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EDITORIAL

The Physician: A Student, Then, Now and Always

This issue of the *Journal* has most of the articles written by medical students or residents, assisted often by more senior physicians. This continues the tradition of encouraging the younger members of the profession in their efforts at publishing.

Students at Dalhousie have not had a student publication to record results of their work since the *Dalhousie Medical Journal* ceased publication in the early 1970s. Although the work may not reach the standards of national or refereed journals it is often useful and timely, educational information.

The Nova Scotia Medical Journal often gives them their first opportunity to publish and a sense of accomplishment that is difficult to duplicate by other means. As we foster this educational process we can be reminded we are all still students or should be. As fellow students these young writers are an important part of our profession and deserve our support and encouragement.

This support and encouragement is being tested on a constant basis. Changing examination and licensing requirements and manpower controls presently being implemented challenge us to be both fair and protective of the student population.

Manpower controls, either now or will in the future, apply to all of us but at present the most severely affected will be the new graduates. These controls challenge our solidarity with our young colleagues and we must attempt to see that their interests are dealt with in a fair manner. After all, they are us.

When they entered medical school, they had expectations that they would have some choice in type and location of practice and at bare minimum an opportunity to work. That "implied" contract existed for anyone entering medical school in the last four years but may not exist any longer for new entrants to medical school. The Medical Society has some responsibility in aiding these students as the contract changes. To deny responsibility has been described as "eating our young" a quote that has been heard from more than one source.

Those currently in the system are owed our support and help as they try to achieve reasonable expectations. We cannot change the reality of decreased budgets but we can make adjustments. We should appreciate our Society's attempt to aid the young of our profession.

Meanwhile, we support and salute the efforts of students everywhere and in particular their efforts in this *Journal.* □

J.F.O'C.

Group A Streptococcus

CHANGING PATTERNS IN DISEASE MANIFESTATIONS

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There is increasing concern about the recent reemergence of group A ß-haemolytic streptococcal (GABHS) infection in a more virulent and invasive form. In the past, this pathogen was responsible for outbreaks of scarlet fever, suppurative infections including puerperal sepsis, endocarditis, empyema, and osteomyelitis, and non-pyogenic sequelae such as rheumatic fever and glomerulonephritis. With widespread use of antibiotics, GABHS infections became limited primarily to pharyngitis and impetigo. However since 1987, there has been an apparent increase in the number and severity of GABHS infections. The incidence of rheumatic fever, invasive infections, and toxin-mediated diseases all appear to be increasing. Each of these manifestations will be reviewed as well as the hypotheses that explain this change in GABHS epidemiology.

ACUTE RHEUMATIC FEVER (ARF)

Until recently, ARF had virtually disappeared from most developed countries. Since the mid-1980s, however, outbreaks of ARF have been reported in Ohio[1], Pennsylvania[2], Utah[3], and in military trainees.[4] In contrast to previous outbreaks in North America and elsewhere, which were associated with lower socioeconomic status and overcrowding[5], these recent outbreaks occurred in middle income children from suburban or rural areas. A nationwide survey in the United States suggested that the increases in ARF were focal, not widespread.[6] No increase in the incidence of ARF has been found in Canada.[7]

INVASIVE DISEASE

The increase in invasive disease caused by GABHS has been more notable and geographically widespread than ARF. Rates of both bacteremia and suppurative complications have risen. Proportionate increases in GABHS bacteremia have been reported from the United States[8-10], Great Britain[11], Sweden[12], and Canada.[13-15] Primary sources of infection were typically skin or soft tissue; mortality reached 35% in some series.[11] Metastatic spread of infection following bacteremia often led to other serious infections including osteomyelitis[9,10,17], septic arthritis[9,10,17], meningitis[10,16,18], myositis[19,20], peritonitis[10,17], and empyema.[10,17] Complications of invasive GABHS infection have been responsible for deaths during outbreaks at several nursing homes.[21] Of greater concern is the increased incidence of GABHS complications in previously healthy individuals. In a 10 year review of GABHS disease in Nova Scotia, a remarkable increase in the proportion of previously well children with invasive GABHS infection was demonstrated although the overall incidence of infection did not change[15].

TOXIN-MEDIATED DISEASE

Scarlet fever is the most common and well-recognized toxin-mediated disease caused by GABHS. GABHS produce three serologically distinct exotoxins (A,B, and C). The toxins share properties of pyrogenicity and the capacity for mediating shock and tissue damage. GABHS exotoxins have many similarities to the staphylococcal toxin that mediates toxic shock syndrome.[22] In 1988, Bartter *et al.* described three cases of what he referred to as "toxic strep syndrome".[19] An additional 20 cases were reported from the Rocky Mountain area of the United States in 1989.[20] Manifestations of the disease included hypotension, altered mental status, lethargy, nausea, fever, renal failure, adult respiratory distress syndrome, erythroderma, and desquamation. The illness was typically associated with a soft tissue focus of infection such as necrotizing fasciitis or myositis; the presence of bacteremia was variable. Similar cases have now been reported from British Columbia[13], Ontario[14], Nova Scotia[15] and elsewhere in the United States.[10,17,23]

PATHOGENESIS

The increased severity of GABHS infection may be a consequence of an increase in the virulence of the organism or changes in host characteristics. ARF always follows a pharyngeal infection with GABHS; serotypes associated with ARF typically are rich in M-protein, contain a large hyaluronate capsule, and have a mucoid colony morphology.[24] GABHS isolates from the recent ARF epidemics were found to be primarily of five serotypes: M-1, M-3, M-5, M-6, and M-18; 45% were identified as mucoid strains.[25] Similarly, a limited number of GABHS serotypes have been associated with bacteremia and other invasive infections. In several series of GABHS bacteremia, M-1, M-3 and M-18 have predominated over the formerly common M-4 and M-12.[26,27] Virulence of the M-1 serotype may also be augmented by production of a protease which enhances spread through tissue.[16]

The recognition of a new toxic shock-like syndrome caused by GABHS may also in part be related to both

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pathogen and host factors. Production of exotoxin A has been demonstrated from the majority of GABHS associated with the toxic shock-like syndrome.[20,28] This exotoxin was the predominant toxin in the pre-antibiotic era but had become relatively uncommon in North America. Production of this exotoxin might be associated with an increase in the virulence of the organism exacerbated by a population without protective antibody against the toxin because of its rarity over the last 50 years. Thus, the combination of a change in predominant GABHS serotypes (M-types) with a coincident change in the predominant exotoxin may produce severe infection in a population without protective serotype and exotoxin antibody.

IMPLICATIONS FOR THERAPY

The changes in GABHS disease have caused concern about the need for changes in treatment and management of GABHS infection. It is unlikely that antibiotic resistance is associated with the resurgence of GABHS disease. Although there have been reports of increasing rates of erythromycin resistance, GABHS remain exquisitely sensitive to penicillin which remains the drug of choice for primary GABHS infections.[29] Eradication of GABHS from the pharynx still requires prolonged rather than high dose penicillin therapy. Most GABHS infections will continue to be mild and easily treated with standard therapy. The practising physician, however, must diligently observe for any manifestations that would suggest more severe infection requiring more aggressive therapy. Soft tissue complications may require incision and drainage, while more serious infections such as fasciitis require aggressive surgical debridement and parenteral penicillin therapy. Toxin-mediated and other invasive infections often require the highly skilled supportive care only available in an intensive care unit.

SUMMARY

Significant changes are occurring in both the incidence and manifestations of GABHS infections. Increases in GABHS invasive disease and toxic syndromes are likely to have been caused because of a shift in predominant GABHS serotypes as well as from the population's susceptibility to these strains. Despite the changing characteristics of GABHS infections, management of streptococcal infections remains the same. However, physicians should be aware of the potential for invasive and fulminant complications and approach GABHS infections with caution. □

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The Impact of Bystander CPR in Out-of-Hospital Cardiac Arrest

(A LITERATURE REVIEW FROM THE ATLANTIC EMA-D NETWORK)

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Cardiovascular disease is very prevalent, especially in Western culture where lifestyles frequently include poor dietary habits and minimal physical activity. One half of all deaths in the United States can be attributed to this disease and predominantly present as sudden cardiac arrest.[1] Furthermore, 60-70% of sudden deaths occur out-of-hospital, away from immediate medical attention, but in the presence of bystanders.[1] In total, it is estimated that each year in the United States, close to 350 000 people die out-of-hospital from cardiac arrest.[2]

The aim of Emergency Cardiac Care (ECC) providers is to resuscitate victims of out-of-hospital cardiac arrest and to ensure the patients' survival with minimal or negligible neurologic impairment. Unfortunately, evidence suggests that only approximately 20% of victims can be successfully resuscitated and are alive upon hospital arrival.[3] Of those that are initially resuscitated, more than half then die in hospital.[3] And of those discharged from hospital alive, there is always the chance that neurologic damage has occurred, generally as a result of global brain ischemia.[4]

Overwhelmingly, research suggests that two factors primarily determine the outcome of cardiac arrest events: first, the response time to defibrillation by the emergency medical services (EMS) team must be adequately short in duration; and second, until EMS personnel arrive, tissue oxygenation must be satisfactorily maintained. Achieving some level of tissue oxygenation improves chances for resuscitation and delays the onset of neurologic damage. Research further suggests that on site cardiopulmonary resuscitation (CPR) is a useful method of ensuring the second requirement is met.

HISTORY OF BYSTANDER CPR

The first published data pertaining to the effects of CPR were animal studies performed in 1960 by Kouwenhoven *et al.*[5] Over a decade later, the American Heart Association advocated the use of CPR in Emergency Cardiac Care.[6] Its utilization in a pre-hospital setting by emergency medical teams eventually led to the training of other health care professionals, and even the general public in CPR administration — hence, the term "bystander CPR" was coined.

Early studies were almost unanimously supportive of bystander CPR and of its positive impact on the survival rates and neurologic outcome of cardiac arrest victims.[4,7,8] Recently, however, its effects have been disputed and this new controversy calls for the accumulation of more data along with a more detailed examination of CPR's effects.

CURRENT RESEARCH

Multiple variables influence survival from out-of-hospital cardiac arrest events. Among them are features of the emergency medical response as well as characteristics of the individual patient. Important features of the response include whether the arrest was witnessed and whether there was early administration of CPR and of Advanced Cardiac Life Support (ACLS). Characteristics of the individual are those such as age, gender, cardiac history, and post-arrest cardiac rhythm.[9] Due to the large number of variables at hand it is difficult to isolate, and discriminate between, the effects of individual factors. However, with accumulating data, the medical literature has consistently found the most powerful determinants of survival to be CPR and defibrillation response times, as well as the state of cardiac rhythm at the time of electric countershock.[10]

Best results appear to occur in arrests that are witnessed (so as to quickly summon medical aid), that have bystander CPR performed, and who receive electric countershock within six to eight minutes of collapse.[2,3,10-13] Furthermore, it is shown that the patient must be experiencing either pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF) at the time of ACLS arrival in order to restore spontaneous circulation through countershock. Those experiencing asystole or electromechanical dissociation (EMD) consistently tend to show poor outcomes.[3,10] Studies statistically show that bystander CPR, in association with quick response ACLS, can increase the chance of survival to hospital discharge from 5% (with medic-initiated CPR only) to 25%.[14,15]

It is important to note the exclusion criteria for studies in out-of-hospital cardiac arrest. All arrests that take place as a result of trauma, disease, hypothermia, hyperthermia, poisoning, or drug overdose should not be considered in the statistical analysis.

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Controversy over the impact of CPR tends to arise from studies of EMS systems with very short response times.[16-17] When ACLS arrival is speedy, bystander CPR appears to have no beneficial effect. In fact, the only apparent determinant of survival is the actual ACLS response time itself. Although this poses a discrepancy with previous findings, the usefulness of bystander CPR should not be readily underestimated. In fact, the basis for the controversy likely can be found if one examines the biological effects of CPR. Combining chest compressions with ventilation maintains a certain amount of oxygenated circulation in the patient and is thought to prolong the time of ventricular fibrillation. Thus, ventricular fibrillation cannot be viewed as an independent variable affecting survival, but as a dependent variable relying on early response time.[8,9] In instances where ACLS is provided quickly, the cardiac rhythm is more likely to still be ventricular fibrillation. When ACLS is delayed, the patient is likely to move from VF to asystole unless bystander CPR is performed. Therefore, as stated by Weaver *et al.*, "It is not who initiates such care, but instead how quickly resuscitative efforts are begun" which affects the outcome.[10]

Other than survival, the neurologic status of survivors is also of great importance. Improved neurologic recovery has been demonstrated with animal studies but the number of human studies that address this issue is small.[8] Nevertheless, it has been noted that patients who received early initiated CPR generally are more alert and have less neurologic dysfunction than their counterparts receiving delayed CPR.[8] In theory, closed-chest CPR should improve cerebral circulation, and should slow the effects of hypoxia on the brain. But in practice, this too has surrounding controversy. Experimentation with animal models shows that cerebral blood flow is not significantly augmented by CPR, thus the biologic basis for improved neurologic outcome has not yet been resolved. Furthermore, some have found no correlation between early intervention and brain function and contest the results that profess otherwise.[8]

FINDINGS IN ATLANTIC CANADA (THE ATLANTIC EMA-D NETWORK)

The Atlantic Emergency Medical Attendant-Defibrillation (EMA-D) Network consists of more than seventeen centres throughout the four Atlantic provinces that administer prehospital defibrillation in cardiac arrest situations. A data collection program has been established where each centre records pertinent information from all pre-hospital cardiac arrest events that meet the inclusion and exclusion criteria previously stated. Records are then submitted to a central data collection agency located in Halifax, Nova Scotia. In place since 1988, this agency acts to coordinate the efforts of individual EMA-D services. Here, data is analyzed and attempts are made to identify the impact of the various factors mentioned on survivorship as well as neurologic outcome.

Results representing the first 311 patients of this program demonstrate that less than 40% of all arrest patients receive bystander CPR. And although bystander CPR is "viewed as critical for improvement in survival" more data is needed to verify any statistical significance of its positive impact.[2]

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FUTURE GOALS IN BYSTANDER CPR

Three criteria must be met in order for on site bystander CPR to be effective. First, the general public must be trained to administer CPR effectively. Second, they must retain these skills. And third, they must be willing to utilize these skills when necessary.[8] The importance of focussing on bystander CPR is that it has been shown to be beneficial and it can be influenced by public policy.[17]

It has been documented that the effectiveness of CPR decreases markedly if it is administered poorly.[8] A more active effort should be made to provide training programs for lay persons to teach them proper CPR technique and to improve retention of what they learn. Furthermore, families of cardiac patients can be encouraged, through media and through the advice of medical professionals, to acquire these skills.[18] Lastly, the willingness to perform CPR, given the opportunity, could be best aided through media programs that promote and applaud bystander involvement in arrest situations.

SUMMARY

Although the impact of bystander CPR has posed somewhat of a controversy in certain instances, its influence on survival rates and on post-arrest neurologic outcome has been generally well documented. In order to globally improve results from cardiac arrest, bystander CPR should be encouraged – not only by future research into its benefits but also by public policy that supports and advises CPR training. □

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Bronchiolitis in Infants

AN UPDATE ON CLINICAL MANAGEMENT AND RESULTS OF A STUDY OF CHILDREN REQUIRING HOSPITALIZATION AT THE IWK 1988-1991

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Bronchiolitis is the most common lower respiratory tract infection in young children. Although only 1% of affected children will be ill enough to require hospitalization, certain underlying problems (eg. cardiopulmonary disease, prematurity, age less than 6 weeks) place some children at risk for serious morbidity. This paper reviews the clinical approach to bronchiolitis and presents the results of a retrospective survey of children hospitalized at the Izaak Walton Killam Children's Hospital with bronchiolitis due to respiratory syncytial virus from 1988-1991.

Bronchiolitis, characterized by rapid respirations, chest retractions and wheezing, is the most common lower respiratory tract (LRT) infection in young children. Epidemics occur yearly in the winter months during which bronchiolitis may account for 40 to 50% of all hospital admissions for respiratory distress in infants. Respiratory syncytial virus (RSV) is the most common cause of bronchiolitis. RSV infection develops in up to 50% of all infants in the first year of life, and by age 3 nearly all children will have been affected. Immunity is of short duration and repeated infections may occur.[7]

This paper will review the clinical approach to bronchiolitis in infants and present the results of a retrospective survey of children hospitalized at the Izaak Walton Killam Children's Hospital (IWK) from 1988-1991 with RSV infection to determine the outcomes in those at high risk for death or complications.

CLINICAL APPROACH TO BRONCHIOLITIS

Bronchiolitis presents initially with upper respiratory tract signs of rhinitis and congestion, followed in a few days by LRT signs of wheezing, hyperinflation, chest retractions and tachypnea. Fever and crackles may or may not be present. Moderately severe hypoxia may exist without cyanosis; tachypnea is the best single clinical indicator of this state. Apnea may occur in up to 20% of infants.

Cytolytic change and inflammation in the respiratory epithelium of the bronchioles constitute the pathophysiology of bronchiolitis. Airway obstruction is due to edema and plugs of cellular debris, fibrin and secretions. These changes result in increased airway resistance, decreased airway compliance, hyperinflation of the lung and hence increased work of breathing.[2]

The etiologic agents in bronchiolitis include RSV, parainfluenza virus 1 and 3, influenza virus, adenovirus, rhinovirus and occasionally Mycoplasma pneumoniae. RSV accounts for 60-90% of all cases. Infection is transmitted by direct or close contact with nasopharyngeal secretions. Viruses can persist for minutes to hours on hands and for several hours on environmental surfaces. Self-inoculation and transmission of infection can be prevented by careful hand washing before and after patient contact. The incubation period for most of the respiratory viruses causing bronchiolitis is usually less than a week.

