersion in amniotic fluid.[15] Spontaneous labor following lightning strike can occur.[15]

PREHOSPITAL CARE

The first responders at the scene of a lightning strike should immediately begin CPR on any victim who is pulseless or apneic.[2] Nonreactive or dilated pupils are not reliable indicators of brainstem hypoxia in lightning strike victims, and resuscitative measures should, therefore, never be withheld based on pupillary findings.[16] Responders to the scene of a lightning strike should persist in their resuscitation, even if the resuscitative effort is delayed.[2] In one study of lightning strike victims, 30% (17/58) suffered cardiopulmonary arrest; and of these, 77% died (13/17) in spite of resuscitative efforts.[5] Other studies have reported a success rate of 56% even if CPR is begun in hospital.[17]

In a lightning strike mass casualty situation, the traditional triage protocol should be altered. Rather than abandoning apparently or near dead victims to allocate limited resources to those showing signs of life, initial attention should be given to the apparently dead.[2] Those in cardiopulmonary arrest should be given priority because if a victim is not in arrest, death is unlikely.[5] Victims showing any signs of consciousness generally recover, although 74% of survivors of lightning strike show some form of permanent sequelae.[5]

EMERGENCY DEPARTMENT TREATMENT

In the Emergency Department, CPR must be continued if the patient has not yet been successfully resuscitated, and the usual ABC's of care for the severely injured instituted.[2] Spinal precautions as well as haemorrhage control and fracture splinting are important.[2] A history should be taken from witnesses of the event to guide pre- and in-hospital treatment.[2] In one reported case, alcohol or drug abuse was suspected and acted upon because of the victim's confusion and disorientation. Treatment changed when a history of lightning strike was available.[18]

The patient should be undressed and, once stabilized, a thorough physical examination should be performed, to ascertain non-life threatening injuries. All distal pulses should be checked and any peripheral sensorimotor deficits should be determined. An accurate rectal temperature should be taken because many victims are found wet and at risk of hypothermia. Vision and hearing should also be assessed.[2]

Victims should all have an EKG performed, as well as a hematocrit, urinalysis (for myoglobin), cardiac isoenzyme levels, and a CBC.[1,2] Radiographs of chest, cervical spine, and pelvis should be performed if there is a possibility of associated trauma.[2] Further x-rays and blood testing (electrolytes, BUN, creatinine, etc.,) should be performed as required by the clinical situation.[2]

Hypotension may be indicative of intrathoracic or intraabdominal haemorrhage, or bleeding from major fractures, spinal shock or fluid loss from burns.[2] Patients with an altered mental status, and suspected or known blunt abdominal injury may require a peritoneal lavage.[19] Initial fluid replacement should be with Ringers lactate.[18] In the presence of raised intracranial pressure, CVP or pulmonary capillary wedge pressure should be used to guide fluid resuscitation,[2] and diuretics, such as mannitol, and hyperventilation can be used to attempt to reduce cerebral oedma.[18] The use of steroids to reduce intracranial pressure is controversial.[18] A CAT scan can provide a definitive diagnosis of the intracranial pathology and in particular identify any surgically correctable lesions.[3] Repeated neurological testing is required to follow mental status or sensorimotor deficits.[3]

Early treatment of the lightning damaged ear is usually restricted to cleaning the external auditory canal.

Operations on tympanic membrane perforations should be delayed at least six months because of the possibility of spontaneous healing. Also oedema or charring of the auditory canal can interfere with tympanoplasty.[6]

Burns should be cleaned and treated in the usual fashion with a topical antimicrobial cream,[6] and tetanus prophylaxis insured.[2,6] Fasciotomy may be required for burns secondary to ignition of clothing or other flammable substances on the patient.[2]

For pregnant victims of lightning strike, obstetrical consultation is advisable.[2]

DISPOSITION

Some patients may require hospitalization (trauma, coma, post successful cardiac resuscitation, EKG abnormalities, enzyme changes, etc.,).[2] Admission with close observation is advised for patients with neurological abnormalities, even though these are usually transient.[2] Patients who had a transient loss of consciousness, amnesia, or confusion, as well as those with problems in hearing and vision can often be treated on an outpatient basis.[2] Because of the often associated permanent sequelae (especially psychiatric), discharged patients should have a 24-hour follow-up and at least one more follow-up several months later.[2]

PREVENTION

Lightning strike is more easily prevented than treated. Steel frame buildings,[6] or buildings with lightning rods[2] are excellent places to seek shelter during a storm. When indoors during a storm, one should also not stand in or near an open doorway or window, take a bath, or use a telephone.[6] An automobile with rubber tires is also a safe refuge.[4] When caught outdoors, curtail activities and seek shelter. Never swim outdoors during a storm. Do not handle metallic sporting equipment and remove metal objects from the head.[2] Avoid isolated tall objects when outdoors during a storm and, if no shelter is available, seek a grove of trees which is shorter than adjacent groves rather than standing out in the open.[2] If caught in the open, seek low ground and bend down with feet close together or lie down in the foetal position to avoid stride potential, preferably on a rubber or plastic raincoat for insulation.[2] Remember that lightning can strike the same location more than once during a single storm.[2]

Most physicians never see or treat a victim of lightning strike[20] even though lightning kills more people than any other natural disaster.[1] The main cause of death is cardiac arrest and injuries found are those expected from an overloading of the body's electrical system (e.g., amnesia, blindness, cardiac dysrhythmias, confusion, tinnitus, and vascular instability).[1] Lightning injuries are associated with a 30% mortality and 74% of survivors experience sequelae.[5] Rescuers on the scene should concentrate on resuscitating the apparently dead (e.g., cardiopulmonary arrest).[2] Follow-up upon discharge is advised for evaluation of late developing or permanent sequelae. □

References

1. Cooper MA. Lightning injuries in Rosen P., eta al. (eds) Emergency Medicine: Concepts and clinical practice. St. Louis: The C.V. Mosby Company, 1988.
2. Cwinn AA, Cantrill SV. Lightning injuries. *J Emerg Med* 1985; 2: 379-388.
3. Craig SR. When lightning strikes. Pathophysiology and treatment of lightning injuries. *Postgrad Med* 1986; 78(4): 109-124.
4. Clore ER, House MA. Prevention and treatment of lightning injuries. *Nurse Practitioner* 1987; 12(12):37, 40-41, 44-45.
5. Cooper MA. Lightning injuries: prognostic signs for death. *Ann Emerg Med* 1980; 9: 134-138.
6. Tribble CG, Persing JA, Morgan RF, Kenney JG, Edlich RF. Lightning injuries. *Comprehens Ther* 1985; 11(2): 32-40.
7. Moran KT, Thupari JN, Munster AM. Lightning injury: pathophysiology and clinical features. *Irish Med J* 1986; 79(5): 120-122.
8. Ohashi M, Kitagawa N, Ishikawa T. Lightning injury caused by discharges accompanying flashovers — a clinical and experimental study of death and survival. *Burns* 1986; 12: 496-501.
9. tenDuis HJ, Klasen HJ, Reenalda PE. Keraunoparalysis, a "specific" lightning injury. *Burns* 1985; 12: 54-57.
10. Campo RV, Lewis RS. Lightning induced macular hole. *Am J Ophthalmol* 1984; 97: 792-794.
11. Johnstone BR, Harding DL, Hocking B. Telephone related lightning injury. *Med J Austrail* 1986; 144: 706-708.
12. Bergstrom L, Neblett LM, Sando I, Hemenway WG, Harrison GD. The lightning damaged ear. *Arch Otolaryngol* 1974; 100: 117-121.
13. Youngs R, Deck J, Kwok P, Hawke M. Severe sensorineural hearing loss caused by lightning. *Arch Otolaryngol* 1988; 114: 1184-1187.
14. Dowling J, Barry OCD, Lennon F, Byrne K, Long JP. Lightning injury. *Irish Med J* 1984; 77(8): 250-251.
15. Pierce MR, Henderson RA, Mitchell JM. Cardiopulmonary arrest secondary to lightning injury in a pregnant woman. *Ann Emerg Med* 1986; 15: 597-599.
16. Abt JL. The pupillary responses after being struck by lightning. *JAMA* 1985; 254: 3312.
17. Morgan KT, Thupari JN, Munster AM. Electric and lightning induced cardiac arrest reversed by prompt cardiopulmonary resuscitation. *JAMA* 1986; 255: 2157.
18. Moser MY. When lightning strikes. *Am J Nursing* 1986; 86: 802-803.
19. Marx JA. Abdominal trauma In: Rosen, P.: et al (eds.) *Emergency Medicine: concepts and clinical practice.* St. Louis: The C.V. Mosby Company, 1988.
20. Amy BW, McManus WF, Goodwin CW, Pruitt BA. Lightning injury with survival in five patients. *JAMA* 1985; 253: 243-245.

"Team up for safety in the '90s"
PLAN TO ATTEND
CANADA SAFETY COUNCIL'S 1990 CONFERENCE JUNE 3-5, 1990
at the NOVA SCOTIAN HILTON INTERNATIONAL, HALIFAX, NOVA SCOTIA

Medical Treatment of Stress Incontinence

Alfred E. Bent, *MD

Long Beach, California

There are several factors which maintain continence during stress, and they may involve one of three urethral components:

1. Intra-abdominal location of the proximal urethra during times of stress (coughing, aerobics, running, etc.).
2. Periurethral skeletal muscle (reflex contraction with stress).
3. Urethral sphincteric mechanism — smooth muscle, mucosa, submucosal vessels, and fibroelastic tissue.

Surgery corrects the first component, the intra-abdominal location of the proximal urethra during stress. Medications and other therapies described in the medical treatment will refer to the second and third continence maintaining components (Table I).

TABLE I

Medical Treatment of Urinary Incontinence

Avoidance techniques
Estrogen therapy
Drug therapy
Kegel's exercises
Biofeedback
Vaginal cones
Electrical stimulation
Pessary
Periurethral collagen injection

AVOIDANCE TECHNIQUES

The simplest methods for preventing urinary stress incontinence involve avoidance techniques which are usually unacceptable to the patient:

a. empty the bladder regularly and prior to physical activity.
b. avoid the activity which causes leakage.
c. discontinue the medication which causes leakage (phenothiazine, antihypertensive).
d. fluid and caffeine restriction prior to planned activities.
e. wear appropriate protection.
f. prevent chronic coughing — asthma treatment, smoking restriction, alteration of allergic environment.
g. job alteration (avoid lifting).

---
* Assistant Professor Obstetrics/Gynecology University of California, Irvine and Assistant Medical Director of Gynecology, Memorial Women's Hospital, Long Beach Memorial Medical Center.

Correspondence: Alfred E. Bent, M.D., 2880 Atlantic Avenue, Suite 130, Long Beach, California, 90806

MEDICAL TREATMENT OF STRESS INCONTINENCE

Estrogen Therapy

The urethral mucosa and submucosal vessels are estrogen dependent. Various reports in the literature indicate increased urethral closure pressure or improved transmission ratio to the proximal urethra during estrogen therapy. Clinically, estrogen is probably best used as a vaginal application (dienestrol vaginal cream 0.01%) 1/3 to 1/2 applicator at bed time three times per week for 12 weeks or use nightly for two weeks followed by maintenance therapy of 1/3 to 1/2 applicator once or twice weekly. Mild stress incontinence in postmenopausal patients may be completely cured. Incontinence of moderate severity may be helped but severe conditions are usually changed little. Some patients may be sensitive to dienestrol cream but may be able to tolerate Premarin® vaginal cream (conjugated estrogen 0.625 mg/g), Ogen® (estropipate 1.5 mg/g), or Estrace® (estradiol 0.01%). An additional benefit from estrogen therapy is the reduced sensitivity of periurethral tissues which helps control irritative symptoms such as urgency, frequency, and nocturia.

Side Effects: Vaginal estrogens are absorbed systemically but small doses utilized have minimal significant side effects while the beneficial effects on the vaginal urethral tissues are maximal. Vaginal estrogens do not prevent osteoporosis and usually do not control hot flushes or sweats. Large vaginal doses may carry the same risk as unopposed oral estrogen therapy. Patients with an intact uterus must be managed appropriately should abnormal bleeding occur (i.e., endometrial biopsy). Recently, oral estrogens have been shown to have efficacy in the treatment of stress incontinence, especially when combined with phenylpropanolamine.[2]

Drug Therapy

Urethral smooth muscle responds to alpha adrenergic stimulation with an increase in tone. Mild to moderate degrees of stress incontinence may respond to alpha adrenergic therapy. Some studies in women with stress incontinence have demonstrated urethral pressure increases following alpha adrenergic therapy. Three drugs of use are imipramine, phenylpropanolamine, and ephedrine.

Imipramine may be started at a dose of 25 to 75 mg at bed time and increased as tolerated according to the response. Elderly patients (i.e., over age 65) require smaller starting doses. Imipramine also has anticholinergic, some antispasmodic, and central sedative effects which inhibit bladder contractility and therefore make

this drug useful for patients with combined genuine stress incontinence and detrusor instability. The side effects of the medication include dry mouth, hypotension, tachycardia, palpitation, weakness confusional states, disorientation, anxiety, restlessness, agitation, insomnia, tingling, numbness and tremor. Although up to fifty percent of patients may experience side effects, a large number are significantly improved and tolerate the medication well.

Pheylpropanolamine is utilized as an appetite suppressant and is available in several over-the-counter forms, the most common being appetite suppressants, Ornade® spansules, and Entex-LA®. Ornade is combined with an antihistamine, chlorpheniramine. The medication is used in the starting dose of 75 mg daily and may be increased up to 150 mg per day. The side effects include drowsiness, dry mouth, nervousness, or insomnia.