Diagnosis is made by observation of the aforementioned clinical findings in young children. A chest roentgenogram usually shows hyperinflation with or without some patchy atelectasis. Specific viral diagnosis is not necessary in children well enough to be cared for as outpatients as management will not be affected. Infants with respiratory rates >60/minute are probably hypoxic; a simple noninvasive method to measure oxygen saturation is pulse oximetry. Outpatient management of the infant with mild respiratory distress (playful, alert child with respiratory rate between 40 and 50/min, no or minimal subcostal retractions) who does not fall into a high risk group (see below), consists of frequent small feedings and monitoring. Infants early in their illness should be reevaluated in 24 to 36 hours. A parental educational sheet to assist in ambulatory management is seen in Appendix 1.[3]

In children requiring hospitalization because of risk factors for a complicated course or support for respiratory distress, specific viral diagnosis is worthwhile. A nasopharyngeal aspirate is taken for antigen detection or viral culture. The tubing and syringe from the aspirate may be submitted immediately to a viral laboratory or inoculated in viral transport media if there is to be a delay in delivery of the specimen. In the child ill enough with bronchiolitis to be hospitalized in whom RSV infection is diagnosed, ribavirin (Virazole® (lyophilized), ICN Canada Ltd., Montreal, PQ) antiviral therapy may be used and these children should be cohorted appropriately to prevent nosocomial transmission. Those recog-

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nized at increased risk of complications such as apnea, respiratory failure or air leaks are children with cardiac or pulmonary disease, immunodeficiency, age less than 6 weeks or prematurity. Children who are hypoxic at first presentation are probably also at increased risk for more serious illness. Therapy is primarily supportive. Hypoxemia should be corrected with warm humidified oxygen supplied by hood, tent or nasal prongs as appropriate. Hydration can be maintained by intravenous fluids if the child is in too much respiratory distress to breast or bottle feed. Children with severe distress should be managed in an intensive care unit in case intubation and assisted ventilation are required.

The role of aerosolized bronchodilators remains unresolved.[4-6] A trial of aerosolized salbutomol or albuterol (0.01-0.03 ml of inhalation solution/kg/dose q 1-4 hours) may be given; if no improvement is noted in 2 doses it should probably be discontinued. Steroids have not been shown to be beneficial. Because bronchiolitis in infants is almost exclusively caused by viral infections antibiotics are of no value.

Children in high-risk groups may benefit from specific antiviral therapy with ribavirin. This drug, administered by a small particle aerosol generator, results in small to moderate improvements in rate of recovery and oxygenation. A 5-day course of ribavirin at the IWK costs about $2,000. Such patients usually require one-on-one nursing care to monitor respiratory distress and precipitation of the drug in tubing and on oxygen hoods. Because improvements with the drug are small, its cost high and the long-term effects unknown, ribavirin is only indicated for infants with severe illness or those considered high-risk.[7]

SURVEY OF CHILDREN REQUIRING HOSPITALIZATION WITH RSV BRONCHIOLITIS AT THE IWK CHILDREN'S HOSPITAL.

RSV is the single most common viral agent causing bronchiolitis in infancy. Although only 1% of RSV infected children will be so severely affected to require hospitalization, morbidity may be considerable, particularly in the high-risk groups previously mentioned. Up to 37% of infants with congenital heart disease may die during an episode of RSV bronchiolitis.[8] Because the original studies documenting severe morbidity and mortality were done in the United States over a decade ago, a group of Canadian pediatric infectious disease specialists, the Pediatric Investigators Collaborative Network on Infections in Canada (PICNIC), decided to complete a retrospective chart review of all children hospitalized with RSV from 1988-1991 at participating pediatric hospitals to determine the outcomes in children at high risk for death or complications.

The following are the results of the review conducted at the IWK Children's Hospital, a university-affiliated pediatric hospital in Halifax, Nova Scotia which serves the Maritime provinces. The combined results of the PICNIC study have been published elsewhere.[9]

METHODS

The charts of all patients discharged from the IWK with a diagnosis of "acute bronchiolitis" between 1 April and 31 March 1991 were reviewed. Patients were included if RSV infection was diagnosed by an antigen detection test or viral culture and one or more of the following risk factors were present: 1) congenital heart disease; 2) chronic lung disease; 3) immunodeficiency; 4) age < 6 weeks; 5) gestational age < 36 weeks; and 6) hypoxia (defined as oxygen saturation < 90% or pO_2 < 60 mmHg). The use and duration of oxygen supplementation, ribavirin, bronchodilator therapy, intensive care placement and ventilatory support were noted. Death within 2 weeks of RSV diagnosis was recorded.

RESULTS

There were 581 children with bronchiolitis admitted to the IWK during the study period. Eight charts could not be obtained; therefore 573 were reviewed. Fifty-seven percent of these had RSV infection (326/573); the rest had bronchiolitis for which an etiologic agent was not found or which was due to another respiratory pathogen. No children with immunocompromise were identified. Of those with RSV 44% (143/326) fulfilled inclusion criteria.

The most common risk factor for morbidity with RSV was hypoxia at presentation (Figure 1). Two or more risk factors were present in 36% of patients (51/143). The average age was 5 months (152.3 days). Underlying types of cardiopulmonary disease are seen in Table I. Only one child with cardiac disease had documented pulmonary hypertension; 50% (15/30) had left-to-right shunt. One patient with chronic lung disease had pulmonary hypertension.

TABLE I

TYPES OF CONGENITAL HEART AND LUNG DISEASE IN CHILDREN HOSPITALIZED WITH RSV INFECTION AT THE IWK APRIL 1988-MARCH 1991

Congenital Heart Disease Lesion	Number
Atrial-septal defect	8
Ventriculoseptal defect	6
Patent ductus arteriosus	7
Outflow tract stenosis	
aortic	2
pulmonic	4
Other	3
	30

Lung Disease	Number
Bronchopulmonary dysplasia	2
Cystic fibrosis	1
Chronic aspiration	1
Other	2
	6

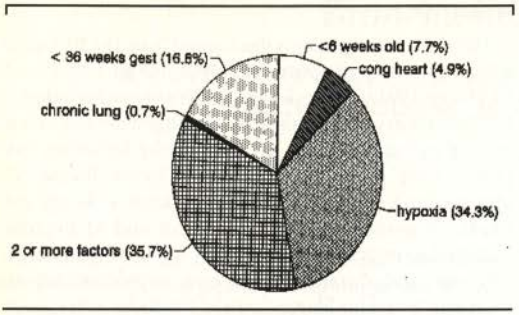

Figure 1

Risk factors for morbidity
with RSV infection: IWK 1988-1991

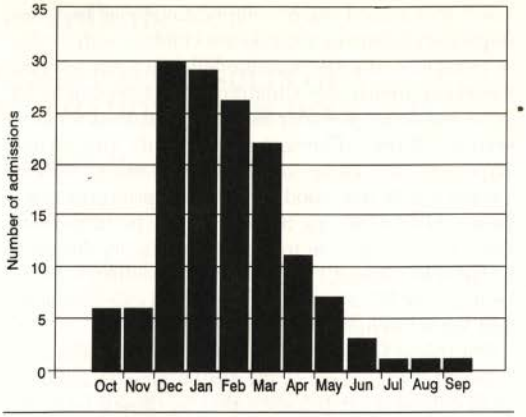

Figure 2

RSV bronchiolitis admissions
to IWK 1988-1991 by month

The seasonal distribution of patients over the 3 seasons is seen in Figure 2. The number of cases per season were 17 in April 88-March 89, 76 in April 89-March 90 and 50 in April 90-March 91.

Children with two or more risk factors compared to children with only one were more frequently admitted to the intensive care unit 12/51 (23.5%) vs. 4/92 (4.3%) requiring of mechanical ventilation 3/193 (2.1%) vs. 0/92 (0%) and days of intravenous fluids 32/143 (29%) vs. 17/92 (18.5%). Twelve of 143 children (8%) were given ribavirin therapy; 8 of these had 2 or more risk factors. There were no deaths within 2 weeks of diagnosis.

DISCUSSION

The age of patients affected and seasonal distribution of RSV infection in Nova Scotia is similar to the rest of North America.

About half of patients with RSV bronchiolitis ill enough to require admission did not have identifiable risk factors for morbidity and mortality. We did not examine the hospital course of these infants as they did not fulfill study

TABLE II

OUTCOME MEASURES IN 143 "HIGH-RISK" CHILDREN WITH RSV INFECTION HOSPITALIZED AT THE IWK 1988-1991

Risk factor	IV fluids (average no. of days)	Oxygen Supplementation (average no. of days)	Admission to Intensive Care Unit	Mechanical ventilation	Average length of stay (average no. of days)
Congenital heart disease (N=7)	4.0	5.9	1	0	8.7
Chronic lung disease (N=1)	0	15*	0	0	30*
Prematurity (N=)					
>6 weeks (N=11)	4.5	2.8	0	0	6.2
Hypoxia (N=49)	3.0	3.1	3	0	4.9
≤ 36 weeks gestation (N=24)	1.7	3.4	0	0	6.7
2 or more risk factors (N=51)	7.0	4.1	12	3	10.4

*This group is distorted because there is only one patient in it.

criteria but it is clear that previously well children with acute RSV bronchiolitis may require oxygen supplementation, intravenous fluids or other support. Careful history and clinical examination with particular attention to the relationship between tachypnea and hypoxia will identify these children in respiratory distress.

The presence of more than one risk factor appears to increase morbidity and requirements for all types of intervention reviewed. The overall mortality rate, however, is much lower than previously reported. No patients died within 2 weeks of diagnosis at the IWK; in the Canada-wide study the mortality rate was 1% (17/1584). Possible explanations for this include underestimation of deaths by the set time limit, or improvements in the treatment of cardiac and pulmonary disease since the publication of earlier studies that allow these infants to better handle intercurrent illness. As well, awareness of the role of respiratory infections in exacerbating chronic cardiopulmonary disease may mean patients are brought to medical attention earlier.

SUMMARY

Bronchiolitis is the most common lower respiratory tract infection in young children and occurs predictably every winter in epidemics of varying severity in Nova Scotia. The child with rapid respirations, chest retractions and wheezing should be assessed for hydration, ability to maintain fluid intake, hypoxia and other signs of respiratory distress and the presence of risk factors for severe disease (cardiac or lung disease, immunocompromise, prematurity, age less than 6 weeks). Previously well children with mild disease can be followed on an outpatient basis and reassessed in 24 to 36 hours for progression of disease. Children with hypoxia or other signs of moderate to severe respiratory distress or risk factors for severe disease should be admitted for supportive care, viral diagnosis and consideration of ribavirin therapy if RSV is identified. A pediatrician should be involved in the care of high risk children. Although only a small percentage of RSV infected children will develop severe enough disease to require hospital care, they are at high risk for respiratory failure, exacerbation of underlying cardiopulmonary disease, apnea and other problems. Early identification of these high risk children can decrease morbidity and mortality. □

APPENDIX I – See pages 168 and 169.

ACKNOWLEDGEMENT

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Continued from page 163.

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INFORMATION FOR PARENTS AND PATIENTS

Bronchiolitis

General Information

Bronchiolitis is an illness of young infants affecting the entire respiratory tract, but primarily the smallest air passages in the lung (the bronchioles). It is caused by several different viruses which also cause colds and flulike illnesses. Respiratory syncytial virus, often called RSV, is the most frequent cause and parainfluenza viruses the second most common.

Young children catch these viruses through close contact with others who are infected. Often these are older children and other family members with mild illness or only a cold. The virus is spread when infected mucous is sneezed or coughed into another child's face or onto table tops or objects such as toys. Infection occurs when the child touches these surfaces and then his/her eyes or nose. Illness begins about 3 to 7 days later.

The Illness

Bronchiolitis usually starts as a cold, accompanied by fever and nasal stuffiness. After 2 to 4 days the virus spreads down to the bronchioles, causing irritation and narrowing of these air passages. This causes the child to cough and produce a whistling sound (wheeze) when breathing out. Some children appear to be having an asthma attack as their breathing becomes more rapid and labored and the cough more juicy or hoarse ("croupy"). Fever may still be present but has often disappeared by this time. Most children have a poor appetite and infants may have difficulty sucking, especially for prolonged periods. Restless sleep with frequent awakening is common.

Wheezing usually gets better after 3 to 5 days; however, nasal stuffiness may last longer and cough may persist for another 1 or 2 weeks.

When To Call Your Doctor

You should call our office if you feel frightened or worried about your child's illness.

Call our office right away if your child has: increasing difficulty breathing; severe sucking in of the spaces between the ribs with each breath; very fast breathing (over 60/minute); a bluish tinge around the lips; difficulty staying awake.

Call us during regular office hours (or on weekends) if your child: refuses to drink; has fever over 102°F (38.9°C); complains of an earache or, in an infant, pulls at the ears or becomes increasingly cranky.

Treatment

Most infants with bronchiolitis do not need specific medications but rather require patience and care to make breathing and drinking less difficult. Using a vaporizer or humidifier in the room where the child sleeps will help keep nasal secretions moist and the nasal passages clear. Cold water vaporizers are preferable; those using hot water can cause burns if pulled over by an inquisitive infant or toddler.

The nose may be suctioned every few hours with a rubber bulb (called an ear syringe), especially prior to feeding and sleeping. Saltwater nose drops (¼ teaspoon table salt in 1 cup of water) placed in the nasal passages before suctioning will help liquify and clear the secretions. Elevating the head of the bed or crib during sleep may also improve the clogging of the nasal passages.

If your child appears to have difficulty feeding or sucking, offer smaller feedings more frequently. It is more important to drink liquids, such as juice or soft drinks, than to eat solid food during the early part of the illness.

Editor: S. Michael Marcy, M.D. Associate Editors: Michael E. Pichichero, M.D. and Richard H. Schwartz, M.D.

Acetaminophen (e.g. Tylenol®, Tempra®, Panadol®, Liquiprin®) can be used for fever over 101°F (38.3°C). Aspirin should not be given to children with viral infections.

In some cases your doctor may prescribe treatment for coughing or wheezing. Do not give your child any medicines for colds or asthma without first checking with your doctor; they could be harmful.

Because bronchiolitis is caused by viruses, antibiotics are of no use. Your child may, however, need antibiotics if s/he has an associated bacterial infection such as an ear infection.

You generally do not need to restrict your child's activity. Most children will adjust their activity according to how they feel.

Contagion

Children with bronchiolitis are most apt to spread the virus to others during the first days of their illness when they have fever and coldlike symptoms, especially sneezing and coughing. Mucous secretions from the nose and mouth can, however, be contagious for a week or more.

Little can be done to prevent young children from spreading or acquiring viral respiratory infections. Disposing of dirty facial tissues promptly and properly, along with good handwashing, can help prevent spread of infection among family members.

Return to Group Activities

Infants and young children can return to day care or other group activities when they have no fever, feed normally and feel well. A lingering cough or runny nose is no reason to keep them at home.

Common Concerns

Coughing helps your child clear the airways and should not be suppressed with strong cough medicines. Overly vigorous use of decongestants for treatment of nasal stuffiness should also be avoided. These drugs may make the secretions thick and can have unwanted side effects in young children.

A small object inhaled into the lungs can occasionally cause wheezing which sounds just like bronchiolitis. If difficulty breathing occurred suddenly or if your child was playing with a small object right before the illness began, let your doctor know.

Recheck Appointment

Make an appointment to be seen in _____ days.

Not necessary _____

Other _____

_____

_____

_____

_____

_____

Further Advice from Your Doctor

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Coal Workers' Pneumoconiosis and Compensation in Nova Scotia

ARE WE USING ALL THE AVAILABLE TOOLS IN ASSESSING DISABILITY?

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Radiography and pulmonary function testing are valuable tools in the assessment of such occupationally related lung diseases as coal workers' pneumoconiosis. However, because of evidence suggesting that correlation between symptoms, pathology, pulmonary function testing and radiography is often less than perfect, concerns have been raised about the exclusive reliance on these tools in the assessment of disability. This review describes the guidelines by which disability claims of coal workers with respiratory complaints are adjudicated in Nova Scotia. Also examined is the validity of the preeminent position occupied by the chest x-ray and resting pulmonary function tests in this process. Finally, our present understanding of the uses and limitations of exercise testing in the compensation process is summarized. As a result of numerous revisions, it is probable that the present system is designed appropriately to serve most miners who seek compensation for respiratory disability. However, in certain instances, when the chest x-ray and PFTs fail to resolve the issue of impairment and disability, exercise testing as a form of assessment might be warranted.

The coal mining industry has had a major influence on the social and economic development of Nova Scotia. Coal mining in the province dates back to 1720, when a mining operation was begun to supply the French fortress at Louisbourg. The first officially recorded export of minerals from Canada followed in 1724 with a shipment of Cape Breton coal to Boston. By 1870 some 20 collieries were in operation in Nova Scotia, and when the industry reached its peak in the mid-1940s, about 10 000 men were employed underground in 49 collieries.[1,2] Since the late 1950s, coal mining in Nova Scotia has experienced a sharp decline as less expensive, alternative forms of energy have replaced coal in many of its traditional and domestic markets. In 1991 only four underground coal mines were in operation, and this number was further reduced recently when an explosion forced the closure of the newly-opened Westray Mine in Plymouth.

The coal mining industry's contribution to the Nova Scotian economy has not been without human cost. Its history is punctuated with stories of tragic accidents and heroic rescue efforts. Equally disturbing, though perhaps less well publicized, is the high prevalence of disabling respiratory disease in workers employed in the mines. Improved ventilation of mine shafts and the adoption of rigorous air quality standards have undoubtedly lessened the risk of developing lung disease. However, the prevalence of respiratory diseases such as pneumoconiosis remains significantly greater in coal workers than in workers not exposed to dusty environments.[3-6]

COAL DUST AND RESPIRATORY DISEASE

Coal workers' pneumoconiosis (CWP) is a disease of the lung interstitium, resulting from the body's response to inhaled coal dust. Workers affected by CWP often present with dyspnea, cough and sputum,[7-9] and may have concurrent chronic bronchitis and emphysema.[10,11] While the pathogenesis of CWP is incompletely understood, a direct relationship has been demonstrated between inhaled dust and the disease that results.[4,11] Dust dose and composition do not appear to account wholly for variation in the prevalence of CWP: other factors such as age, health and even ethnicity of the coal worker may also be involved.[9]

The characteristic pathological hallmark of CWP is the *coal macule*, which consists of a reticulin mesh enclosing a mass of dust particles, fibroblasts and dust-laden macrophages.[7,12,13] It has been postulated that, combined with interstitial thickening and desquamation, this may account for the reported diffusion impairment and airway obstruction seen in patients with CWP.[14-17] Respiratory symptoms have also been attributed to the involvement of smaller, peripheral airways, [6,18] and to ventilation/perfusion discrepancies. [19,20] Mechanical changes, such as reduced elastic recoil of the lungs, have also been implicated.[21,22]

Physical signs of CWP are non-specific, and consequently the diagnosis is usually made on the basis of radiographic appearance.[13,23] For clinical purposes, coal workers' pneumoconiosis is categorized as either *simple* (with nodular interstitial disease) or *complicated* (with progressive massive fibrosis). The distinction is made on the basis of the chest x-ray, according to guidelines set out in 1980 by the International Labour Organization.[24] Since there is a definite relationship between the extent and profusion of the radiographic opacities in the lung fields and the coal dust content of the lungs, the chest

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radiograph offers a means of assessing the amount of dust retained by the lungs.[25] Simple CWP refers to the presence of small, diffuse opacities on chest x-ray films, and is further subdivided into three radiological categories based on the size and extent of nodulation present. *Progressive massive fibrosis* refers to a more severe form of the disease in which the interstitium of the lung is replaced with extensive scar tissue, appearing on the chest x-ray as large dense masses in the upper lobes. This type of pneumoconiosis can be complicated by tuberculosis and may progress even after exposure to coal dust ceases.[26]

COAL WORKERS' PNEUMOCONIOSIS AND COMPENSATION

Because of the various respiratory complaints that arise from occupational exposure to coal dust, coal workers often seek disability benefits. Consequently, physicians who deal with occupationally-related disease must have a clear understanding of the distinction between impairment and disability.[23] *Impairment* is defined as a reduction in organ function, and in the case of the lungs refers to measurable abnormalities of pulmonary function. Evaluation of patients with respiratory complaints usually entails the use of resting pulmonary function tests (PFTs) to determine whether or not a significant degree of respiratory impairment is present.