Ephedrine is a general adrenergic stimulator effective in patients with mild stress incontinence. The dose is 15 to 60 mg three to four times per day and the side effects include restlessness, insomnia, anxiety, tension, tremor, dizziness and palpitations.

The various forms of medical therapy carry an improvement rate as high as 40 to 60% in selected patients and in some patients have provided cure of their difficulty. Long-term effects may not be as encouraging in many patients and often these patients are also not good candidates for surgery. A small number of patients may continue to be managed conservatively as opposed to proceeding with surgery.

Kegel's Exercises

The pubococcygeal muscle (part of levator ani) and periurethral striated sphincter contain fibres which are the main voluntary support structure for the urethral sphincteric mechanism. It is thought that part of the pubococcygeus muscle contains fast twitch muscle fibres which allow rapid increase in intraurethral pressure during coughing, which helps promote maintenance of continence and accounts for pressure transmission ratios during coughing which may be greater than 100%. The pubococcygeal and urethral striated sphincter are weakened by child bearing, aging, obesity, and trauma. The pubococcygeus muscle is identified by stopping urine midstream without straining or squeezing the buttocks. Inserting a finger or tampon in the vagina may help identify muscle contraction. There is also a commercial device, a perineometer, which can be placed in the vagina to measure the strength of muscular contractions. This was originally the method described by Kegel in his initial paper.[3]

There are many formulas for exercising according to the method of Kegel. One method is to have the patient contract the pubococcygeus muscle for three seconds, then release for three seconds, and repeat this for a total of ten times in one minute. The next set of exercises are performed by quickly holding and releasing as many times possible within one minute. The exercises are repeated for four minutes five or more times a day depending on the patient's motivation and challenge. The many places to exercise include waiting in lines, waiting at stop lights, and while commuting. The reported cure/improvement rate from Kegel's exercises has been as high as 70% or greater in some studies.[4]

Biofeedback

Biofeedback is an extended form of Kegel's exercises where a patient is initially taught the correct maneuver for the exercise while observing pressure generated on a perineometer. Another sensor detects and alerts the investigator to the presence of abdominal straining. The patient can see the strength of the contraction on a graded sensing device. There is also a work-rest cycle so the patient knows when to contract and when to release her muscles during the exercise. The patient is able to practise the Kegel's exercises at home, as they have been trained in the laboratory setting by the biofeedback technique. On occasion, patients also take the perineometer units home in order to do their form of biofeedback therapy. The cure rate from biofeedback for urinary stress incontinence has been reported to be as high as 50%.

Vaginal Cones[5]

A set of weighted vaginal cones increasing in weight from 20 to 100 grams has been designed to exercise the pelvic floor muscles in patients with genuine stress incontinence. Patients place the weighted cones in the vagina, stand up and walk for one minute and by pelvic muscle contraction, are gradually able to maintain progressively heavier weighted cones. The effectiveness of the exercises in reducing incontinence was as high as 70%. However, greater than 1/3 of patients opted for surgery after training with the vaginal cone device.
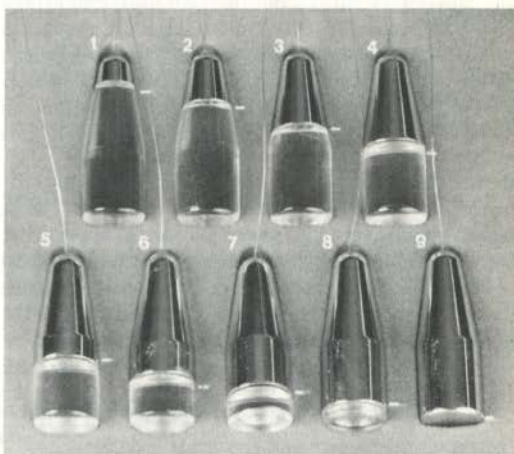
Fig. 1 Vaginal cones. Progressively heavier vaginal inserts are used to gradually strengthen pelvic floor muscles, and thereby improve urinary control.

Functional Electrical Stimulation

Electrical stimulation aids in the restoration of continence through stimulation of the pudendal nerve. This causes contraction of the pelvic floor and periurethral striated muscles. This passive type of Kegel's exercises hypertrophies the periurethral fast twitch fatigue resistant muscle fibres. The device is suited either for wearing in the vagina or rectum over a short period of time but more frequently now, it is being used as a vaginal probe which is inserted in place for 15 minutes two times daily in order to treat the patient. Patients usually respond within six weeks of therapy and there has been a success rate as defined by improved or cure, in the range of 70%.[6] The major questions involve how long therapy should be continued and whether patients maintain the good effect once therapy is discontinued.

Fig. 2 Microgyn II. Vaginal delivery system for functional electrical stimulation. (Courtesy of InCare Medical Products, a Division of Hollister, Incorporated)

Pessary Use

Vaginal pessaries are usually applied in patients with marked pelvic relaxation who are poor operative risks. It has been shown that stress incontinence may be unmasked in some of these patients with large prolapse in the form of cystocele or procidentia. It has been shown that some patients with urinary stress incontinence are actually improved with placement of a pessary. In patients with severe pelvic relaxation, some become incontinent once the pessary is inserted. In terms of beneficial effects from the pessary related to urinary incontinence, it is felt that the pessary stabilizes the urethra and urethrovesical junction under conditions of stress. Inflatable, Smith-Hodge, soft doughnut, or ring-diaphragm type pessaries may be utilized along with estrogen cream in order to maintain healthy vaginal tissues.

Periurethral Collagen Injection

Recently described is the technique of injecting Teflon into the periurethral tissues under endoscopic guidance.[7] Twenty-six patients had the procedure performed with good or moderate results in 50% of patients. This procedure has subsequently been performed with periurethral glutaraldehyde cross-linked collagen injections (GAX collagen) which avoids some of the problems of the original Teflon. The cure or improved rate in some studies was higher than 60% although much of this material is not yet available for publishing. The injections can be repeated in order to further improve patients who do not fully respond to initial therapy. The collagen is biodegradable and is gradually replaced with the patient's own collagen.

SUMMARY

Patients with severe symptoms of genuine stress incontinence usually do poorly with the nonsurgical treatments. Those at increased surgical risk, though not cured, may certainly benefit from a trial of conservative therapy although on many occasions, they are also good candidates for surgical intervention. A well motivated patient will benefit most from a trial of physiotherapy techniques such as biofeedback, electrical stimulation, or pelvic floor muscle exercises. ☐

References

1. Bergman A. Nonsurgical treatment of stress urinary incontinence. *Gynecologic Urology and Urodynamics: Theory and Practice*, 2nd edition, Editor Donald R. Ostergard. Baltimore: Williams and Wilkins, 1985, pp 419-435.
2. Kinn AC, Lindskog M. Estrogens and phenylpropanolamine in combination for stress urinary incontinence in postmenopausal women. *Urology* 1988; **32:** 273-280.
3. Kegel AH. Progressive resistance exercises in the functional restoration of the perineal muscles. *Am J Obstet Gynecol* 1948; **56:** 238-248.
4. Henalla SM, Kirwan P, Castleden CM, Hutchins CJ, Breeson AJ. The effect of pelvic floor exercises in the treatment of genuine urinary stress incontinence in women at two hospitals. *Br J Obstet Gynecol* 1988; **95:** 602-606.
5. Peattie AB, Plevnik S, Stanton SL. Vaginal cones, a conservative method of treating genuine stress incontinence. *Br J Obstet Gynecol* 1988; **95:** 1049-1053.
6. Fall M. Does electrostimulation cure urinary incontinence. *J Urol* 1984; **131:** 664-667.
7. Palle JO, Rohl H. Female urinary stress incontinence treated with Teflon injections. *Acta Obstet Gynecol Scand* 1987; **66:** 333-335.

Innumeracy, an inability to deal comfortably with the fundamental notions of numbers and chance, plagues far too many otherwise knowledgeable citizens.

John Allen Paulos,
Professor of Mathematics, Temple University

School Absenteeism in Halifax, Nova Scotia

Lynn McIntyre, * MD, MHSc, FRCPC

Halifax, N.S.

School and learning problems leading to school failure have a well-recognized association with excessive absenteeism as do psychosocial problems and socioeconomic status of the primary caregiver. Children aged 6-16 years are reported to miss between 4.9 and 11.1 days of school per year. The purpose of this study was to ascertain normative data of school absenteeism in Halifax, Nova Scotia schoolchildren using 1987 Principal's Summary of Attendance reports.

Of 40 public schools in Halifax, the mean monthly absenteeism per school was 6.0% (sd 2.0) and the average child missed 11.1 days of school per year (sd 3.3). School absenteeism was significantly related to junior high school enrollment (p = .05) and lower mean income (p = .003). The monthly pattern of absenteeism showed highest absenteeism in the winter months for all grades and census areas.

School attendance is a sensitive measure of the functioning level of a child. Excessive absence from school is a hindrance to progress in basic educational and social skills. It contributes to a child's problems in coping with school and interferes with learning skills necessary to achieve career productivity in adulthood.[1] School and learning problems leading to school failure have a well-recognized association with excessive absenteeism.[2,3,4]

The majority of school absences are related to acute infectious processes, such as upper respiratory tract infections or gastroenteritis. In studies of the general child population, 70 to 80 percent of all absences are attributed to health-related problems.[5]

Socioeconomic status affects school absenteeism. Weitzman *et al.* found that otherwise healthy children in a poor neighbourhood were absent 11.6 days in the year compared to 6.4 days for their wealthier neighbours (p < .001).[3] Other studies have found that the predicted number of days absent decreases as the education level of the primary caretaker increases.[1]

Normative data on school absenteeism are largely lacking and are quite variable when reported. According to data from the 1981 National Health Interview Survey, children in the United States, aged 6-16, were absent an average of 4.9 days from school per year.[6] Parcel *et al.* found an average absence rate of 5.9% for non-asthmatic students[7]; for a school year of 188 days, this represents

11.1 days. Weitzman *et al.* found that healthy children were absent from school an average of 5.8 days in the previous year.[3]

The purpose of this study was to ascertain normative data on school absenteeism in Halifax, Nova Scotia public schoolchildren and to examine absenteeism differences by season, grade level and by mean income of the schools' surrounding census tracts.

METHODS

Principal's Summary of Attendance Reports for all schools in Halifax for the school year September 1986 to June 1987 inclusive were received from the Halifax Board of Education. These reports indicate the actual days the school was open, active monthly enrollment, pupil days lost and actual total attendance. All data were reported by school only rather than by grade, classroom or by child. Private schools were not included.

Census maps were used to plot the schools by census tract. Statistics Canada releases were obtained for average income data by census tract for families in private households based on a 20% sample of 1981 census responders. Median income data were unavailable.

From the Principal's Attendance Reports, the percentage of days absent per month and year and the average number of school days absent were calculated. The percentage of days absent was derived by dividing the number of days absent by the number of days in the school month or year.

Spearman's rank correlation was used to assess the relationship between absenteeism and mean income level of the census tract surrounding the school. Analysis of variance evaluated mean differences in absenteeism by season and by grade grouping. Analysis of covariance studied these relationships by adjusting for mean income level of the census tract. The level of significance chosen was p < .05.

RESULTS

There are 40 public schools in Halifax offering classes from Grades Primary to 12. The school system's average enrollment for the 1986-1987 188-day school year was 13,637 students.

According to the attendance data obtained from the Principal's Reports, the mean monthly absenteeism per school was 6.0% (s.d. 2.0) and the average child missed 11.1 days of school per year (s.d. 3.3).

Average school absenteeism was significantly related to lower mean income of the census tract surrounding the school (p = .003). The city is divided by school authorities into three sectors and the lower income sector

* Hospital Epidemiologist, IWK Children's Hospital, Assistant Professor, Department of Community Health & Epidemiology, Dalhousie University, Halifax, N.S.

Correspondence: Dr. L. McIntyre, IWK Children's Hospital, Box 3070, Halifax, N.S. B3J 3G9

compared to the higher income sector also had significantly higher absenteeism rates (p = .04, Student's *t*-test).

When grade levels were adjusted for income, analysis of covariance revealed that school absenteeism was significantly related to junior high school enrollment, (p = .05). Students in Grades 7-9 were absent an average of 13.2 days (s.d. 4.3) per year compared with other children who were absent an average of 10.8 days (s.d. 1.3). [Table I]

TABLE I

SCHOOL ABSENTEEISM BY GRADE GROUPING

Grades	Number of Schools	Average School Absenteeism (s.d.)	Total School Days Absent per Child per Year (s.d.)
P*-6	17	5.1 (1.1)	9.5 (2.1)
7-9	5	7.1 (2.4)	13.2 (4.3)
P*-9	14	6.7 (2.4)	12.1 (3.7)
10-12	4	6.4 (1.5)	11.9 (2.8)
Total	40	6.0 (2.0)	11.1 (3.3)

*P = primary

When the school year was divided into fall (September to November), winter (December to March) and spring (April to June), one-way analysis of variance demonstrated that average absenteeism was significantly related to season (p < .008). Winter's mean absenteeism was 7.1%, spring absenteeism was 6.4% and fall's absenteeism was 4.3%.