Disability on the other hand, refers to the loss of ability to work and carry out the normal duties of one's job. In effect, occupational disability is equated to a loss of earning power. While assessment of impairment focuses on physiologic abnormality, the assessment of occupational disability must consider multiple factors, such as job requirements, social and economic conditions, age and gender of the worker, and the workers' compensation program under which the patient may be entitled to receive benefits.[27] One of the many challenges faced by the architects of the compensation process, then, is to decide the level of impairment that constitutes disability, and to define how such impairment will be measured.

In Canada, workers' compensation falls under provincial jurisdiction and the guidelines differ somewhat from province to province. Disability claims in Nova Scotia are adjudicated by the Workers' Compensation Board (WCB) of Nova Scotia according to regulations contained in the WCB policy statement, and the *Workers Compensation Act* (revised, 1981). Currently, a miner in Nova Scotia who wishes to file a claim for disability resulting from coal dust exposure may proceed in one of two ways. The first of these is defined by Section 9A of the *Workers Compensation Act*, the so-called "Automatic Assumption" clause. Section 9A was amended to its present form in 1981, and states that any miner who has a twenty year history of coal dust exposure, as well as a *demonstrable loss of lung function*, will be compensated according to his disability. In order for loss of lung function to be established, the applicant must display a certain degree of abnormality on pulmonary function testing.

For compensation purposes, the forced vital capacity (FVC) and the ratio of forced expiratory volume in one second to forced vital capacity (FEV_1/FVC) are the principal pulmonary function parameters of interest. The FVC is reported both as an absolute value and as a percentage of the normal expected value, taking into account the subject's age, sex and height. Many pathologic processes can lead to a decrease in FVC, including obstructive and restrictive lung disease. The FEV_1/FVC is reported as a percentage, and in individuals with normal lung function it is usually in excess of 75 %. Lower values suggest the presence of obstructive lung disease.

The disability pension itself is decided on the basis of a formula that takes into account the most recent yearly salary earned while working underground. For disease of obstructive origin, the award is based on the numerical value of FEV_1/FVC. Coal miners with restrictive disease receive benefits based on the level of their FVC, as a percent of the predicted value (Table I).

TABLE I

NOVA SCOTIA DISABILITY AWARDS FOR COAL WORKERS WITH PNEUMOCONIOSIS UNDER THE AUTOMATIC ASSUMPTION CLAUSE

FEV_1/FVC	FVC	Class	Level of Impairment	Award*
≥ 70%	≥ 85%	I	none	0
60-70%	80-85%	II	mild	10%
55-60%	75-80%	III	moderate	20%
50-55%	60-75%	IV	severe	35%
<50%	<60%	V	very severe	60%

*refers to percent of most recent yearly salary while employed underground in coal mine

If the miner does not qualify under the Automatic Assumption criteria, he may still file for a disability pension according to General Claim guidelines outlined in Sections 74 to 82 of the *Workers' Compensation Act*, and further described in the WCB policy manual. Under these guidelines, there is no minimum exposure period to coal dust, but a miner must exhibit evidence of at least category 1 nodular markings on chest x-ray. If either the WCB physician or one of two consulting chest physicians judge that category 1 nodularity is present on the claimant's x-ray, the miner is awarded a disability pension. The amount of the award varies and, unlike claims disbursed under the Automatic Assumption clause, is not based solely on impairment of pulmonary function. If there is no PFT abnormality, a disability pension of 10 % is often given regardless. If neither the WCB physician nor the two chest physicians find evidence of category 1 nodular markings, the coal worker's claim is dismissed.

Since the eligibility for compensation depends on x-ray appearance and on the results of pulmonary function testing, the adequacy of the present compensation guidelines, in ensuring that all those with occupationally-related respiratory disability have an opportunity to receive appropriate compensation benefits, has been called

into question. Automatic assumption is limited to miners with at least 20 years of mining exposure, and those who do not meet the 20-year criterion must demonstrate radiographic abnormalities to qualify for compensation. For many miners, therefore, the outcome of the compensation claim is dependent entirely on the radiographic diagnosis of CWP. If nodular opacities are present, then symptoms are attributed to pneumoconiosis. If the opacities are absent, symptoms are frequently attributed to non-occupational factors. These include smoking which, because of its high prevalence in the coal miner population, often confounds the assessment of occupationally-induced respiratory disability.[28]

While many symptomatic coal miners exhibit some abnormality on chest films or PFTs, some complain of dyspnea and cough but fail to meet WCB criteria for x-ray abnormality or PFT impairment. For these individuals, this lack of objective findings can lead to financial hardship, since many feel that they are unable to continue working underground.

RADIOGRAPHIC INVESTIGATION

While the plain chest x-ray has long been accepted as a suitable tool for the diagnosis of CWP, it has been suggested that exclusive reliance on radiography may not be appropriate. For example, the International Labour Organization (I.L.O.) convention that physicians use to assess radiographs for the presence of pneumoconiosis was originally developed for the purpose of standardizing epidemiological investigations.[24] It was never intended to be used for individual case determinations in compensation proceedings, and "a logical dilemma is thus raised through application of a standard in one context (the compensation environment) when it was intended for use in another (investigation of disease occurrence in populations)".[13] Consequently, many authors have called for a revision of the I.L.O. classification, citing such problems as ambiguous instructions for x-ray readers and poor definition of normal/abnormal films.[24,29] Since chest x-ray interpretation can be very subjective, readers frequently disagree among themselves, and even with their own interpretations when films are viewed at different times.[24,31] To quote one author, "CWP, like beauty, is in the eye of the beholder".[30] Other factors related to the subject, such as age, obesity and inspiratory effort also influence x-ray appearance, as do film quality and exposure.[12]

In addition to inconsistencies that arise from x-ray interpretation, there is evidence to suggest that interstitial lung disease may be present in subjects with apparently normal chest films. In a study of 219 insulation workers who died of lung cancer following development of asbestosis, 18 % showed post-mortem evidence of interstitial pulmonary fibrosis even though fibrosis had not been detectable radiographically.[32] Similar findings were reported in a group of patients with various occupationally-induced interstitial disorders. Of 458 patients with histologically confirmed interstitial fibrosis, 44, or 9.6 per cent, had pre-biopsy films that were read as "normal".[12] A review of ten other studies that examined this question revealed similar findings: pathological evidence of parenchymal lung disease is not infrequent in patients who have apparently normal lung fields on the chest x-ray.[33,34]

Several explanations have been proposed for the finding of normal radiographs in patients with histologically recognizable diffuse interstitial disease.[12] There is the possibility that multiple isolated lesions individually are too small to cast a radiographic density unless a minimum number and size of nodules has developed, or that diffuse interstitial thickening characteristic of occupational pulmonary disorders is not easily appreciated visually on x-ray films. Therefore, radiographs lacking evidence of interstitial disease do not necessarily exclude the presence of early interstitial fibrosis. In light of these findings, the reliance on x-rays as a means of assessing occupational lung disease disability claims may deserve re-evaluation.

PULMONARY FUNCTION TESTING

The presence of impaired pulmonary function in coal workers has long been recognized, and a direct correlation between respirable dust levels and FEV_1 has been documented.[4,10,17] It has also been suggested that PFTs may be sensitive indicators of dust-induced alterations of lung biology prior to the development of radiographic abnormalities.[13,33] A reduced diffusing capacity in non-smoking miners, for instance, has been demonstrated even though chest films showed no radiographic evidence of pneumoconiosis.[6] Similar discrepancies in correlation have been noted with other inhaled irritants such as asbestos fibres and carbon black dust.[5,35,36] It therefore seems likely that in certain instances, the decline in pulmonary function is unrelated to radiographic evidence of interstitial disease.[28]

Although there is little doubt that PFTs play an important part in evaluating disability in coal miners who file claims, pulmonary function testing is not without its limitations. There is, for instance, ongoing disagreement as to which measure of pulmonary function correlates best with disability, and which level of functional impairment constitutes disability. Since many PFT indices are effort-dependent, there is the possibility that workers applying for disability compensation will attempt to malinger PFT measurements. Moreover, the relative effects of cigarette smoking and occupational dust on pulmonary function remain controversial.[6,15] Because cigarette smoking is still perceived as a significant contributor to the respiratory impairment seen in coal miners, it seems likely that pulmonary function testing will continue to enjoy only a limited role in the disability assessment process.

In spite of its possible limitations, it is clear that pulmonary function testing can reveal information about respiratory fitness that radiography cannot. The reverse is likely also true, and there are certain to be instances in which neither PFTs nor radiography show perfect correlation with disability. It has been suggested, therefore,

that the evaluation of an individual's disability claim should make use of all appropriate diagnostic procedures without placing any one procedure in a preeminent position.[13] With this in mind, the role of exercise testing in disability assessment may deserve more careful consideration.

EXERCISE TESTING

Exercise testing is used frequently in the evaluation and quantification of disease involving both cardiovascular and respiratory systems, since functional impairment in either system may affect overall exercise performance.[37] Since exertional symptoms during exercise precede symptoms at rest, exercise testing can also uncover cardiovascular and lung disease at an early stage. In addition, exercise testing is useful in isolating the causes of common complaints such as exertional dyspnea when they are not obvious from routine clinical and laboratory assessment.[20]

The response to exercise in subjects with interstitial lung disease has been the focus of several recent studies. In a large group of patients with various forms of interstitial and destructive lung disease, exercise testing demonstrated that the most striking abnormality imposed was a decrease in oxygen transfer.[16] Individuals studied showed dramatic reductions in PaO_2 induced by exercise, and demonstrated marked exercise-induced changes in the alveolar-arterial oxygen pressure difference ($AaDO_2$). Similar results have been reported in groups of patients affected by asbestosis and silicosis.[39,40] Moreover, the response to exercise in subjects with silicosis was found to be essentially identical in smokers and non-smokers.[40]

It has also been suggested that "in the early roentgenographic stages of pulmonary silicosis, no relationship exists between exercise capacity and resting pulmonary function, symptoms, x-ray profusion or smoking habit".[40] These findings deserve closer scrutiny for two reasons. First, they suggest that certain measurements obtainable by exercise testing may be independent of the effects of smoking history. In addition, these data indicate that impaired capacity to perform exercise can be present even in the absence of significant x-ray and PFT abnormality. Therefore, instances when exercise testing is a more effective indicator of disability than either PFTs or x-rays probably exist. For this reason, exercise testing may have a role in the assessment of workers who complain of respiratory symptoms but whose chest x-ray and baseline studies of pulmonary function show no significant abnormalities.

In Nova Scotia, exercise testing is not used routinely to evaluate respiratory impairment in patients with symptoms of pneumoconiosis. To a certain extent, this is probably because it has been assumed that the maximum exercise performance is difficult to assess in patients seeking compensation.[40] While poor motivation during testing is a frequently suspected phenomenon, it has been argued that its occurrence is usually overestimated and can often be detected.[39] A recent review of exercise testing in assessing occupationally-related respiratory impairment addressed some of these concerns. According to the authors, exercise testing "may be superior to static tests for determining whether a worker can perform a specific job, since it measures the ability of the body to meet O_2 demands as they vary with exertion".[8] For this reason, the American Medical Association has advocated exercise testing for patients who complain that they are unable to meet the demands of their specific job.[8]

Although a sensitive means of detecting early disease, exercise testing too has its disadvantages. It is time-consuming, expensive and requires considerable expertise in its interpretation.[40] Certainly, it is not to be advocated in all cases where occupationally-induced respiratory disability is suspected. In selected circumstances, however, when conventional radiographic and resting pulmonary function studies have failed to clarify the cause of the miner's respiratory symptoms, exercise testing may help to establish the presence, nature, and extent of any underlying disturbance in cardiopulmonary physiology.[41]

CONCLUSION

Since its beginnings, the compensation process for Nova Scotian coal miners with respiratory complaints has undergone significant revision. The introduction in 1981 of the Automatic Assumption clause widened access to disability pensions for miners who lacked clear-cut radiographic evidence of pneumoconiosis and had at least a 20-year history of exposure. Combined with compensation awarded under the General Claim guidelines, it is probable that the present system is designed appropriately to serve most miners who seek compensation for respiratory disability. Indeed, among certain physicians and administrators, it is felt that the present guidelines may even be too generous and that certain individuals may receive benefits in the absence of genuine disability.

However, in light of evidence that radiography and resting pulmonary function tests show less-than-perfect correlation with early disease and pathology, reliance on the chest x-rays alone as a means of assessment could lead to rejection of a valid disability claim in a miner complaining of respiratory symptoms. Consequently, it may be advisable to reexamine the pre-eminent position of the chest x-ray in the present compensation process. In certain instances, when the chest x-ray and resting PFTs fail to resolve the issue of impairment and disability, exercise testing as a form of assessment might be warranted. □

The opinions expressed in this article are those of the authors and do not necessarily reflect those of the Nova Scotia Lung Association.

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Correspondence

To the Editor:

MELANOMA

I enjoyed the article on melanoma by Drs. Murray and Miller in your June 1993 issue. They drew attention to the Pigmented Lesion Clinic at Camp Hill Medical Centre which is essentially a screening clinic for melanoma.

I would like to draw your readers attention to the facilities for treating patients with melanoma in the Nova Scotia Cancer Centre. This is the clinical arm of the Cancer Treatment and Research Foundation of Nova Scotia and it is housed on the ground floor of the Dickson Building of the Victoria General Hospital. Weekly clinics are available for multidisciplinary opinions on the management of new patients and for non-surgical treatment by radiotherapy, chemotherapy or immunotherapy. The melanoma group participate in clinical trials that are designed to improve treatments and identify optimum care. Patients may be referred to the clinic by telephone or mail and all patients should be accompanied by a letter outlining the essential points in the history and management to date, when practical. Treatment is managed through physicians in the local community.

Yours sincerely,

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Evaluation of Risk Scoring in Predicting Preterm Birth

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A risk scoring system developed by Robert Creasy based on demographic, social, and medical factors, has shown variable success in predicting preterm birth. The purpose of this study was to assess the applicability of such a risk scoring protocol in predicting preterm birth in pregnant women of Nova Scotia. A retrospective chart review was performed to compare fifty women who delivered prior to 36 weeks gestation with fifty controls who delivered at term. In the patients studied, twin gestation, premature rupture of the membranes (PROM), previous abortions, smoking, pre-eclampsia, and bacteriuria were particularly important predictors of preterm birth. Calculated risk scores were significantly higher in women who delivered prematurely than in those who delivered at term. Our data suggest that the Creasy risk scoring protocol can be successfully utilized to identify Nova Scotian women at risk for preterm birth.

Preterm birth is responsible for over 60% of perinatal mortality and morbidity in North America. In the United States, the average incidence of preterm delivery is 5-10%, yet it accounts for 75-85% of neonatal deaths not due to lethal anomalies.[1] Despite the advancements in tocolytic treatment, the incidence of preterm birth has remained unchanged. The effect of tocolytic therapy has been limited because most patients who develop preterm labor fail to seek medical care until they have ruptured membranes or advanced cervical dilation, thus decreasing the potential benefits of long-term tocolysis.[2] Recognition of the importance of early detection of preterm labor led to the development of preterm delivery prevention programs that stress identification of high-risk patients, patient education, self-palpation for uterine contractions, and frequent cervical examinations.[1,2] Studies using such prevention protocols in obstetric populations similar to that of Nova Scotia, have demonstrated an overall decrease in the incidence of preterm delivery.[1,3,4,5]

The purpose of this retrospective case-control study was to:

1. Compare the occurrence of risk factors known to be associated with preterm birth between a group of women who delivered prior to 36 weeks gestation and a group of women who delivered at term.
2. Evaluate the efficacy of the Creasy risk scoring protocol[6,7] in predicting preterm birth in Nova Scotian women.
3. Develop a preterm birth risk scoring protocol specific to our pregnant population.

In the Creasy risk scoring protocol, each patient is evaluated for the presence of several risk factors, each of which is assigned a numerical point value. A value of *10* or more indicates that the patient is at high risk for preterm delivery. The risk of preterm delivery scoring system developed by Creasy is depicted in Table I.

MATERIAL AND METHODS

Using a randomized, retrospective chart review, an investigation was undertaken to determine the value of the Creasy risk of preterm delivery scoring system in predicting the incidence of preterm birth in women delivering at the Grace Maternity Hospital in Halifax, Nova Scotia, during 1989. Fifty charts of women who delivered at or less than 36 weeks gestation were compared with fifty charts of controls who delivered at 37 weeks gestation or greater. Based on available chart information, a risk score was assigned to each patient using Creasy's method. In addition, other factors not mentioned in Creasy's system were noted, including PROM, anemia, and a diagnosis of pre-eclampsia.

The statistical calculations performed on the data were the following:

1. *Chi-squares* to compare frequencies of occurrence for all variables between the preterm and term groups.
2. *Pool variance t-tests* to compare preterm risk scores between groups.
3. *Relative risk ratios* on all variables except where the denominator was zero.
   $P \leq 0.05$ was required for statistical significance.

RESULTS

The population studied consisted of predominantly white, middle class Nova Scotian women.

Comparison of demographic and clinical variables between groups were not significantly different. (Table II). Tables IIIa-IIId indicate the various risk factors identified in the Creasy risk of preterm delivery scoring system. For each risk factor listed, the frequency of occurrence is shown for both preterm and term groups. The p-values and the relative risk ratios are found in the last two columns.

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TABLE I

CREASY RISK OF PRETERM DELIVERY SCORING SYSTEM

Points	Socioeconomic status	Past History	Daily habits	Current Pregnancy
1	2 children at home	1 abortion Less than 1 year since last birth	work outside home	Unusual fatigue
2	Younger than 20 years Older than 40 years Single parent	2 abortions	More than 10 cigarettes per day	Less than 5 kg gain by 32 weeks' gestation Albuminuria Hypertension Bacteriuria
3	Very low socioeconomic status Shorter than 150 cm Lighter than 45 kg	3 abortions	Heavy work Long tiring trip	Breech at 32 weeks Weight loss of 2 kg Head engaged Febrile illness
4	Younger than 18 years	Pyelonephritis		Metrorrhagia after 12 weeks' gestation Effacement Dilatation Uterine irritability
5		Uterine anomaly Second-trimester abortion DES exposure		Placenta previa Hydramnios
10		Prior premature delivery Repeated second-trimester abortion		Twins Abdominal surgery

As shown in Tables IIIa-IIId, several factors were associated with preterm delivery (in order of significance):

- Twins
- Premature rupture of the membranes
- One previous abortion
- Smoking more than 10 cigarettes per day
- Bacteriuria
- Pre-eclampsia

Risk factors found to be approaching significance were:

- Hydramnios
- Hypertension

Using the Creasy risk of preterm delivery scoring system, the mean total scores of the preterm (study) and term (control) groups were determined to be 12.0 and 8.1 respectively. When other risk factors such as anemia, other substance abuse, nutrition, PROM, and pre-eclampsia were considered (modified Creasy scoring system), the mean total scores of the study and control groups were 14.3 and 9.4 respectively (Table IV).

TABLE II

DEMOGRAPHIC AND CLINICAL VARIABLES

Variables	Preterm delivery (mean)	Term delivery (mean)	p-value
Age	25.7	26.4	NS
Parity	0.3	0.5	NS
Pre-pregnancy wgt	133.4	134.4	NS

TABLE IIIA

INCIDENCE OF RISK FACTORS FOR PRETERM BIRTH – SOCIOECONOMIC STATUS

Risk factor	Preterm delivery (N=50)	Term delivery (N=50)	p-value	Relative risk
Younger than 18 years	2	4	NS	0.5
Low socioeconomic status	5	2	NS	2.5
Shorter than 150 cm	0	2	NS	0
Lighter than 45 kg	4	4	NS	1
Younger than 20 (but >17)	4	3	NS	1.33
Older than 40	0	0	NS	–
Single parent	13	10	NS	1.3
2 children or more at home	3	8	NS	0.38

TABLE IIIB

INCIDENCE OF RISK FACTORS FOR PRETERM BIRTH– PAST HISTORY

Risk factor	Preterm delivery (N=50)	Term delivery (N=50)	p-value	Relative risk
Repeated 2nd trimester abortion	1	0	NS	–
Prior preterm delivery	0	0	NS	–
Uterine anomaly	3	0	.079	–
Second trimester abortion	0	1	NS	0
Diethylstilbestrol exposure	0	0	NS	–
Pyelonephritis	1	1	NS	1
3 abortions	0	1	NS	0
Anemia	8	6	NS	1.33
2 abortions	1	1	NS	1
1 abortion	11	3	.021	3.67
Less than 1 yr since last birth	0	0	NS	–

TABLE IIIC

INCIDENCE OF RISK FACTORS FOR PRETERM BIRTH–DAILY HABITS

Risk factor	Preterm delivery (N=50)	Term delivery (N=50)	p-value	Relative risk
Heavy work or excess physical activity	1	0	NS	–
Long tiring trip	0	0	NS	–
Inadequate nutrition	2	2	NS	1
Other substance abuse	6	7	NS	0.86
Absent prenatal care	0	0	NS	–
More than 10 cigarettes per day	22	12	.035	1.83
Work outside home	14	13	NS	1.08

TABLE IIID

INCIDENCE OF RISK FACTORS FOR PRETERM BIRTH–CURRENT PREGNANCY

Risk factor	Preterm delivery (N=50)	Term delivery (N=50)	p-value	Relative risk
Twins	9	0	.002	≥9
Abdominal surgery	1	0	NS	–
PROM	23	10	.002	2.3
Pre-eclampsia	9	2	.046	4.5
Placenta previa	1	0	NS	–
Hydramnios	9	3	.065	3
Metrorrhagia after 12 wks	9	5	NS	1.8
Effacement and/or dilatation	19	27	NS	–
Uterine irritability	3	2	NS	1.5
Breech at time of labor	7	3	NS	2.33
Weight loss of 2 kg	0	0	NS	–
Head engaged	3	16	NS	0.19
Febrile illness	2	0	NS	–
Less than 5 kg gained by time of delivery	18	15	NS	1.2
Albuminuria	8	6	NS	1.33
Hypertension	16	8	.061	2
Bacteriuria	14	6	.046	2.33
Unusual fatigue	3	0	.079	–

TABLE IV

CALCULATED RISK SCORES

Risk Scoring System	Preterm delivery (mean)	Term delivery (mean)	p-value
Creasy	12.0 ± 7.1	8.1 ± 4.8	.001
Modified Creasy	14.3 ± 7.1	9.4 ± 5.3	.000

DISCUSSION

The primary objective of this ongoing project is to establish a risk-scoring system which is specific for childbearing women of Nova Scotia. This retrospective chart review has shown several risk factors to be quite significant in predicting preterm birth. These factors are twins, PROM, one prior abortion, smoking more than 10 cigarettes per day, bacteriuria, and pre-eclampsia. Other risk factors were found to approach but not reach significance, namely uterine anomaly, hydramnios, hypertension, and unusual fatigue. In addition, breech presentation at the time of labor and low socioeconomic status had a relative risk of >2, and therefore may be an important predictor of preterm birth. However, the lack of significance associated with these latter risk factors may be due to the small sample size of this study.

Prior preterm birth, which is known to be a strong determinant of premature delivery was not identified in either group. This is also undoubtedly due to the small sample size, which is the major deficiency of this study. Other risk factors such as diethylstilbestrol (DES) exposure, absent prenatal care and maternal age ≥40 were also absent but are seldomly encountered factors in Nova Scotian parturients.

It is realized that further work needs to be done in this area in order to ensure an effective and successful preterm birth prevention program for childbearing women of Nova Scotia. Future studies should be aimed at evaluating risk factors associated with preterm birth in a large sample of women who delivered preterm infants compared to women who delivered at term. Secondly, a risk scoring system should be developed which is specific to Nova Scotian women. Lastly, a preterm birth prevention program should be established, including prenatal risk assessment and patient education, for study in a prospective randomized fashion.

In summary, this study has demonstrated the following:

1) The Creasy risk of preterm delivery scoring system may be successfully utilized to identify Nova Scotian women at risk for preterm birth.

2) Certain factors have been identified as particularly important indicators of preterm birth in Nova Scotia women.

3) The conclusions which can be made from this study are limited because of the relatively small number of cases which were studied. □

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Lasers in Surgery and Medicine

PART 2

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The second part of this article examines typical laser applications in Ophthalmology, Gynecology, Otolaryngology/Head and Neck Surgery, Dermatology/Plastic Surgery, Neurosurgery, Urology, G.I. Surgery and Pulmonary Surgery. More recent laser applications and experimental results are presented as well. Information concerning laser use in Halifax hospitals and clinics has also been included.

APPLICATIONS OF LASERS IN MEDICINE

In ophthalmology, argon and krypton lasers are used to photocoagulate bood vessels of the retina. This is used in treating diabetic retinopathy where proliferation of retinal blood vessels occurs in diabetics due to ischemic conditions. These vessels tend to be fragile and often hemorrhage, leading to blindness. Photocoagulation causes a diminution in the number of such vessels, reducing the chance of hemorrhage and possibly stopping the progression of the disease. The reduction in severe vision loss is about 60%.[1]

Lasers may also be used to perform iridotomies to improve fluid flow between anterior and posterior chambers. This is effective in treating closed angle glaucoma. An argon laser is also used for this, but in the case of a pale-coloured iris, an Nd:YAG laser is more effective. Open-angle glaucoma may also be treated by creating lesions around the periphery of the iris into the trabecular meshwork using a laser and a special contact lens.

The laser is also used by ophthalmologists for posterior capsulotomy. Often, the posterior capsule opacifies after cataract removal and intraocular lens replacement. In this procedure, the Nd:YAG laser is used at a special setting where pulses of intense energy are delivered in very short periods of time such that atoms are ionized, creating a mini sonic boom of acoustical energy that causes the membrane to burst open. This technique may also be used to crack open a hard cataract, making it easier to remove by traditional surgical means.

Gynecology is another field where lasers are used very frequently. The CO_2 laser is used both colposcopically and laparoscopically. Endometriomas may be vaporized or coagulated while adhesions may be conveniently dissected. The CO_2 laser is useful for infertility work since obstructed tubes and ovaries may be easily reopened. Also, abdominal cysts and tumours may be excised with minimal damage to surrounding tissue.

A common application of the CO_2 laser is in the treatment of cervical intraepithelial neoplasia (CIN) using a colposcope. This may also be performed using the Nd:YAG laser with contact probe, though more slowly. The advantages of laser treatment of CIN over knife conization, cauterization or cryotherapy include leaving the cervix in a more viable condition with no stenosis and minimal scarring. Vaginal intraepithelial neoplasia and genital warts may also be treated advantageously using laser.

The CO_2 laser is used commonly in Otolaryngology/Head and Neck Surgery to excise vocal cord nodules and polyps. It is also used to treat hyperkeratosis, granulomas, Quincke's edema, laryngeal stenosis and to remove cysts and webs and to perform arytenoidectomy. Precision, hemostasis and excellent healing properties make the laser very useful for these applications. The low post-operative swelling and scarring also make it particularly convenient for pediatric Otolaryngology/Head and Neck Surgery.

The CO_2 laser is also used for intranasal work such as turbinectomy, treatment of choanal atresia and telangiectasia. Rhinophyma, polyposis, synechia and granulomas may also be treated by laser. Tonsillectomy and hemiglossectomy performed by laser result in minimal blood loss and little post-operative pain. Lesions of the oral cavity such as leukoplakia and tongue releases may also be treated. Lasers have also been used in otology for procedures such as stapedotomy and tympanoplasty.

Successful laser applications in Dermatology/Plastic Surgery include photocoagulation of pigmented cutaneous lesions such as portwine stains, capillary hemangiomas, telangiectasia, strawberry marks, Campbell DeMorgan senile angiomas and acne rocasea. Tattoo removal is also a common application producing good results. The laser is not as useful for treating keloid scars, subcutaneous varicose veins, road skid burns, moles and warts. The CO_2 laser can cause destruction of skin regardless of its colour, whereas the argon laser and others having a wavelength in the visible spectrum exert effects that depend upon skin colour.

In Neurosurgery, the CO_2 laser is often used to excise tumours when precision is of utmost importance. For example, a meningioma may be 'peeled away' from its dural attachment and acoustic neuromas may be excised without harming the acoustic nerve. Similarly, the laser is useful in removing tumours around the optic chiasma and nerve. Spinal tumours may be conveniently removed without any manipulation that might injure the spinal cord and nerve roots.

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The Nd:YAG laser is used most often in Urology for transurethral destruction of bladder tumours and for treating urethral stricture. The CO_2 laser is used to vaporize condylomata and other external lesions. Partial nephrectomy may also be performed using this laser. Kidney stones are sometimes fragmented using a dye laser, but mechanical shock waves are actually the active forces involved.

In GI Surgery, the Nd:YAG laser is used to treat bleeding peptic ulcers. It may also be used to destroy GI tumours. Hemorrhoids have been successfully treated using lasers. The CO_2 laser is used for external hemorrhoids and anal tags while the Nd:YAG is more useful for internal hemorrhoids. Unfortunately, lasers have not produced good results in treating esophageal varices. Recanalization of tumour-obstructed GI tract as a palliative measure is a very useful application. This may relieve symptoms such as dysphagia and fecal incontinence. Similarly, in Pulmonary Surgery, a tumor-obstructed trachea or bronchus may be recanalized to permit easier breathing.

Lasers are not commonly used by General and Orthopedic Surgeons. Potential applications include tumour resection, muscle incision and practically any sort of organ work. In fact, lasers may generally be used in any situation where ordinary scalpels are used.

NEW DEVELOPMENTS

There are many new surgical applications being developed using the laser. A technique known as 'photodynamic therapy' (PDT) involving lasers has given hope in the treatment of certain difficult GI and Head and Neck cancers. It involves injection of a hematoporphyrin derivative (HPD) intravenously which then localizes in malignant tumours and inflamed tissue. A laser may be chosen such that its wavelength matches that of maximum HPD absorption and selective necrosis of the tumor may be achieved. Usually an argon or dye laser tuned to 630 nm is used. Gold vapour lasers are also useful for this purpose.

Spinelli *et al.* have performed PDT from 1982 to 1990 on 41 patients most of whom had esophageal, stomach and rectal tumors.[2] In all cases of advanced tumors, greater than 50% reduction in tumor size was reported with no serious post-operative complications. Complete resection was reported in the majority of early stage tumors.

Laser PDT will probably become an adjuvant form of cancer therapy and in certain neoplasms, the primary mode of treatment. Previous or subsequent radiotherapy or chemotherapy do not seem to affect the mechanism of PDT. One of the major complications of PDT is that the patient remains sensitive to ordinary light for approximately a month following HPD injection.

In 1990, laser prostatectomy was shown to be feasible in canine models. It was performed using a Nd:YAG laser transurethrally-guided by ultrasound. Necrosis of cancerous tissue was achieved without bleeding, and dogs were able to urinate immediately following the operation. More recently, this procedure has been performed on human subjects in Australia and the US with excellent results. In comparison to standard transurethral prostatectomy, laser prostatectomy is half as time-consuming and there is less associated morbidity such as incontinence and retrograde ejaculation. The patient is usually discharged on the same day, compared to the average 10 day hospital stay following standard prostatectomy.

The Nd:YAG laser equipped with a sapphire contact probe has been used to resect capillary/cavernous hemangiomas. The hemostatic properties of the laser make it ideal for this purpose whereas using an ordinary scalpel could result in massive blood loss. The contact probe allows fine control which is useful for cosmetic purposes. In fact, there are a variety of contact probes with different geometrically-shaped tips so that the pattern of incision or coagulation may be chosen.

Lasers may be used at low powers to actually weld tissue together. Blood vessel anastomosis using laser is a new application with great potential. Kuroyanagi *et al.* used an argon laser to anastomose medium-sized vessels and followed these patients for one year.[3] Their results show excellent vessel patency for medium-sized vessels of 3-8 mm diameter and no occurrence of pseudoaneurysms. This technique is also faster than conventional surgery. Results were not very good for small vessel anastomosis.

Other developing applications include excimer laser refractive surgery where the corneal shape of the eye is altered to treat myopia. Blood vessel recanalization, meniscectomy, treatment of ectopic tubal pregnancies and raising skin flaps are also among the latest applications of lasers.

CONCLUSION

The laser has unquestionably become a very important addition to the surgical armamentarium. Not only are there diseases untreatable without laser technology, but there are many others which are better managed by lasers. The laser has become the primary method of treatment for such conditions as tracheal stenosis, port wine stains, cervical intraepithelial neoplasia and obstructive carcinomas of the GI tract. It has also become a very cost-effective mode of therapy mainly due to its efficiency and minimally-invasive nature. During the 1990s, it is expected that many new applications of the laser will be implemented and it will be used more and more often in treating diseases in virtually all fields of medicine.

More on laser use in Halifax hospitals on page 180.

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LASERS IN HALIFAX HOSPITALS AND CLINICS

There are approximately 20 medical lasers being used in Nova Scotia, with the highest concentration in Halifax. There are five at the Halifax Infirmary: two argon, a CO_2, a Nd:YAG and an argon-krypton combined laser. There are four at the Victoria General Hospital: three CO_2 and a Nd:YAG laser. Other communities with medical lasers include Amherst, Antigonish, Sydney, Truro, Kentville and Aberdeen, most of which are argon or CO_2 lasers. There is a laser committee based in Halifax which meets quarterly to discuss safety issues, establish policy and discuss new acquisitions and new applications.

Ophthalmology at the Halifax Infirmary has been using lasers since the mid-1970s. The argon or krypton laser is used in treating diabetic retinopathy as well as retinal tears/detachment and macular diseases. A Nd:YAG laser is used for posterior capsulotomy, uncontrolled glaucoma and iridotomy. Such laser procedures are performed very frequently, particularly in the clinic. A Holmium:YAG laser may be acquired in the near future. This is similar to a Nd:YAG except that the YAG crystal is doped with Holmium instead of Niodymium. This would be used to perform sclerostomies with the advantage of less scarring.

Lasers have been used for the past 8-10 years in Gynecology at the VG Hospital and Halifax Infirmary. They are used practically every day treating endometriosis and infertility patients laparoscopically, while precancerous changes of the lower genital tract are normally taken care of in clinics. The CO_2 laser is used for all cases. There are no plans to introduce new lasers or new applications in gynecology at present, but current procedures are being performed more and more frequently and seem to be working well.

The Otolaryngologists/Head and Neck Surgeons at the Halifax Infirmary have been using lasers for the past 10-15 years. They are used about three times per week and the most common applications involve laryngoscopic surgery such as vocal cord polyp removal and treatment of laryngeal stricture. A recently-acquired CO_2 laser is used and Nd:YAG and argon lasers may soon be tried out. The laser is not used for otological purposes.

Over the past five years, plantar warts, port wine stains, benign skin tumours, actinic chelitis and tattoos have been treated by Dermatologists at the VG Hospital using a CO_2 laser. There has been an increase in demand for laser treatment by patients with skin lesions. The Dermatology department hopes to acquire a tunable dye laser in the near future.

The Nd:YAG laser has been used twice per week on average in Thoracic Surgery at the VG Hospital over the past five years. The most common application has been tracheal/carinal tumour ablation as a palliative measure. Newer applications include treatment of pneumothorax and in open chest surgery.

Neurosurgery at the VG Hospital has been using a CO_2 laser for the past ten years. It is used much less frequently than in the other specialties mentioned but it serves some purpose in ablating tumours that are hard to reach with an ordinary scalpel.