DISCUSSION

Halifax's school absenteeism is similar to results reported from attendance rolls in Galveston[10] but much higher than those reported in studies which used parent reporting[3] or a health interview survey.[9] Parents frequently underreport school absences and Cook *et al.* have emphasized the importance of obtaining attendance data directly from the schools.[1]

"My child misses a lot of school" is a common complaint of parents of both normal children and children with chronic conditions. Health professionals and teachers may interpret a child's actual absenteeism by comparing it with normative results. Using data obtained in this study, an excessively absent student may be one who is in the 95th percentile for days missed for his or her cohort. This would be 18 days absent per year compared to the 11 days expected for an average child.

Asthma is the principal cause of school absences due to chronic disease in childhood, accounting for 20% of school days lost in elementary and high schools.[8] In a comparative study, children with asthma missed one and a half times as many days as non-asthmatic children.[7] In Halifax, an "average" asthmatic child would be expected to miss 16.7 days per year. Cook *et al.*

found that the average school absence for children with various types of chronic conditions was 16.9 days per year.[1]

This study was consistent with others which have demonstrated an association between absenteeism and ascending age and grade. However, in Halifax, the highest absenteeism was found in junior high, rather than high school. Age and grade-related absenteeism is postulated to reflect a diminution of educational motivation which increases with grade, particularly in the post-primary school years.[3] High school entry in our school system may renew educational commitment.

The seasonality of absenteeism is likely explained by the higher occurrence of acute infectious diseases in the winter compared to other times of the year.

The strong association between higher absenteeism and lower socioeconomic level of the surrounding census tract was expected.[1,3] School populations in lower income areas may require specific initiatives to reduce absenteeism. ☐

References

1. Cook BA, Schaller K, Krischer JP. School absence among children with chronic illness. *J School Hlth* 1985; 55: 265-267.
2. Weitzman M, Alpert JJ, Klerman L et al. High-risk youth and health: the case of excessive school absence. *Pediatrics* 1986; 78: 313-322.
3. Weitzman M, Walker D, Gortmaker S. Chronic illness, psychosocial problems and school absence. *Clin Ped* 1986; 25: 137-141.
4. Rozelle RM. The relationship between absenteeism and grades. *Educ Psychol Measurement* 1968; 28: 1151-62.
5. Rogers KD, Reese G. Health studies — Presumably normal high school students. *Am J Dis Child* 1965; 109: 9-19.
6. National Center for Health Statistics. Current estimates from the Health Interview Survey 1981, series #141, Hyattsville, Md. 1983.
7. Parcel GS, Gilman SC, Nader PR et al. A comparison of rates of elementary schoolchildren with asthma and nonasthmatic schoolmates. *Pediatrics* 1979; 64: 878-881.
8. Richards W. Allergy, asthma, and school problems. *J School Hlth* 1986; 56: 151-152.

ERRATUM

February 1990, Volume 69, page 24.

CENTRAL (HYPOTHALAMIC) PRECOCIOUS PUBERTY MANIFESTATIONS AND MANAGEMENT OPTIONS
by S. Salisbury, MD and S. Baker, RN.

Fig. 4 - Case 2

Data relevant to this case was not shown in this Figure. If you wish to receive further information please contact Dr. Salisbury.

A Pathologist's Viewpoint
'GO WITH THE FLOW'

Annette Foyle,* MD, CM, FRCP(C)

Halifax, N.S.
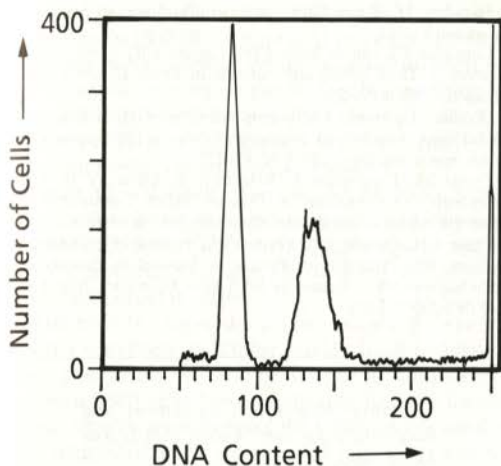
Fig. 1 Flow cytometry DNA analysis: Histogram of breast cancer from a 64 year old woman. What do the peaks signify?

Fig. 2 Diagram of a flow cytometer. The instrument illustrated can measure side light scatter (cell granularity), forward light scatter (cell size) and three colors of fluorescence. It can measure 5 parameters per cell simultaneously if desired.

What is a flow cytometer? And what does it mean to your patient with breast cancer? In previous publications of the *Journal*, the classic role of the anatomic pathologist in the diagnostic study of antemortem and postmortem tissues was addressed. The processing of

*Assistant Pathologist, Victoria General Hospital, Assistant Professor, Dalhousie University, Halifax, N.S.

Fig. 3 Histogram and cell cycle of a normal diploid cell population. G0 resting phase; G1 pre DNA synthesis; S synthesis; G2 post DNA synthesis, M mitosis.

tissue for examination with the light microscope is a relatively lengthy procedure that has been established for over a hundred years. More recently, it has become possible to screen several hundred individual tissue cells, spread out on a Pap smear, in about ten minutes. Now technology has advanced such that various parameters of 10,000 cells can be analyzed within five minutes. The instrument capable of this feat is called a flow cytometer. In this communication we will briefly outline how the flow cytometer works, and describe how one cell parameter, DNA, can be measured. In particular, we will focus our attention on the utility of DNA analysis in breast cancer.

A *flow cytometer* is an *instrument* which can measure features of individual *cells* as they *flow* through it in a stream.[1,2] The measurements of each cell that can be obtained are its size, its cytoplasmic granularity and its fluorescence intensity. In the flow cytometer cells are introduced and positioned in a single file in the center of a fluid stream (Fig 2). As the separate cells pass by a laser beam, they scatter the light and this light scattering gives information on the cell's size and granularity, which are its intrinsic parameters.

Other parameters, however, are measured with the aid of extrinsic fluorescent dyes. The DNA content of cells in solid tumors has been extensively studied in diagnostic pathology. In a cell suspension made from either fresh, frozen or formalin-fixed, paraffin-embedded tissue, the cell nuclei are stained with a fluorescent dye. The cells then fluoresce individually as they pass the laser beam. The degree of fluorescence of each individual cell is proportional to its DNA content. This fluorescence is monitored and analyzed by a computer so

that DNA content of 10,000 cells can be measured in less than 5 minutes.

The mitotic cell cycle is illustrated in Figure 3. Cells in the resting phase of the cycle (G0) and cells in the pre-synthesis phase (G1) have a normal, diploid content of DNA. The majority of cells in the population are in this phase of the cycle, and produce the largest G1/G0 peak on the DNA histogram. During the synthesis (S) phase, cells are making increasing quantities of DNA. In the post synthesis phase (G2) and during mitosis (M), cells have twice the normal diploid content of DNA which produces a second small peak on the histogram.