Urology in Nova Scotia has not seen laser use as of yet. However, there is a laser prostatectomy clinical trial underway for the first time in Canada. Halifax, in addition to Hamilton and Vancouver will soon be treating the first group of patients. The Nd:YAG laser at the VG Hospital will be used and bladder tumours may also be treated in the near future. □

Controversies and Contraindications Regarding Pertussis Immunization

TO DPT OR NOT TO DPT

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Recent controversies surrounding the pertussis vaccine have confused the issue of eligibility for immunization. Media attention, court cases, and an association of parents of children allegedly damaged by the vaccine have made many families reluctant to accept the vaccine for their children. Many physicians are also uncertain about the use of the vaccine in light of allegations that it may cause encephalopathy or brain damage.

The purpose of this review is to attempt to dispel the myth that the pertussis vaccine damages the nervous system, outline the known side effects of the vaccine, and to offer guidelines to Maritime physicians about contraindications to Pertussis immunization.

CONCERNS ABOUT THE PERTUSSIS VACCINE

Public concerns over the pertussis vaccine were first sparked in 1974, following a British television documentary which showed a number of children thought to have been damaged by their pertussis immunization. The story was based on a reported association between 36 cases of long-term neurological illness after Diphtheria-Pertussis-Tetanus (DPT) immunization.[1] The resultant outcry helped to drop the British rate of pertussis immunization from 80% in 1974 to 31% in 1978. Unfortunately, from 1977-79 there was a pertussis epidemic which caused at least 36 deaths. A similar situation occurred in Japan. Based on two deaths thought to be related to encephalopathy secondary to the DPT vaccine, the Japanese government stopped issuing the pertussis vaccine in 1975, and in 1979 a pertussis epidemic claimed the lives of at least 41 children.[2]

The pertussis vaccine controversy crossed over to North America in 1982 when the "Today Show" telecast a feature showing children allegedly damaged by the DPT immunization. Thereafter, litigation against manufacturers of DPT vaccines rose from one case in 1978 to 73 cases in 1984. In order to guard against potential claims, American manufacturers raised the 1982 price per dose of the vaccine from 45 cents to $11.40 in 1986, with $8.00 going to a litigation fund.[3]

Fortunately, this controversy has not become epidemic in Canada and to date, there has been only one major case of litigation in Canada. From October 1987 to June 1988, the case of a nine year old boy with epilepsy, microcephaly, cortical blindness and spastic quadriplegia allegedly due to the pertussis vaccine, was heard by a judge in the Ontario Supreme Court. The defendants were the physicians who administered the vaccine, the manufacturer of the vaccine, and the Province of Ontario. The judge ruled, based on medical evidence and some of the studies later reviewed in this paper, that the boy's brain damage was not due to the pertussis vaccine. Furthermore, he observed that there is no clearly recognizable entity such as DPT-encephalopathy (Rothwell vs. Raes, 1988).[4]

Pertussis vaccine and damage to the nervous system

Most of the apprehension about pertussis immunization relates to a concern that the vaccine might cause to brain damage. It has been implied that the vaccine can produce a broad scope of neurological conditions ranging from seizures, epilepsy and infantile spasms to encephalopathy. As well, the vaccine has also been challenged as a cause of the Sudden Infant Death Syndrome (SIDS). In fact, the pertussis vaccine has often become suspect whenever one of these conditions occurs without apparent cause.

One can understand why these conditions have been associated with the pertussis vaccine. Since the vaccine is given to most children 4 times in the first 1½ year of life, there is a 1/19 chance that any event occurring during these first 18 months will be within one week of pertussis immunization. This temporal association of unexplained neurologic events and pertussis vaccination has lead to an extraordinary misunderstanding.

Does Pertussis Vaccine cause febrile seizures?

Febrile seizures and most immunizations are events of infancy and early childhood. Besides this temporal relation, immunization often causes a fever which can provoke a febrile seizure. The DPT vaccine is no exception. The National Collaborative Perinatal Project of the National Institute of Neurological and Communicative Disorders and Stroke followed the children born to 54000 pregnant women until age seven, looking for the development of neurologic disease. Of the 2766 children found to have seizures, only eight had a seizure within two days of DPT vaccination. Almost all were febrile

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seizures, and no child went on to have a seizure disorder at the end of the study.[5]

Does Pertussis Vaccine cause epilepsy and/or infantile spasms?

A few studies have addressed this issue well. In a study where children who had a seizure within 48 hours of DPT immunization were reassessed six or seven years later, Baraff *et al.* found no cases of epilepsy[6]. Two interesting reports have arisen from the 1970 change in the Danish schedule for immunization, as investigators were thus given the opportunity to watch for changes in the age of onset of seizure disorders. With the change in DPT immunization schedule from 5, 6, 7 and 15 months of age, to 5 and 9 weeks and 10 months, Melchior found no shift in the onset age for infantile spasms,[7] and Shields *et al.* likewise found no difference for onset of epilepsy.[8]

Does Pertussis immunization cause brain damage/ encephalopathy?

Many neurologic disorders of unknown etiology become manifest in the first year of life, and some will certainly appear, by temporal relation, shortly after immunization. The issue of potential brain damage from the pertussis vaccine has been the most controversial. Case studies have fuelled this suspicion, but there is now an overwhelming body of evidence that the pertussis vaccine does not cause encephalopathy.

The most well known study of the possible link between the pertussis vaccine and encephalopathy is the British National Childhood Encephalopathy Study (NCES), which ran from 1976 to 1979.[9] During this period, children admitted to hospital with serious acute neurologic events were compared to children without neurologic complaints and reassessed for brain damage at one year follow-up. From this study, the oft quoted rates for acute and permanent brain damage per DPT immunization are 1:110,000 and 1:310,000 with respective confidence intervals of 1:360,000 to 1:44,000 and 1:5,310,000 to 1:54,000. Subsequent reanalysis of this data excluded some children from the group of nine children with long-term neurologic damage after one year, including those with coxsackie virus infection and infantile spasms. In fact, only one child who had been normal before immunization was found to have an encephalopathy of unknown origin and long-term neurologic impairment.[10,11]

More recently, the participants of a workshop reconsidering the NCES criticized the study on design and methodology, and stated that: "the NCES is not informative with regard to long-term outcome of acute neurological illness associated with recent DPT vaccination".[12]

Another British study, performed from 1975 to 1981, compared 134,700 children who received the DPT vaccine with 133,500 who were immunized against diphtheria and tetanus only.[13] The results showed that from over 400,000 DPT immunizations, there was no associated neurologic disease or encephalopathy.

Overall, if the pertussis vaccine can cause severe neurologic damage, this has not been borne out in numerous studies. If encephalopathy is indeed a complication of pertussis immunization, the extremely low rate would make detection very difficult.

Based on a review of the literature the Child Neurology Society of the United States has overwhelmingly adopted a 1990 position paper, which states:

1) Administration of pertussis vaccine is associated with a short term increase of seizures, most of these being febrile seizures and complete recovery is expected.
2) Case reports have raised the question as to whether there is an association between pertussis vaccine and progressive or chronic neurologic disorders, but controlled studies have failed to prove such an association.
3) At the present time there is no means by which a diagnosis of pertussis vaccine-encephalopathy can be established in an individual case.

A similar position statement was put out by the American Academy of Neurology in 1991:

1) DTP immunization is an important public health measure. Acute reactions warrant support for development of improved vaccines.
2) There is no clinical or neuropathologic syndrome associated with DPT vaccine, and no means by which a diagnosis of brain damage due to DPT immunization can be established in an individual case. Children whose neurologic problems begin soon after immunization warrant a full diagnostic workup, not an assumption that the symptomatology is explained by its onset shortly after immunization.
3) Controlled epidemiologic studies have failed to support the suggestion that permanent neurologic injury results from DPT immunization. Temporal association alone does not establish causation.

We agree with these statements, and conclude that the pertussis vaccine does not cause brain injury.

Does Pertussis vaccine cause Sudden Infant Death Syndrome?

The possibility that the pertussis vaccine causes SIDS arose from case studies, and was forwarded by the media.[14] However, several investigations have indicated the contrary. Over a period of eight years, Solberg (1985) found that of 53 cases of SIDS within one month of DPT immunization, none were attributable to the vaccination.[15] In a case-control study of 800 SIDS children, Hoffman *et al.* did not identify the pertussis vaccine as a significant etiologic factor.[16] Similarly, Griffen *et al.* showed, in a ten year study, that there is no increased risk for SIDS following DPT immunization.[17]

SIDE EFFECTS OF PERTUSSIS VACCINE

Since the pertussis vaccine is usually given mixed with immunizations for diphtheria and tetanus, it can be difficult to ascribe side effects to any particular component of the inoculant. In 1981, Cody *et al.* designed a study to compare the side effects of DTP versus DT vaccines. While it was not carried out entirely in a double-blind fashion, a subset of the study group was double-blinded, and no differences in the data were seen between the blinded and non-blinded groups. The results clearly demonstrate that the pertussis component of the vaccine is responsible for the majority of the side effects from DPT immunization. The local reactions observed were pain, swelling and redness in the immunized limb in 51%, 41%, and 37%, respectively. The systemic reactions noted were: fretfulness (53%), fever >38°C. (47%), drowsiness (32%), anorexia (21%), vomiting (6%) and persistent crying (3%).[18]

Episodes of hypotonia and hyporesponsiveness wherein a febrile and irritable child becomes pale, limp, unresponsive, with shallow respiration were reported by Hopper in (1961).[19] These rare events of unknown etiology usually occur within 12 hours of DPT vaccination, and are understandably frightening for both parents and physicians. Fortunately the outcome has been benign.[6] It is possible that these events are not due to pertussis immunization *per se*, as Pollock found similar episodes following the DT vaccine.[13]

Allergic hypersensitivity to any vaccine is a remote possibility, and has been reported for the DPT vaccine.[14] The symptoms of allergic hypersensitivity range from generalized pruritus and urticarial lesions through to anaphylaxis. The earliest symptoms of anaphylaxis are generalized pruritus, erythema, and warmth, which may be followed by nausea, vomiting, abdominal pain, diarrhea, diaphoresis, wheezing, dyspnea, stridor, syncope, hypotension, and cardiovascular collapse.

For this reason, it is generally recommended that patients remain in the physician's office for at least 20 minutes following any immunization. For infants and children, the treatment of anaphylaxis involves control of the airway, as appropriate, and 0.01 ml/Kg SQ/IM of a 1:1000 solution of adrenaline.[20]

CONTRAINDICATIONS TO PERTUSSIS IMMUNIZATION

Adverse reactions to immunization with a pertussis vaccine do not necessarily constitute absolute contraindications to re-immunization against pertussis. The 1991 *Red Book Report of the Committee on Infectious Diseases of the American Academy of Pediatrics* has proposed that certain events be considered relative contraindictions to re-immunization with the pertussis vaccine.[21] These recommendations follow, along with our impressions:

A severe neurologic event such as severe acute encephalopathy of unknown cause.

As described earlier, data to date questions the existence of pertussis encephalopathy. If it indeed exists, it is extraordinarily rare.

Persistent, severe, inconsolable crying or screaming for three hours, or unusually high pitched cries within 48 hours of immunization.

The study of side effects by Cody *et al.*, showed that children given the DPT versus the DT vaccine cry longer and more episodically.[18] However, doubt has been cast on this idea, since delayed crying might be related to the act of immunization itself, as Pollock found no difference in crying following the DPT and DT vaccines.[13]

A convulsion, regardless of body temperature, up to three days following immunization with the DPT vaccine.

It has been repeatedly shown that the pertussis vaccine does not cause afebrile seizures.

A fever of 40.5° C, or greater, that cannot be explained by any other cause, within 48 hours of DPT immunization.

Any vaccination can bring about fever, which can precipitate a febrile seizure. Thus, measures taken to control immunization related fever should reduce the risk for febrile seizures.

An anaphylactic response to the vaccine.

Although rare, this is a true phenomenon, and valid contraindication.

A hypotonic-hyporesponsive episode within 48 hours of immunization.

The relationship of the pertussis vaccine to these episodes is not clear, as they may also occur with DT immunizations alone.

It is our opinion that the only relative contraindications for non-primary pertussis vaccination are anaphylaxis, due to its potential morbidity and mortality, and hypotonic-hyporesponsive episodes, due to their rarity and our incomplete understanding of the phenomenon.

The decision to repeat immunization with a pertussis vaccine, following any of these situations is difficult for both the physician and the family. Nonetheless, the decision must be made with consideration of the prior adverse event, the likelihood of pertussis exposure, and the potential benefits and risks of pertussis immunization. This is particularly important in Nova Scotia, as pertussis remains a significant health risk for this province.[22] If doubts persist, discussions of individual cases with either a Pediatrician or Pediatric Neurologist should help to clarify the issue. Children with a personal history of febrile convulsions may be at increased risk for a seizure following any immunization. Since a febrile reaction following immunization may predispose such children to having a convulsion, prophylactic acetaminophen

is recommended. The suggested dose is 10-15 mg/Kg just prior to immunization, and then every four hours for the next 48-72 hours.[23] Notably, febrile seizures do not significantly increase one's overall risk for epilepsy or brain damage.[24].

A family history of seizure disorder should not be considered a contraindication to pertussis immunization.[23] While such children may be at increased risk for a febrile seizure following any immunization, antipyretic prophylaxis should control the febrile response. It has been thought that if a child has a progressive neurologic or developmental disorders, immunization should be deferred as it would be difficult to ascribe any changes in condition to the disorder or the vaccine.[14]. However, with current evidence that the pertussis vaccine does not cause brain damage, there is no longer any need to withhold the vaccine from children with neurologic or developmental disorders.

SUMMARY

The decision whether to immunize a child against pertussis has recently been complicated by concerns about brain damage as a side effect of the vaccine; court cases and media attention have served to make both parents and physicians apprehensive about immunizing against pertussis. Unfortunately, suspicion about the vaccine arose mainly from the temporal relation between pertussis immunization and febrile seizures, infantile spasms, encephalopathy and sudden infant death. Data in the defense of the pertussis immunization has mounted from clinical studies, and it is now clear that the pertussis vaccine does not cause any of these conditions. While it is true that immunization of infants and young children can cause a febrile response which carries the risk for febrile seizures, any such seizure cannot be attributed directly to the vaccine. It is important to note that single neurologic events following pertussis immunization are not necessarily indicators or harbingers of neurologic disease.

There are no contraindications, beyond that relating to acute febrile illness, for the *first* pertussis immunization in the infant. In children with a history of febrile seizures or a family history of seizures, prophylactic administration of acetaminophen for 48-72 hours should protect against fever and help avoid febrile seizures.

For subsequent pertussis immunizations, the contraindications relate, again, to general principles of immunization in addition to a past episode of anaphylaxis or hypotonic-hyporesponsive event. As always, the decision for immunization must be made with the benefits of disease prevention weighed against the potential for injury from the disease or the side-effects of immunization. The ultimate decision regarding pertussis immunization still rests in the hands of the parents and physician, and hopefully, the data presented in this paper will make this decision somewhat easier. □

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The Ratio of the Length of the Thoracic-kyphotic Curve to that of the Lumbar-lordotic Curve in Relation to Low-Back Pain

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The purpose of the study was to test the hypothesis that men with chronic low-back pain (LBP) tend to have a greater proportion of their spine occupied by the lordotic curve, compared with men who do not have problems with LBP. Patterns were made of the back curves of 202 men who, for at least two years, had suffered LBP that their physicians had diagnosed as mechanical in origin, and of the backs of 120 men who reported they had never been troubled with LBP. The average kyphotic-lordotic ratio of the pain-free men was significantly greater than that of LBP sufferers. The data were further analyzed to identify the kyphotic-lordotic ratio that best predicted whether a subject would be in the pain-free group or the group with LBP. It was 1.435. In light of these findings, the authors recommended parallel research on women and clinical trials to evaluate exercise programs designed to increase the kyphotic-lordotic ratio of persons who suffer LBP of mechanical origin.

Low-back pain (LBP) is a common condition, affecting almost 80% of the world's population at one time or another.[1,2] It is the common cause of limitations to physical activity,[3] frequently causing loss of time from work, and exacting a tremendous toll on the economy.[4] Although it is clear that biomechanical factors are important in its etiology, there continues to be controversy about the specific ways these factors are involved. Lack of lumbar lordosis,[5] too much lumbar lordosis,[6,7,8] muscular weakness,[9,10] too much spinal mobility,[1] and inflexibility[11,12] have all been proposed as causes of LBP. These different propositions have different implications for treatment[13,14,5,8] Nachemson has lamented that, because the etiology of LBP in most cases is unknown, treatment is inefficient and only symptomatic.[15]

On carefully studying the spinal curves of women and men who suffered from chronic LBP and noting their response to a specifically developed exercise program, the senior author started to document their patterns of spinal kyphosis and lordosis. It appeared that, within three to six months, those who persevered with the exercise program usually manifested characteristic changes in their back curves, coincident with obtaining relief from their LBP.

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These apparently characteristic changes gave rise to the empirically based hypothesis that persons who are troubled by chronic LBP tend to have a lower ratio of the length of their thoracic kyphosis (hereafter referred to as the *kyphotic curve*) to their lumbar lordosis (hereafter referred to as the *lordotic curve*), compared to persons who do not suffer from chronic LBP.

METHODS

Subjects were instructed to stand with their legs straight, with their trunk and shoulder girdle relaxed. After a subject assumed this relaxed stance, a 24-inch-long "French flexible curve" (a tool that is commonly used for patternmaking and drafting) was placed and molded along the spinous processes between C7 and the median sacral crest (see Figure 1). The bony landmarks were found by palpation. With care not to alter the shape of the flexible curve that resulted from this fitting, the flexible curve was laid upon a paper. A tracing of its outline resulted in a record of the subject's kyphotic and lordotic curves.

Figure 1

Molding the French flexible curve to the spinous processes.

To assess the reliability of the tracings, two consecutive fittings were made of the back curves of 10 standing subjects, who changed posture between fittings by sitting in a chair. Tracings of the French flexible curve were recorded on separate sheets of paper. With the bottom edge of a sheet of transparent graph paper serving as an *x* axis, the graph paper was laid over each subject's first tracing. The *x* axis was placed so it touched the caudal end of the tracing. A *y* axis, which was drawn near the right-hand edge of the graph paper, was placed approximately 10 cm from the tracing. Every 10mm, the length of the horizontal coordinate (i.e., the distance from the *y* axis to the tracing at that level) was measured and recorded in mm. (For a tracing that was 55 cm long, this would result in a series of 56 measurements.) This procedure was repeated with each subject's second tracing. After computing Pearson's *r* for the two sets of measurements for five of the subjects, the authors concluded there was no point to computing the coefficients for the other five sets. The five coefficients were .998, .999 and .999, 1.0, and .999. Inspection of the other sets indicated they were also virtually identical.

Work published by others also indicates that the methods involved in this study yield reliable data. Stagnara *et al.* found that the kyphotic and lordotic curves are remarkably stable, even over the course of several years, provided the subject is clearly told how to stand when measurements are being made.[16] Also, Salisbury and Porter[17], and Stokes, Bevins, and Lunn[18] found that the French-flexible-curve method produced reliable data from both the flexed and the extended lumbar spine.

The ultimate measurements in this study were the ratios of the length of each subject's kyphotic curve to the length of his lordotic curve. The reliability of these measurements depends not only on the reliability of the tracings of the back curves, but on the reliability with which one can determine the inflection point, which is that point at which the lumbar curve ends and the kyphotic begins.[19]

The inflection point was located by examining the tracing with the assistance of a sharp-edged ruler. Using an ALVIN 1112 opisometer (this is a Swiss made instrument used by cartographers to measure distances along curves), the distance from the caudal end of the tracing to the inflection point was measured in cm. Such a measurement was made on two tracings for each of 20 subjects (the pairs of tracings were obtained by the method described above). Pearson's *r* for the two sets of measurements was .995, indicating high reliability.

In the study proper, the inflection point was located on each tracing and the lengths of the kyphotic and lordotic curves were measured with the opisometer. The ratio of the two curves was obtained by dividing the length of the kyphotic curve by that of the lordotic.

SUBJECTS FOR THE STUDY

The study involved data from 322 volunteer, male subjects. This number included 202 who were referred to the study by general-practice physicians. All had suffered for at least two years from LBP that their physicians had diagnosed as mechanical in origin. *Potential subjects were not admitted to the study if they were suffering from an acute bout of LBP* or if they had:

1. LBP when performing the Valsalva manoeuver (including pain upon coughing, straining, sneezing),
2. infective, neoplastic, or inflammatory disease,
3. significant or uncontrolled ischemic heart disease or hypertension,
4. radiographic appearance of osteoporosis, or
5. a history of spinal compression fracture or discectomy.

To obtain a group of comparison subjects, the back curves of approximately 150 volunteers who were visiting a gymnasium on a recreational basis were measured. After their measurements were obtained, the volunteers were asked whether they had ever suffered from LBP. Those who reported that they had experienced LBP were excluded from the study. This procedure yielded a comparison group of 120 LBP-free men, on whom measurements had been obtained in "blind fashion" (this is in contrast to the measurements on the LBP sufferers, whose back-pain status was known when the measurements were made).

The mean age of the men with LBP was 37.55 years (95% confidence interval: 36.54 to 38.55). The comparison group of men who were free of LBP averaged 30.39 years, which was significantly younger (95% confidence interval: 29.61 to 31.17). Examination of the groups' age distribution indicated that the younger average age of the group who were free from LBP was due to a greater proportion who were under 20 years of age (13.3% vs. 3.9%).

The mean height of the men with LBP was 177.24 cm. (95% confidence interval: 174.67 to 179.81). For those who were pain free it was 179.99 cm., which was not significantly different (95% confidence interval: 179.05 to 180.93).

The mean weight of the group with LBP was 79.02 kg. (95% confidence interval: 75.19 to 78.85). The 77.78 kg. mean weight of the pain-free group was not significantly different (95% confidence interval: 76.49 to 79.07).

RESULTS

The mean kyphotic/lordotic ratios of the groups were significantly different ($p \leq .0005$). That for the LBP group was 1.16 (99% confidence interval: 1.11 to 1.22), and that of the pain-free group was 1.62 (99% confidence interval: 1.54 to 1.69). Clearly, as was hypothesized, the men with LBP tended to have a greater proportion of their spine occupied by the lordotic curve, compared to those who were pain free. The strength of this relationship was indicated by a .882 ($p \leq .0000$) correlation between the back-curve ratios and the presence or absence of LBP.

Because the groups differed in average age, a two-way analysis of variance was performed to see whether age was related to the proportion of the spine occupied by the kyphotic and lordotic curves, and whether age and the presence or absence of LBP interacted in their relationship to the back curves. As can be seen in Table I, only LBP was significantly related to the ratio of the back curves. Thus, the fact that the convenience samples of subjects differed significantly in age had no bearing on the results of this study.

TABLE I

SUMMARY OF TWO-WAY ANOVA FOR RELATIONSHIPS BETWEEN THE PRESENCE OR ABSENCE OF CHRONIC LBP AND THE KYPHOTIC/LORDOTIC RATIO, AND BETWEEN AGE AND THE KYPHOTIC/LORDOTIC RATIO

Source of variation	df	Sum of squares	Mean square	F	P
Presence or absence of LBP	1	5.050	5.050	54.645	.0000
Age*	4	.135	.034	.365	.8331
Interaction	4	.142	.036	.385	.8194
Error	312	28.830	.092		

*Age categorized into 13-24, 25-34, 35-44, 45-54, and 54-68 years.

Additional analysis was conducted to identify the kyphotic/lordotic ratio that was best able to predict whether a subject would be in the LBP group or the pain-free group. With each group's ratios ranked independently, the ratio above which the fewest LBP subjects fell and below which the fewest pain-free subjects fell was identified. The critical ratio for this set of data fell between 1.43 and 1.44. Eighty-seven percent ($n = 176$ of 202) of those with LBP had a ratio <1.44, but only 25% ($n = 30$ of 120) of the men who were pain free had a ratio <1.44.

DISCUSSION

Because this study included only male subjects, the findings cannot be generalized to women. Clearly, research stimulated by this study should include female subjects.

Although the average length of the kyphotic curve in relation to the length of the lordotic curve was less among men who suffered chronic LBP than it was among those who did not, the ranges of the groups overlapped. Thus, there is not a critical ratio above which men will assuredly be free of LBP, and below which they inevitably will suffer chronic LBP. Other factors, such as the size of the lumbosacral angle, the acuity of the lordotic curve, and the presence or absence of disease (such as arthritis and degenerated discs), are also related to LBP.

To try to account for chronic LBP merely on the basis of the kyphotic/lordotic ratio would appear to be a gross oversimplification of a complex problem. However, in light of the strong association found in this study between the presence or absence of chronic, mechanical LBP and the ratio of the length of the kyphotic curve to that of the lordotic curve, it would also be a mistake not to take that ratio into consideration.

The research described herein does not allow one to judge whether the relatively low kyphotic/lordotic ratios found in the LBP group made them especially liable to develop chronic LBP. Indeed, even the hypothesis, which we do not find highly plausible, that the relatively low ratios found in the LBP group were a characteristic response to the pain, rather than something that preceded the LBP, has not been subjected to a systematic test.

Based on the data from the present study and the aforementioned clinical impressions, we recommend controlled clinical trials to test the hypothesis that LBP will diminish in a group of chronic LBP sufferers who perform an exercise program that gradually increases the kyphotic/lordotic ratio. If this hypothesis is supported, there would be grounds for the dissemination of exercise programs that can reduce mechanical causes of LBP, with all that this implies for the promotion of personal and community health.

If such clinical trials indicate the efficacy of an exercise program that increases the kyphotic/lordotic ratio, they would logically lead to other questions. One of them would require a large-scale, long-term study to determine whether exercise regimens that increase the kyphotic/lordotic ratio are effective not only in treating, but in preventing, the all-too-common, all-too-costly affliction of chronic LBP. □

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Practical Approaches to the Laboratory Diagnosis of Common Sexually Transmitted Diseases

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Methods used for the laboratory diagnosis of sexually transmitted diseases continue to evolve. It is important that clinicians understand current testing methods so they can give patients high quality care, without inappropriately utilizing increasingly scarce laboratory resources. For each of the tests used in the diagnosis of sexually transmitted diseases there is a *false positive* and *false negative* rate which influences the treatment choices and the advice given to individual patients. In this article we will review the diagnostic methods currently employed in our laboratory, outlining test selection, specimen collection and transport, methods employed and their performance characteristics.

NEISSERIA GONORRHOEAE

The incidence of gonorrhea appears to be declining at a significant rate. In our laboratory, the proportion of submitted cultures positive for *N. gonorrhoeae* has fallen from 5.6% in 1986 to 0.97% in 1991. During the same time period, the number of cultures submitted increased from 16,388 to 18,002. Physicians are therefore testing even more individuals without infections than was previously the case. As a general rule, the number of organisms present in specimens from asymptomatic patients is considerably less than from symptomatic patients. The lower inoculum and the low prevalence in asymptomatic individuals have a significant effect on the performance characteristics and interpretation of test results.

Examination of Gram Stain Smears

In symptomatic males, the sensitivity of a Gram stain smear is between 90% and 95% in the hands of a well trained technologist who uses as a definition of positivity, the presence of typical gram negative diplococci within or closely associated with polymorphonuclear cells. When this definition is applied, the specificity should also be between 95% and 100%. In men with copious purulent urethral discharge, this high specificity rate, accompanied by a high pre-test probability (ie. the physician judges that it is probable that the patient has gonorrhea) enables the predictive value of a positive Gram stain to approach 100%. In asymptomatic males, there are usually fewer organisms and, in these circumstances, the sensitivity is between 50% and 70% only. If the above criteria are used to define a positive Gram stain, the specificity may also be between 95% and 100%. However, when specimens are collected from patients in whom infection is considered unlikely, the sensitivity and positive predictive value of a positive Gram stain result is unacceptably low. Because of this observation, we actively discourage use of the Gram stain in the investigation of asymptomatic males.

In women, the sensitivity of a cervical swab examined by Gram stain is between 50% and 70%. Although the specificity can reach 95% when intracellular gram negative diplococci are seen, the specificity is low when only extracellular organisms or morphologically atypical gram negative diplococci are present. Because of the lower sensitivity and positive predictive values in women, we do not recommend Gram stains in this patient population except in patients with high clinical probability of disease in whom positive Gram stain result reported immediately will result in a change in therapy.

Although the use of Gram stain to diagnose pharyngeal gonorrhea has not been well studied, it is almost certain that the test would not perform well in this setting and we discourage its use. The sensitivity of the Gram stain in patients with anorectal gonorrhea depends on whether the swabs were collected blindly or with the use of an anoscope – the sensitivity in the former being approximately 50%, and in the latter, 75%. When purulent material is encountered, Gram stain may be quite specific in this setting; however, specificity likely suffers considerably when no exudate is present.

For the diagnosis of *N. gonorrhoeae*, it is best to submit a swab of the purulent exudate itself (in the case of *Chlamydia trachomatis*, it is better to use the swab to lightly debride the urethral and the cervix to dislodge infected epithelial cells). The smear is best made at the bedside by rolling the swab over the surface of the glass slide rather than asking the laboratory to prepare the slides from submitted swabs. In our laboratory, Gram stains for investigation of gonorrhea of sites other than male urethral discharge is not performed routinely, but can be arranged for specific cases by discussion with a microbiologist.

Neisseria gonorroheae cultures

Culture remains the gold standard test for the diagnosis of genital urinary infections due to *N. gonorrhoeae*. The positivity rates of cultures relate to whether patients are

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symptomatic or asymptomatic (symptomatic patients have a larger number of organisms and are therefore more frequently culture positive). In a study by Handsfield *et al*, 96% of women with uncomplicated gonorrhea had positive endocervical cultures.[1] Of the 162, seven women were positive only from anal canal or pharyngeal cultures. Heterosexual males with gonorrhea will almost always have positive urethral cultures although a small proportion (6%) in the Handsfield study also had positive pharyngeal cultures. Among homosexual men with uncomplicated gonorrhea, only 58% had positive urethral cultures. Fifty percent had positive anal canal cultures, and in 31%, only this site was culture positive. In the same population, 17% had positive throat cultures and in 5% the throat was the only site positive for *N. gonorrhoeae*. These studies need to be done again in the AIDS era.

The recovery rate may be lower when inadequate specimens are submitted, when cotton swabs (which may be toxic to *N. gonorrhoeae*) are used, when specimens are not transported to the laboratory in appropriate transport medium, or when the specimens are delayed in transit. For clinics close to the VGH, we supply JEMBEC plates so that the specimen can be inoculated onto the media right in the clinic. This system uses a small carbon dioxide generating tablet (sodium bicarbonate and citric acid) to provide an appropriately moist and CO_2 enriched environment for the organism. Ideally, plates should be incubated overnight before being sent; however, same day transport to the laboratory for incubation is acceptable.

In the laboratory, plates are examined after 24, 48, and 72 hours for characteristic colonies and their identification confirmed by Gram stain, oxidase reaction and by direct immunofluorescence using a monoclonal antibody. There have been occasional reports of other Neisseria sp. being mistakenly reported as *N. gonorrhoeae* because the organisms may appear quite similar. In order to avoid such a potentially catastrophic occurrence, we confirm the identification of all *N. gonorrhoeae* from children using a biochemical system in addition to our routine antigen based system.

Strains are tested for production of beta-lactamase using a spot test for which results are immediately available and susceptibilities are performed to penicillin and tetracycline. Isolates are sent to Ottawa for further testing as part of a national survey program. At the present time, beta-lactamase producing strains of *N. gonorrhoeae* are recovered very infrequently in Nova Scotia. In the last year, only 4 of the 175 isolates that we examined produced beta-lactamase, and these are usually acquired outside the province or from sexual contacts from out-of-province.

Chlamydia trachomatis

The prevalence of *C. trachomatis* in Nova Scotia also appears to be declining. At the present time, approximately 3% of specimens submitted to our laboratory are positive for *C. trachomatis*. As with *N. gonorrhoeae*, the proportion of tests done in asymptomatic patients is increasing and the number of organisms present in asymptomatic patients tends to be less than in those with symptoms. Testing for *C. trachomatis* is generally indicated for the investigation of patients with urethritis, mucopurulent cervicitis or upper genital tract infections.

In asymptomatic cases, the decision to perform laboratory testing should take into consideration factors that lead to an increased index of suspicion for chlamydia infection. Recently published guidelines recommend testing of asymptomatic women under the age of 25 who: a) have had two or more sexual partners in the last year; b) have had a new sexual partner in the last two months; c) do not use contraception or who use a non-barrier method; and d) or in whom bleeding is induced when an endocervical swab is performed.[2] STD patients not given anti-chlamydia treatment and other groups in whom the prevalence exceeds 7% should also be tested.[2] At present, screening of asymptomatic homosexual men is not recommended. There is insufficient data to recommend testing of asymptomatic heterosexual men. In addition, sexual contacts of patients with either chlamydia or gonococcal infections should also have chlamydia screening tests performed.

Culture

Chlamydia culture, although considered by many as the gold standard test for the diagnosis of *C. trachomatis* infections, may in fact be less sensitive than the new tests designed for the detection of antigen.[3] Cultures require appropriate collection of specimen into special transport media containing sucrose-phosphate-glutamate, prompt transport to the laboratory, and are generally costly and slow. We reserve the use of cultures to the investigation of possible infection in children and in circumstances where medico-legal issues exist.

When collecting specimens for the isolation of *C. trachomatis*, it is important to recognize that it is an obligatory intracellular parasite and that the culture will have increased sensitivity if care is taken to debride the urethra or endocervix to assure that a large number of epithelial cells are submitted with the specimen.

Chlamydia cultures are performed by inoculating specimens into shell vials containing McCoy cells pretreated with cycloheximide. The inoculated cultures are incubated at 37°C under 5% CO_2. Cultures are fixed after 72 hours and stained with a monoclonal antibody tagged with fluorescein isothiocyanate. The monolayer is examined for fluorescing inclusions. The sensitivity of a single attempt to isolate *C. trachomatis* is not known but is probably approximately 75% in women and 90% in men. In asymptomatic patients, it is likely that the sensitivity declines further. The specificity of *C. trachomatis* culture should be 100%.

EIA

Enzyme immunoassays now represent the most frequently used diagnostic test for *C. trachomatis* in North

America and in the Province of Nova Scotia. In our hands, the specificity of newer assays now appear to be greater than 99%. Manufacturers have been able to improve the specificity substantially by providing a second confirmatory test methodology to re-test initially positive EIA results. The sensitivity of EIAs, when compared with cell culture for detecting endocervical *C. trachomatis* infection, ranges from 70% to 100%.[3] When EIAs have been compared with cell culture for detecting urethral *C. trachomatis* infections in men, the sensitivity has ranged from 67% to 92% (3). Currently, the Syva MicroTrak® EIA (not to be confused with the Syva MicroTrak® DFA test) is used in our laboratory. Other laboratories have chosen to use the Kallestad Pathfinder® EIA. Swabs are obtained by using fibre-tipped swabs and appropriate transport media supplied by the manufacturer. Packaged kits include two larger swabs and a smaller male urethral swab. Removing the ectocervical muco pus before obtaining the endocervical sample decreases the rate of bacterial contamination and may increase both the sensitivity and specificity of the EIA test. Care should be taken to sample large numbers of epithelial cells by rubbing the lateral aspect of the collecting swab against the urethra or endocervical canal. It is suggested that specimens be stored at 2-25°C and transported to the laboratory within 24 hours of collection. The chlamydia EIA test will occasionally be positive when the culture is negative. It is not clear what proportion of these positive tests are true positives and what proportion represent false positives. It is likely that the proportion varies from laboratory to laboratory, depending on the experience and skills of the laboratory technologists.

Direct fluorescence antibody staining

Because of the increased volume of testing and the availability of a confirmatory when using the EIA, we no longer perform DFA examinations of genital specimens. Because of the subjective nature of the microscopic examination, these tests must be performed by highly trained and experienced technologists. These tests are perhaps as sensitive as EIA but are likely to be less specific in the absence of a supplementary test.

HERPES SIMPLEX

No good data exist on the incidence of *H. simplex* (HSV) genital infections in sexually active Nova Scotia men and women, although it is likely that the incidence is declining in a manner similar to that of *C. trachomatis* and *N. gonorrhoeae.* Specimens are usually submitted to our laboratory in order to confirm a clinically active infection. Prior to the recently published article by Prober *et al,* many asymptomatic and pregnant women with a history of HSV infections were being tested during pregnancy for HSV.[4] In their study, they found that only 0.2% of women had positive HSV cultures at the time of delivery and only 1 of the 14 had a history of genital herpes. None of the infants born to the 12 women with serologic evidence of previous herpes infection contracted neonatal herpes. The only case of neonatal herpes which occurred in the 6904 deliveries was in an infant born to a mother having her first HSV infection. These results suggest that testing of asymptomatic women with a prior history of *Herpes simplex* is unlikely to be helpful to the physician managing the patient. These findings were confirmed in another study by Brown *et al*; neonatal HSV infection developed in only 1 of 34 infants born to women with reactivation of their HSV.[5] Not only are women with recurrent HSV unlikely to have positive cultures at the time of pregnancy but, if positive, their infants are unlikely to become infected.