Breast cancer is the most frequent type of cancer in women in Canada today.[3] The diagnosis is established by excisional biopsy of the tumor with microscopic examination of the tissue.[4] After diagnosis, a number of further studies may be performed to evaluate the disease. Prognostic factors in breast cancer are listed in Table I. Relatively favorable prognostic factors include: age greater than 50 years; low stage disease; well-differentiated carcinoma, with a positive estrogen receptor and progesterone receptor assay; a diploid content of cellular DNA and a low synthesis (S) fraction. Recently, these last two flow cytometric markers (DNA content and S phase) have been identified as predictors of tumor recurrence and/or overall survival in women with breast cancer.[5-8] Figure 1 illustrates a population of cells with a normal diploid content on the left. These are normal stromal cells and inflammatory cells associated with the breast tumor. The peak on the left is from a population of cancer cells with an abnormal DNA content or aneuploidy. Abnormal DNA content (aneuploidy) is associated with a higher risk of tumor recurrence and worse prognosis for survival. A high S phase fraction also may indicate a worse prognosis.

TABLE I

BREAST CANCER PROGNOSIS

- Age
- Stage
- Histopathology
- ER/PR status
- Ploidy
- S phase fraction

A flow cytometer is an instrument which can measure a number of parameters in a single cell. The cells pass through the instrument in a fluid flow. One of the parameters the flow cytometer can measure is the DNA content (ploidy) of individual cells. Abnormal ONA content (aneuploidy) in tumor cells from breast cancer is generally indicative of a worse prognosis. Also, cancers with a high synthesis phase fraction carry a poorer prognosis. Therefore, performing DNA analysis in breast cancer may be a useful adjunct in the initial evaluation of a patient with breast cancer. □

References

1. Horan PK: Single Cell Analysis Enters the Space Age. *Diagnostic Medicine,* October 1981, 63-85.

2. Coon JS, Landay AL, Weinstein R. Biology of Disease: Advances in Flow Cytometry for Diagnostic Pathology. *Laboratory Investigation* 1987; 5: 453-479.

3. Hagensen CD. *Diseases of the Breast.* Third Edition. Philadelphia: WB Saunders, Chapter 26: 394-397.

4. Stockdale FE. Breast Cancer in Scientific American Section 12, Subsection V11:2-4.

5. Cornelisse CJ, van de Velde CJH, Caspers RJC, Moolenaar AJ, Hermans J. DNA Ploidy and Survival in Breast Cancer Patients. *Cytometry* 1987; 8: 225-234.

6. Dressler LG, Seamer LC, Owens MA, Clark GM, McGuire WL. DNA Flow Cytometry and Prognostic Factors in 1331 Frozen Breast Cancer Specimens. *Cancer* 1988; 61: 420-427.

7. Ewers SB, Langstrom E, Baldetorp B, Killander D. Flow-Cytometric DNA Analysis in Primary Breast Carcinomas and Clinicopathological Correlations. *Cytometry* 1984; 5: 408-419.

8. Clark GM, Dressler LG, Owens MA, Pounds MT, Oldaker T, McGuire WL. Prediction of Relapse or Survival in Patients with Node-Negative Breast Cancer by DNA Flow Cytometry. *New Eng J Med* 1989; 320: 627-633.

To know how much we know, and how much we don't know, that is true knowledge.

Zoroaster (*c.*630-*c.*553 B.C.)

Non-Relative or Third-Party Adoptions

Susan M. Drysdale, RSW

With the decreasing number of infants available for adoption through agencies, some families are pursuing private arrangements to adopt a child. These adoptions are known as private non-relative or third-party adoptions. In 1987-88, there were 31 private non-relative adoptions in Nova Scotia. While the actual numbers are few, there is increasing activity by families searching for expectant mothers willing to place privately. Medical practitioners may be approached by families or expectant mothers to act as an intermediary.

Under the *Children's Services Act* in Nova Scotia, there are specific requirements to be met in private non-relative adoptions. These requirements were implemented primarily to protect children but also serve to protect the parties involved.

Historically, these adoptions occurred between families who knew each other. Today, more frequently, these placements are arranged between families unknown to each other but brought together by a lawyer, doctor, clergyman or friend of the family. Some families have networked across the province or country seeking out expectant single mothers rather than face lengthy agency waiting lists.

The *Children's Services Act* stipulates the following requirements for these placements:

14 A (1) No person shall place or receive a child for the purpose of adoption unless:
   a) any necessary consent for adoption has been obtained and, if necessary, confirmed;
   b) each person who has consented to the adoption has, before giving the consent, received professional counselling by a person or a member of a class of persons approved for that purpose by the Minister;
   c) a social and medical history respecting the natural parents has been prepared, if the parents or either of them are known and available, by a person or a member of a class of persons approved for that purpose by the Minister; and
   d) the home where the child is to be placed has been identified to an agency in the area where the proposed adopting family resides,

except where
   e) the child is placed by a child placing agency;
   f) the child is placed by the mother or father with a person related to either the mother or father by blood or marriage;
   g) one of the applicants for adoption is a natural parent of the child.

(2) A person who places or receives a child for the purpose of adoption and who does not comply with subsection (1) is guilty of an offence and on summary conviction is liable to a fine of not more than ten thousand dollars or to imprisonment for a term of not more than one year, or to both.

A birth parent's consent is not valid unless it was signed 15 days after the birth of the child not including the date of birth and the date of signing the consent. This is the minimum period and may be extended if necessary. The child cannot be placed in the prospective adoption home until the consent is signed; therefore, an interim placement arranged by the birth parent or prospective adoptive parents with family or friends is often chosen. The purpose of this waiting period, before the child can reside with the adoptive parents, is to provide the birth parent the opportunity to make a decision regarding the adoption placement without the pressure of knowing the child is already with the prospective adoptive family.

Prior to a consent for adoption, 14 A (1), (b), (c) and (d) must also be complied with.

Professional counselling is provided by designated caseworkers with the local Children's Aid Society, Family and Children's Services Agency or District Office of the Department of Community Services upon request. These workers have the knowledge and expertise in the area of adoption counselling and support to birth parents. A social and medical history is prepared by the caseworker with the birth parents, which is maintained on the permanent adoption record if an adoption occurs. The worker ensures the birth parent is aware of the options available, and the consequences of signing a consent to adoption to assist the birth parent to make an informed decision. The medical and social history, if not shared with the prospective adoptive family by the birth parent, may be obtained form the Department of Community Services under the Release of Adoption Information Policy. This policy permits the release of non-identifying information.

Prior to any plans to proceed with a private non-relative adoption, prospective adoptive parents must be identified to the Children's Aid Society, Family and Children's Services Agency or District Office in the area where they reside. This would include their address, phone number and if possible, the identity of the birth parent, address, phone number and child's date of birth. This will assist the agency in determining if the family is previously known to an agency as a family who had a homestudy completed and were subsequently turned down for adoption for valid reasons or under child protection services. Since a home study on the prospective family in private non-relative adoptions is not initiated until after the placement of the child in the home and notices of proposed adoption are filed, it is the only means to determine if the child could be at risk.