The sensitivity of diagnostic tests for HSV depends, to a great extent, on the quality of the specimen submitted and the stage of the infection and the prior use of antiviral agents. The mean duration of virus shedding from lesions from men with recurrent genital herpes is 4.4 days (range 1-20). In females with recurrent genital herpes, the mean duration of viral shedding is 4.1 (range 2-14).

Electron microscopy

Electron microscopy is most helpful when the patient still has vesicular lesions. At least one million viral particles per 1 ml of specimen are required to allow detection of the virus by electron microscopy. Vesicle fluid can be collected by aspiration with a needle attached to a small tuberculin syringe. Rather than transport the needle and syringe to the laboratory, the specimen can be spotted onto a glass slide and allowed to air dry. On receipt in the laboratory, the virus can be eluted from the slide by adding a small drop of distilled water. Although the virus can be detected in vesicle fluid in approximately 50% of patients early in the course of the disease, the various herpes viruses cannot be distinguished one from the other by EM.

Culture

Vesicle fluid is best collected by breaking the top of a vesicle with a scalpel blade or 18 gauge needle. The fluid that oozes out can be collected with a swab and transported to the laboratory in the transport media available from our laboratory. If the specimen cannot be delivered directly to the laboratory, it should be refrigerated at 4°C or frozen at -70°C. Specimens for virus culture should not be left at room temperature for more than two hours and should never be frozen at -20°C.

In the laboratory, the specimen is inoculated into a cell culture and examined daily for cytopathogenic effect (CPE). The turnaround time can be 1 to 7 days, depending on the initial concentration of virus in the specimen. In our laboratory, the identity of the virus is confirmed by immunofluorescence using monoclonal antibodies to *Herpes simplex* type 1 or *Herpes simplex* type 2. This scheme allows for both culture confirmation and serotyping of isolates.

Serotyping may not add significantly to patient management, although patients often infer, perhaps cor-

rectly, that HSV 1 infection is more likely to have been acquired as a result of oral genital contact and is perhaps less likely a marker of sexual infidelity between partners. There is evidence that disease caused by *Herpes simplex* type 1 is less likely to recur and that attacks are less severe.

BACTERIAL VAGINOSIS

Bacterial vaginosis is a condition characterized by a disturbance of the micro flora of the vagina, resulting in malodour and a change in the predominant organisms present. This condition is referred to as a "vaginosis" rather than as a "vaginitis" because of the lack of an inflammatory response. The clinical presentation is often similar to vaginitis caused by yeast or *Trichomonas vaginalis*. Bacterial vaginosis is one of the most common causes of presentation of patients with vaginal complaints. In a 1982 study of a sexually transmitted disease clinic at the Victoria General Hospital, 37% of women had bacterial vaginosis; in a Halifax family planning clinic, the prevalence was 23%; and 23% of women attending the prenatal clinic at the Grace Maternity Hospital had bacteria vaginosis.[10] The transmission of bacterial vaginosis has been studied carefully and it is generally felt that the condition is not sexually transmitted, although correlation of the occurrence of bacterial vaginosis has been established with the number of sexual partners in the previous 30 days and with the lifetime number of sexual partners. It is likely that a number of factors contribute to the development of this condition, and the introduction of newly acquired bacteria may have a role.[9] At one time, the cause of the condition was thought to be *Gardnerella vaginalis*; however, it has been shown that this organism is found in up to 30% of asymptomatic patients. More recently, anaerobic organisms have been implicated, in particular, Mobiluncus species. These are found at a very low frequency (4%) in asymptomatic controls. Unfortunately, Mobiluncus sps. are not easily cultured. Organisms associated with bacterial vaginosis have been isolated from the gastrointestinal tract. The initial trigger that causes the perturbation of the normal flora is not clear. Hydrogen peroxide production appears to be the important factor in preventing other bacterial growth, especially in combination with low pH. A predominance of lactobacilli that do not produce hydrogen peroxide is seen in affected patients.

Diagnosis

Clinically, the diagnosis of *bacterial vaginosis* has been made based on the presence of three of the following four clinical signs: vaginal discharge which is thin and homogenous; vaginal pH of greater than 4.5; amine or fishy odour when potassium hydroxide (KOH) is mixed with the discharge; and presence of clue cells on microscopic examination. Clue cells are cells covered with bacteria so that the borders of the cell are obscured. It is with these clinical criteria that laboratory based methods have been compared. Gram stain to detect the change in the flora in bacterial vaginosis has been used for a number of years. The sensitivity and specificity of Gram stain has varied considerably in different studies with sensitivities as low as 62% and specificity at times as low as 56%, and both as high as 100%. By looking for 20% of cells to be clue cells, the sensitivity was reduced from 93% to 80%, but the specificity was increased from 85% to 95%.

Recently, a system has been devised to increase the reproducibility of the Gram stain. This is the method that is in use in our laboratory and we term it the "Nugent Score". The score is derived from the number of organisms resembling lactobacilli, as well as the number of organisms that resemble either Gardnerella and Bacteroides or curved gram variable rods. Comparison between centres has shown that this method is highly reproducible. As a general rule, it is agreed that the Gram stain is more sensitive than the production of the fishy odour with KOH and it is more specific than clue cells or culture for *G. vaginalis*. It is also more objective than the clinical criteria, with the exception of measurement of the pH, and allows the opportunity to review results and to conduct quality assurance.

We do not recommend that vaginal secretions be cultured for *G. vaginalis*, because of its low specificity, and in our laboratory we use the "Nugent Score" from slides prepared at the bedside.

TRICHOMONIASIS

Trichomonas vaginalis is a frequent cause of symptomatic vaginal infections. It is one of the most common of the sexually transmitted infections. In the past year, 15,615 vaginal smears were submitted to our laboratory. We are not able to determine how many were from women who had signs or symptoms suggestive of a diagnosis of vaginitis. Of these, *T. vaginalis* was identified in 205.

Extrapolating from American studies, as many as 250,000 cases of trichomoniasis are treated annually by physicians in Canada. The diagnosis of trichomoniasis was made in 10.4% of American women attending STD clinics. The World Health Organization estimates that 180 million cases of trichomonal infections occur annually worldwide.

Laboratory diagnosis

The accurate diagnosis of trichomoniasis is not easily achieved. The "classical" clinical symptoms and signs of trichomoniasis, ie. vulvovaginal irritation and a yellowish or greenish frothy discharge, is found in only 12% of infected women and correctly predicts the presence of trichomoniasis in only 71% of the time.[8] The presence of discharge, odour, irritation or dysuria have little discriminating value.[8]

The microscopic diagnosis is most frequently based upon the microscopic examination of vaginal discharge. Unfortunately, the wet mount procedure is quite insensitive and at least 25% of women with trichomoniasis will not be diagnosed correctly if it is used alone. Physicians

are often reluctant or unable to perform wet mounts in the office and it is usually not possible to transport the specimen to the clinical laboratory in a timely fashion.

Various stains have been used for the diagnosis of trichomoniasis. Smears stained using the Papanicolaou stain may be both insensitive and non-specific.[11] The use of immunofluorescence methods, while sensitive, is too costly for routine laboratory use. Many laboratories continue to use the Gram stain in the investigation of vaginitis.[7] Gram stains have the advantage that other causes of vaginitis may be recognized at the same time, ie. Candida and bacterial vaginosis. Therefore, only one glass slide needs to be submitted and laboratory costs are minimized. Gram stain examination for the purposes of diagnosis trichomoniasis has been used in our laboratory for approximately 10 years. We believe that in the hands of our experienced laboratory technologists both the sensitivity and specificity of the Gram stain may be higher than previously recognized. We are about to undertake a study to determine the sensitivity, specificity, intra- and interobserver error associated with reading smears for *T. vaginalis*. □

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Continued from page 187.

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Medical Humanities

T.J. Murray,* OC, MD, FRCPC, FACP

Halifax, N.S.

Crazywater

"My mother died because of alcohol. She was bludgeoned to death when I was about five. She used to drink quite a bit. She was only thirty-three and had four kids and was living on welfare in the north end of Winnipeg at the time. She used to like to drink and party. One night, one of her boyfriends, they were drinking and she was passed out in the bed. He just bludgeoned her to death with a gun and in the morning we got up and found her there."

Sue, a thirty-one year old Metis

When the medical meeting was over, a friend and I walked down an Edmonton street on a bright sunny morning, and saw legs sprawled out from a doorway, the attire and features telling us this was an unconscious Indian. Feeling a flush of embarrassment we step by, trying not to stare, and pretending we did not see. When I was a child I used to hear of people going to far off lands and getting used to bodies dead and dying in the streets. I could not believe that people would just step over bodies. Now I was doing it.

Brian Maracle in *Crazywater: Native Voices on Addiction and Recovery,* (Toronto: Viking Penguin, 1993) indicates that the "drunken Indian" image has been hurtful, because there are many who lead hard working responsible lives, but he also makes it clear that this image does represent a real and tragic problem. His 250 brief interviews tell an tragic story of drunkenness, despair, violence and death, but also a glimmer of hope and a new spiritual awakening among First Nations people.

I do not have solutions to the many distressing and agonizing problems faced by our native Canadians. I know only one thing for sure – one hundred years of federal paternalism and public neglect have worsened their plight, with little prospect of viable solutions under the current system. Until recently, we blocked every argument the native community brought forward for self-government. Our collective paternalism says that they could never manage themselves (as they did for many thousands of years) and, besides, they will just want back what we took from them.

I wonder what the future is going to say about us?

To sense the disintegration of native customs, traditions, culture, families and personal self respect you just have to read through a handful of the brief interviews. They are painful to read. Maracle travelled over Canada and the United States interviewing 250 Native people about their experiences with alcohol. In clear, resigned, and often majestically simple conversations they talk about the alcoholism, the violence and social disruption in their families, and their easy steps into the same alcoholism. In many instances it is not just a page out of the life of an alcoholic – it is a piece of the story of an alcoholic with an alcoholic father, alcoholic mother, alcoholic siblings and cousins and uncles and neighbours.

Just as it seems too painful to read another tragic story, there is a glimmer of hope. In some a rediscovery of their self respect and a sense of spiritualism is leading to recovery. But for every recovery there are still many more seeking only their next drink, lying in the doorways of buildings, stepped over by a public who does not want to see.

Brian Maracle focuses on alcoholism, but recent publicity about native communities indicates there are other equally serious problems – gasoline and glue sniffing, drugs, suicide and family violence.

Somehow the native and white communities and Government must come together to address the many problems facing the First Nations. Failure to do so will bring more personal tragedy and can lead only to confrontation and revolution.

Talk, Talk, Talk

When I was first studying neurology, I found it difficult to understand the concepts of normal speech and the various dysphasias that resulted from lesions in the brain. The terminology used by the many disciplines interested in speech and communication made it even more difficult. To get a grasp on the field, so that I could apply it to the understanding of my patients, I evolved rather simplistic concepts and approaches. Although this was comforting and practical, I was aware that the field was continuing to expand and change, the controversies and discoveries were multiplying, and the terminology was becoming even more foreign to me.

Jay Ingram reviews the broad field of speech and language in a new book, *Talk, Talk, Talk* (Toronto: Viking Press, 1992, $19.99) and outlines the current concepts, beliefs, arguments and unexplained puzzles. It is not a book to understand specific clinical disorders, but one that takes a broader view of language, and some of

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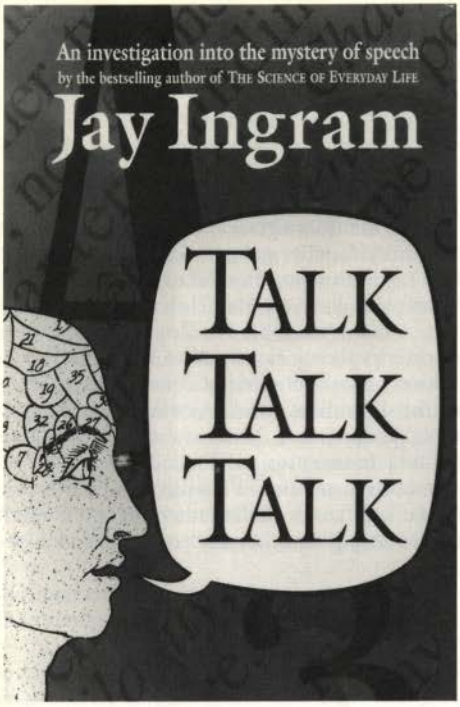

the interesting clinical and experimental observations on the development of speech.

I found it a most entertaining and informative read, and it rekindled an interest in speech, which I had lost as I became more distant from the developments in the field. Ingram is a great interpreter of science, and I always enjoyed him as the easy-going, informed and enthusiastic host of "Quirks and Quarks" on CBC each Saturday. Ingram is a member of the small group of outstanding science writers who can view a complex subject for a broad audience, and deliver understanding with clarity, enthusiasm and a respect for science. There are many writers who exploit science and medicine for easy stories that tickle the fancy of the public, but contribute little to understanding or interpretation.

Although scientists have not always been kind to the popular interpretation of science, it is an important aspect of science communication. Science, and medical science in particular, are expected to benefit society, and are for the most part supported by society, so it is reasonable to expect that we take seriously the translation of science for general understanding. There is a long tradition of those who interpreted science to the masses, Thomas Huxley in the Victorian Age, and Lewis Thomas in ours, but there is a growing band of excellent science writers, many found on the staff of major newspapers and magazines.

Ingram's book rekindled my interest in an area I had neglected, and I am now interested in doing a lot more reading on the subject. That is a pretty good recommendation for a book.

Robert Joy – History of Antibiotics

Visiting Dalhousie on Friday, September 17 will be Dr. Robert Joy, Professor of The History of Medicine at the Uniformed Services University in Bethesda, Maryland. Dr. Joy is one of the outstanding medical historians, and oversees the largest History of Medicine Program in any North American medical school. His area of expertise is the history of military medicine, but he will speak as the first Dr. T.J. Murray Visiting Scholar in the Medical Humanities, on "The History of Antibiotics". This lecture will be the first in the Friday-at-Four series in the 1993-94 academic year.

Ethics in Film

In the next year at Dalhousie Medical School there will be a series of six films that illustrate important ethical issues. Following the films there was an open discussion by the audience. The first film was shown on Sunday evening, September 26 at 7 pm in Theatre A at the Sir Charles Tupper Medical Building. The film was *Arrowsmith* based on the novel by Sinclair Lewis, hosted by Dean John Ruedy.

On October 24 the film will be *One Flew Over the Cuckoo's Nest* hosted by Dr. Jock Murray.

On November 28 the film will be *Hospital* hosted by Dr. Richard Goldbloom.

There will also be films on January 30, February 27 and March 27. All are welcome.

Out of One's Tree

Side effects occur with any drug, we tell our patients when they ask if the drug we are prescribing has any complications. We can often list the adverse symptoms – insomnia, mood change, dizziness, drowsiness, muscle pains and so on. But, do we ever really think about the experience of these side effects? Recently, there has been considerable discussion in the literature of medicine, indicating the need to understand the "the patient's story". We are familiar with the physician's story, as written it in charts, and as heard in presentations and rounds. These are the perception of disease and suffering from the physician's point of view. To look at some patient stories, the first *Reading Weekend* will take place on October 15-17 at the Tattinghouse Inn in Wolfville, where physicians, spouses and friends will discuss books, essays and poems that elucidate the patient's story. (Call 494-2514 if you are interested in further information on this or later Reading Weekends.)

In the January, 1993 issue of *Harper's Magazine*, currently my favourite magazine, there is a very poignant essay by Stanley Elkin about his experience with side effects from steroids, which he was prescribed because of symptoms of multiple sclerosis. The fact that we have recently learned that oral steroids are not beneficial in MS, and may even be deleterious, adds irony to Elkin's experience of emotional and psychotic changes due to to the drug. The patient's story was one of a very disturbing descent into madness, the worst experience of his life.

He raged at his children and wife, shocked his friends on the phone, embarrassed himself in front of strangers, and accused his physician of attempting to seduce his wife. He hurled abuse at nurses and hospital staff, ate his food with hands, and pulled the IVs out of his arms. After discharge from hospital it was a further month before he felt he was thinking clearly, and reflected on the "worst, most difficult, most agonizing days and nights of my life".

The physician's story was probably "Experienced side effects of steroids. Emotional lability. Drug discontinued".

Recommended Reading

Dr. Charles G. Roland, Hannah Professor of the History of Medicine at McMaster has just published a book on medical practice in the Warsaw Ghetto during World War II, entitled *Courage Under Seige.* He outlines the struggle of Jewish doctors and nurses attempting to provide support and care to the half million Jews confined to the Ghetto. They even started a clandestine medical school to train 500 students. They had few resources but tragically had more than enough clinical material due to disease and starvation.

Allan Marble, Research Director in the Department of Surgery at Dalhousie, has published a new book entitled, *Surgeons, Smallpox and the Poor: A History of Medicine and Social Conditions in Nova Scotia, 1749-1799.* Dr. Marble has spent years studying the physicians and medical practice in 18th and 19th Century Nova Scotia.

Like automobile design, it is tempting to consider that medical art of the past far surpasses modern versions. Dr. Ken Roberts and Dr. JDW Tomlinson of Memorial University have published a magnificent book on anatomical illustration which is beautifully printed and illustrated. (*The Fabric of the Body: European Traditions of Anatomical Illustration.* New York: Oxford University Press, 1992, $125.00).

Dr. Susan Sherwin of the Department of Philosophy at Dalhousie, has published a well received and well reviewed book, *No Longer Patient: Feminist Ethics and Health Care* (Philadelphia: Temple University Press, 1992, $39.95) Dr. Sherwin is a respected medical ethicist, and outlines some of the concerns about standard medical ethics and the organization of our health system that require reform.

In an interesting series of essays on the development of the Canadian Health Care system over the past century, *Canadian Health Care and the State,* edited by C. David Naylor (Montreal and Kingston: McGill-Queens University Press, 1992, $21.95) there is a chapter by Dr. Colin Howell of St. Mary's University on the life and ideas of Dr. A.P. Reid. Dr. Reid was the first Dean of Dalhousie Medical School who had a strong vision of a social welfare system for all Canadians. □

OBITUARIES

Dr. Charles MacDonald (70) of Halifax, Nova Scotia died on July 29, 1993. Born in New Waterford, he received his medical degree from Dalhousie University in 1953, and he practised medicine in Halifax from 1958 to 1993. He was an avid sports fan and volunteered his medical services to the Saint Mary's University Athletic Department for 25 years. He served on the Nova Scotia Highway Safety Advisory Board. He is survived by his wife, and two daughters, to whom the *Journal* extends sincere sympathy.

Dr. William A. Condy (70) of Halifax, Nova Scotia died on September 2, 1993. Born in Springhill he received his medical degree from Dalhousie University in 1954. He figured prominently in the 1956 Springhill mine disaster when he went into the mine and offered medical assistance. He was former chairman of the Halifax School Board. He is survived by three daughters. The *Journal* extends sincere sympathy to his family.

ADVERTISERS' INDEX

Atrovent®

(ipratropium bromide)

INHALATION SOLUTION

THERAPEUTIC CLASSIFICATION

Bronchodilator

INDICATIONS AND CLINICAL USES

Atrovent (ipratropium bromide) solution is indicated for the therapy of acute exacerbations of chronic bronchitis. Atrovent solution, when used in conjunction with a β_2-adrenergic stimulant solution such as fenoterol or salbutamol, is indicated for acute asthmatic attacks. It is to be administered by compressed air or oxygen driven nebulizers.

CONTRAINDICATIONS

Known hypersensitivity to Atrovent (ipratropium bromide), to any of the product ingredients, or to atropinics.

WARNINGS

Atrovent (ipratropium bromide) solution in the 20 mL multidose bottle contains preservatives (benzalkonium chloride and disodium ethylene diamine tetraacetic acid-EDTA-disodium). It has been reported that these preservatives may cause bronchoconstriction in some patients with hyperreactive airways.

The 2 mL unit dose vial (250 mcg/mL, 125 mcg/mL) does not contain preservatives.

Atrovent should not be used alone for the abatement of an acute asthmatic attack since the drug has a slower onset of effect than that of an adrenergic β_2 agonist.

Care should be taken to ensure that the nebulizer mask fits the patient's face properly and that nebulized solution does not escape into the eyes. There have been isolated reports of ocular complications (i.e., mydriasis, increased intraocular pressure, angle closure glaucoma) when nebulized ipratropium bromide either alone or in combination with an adrenergic β_2 agonist solution has escaped into the eyes. In the event that glaucoma is precipitated or worsened, treatment should include standard measures for this condition.

PRECAUTIONS

General:

- Patients should be instructed in the proper use of the nebulizer.
- Caution is advised against accidental release of the solution into the eyes.
- In patients with glaucoma, prostatic hypertrophy or urinary retention, Atrovent (ipratropium bromide) should be used with caution.
- If a reduced response to Atrovent becomes apparent, the patient should seek medical advice.
- Atrovent solution, when administered to patients with acute severe asthma, should be used with concomitant β_2-adrenergic stimulant therapy.

Use in Pregnancy:

The safety of Atrovent in pregnancy has not been established. The benefits of using Atrovent when pregnancy is confirmed or suspected must be weighed against possible hazards to the fetus. Studies in rats, mice and rabbits showed no embryotoxic nor teratogenic effects.

Use During Lactation:

No specific studies have been conducted on excretion of this drug in breast milk.

Benefits of Atrovent use during lactation should therefore be weighed against the possible effects on the infant.

Use in Children:

The efficacy and safety of Atrovent in children younger than 5 years has not been established.

Use with Other Drugs:

In patients receiving other anticholinergic drugs, Atrovent should be used with caution because of possible additive effects.

In patients with glaucoma or narrow anterior chambers, the administration by nebulizer of a combined Atrovent-β_2 agonist solution should be avoided unless measures (e.g., use of swimming goggles) are taken to ensure that nebulized solution does not reach the eye. Exposure of the eyes of such patients to a nebulized combination of Atrovent and a β_2 agonist solution has been reported to result in increased intraocular pressure and/or acute angle closure.

Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglycate, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

ADVERSE REACTIONS

The frequency of adverse reactions recorded in 214 patients receiving Atrovent (ipratropium bromide) solution was as follows, given by percentage of patients reporting: Dry mouth or throat, 9.3; Bad taste, 5.1; Tremor, 4.2; Exacerbation of symptoms, 4.2; Burning eyes, 0.9; Nausea, 0.9; Sweating, 0.9; Cough, 0.9; Headache, 0.5; Palpitations, 0.5.

The adverse effect judged to be most severe was exacerbation of symptoms. This occurred in 8 patients treated with Atrovent solution alone, 6 of whom withdrew from the clinical studies. Bronchospasm occurred in 3 patients with acute severe asthma who received Atrovent solution alone. In two patients, this was reversed after therapy with a β_2 sympathomimetic solution. The third patient received no other therapy.

The following table compares the incidence of adverse effects of the combination of Atrovent and a β_2 agonist (either fenoterol or salbutamol) solution with that of the β_2 agonist alone.

ADVERSE EFFECT	ATROVENT + β_2 AGONIST (% of 94 patients)	β_2 AGONIST (% of 96 patients)
Tremor	31.9	26.0
Dry mouth	16.0	28.1
Bad taste	16.0	13.5
Vomiting	2.1	2.1
Palpitations	2.1	1.0
Headache	1.1	2.1
Cough	1.1	0.0
Flushing	1.1	0.0
Dizziness	0.0	1.0
Numbness in leg	0.0	1.0

There have been isolated reports of ocular effects such as mydriasis, increased intraocular pressure, and acute glaucoma associated with the escape of nebulized ipratropium bromide–alone or in combination with a β_2 agonist solution into the eyes.

DOSAGE AND ADMINISTRATION

In adults, the average single dose of Atrovent (ipratropium bromide) solution is 250-500 µg of ipratropium. In children, aged 5-12 years, the recommended dose is 125-250 µg of ipratropium bromide solution. This should be diluted to 3-5 mL with preservative free sterile Normal Saline [Sodium Chloride Inhalation Solution, USP 0.9%] or with a bacteriostatic sodium chloride solution, 0.9% preserved with benzalkonium chloride (see PHARMACEUTICAL INFORMATION). Nebulization should take place using a gas flow (oxygen or compressed air) of 6-10 L/minutes and the solution nebulized over a 10-15 minute period. The Hudson Updraft™, Bennett Twin Jet® and Inspiron Mini-Neb® nebulizers, with facemask or mouthpiece have been used. The manufacturers' instructions concerning cleaning and maintenance of the nebulizer should be strictly followed. Treatment with Atrovent solution may be repeated every 4-6 hours as necessary.

PHARMACEUTICAL INFORMATION

Stability and Storage Recommendations:

20 mL Bottle: Unopened bottles of Atrovent (ipratropium bromide) solution should be stored at controlled room temperature (below 30ºC). Solutions diluted with presevative free sterile Sodium Chloride Inhalation Solution, USP 0.9% should be used within 24 hours from time of dilution when stored at room temperature and within 48 hours when stored in the refrigerator. Dilutions may also be made with a bacteriostatic sodium chloride solution 0.9% which contains benzalkonium chloride as the bacteriostatic agent (see WARNINGS). This diluted solution may be stored at room temperature and used within 7 days.

Controlled laboratory experiments using mixtures of Atrovent solution with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6mg/mL preserved with benzalkonium chloride) solutions and diluted with a sterile bacteriostatic sodium chloride solution 0.9% (i.e. normal saline), preserved with benzalkonium chloride, indicated that such mixtures were stable for 7 days at room temperature. For the preparation of such mixtures, it is recommended that only sterile solutions of bacteriostatic sodium chloride 0.9% preserved with 0.01% benzalkonium chloride be used to maintain the level of preservative in the mixture.The safety of preservatives other than benzalkonium chloride has not been established.

Incompatibilities: Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglygate solution, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

2 mL Unit Dose Vials (250 mcg/mL and 125 mcg/mL):

Unopened unit dose vials of Atrovent solution should be stored at controlled room temperature (below 30ºC) and protected from light. If required, the solution should be diluted with a preservative free sterile sodium chloride solution 0.9% and used immediately. Any solution remaining in the vial must be discarded.

The solution is physically compatible with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6 mg/mL) solutions. If such mixtures are prepared, they should be diluted with preservative free sterile sodium chloride solution 0.9% and used immediately. Any unused portion of such combined solutions must be discarded.

AVAILABILITY

20 mL Bottle: Atrovent (ipratropium bromide) solution is provided as 20 mL clear, colourless or almost colourless solution containing 250 µg/mL (0.025%) Atrovent in isotonic solution. This solution is preserved with benzalkonium chloride 250 µg/mL and EDTA-disodium 500 µg/mL at pH 3.4 in an amber glass bottle with screwcap.

***2 mL Unit Dose Vial:* 250 µg/mL** Atrovent solution is also provided as 2 mL of clear, colourless solution containing 250 µg/mL (0.025%) ipratropium bromide in isotonic solution, presented in a plastic single use vial. One vial contains a total of 500 µg of ipratropium bromide.**125 µg/mL** Atrovent solution is also provided as 2 mL of clear colourless solution containing 125 µg/mL (0.0125%) ipratropium bromide in isotonic solution, presented in a plastic single use vial. Each vial contains a total of 250 µg of ipratropium bromide.

The complete Product Monograph for Atrovent (ipratropium bromide) Inhalation Solution is available to health professionals on request. Patient Information/Instructions are provided with the solution.

REFERENCES:

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Boehringer Ingelheim

Boehringer Ingelheim (Canada) Ltd./Ltée
5180 South Service Road., Burlington, Ontario L7L 5H4

Dedicated to Respiratory Care

PAAB

N-4225/93

once daily Nasacort®

(Triamcinolone Acetonide Nasal Inhaler)

THERAPEUTIC CLASSIFICATION

Corticosteroid for nasal use

ACTIONS AND CLINICAL PHARMACOLOGY: Triamcinolone acetonide is a potent anti-inflammatory steroid with strong topical and weak systemic activity. When administered intranasally in therapeutic doses, it has a direct anti-inflammatory action on the nasal mucosa, the mechanism of which is not yet completely defined. The minute amount absorbed in therapeutic doses has not been shown to exert any apparent clinical systemic effects.

INDICATIONS AND CLINICAL USE: Nasacort™ (triamcinolone acetonide) nasal inhaler is indicated for the topical treatment of the symptoms of perennial and seasonal allergic rhinitis unresponsive to conventional treatment.

CONTRAINDICATIONS: Active or quiescent tuberculosis or untreated fungal, bacterial and viral infection. Hypersensitivity to any of the ingredients of Nasacort™ (triamcinolone acetonide).

WARNINGS: In patients previously on prolonged periods or high doses of systemic steroids, the replacement with a topical corticosteroid can be accompanied by symptoms of withdrawal, e.g. joint and/or muscular pain, lassitude, and depression; in severe cases, adrenal insufficiency may occur, necessitating the temporary resumption of systemic steroid therapy. Careful attention must be given to patients with asthma or other clinical conditions in whom a rapid decrease in systemic steroids may cause a severe exacerbation of their symptoms.

Pregnancy: See Precautions.

PRECAUTIONS:

1) The replacement of a systemic steroid with Nasacort™ (triamcinolone acetonide) has to be gradual and carefully supervised by the physician. The guidelines under "Administration" should be followed in all such cases.
2) During long-term therapy pituitary-adrenal function and hematological status should be assessed.
3) Patients should be informed that the full effect of Nasacort™ therapy is not achieved until 2 to 3 days of treatment have been completed. Treatment of seasonal rhinitis should, if possible, start before the exposure to allergens.
4) Treatment with Nasacort™ should not be stopped abruptly but tapered off gradually.
5) Corticosteroids may mask some signs of infection and new infections may appear. A decreased resistance to localized infections has been observed during corticosteroid therapy; this may require treatment with appropriate therapy or stopping the administration of Nasacort™.
6) The long term effects of Nasacort™ are still unknown, in particular, its local effects; the possibility of atrophic rhinitis and/or pharyngeal candidiasis should be kept in mind.
7) There is an enhanced effect of corticosteroids on patients with hypothyroidism and in those with cirrhosis. Acetylsalicylic acid should be used cautiously in conjunction with corticosteroids in hypothrombinemia.
8) Because of the inhibitory effect of corticosteroids on wound healing, in patients who have had recent nasal surgery or trauma, a nasal corticosteroid should be used with caution until healing has occurred.
9) Patients should be advised to inform subsequent physicians of prior use of corticosteroids.
10) Until greater clinical experience has been gained, the continuous, long-term treatment of children under age 12 is not recommended.
11) **Pregnancy:** The safety of Nasacort™ in pregnancy has not been established. If used, the expected benefits should be weighed against the potential hazard to the fetus, particularly during the first trimester of pregnancy.
Like other glucocorticosteroids, triamcinolone acetonide is teratogenic to rodents and non-human primates (see under TOXICOLOGY). The relevance of these findings to humans has not yet been established. Infants born of mothers who have received substantial doses of glucocorticosteroids during pregnancy should be carefully observed for hypoadrenalism.
12) **Lactation:** Glucocorticosteroids are secreted in human milk. It is not known whether triamcinolone acetonide would be secreted in human milk, but it is suspected to be likely. The use of Nasacort™ in nursing mothers, requires that the possible benefits of the drug be weighed against the potential hazards to the infant.
13) **Children:** Nasacort™ is not presently recommended for children younger than 12 years of age due to limited clinical data in this age group.
14) Fluorocarbon propellants may be hazardous if they are deliberately abused. Inhalation of high concentrations of aerosol sprays has brought about cardiovascular toxic effects and even death, especially under conditions of hypoxia. Aerosols are safe when used properly and with adequate ventilation, but excessive use should be avoided.
15) To ensure the proper dosage and administration of the drug, the patient should be instructed by a physician or other health professional in the use of Nasacort™ (see Patient Instructions).

ADVERSE REACTIONS: Adverse reactions reported in both controlled and uncontrolled studies involving 1148 patients who received Nasacort™ (triamcinolone acetonide) are provided in the following table:

Adverse Experience	Nasacort % (n = 1077)	Placebo % (n = 545)
Headache	20.4	19.4
Upper Respiratory Infection	5.3	8.1
Nasal Irritation	5.1	4.2
Throat Discomfort	4.6	3.3
Dry Mucous Membranes	3.5	2.2
Epistaxis	4.6	6.6
Sneezing	3.1	5.5
Sinusitis	2.1	3.7

When patients are transferred to Nasacort™ from a systemic steroid, allergic conditions such as asthma or exzema may be unmasked (see Warnings).

SYMPTOMS AND TREATMENT OF OVERDOSAGE: Like any other nasally administered corticosteroid, acute overdosing is unlikely in view of the total amount of active ingredient present. However when used chronically in excessive doses or in conjunction with other corticosteroid formulations, systemic corticosteroid effects such as hypercorticism and adrenal suppression may appear. If such changes recur, the dosage of Nasacort™ (triamcinolone acetonide) should be discontinued slowly consistent with accepted procedures for discontinuation of chronic steroid therapy. (see Administration). The restoration of hypothalamic-pituitary axis may be slow; during periods of pronounced physical stress (i.e. severe infections, trauma, surgery) a supplement with systemic steroids may be advisable.

DOSAGE AND ADMINISTRATION: See Warnings.

Nasacort™ (triamcinolone acetonide) is not recommended for children under 12 years of age.

Careful attention must be given to patients previously treated for prolonged periods with systemic corticosteroids when transferred to Nasacort™. Initially, Nasacort™ and the systemic corticosteroid must be given concomitantly, while the dose of the latter is gradually decreased. The usual rate of withdrawal of the systemic steroid is the equivalent of 2.5 mg of prednisone every four days if the patient is under close supervision. If continuous supervision is not feasible, the withdrawal of the systemic steroid should be slower, approximately 2.5 mg of prednisone (or equivalent) every ten days. If withdrawal symptoms appear, the previous dose of the systemic steroid should be resumed for a week before further decrease is attempted.

The therapeutic effects of corticosteroids, unlike those of decongestants, are not immediate. Since the effect of Nasacort™ depends on its regular use, patients must be instructed to take the nasal inhalations at regular intervals and not as with other nasal sprays, as they feel necessary.

In the presence of excessive nasal mucus secretion or edema of the nasal mucosa, the drug may fail to reach the site of action. In such cases it is advisable to use a nasal vasoconstrictor for two to three days prior to Nasacort™ therapy. Patients should be instructed on the correct method of use, which is to blow the nose, then insert the nozzle firmly into the nostril, compress the opposite nostril and actuate the spray while inspiring through the nose, with the mouth closed.

An improvement of symptoms usually becomes apparent within a few days after the start of therapy. However, symptomatic relief may not occur in some patients for as long as two weeks. Nasacort™ should not be continued beyond three weeks in the absence of significant symptomatic improvement.

Adults and Children 12 years of age and older: The recommended starting dose of Nasacort™ is 400 μg per day given as two sprays (100 μg/spray) in each nostril once a day. If needed, the dose may be increased to 800 μg per day (100 μg/spray) either as once a day dosage or divided up to four times a day, i.e., twice a day (two sprays/nostril), or four times a day (one spray/nostril).

After the desired effect is obtained, patients may be maintained on a dose of one spray (100 μg) in each nostril once a day (total daily dose: 200 μg per day).

AVAILABILITY: Nasacort™ (triamcinolone acetonide) is a metered-dose aerosol unit containing a microcrystalline suspension of triamcinolone acetonide in the propellant dichlorodifluoromethane and dehydrated alcohol USP 0.7% w/w. Each canister contains 15 mg triamcinolone acetonide. Each actuation releases approximately 100 μg triamcinolone acetonide of which approximately 55 μg are delivered from the nasal actuator to the patient (estimated from in-vitro testing). There are at least 100 actuations in one Nasacort™ canister. The device should not be used after 100 inhalations, since the amount delivered thereafter per actuation may not be consistent. It is supplied with a nasal adapter and patient instructions: Box of one.

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1. Storms W.W. et al., Annals of Allergy, 1990; 66: 329-334.
2. Tinkelman D. et al., Annals of Allergy, 1990; 64 (Feb. Part II): 234-240.
3. Spector S. et al., Annals of Allergy, 1990; 64: 300-305.
4. Data on File: Rhone-Poulenc Rorer; 5029-02.
5. Data on File: Rhone-Poulenc Rorer; 5029-01.

unresponsive to conventional treatment

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