The legislation pertaining to adoptions is under provincial statutes, therefore, the requirements may vary

Current Topics in Community Health

Selected by: Dr. Lynn McIntyre
Department of Community Health & Epidemiology
Dalhousie University, Halifax, N.S.

THE NEED FOR A PROVINCIAL HEALTH PROFILE

Terry Delmore, Epidemiologist of the Regional Niagara Health Services Department in Southern Ontario, sent me a copy of his 1989 report, *Mortality in Niagara 1979 to 1986* which provides an indepth review of mortality patterns in the health unit.[1] The report is a neatly bound 32 pages with multiple graphs and tables, produced by a desk-top publishing package.

The report presents perinatal, neonatal and infant mortality rates and identifies the major causes of mortality by age and gender and potential years of life lost by gender. It also compares standardized mortality ratios over different time periods for the health unit population. While findings of the report are unremarkable, it is remarkable that the report exists at all. This small health unit, unattached to a university centre, is able to produce a health status profile of its citizens while Nova Scotia has no such document.

The Canadian Institute of Child Health's recently released report, *The Health of Canada's Children: A CICH Profile* presents available health status information about Canada's children.[2] We can learn something about the demography of Nova Scotia children in the report. For example, about 28% of the population is under 19 years of age; 46% of Nova Scotians live in rural areas; about 2% of families in Nova Scotia speak a language other than English or French in the home; our total fertility rate is 1.6; and 0.6% of children live in protective care.

For birth and infancy outcomes, the report presents Nova Scotia male life expectancy at birth as 72.2 years, seven years shorter than Nova Scotia females; infant mortality is 8 per 1000 live births; perinatal mortality is 2.9 per 1000 total births; low birth weight (<2500 g) and very low birth weight (<1500 g) proportions of live births are 5.5 and one percent respectively; 38% of infants are hospitalized; infancy child restraint use is 38%; and the smoking risk is 31% for women of reproductive age. These data are similar to Canadian experience, except for more frequent hospitalization of infants and for higher smoking by Nova Scotia women. Few definitive comments can be made, however, since the data often represent one year's experience and intraprovincial disparities in health outcomes are not examined.

The 1985 death rate in Nova Scotia for children aged 1 to 4 years was about 35 per 100,000; hospitalization of that age group was 15%; and child restraint use was about 52%. There is otherwise a paucity of health status information about this age group.

Nova Scotian school-aged children, aged 5 to 14 years, experienced a death rate of 27 per 100,000, of which 59% was injury-related; hospitalizations were about 7%; and child restraint use was 78%. Again, the data are sparse for this age group.

The death rate for adolescents, aged 15-19 years, in Nova Scotia for 1985 was 80 per 100,000 of which 75% was injury-related; 1985 suicide rates by male and female gender were 23.9 and 2.8 per 100,000 respectively (male rates were second highest of the provinces); hospitalizations rose to 9%; 4% of adolescent women became pregnant in 1985; and 19% smoked. The profile reminds us about how much we do not know about the state of adolescent health in the province.

The health status of poor children in Canada was examined separately in the CICH Profile. Poor children experience much higher rates of mortality and morbidity at all ages. Babies born into poor families are more likely to weigh less, be premature or have a growth abnormality. Since low birth weight is the greatest risk factor for infant mortality and disability, poor children bear this burden as well.[2] Income disparities in Nova Scotia are significant. We do not know the health impact of poverty in the province.

The *Report of the Nova Scotia Royal Commission on Health Care* devotes six pages to health indicators of Nova Scotians, four pages of which present graphs.[3] Its research study, *Health Status Indicators and Demographic Trends for Nova Scotia*[4] compiles vital statistical information on life expectancy, age-standardized death rates and potential years of life lost with survey data on disability, cigarette smoking, weekly alcohol consumption, and *Health Attitudes and Behaviour Study* data for Grades 4, 7 and 10.[5] This jumble of graphs hardly constitutes a provincial health profile.

The Royal Commission has recommended a new strategy for our health system whose guiding principles include health policies oriented to healthy outcomes and matching resources to health needs.[3] Without a provincial health profile evaluating at least 5 years' data at the county and health unit level, how can we know what health outcomes we are targeting; how can we know what health needs exist at the local level?

Health needs in the Western world are rarely identifiable through death certificate review. A provincial health profile could compile readily available data on hospitalizations; other institutionalization and agency use; physician and drug utilization; disability patterns; home care, social services and public health case loads; morbidity patterns by age and gender; and risk factor information from recently conducted surveys.[6,7]

We need a provincial health profile to tell us where we are and where we need to go. □

References

1. Delmore T. *Mortality in Niagara 1979 to 1986*. Regional Niagara Health Services Department, August 1989.
2. Avard D, Hanvey L. *The Health of Canada's Children: A CICH Profile*. Canadian Institute of Child Health, Ottawa, 1989.
3. *The Report of the Nova Scotia Royal Commission on Health Care, Towards a New Strategy*. Nova Scotia Royal Commission on Health Care, Halifax, December 1989.
4. Tregunna KT, Angus DE, Manga P. *Health Status Indicators and Demographic Trends for Nova Scotia, Final Report* in The Report of the Nova Scotia Royal Commission on Health Care, Towards a New Strategy, Research Studies, Volume I, Nova Scotia Royal Commission on Health Care, Halifax, December 1989.
5. Social Program Evaluation Group. *Canada Health Attitudes Behaviours Survey, 9, 12, 15 Year Olds: Nova Scotia Report, 1984-85*. Ottawa: Health and Welfare Canada, 1986.
6. Nova Scotia Department of Health and Department of National Health and Welfare. *Report of the Nova Scotia Heart Health Survey* (1986), Halifax, June 1987.
7. King AJC, Beazley RP, Warren WK *et al. Canada Youth and AIDS Study, Nova Scotia Report*. Ottawa: Federal Centre for AIDS, Health and Welfare Canada, 1989.

NON-RELATIVE OR THIRD-PARTY ADOPTIONS

Continued from page 67.

from one province to another. In some provinces, private non-relative adoptions are not permitted. If another province is involved, parties are advised to contact the Department of Community Services, Coordinator of Adoption Services for assistance prior to initiating any placement plans.

For further information, please contact Susan M. Drysdale, Coordinator of Adoption Services (424-3205) or Alex Shaw Q.C., Departmental Solicitor (424-3201), Department of Community Services.

If finding an approprite lawyer to handle this type of situation poses a problem, there is a lawyer referral service, provided via the Nova Scotia Barristers' Society by telephoning 429-9898. □

Obituaries

Dr. Donal B. Sheehan, (73) of Lower Clark's Harbour, N.S. died on January 17, 1990. Born in Ireland, he was a veteran of the Second World War serving in the Royal Army Medical Corps. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association until his retirement in 1984. He is survived by his wife, three sons, two stepsons, five daughters, and two stepdaughters. The *Journal* expresses sincere sympathy to his family.

Dr. Charles I. Cohen, (65) of Dorset, England died on January 18, 1990. Born in London he received his medical degree in London in 1947. He practised medicine in Nova Scotia for approximately ten years and continued his membership with The Medical Society of Nova Scotia and the Canadian Medical Association. The *Journal* extends sincere sympathy to his family.

Dr. John W. MacIntosh, (63) of Halifax, N.S. died on March 13, 1990. Born in Prince Edward Island he received his medical degree from Dalhousie Medical School in 1951 and has practised in Halifax since that time. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by his wife, and five sons, to whom the *Journal* extends sincere sympathy.

Dr. G. Richie Douglas, (76) of New Glasgow, Nova Scotia died on March 31, 1990. Born in New Glasgow, he received his medical degree from Dalhousie Medical School in 1939 and completed his fellowship in surgery in 1951. He was president of the Pictou Branch Medical Society and of the medical staff of Aberdeen Hospital. In 1977, he received the Queen's Silver Jubilee medal. In 1984, he received senior membership in The Medical Society of Nova Scotia and in 1987, he received senior membership in the Canadian Medical Association. He is survived by his wife, a son, and two daughters. Sincere sympathy is extended to his wife and family. □

THE MEDICAL SOCIETY OF NOVA SCOTIA

NOVA SCOTIA DIVISION OF THE CANADIAN MEDICAL ASSOCIATION

MEMBERS OF EXECUTIVE COMMITTEE

OFFICERS

President	P.D. Jackson
President-Elect	W.D. Canham
Past President (Immediate)	V.P. Audain
Chairman of the Executive Committee	G.A. Ferrier
Vice-Chairman	D. Ryan-Sheridan
Treasurer	R. Brewer Auld
Honorary Secretary	S.M.T. Leahey
Executive Director	H.M. Epstein

OBSERVERS AND STAFF

Economics Committee Chairman	R.J. Gibson
Director, Economics Department	R.J. Dyke
Director of Communications	D. Grant

BRANCH SOCIETY REPRESENTATIVES

Antigonish-Guysborough	M.A. MacKenzie
Bedford-Sackville	B.M. O'Hearn
Cape Breton	P.W. Littlejohn
Colchester-East Hants	G.A. Corbett
Cumberland	J.P. Donachie
Dartmouth	C. Gallant & M.F. Moriarity
Eastern Shore	D.R. Barnard
Halifax	J.J.P. Patil & D.C.S. Brown
Inverness-Victoria	C.W. Chow
Lunenburg-Queens	B.N. Chutskoff
Pictou	H.P. MacDonald
Shelburne	Gordon Hollway
Sydney	J.R. LeMoine
Valley	C.L. Smith & P.D. MacLean
Western	M.A.C. Duggan
Student Members	Diane McLennaghan, Sarah Kerr & Alexandros Alexiadis
I.R.A. Representatives	Andrew Orr, Daniel Vaughan

OBSERVERS

Editor-in-Chief — The Nova Scotia Medical Journal	J.F. O'Connor
Representative to Provincial Medical Board	
Medical Director M.M.C. Inc.	A.C. Watson
President & C.E.O. M.M.C. Inc.	D.L. McAvoy
C.M.A. Board of Directors	V.P. Audain
C.M.A. Council on Health Care	A.H. Patterson
C.M.A. Council on Economics	P.D. Muirhead
C.M.A. Council on Medical Education	G.L. Myatt
M.D. Management Limited	G.A. Sapp

STANDING COMMITTEES

	Chairman
Allied Health Disciplines	A.H. Shears
Archives	D.B. Hogan
Building	J.F. Hamm
By-Laws	P.W. Littlejohn
Child Health	N.P. Kenny
Community Health	D.B. Langille
Environment	D.B. Shires
Ethics	D.F. Fay
Gynecological Cancer (Screening Program)	R.C. Fraser
Home Care	D.P. Cudmore
Hospital & Emergency Services	D.M. Maxwell
Liaison Committees:	President
(i) Minister of Health	
(ii) Workers' Compensation Board	
(iii) Registered Nurses' Association	
(iv) Faculty of Medicine	
Maternal & Perinatal Health	A.C. Allen
Mediation	President
Medical Education	A.H. Murray
Membership Services	G.L. Myatt
Mortality Review (Appointed by and Reports Through Section of Anaesthesia)	K.W. Fairhurst
Occupational Health	R.E. Marchant
Pharmacy	S.G Carruthers
Professionals' Support Program	W.G. Gill
Risk Management	J.P. Anderson
Senior Advisory Committee	J.D.A. Henshaw

BRANCH SOCIETIES

	President	Secretary
Antigonish-Guysborough	J. Hamilton	S. Rappard
Bedford-Sackville	C.A. Forbes	J. Fraser
Cape Breton	D.P. Hickey	P.W. Littlejohn
Colchester-East Hants	G.A. Corbett	D.B. Langille
Cumberland	A. DeCroos	W. Smolonski
Dartmouth	G.G.R. Stewart	M.W. Ellis
Eastern Shore	D.P. Sinha	R.J. Fraser
Halifax	J.J.P. Patil	D.A. Stinson
Inverness Victoria	K.R. Murray	R.J. Genge
Lunenburg-Queens	D.W. Himmelman	G.W. Jansen
Pictou	A.K. Clark	M.N. MacDonald
Shelburne	I.C. Cree	Mark Riley
Sydney	F. Buhariwalla	M.A. Campbell
Valley	R.G. Bustin	R.C. Jung
Western	W.M. Gorman	A.M. Cottreau

SECTIONS

Anaesthesia	A.J.M. Clark	A. Finlayson
Emergency Medicine	D.E. Sinclair	L.M. Costello
General Practice	M.E. Lynk	S.A. Canham
Internal Medicine	J.L. Sapp	N.L. MacDonald
Internes & Residents	P.C. Champion	C. Zip
Obstetrics and Gynaecology	R.H. Lea	
Ophthalmology	D.M. Keating	F.J. Carpenter
Orthopaedic Surgery	J.L. Leahey	E. Howatt
Otolaryngology	C.C. Cron	G.F. Martin
Paediatrics	A.F. Pysemany	A.E. Hawkins
Laboratory Medicine	A.I. Bernardo	D.F. Glasgow
Psychiatry	J.V. O'Brien	P.A. Moss
Radiology	W. F. Barton	M.D. Riding
Surgery	A.J. Bodurtha	P.D. Roy
Urology	S.G. Lannon	R.W. Norman
Vascular Surgery	J.A.P. Sullivan	R.S. Dunn

GENERAL INDEX
VOLUME 68, 1989

The underlying value in the practice of science is truth; what achieves truth most efficiently is the most valuable. But the underlying value in the administration of science is utility; what is most useful, in addition to being true, is the most valuable.

Alvin Weinbert (1915-   )
Nuclear physicist