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Manpower, Womanpower and Government Power

Recent comments by the Minister of Health of Nova Scotia regarding manpower make real all the worst fears concerning the medical profession, and its loss of many vital characteristics such as independence and self regulation.

While the Minister's comments have been about manpower, due to the ever increasing expense of our health care system, his real concern is about power to control his own budget. He has little control over utilization of the system and any control he might wish to exercise is politically unpopular. He cannot control hospitals easily in this province, since they have become in some instances, a part of the patronage system or ways of subsidizing small communities. Besides, limiting hospitals leaves his government open to accusation of cutting services. He cannot control the medical school without stirring up a hornet's nest. (Medical education is not directly funded by the Health Department and any further cuts to the school would guarantee a loss of quality in medical care and research that probably would not and should not be tolerated.) The Minister cannot control other allied health services when the major move of his government must be towards a home care service utilizing more nursing service, physiotherapists, respiratory technicians, community care workers and social workers.

However, he can control the number of doctors that seems to be the source of all the spending and expense. He can do this because of the many myths that are currently alive and well in our community — some of which are well known.

1) Doctors make too much money.
2) Doctors' incomes are not related to care provided but to their need of an excessive income.
3) There are too many doctors for our population. (Manpower statistics being so confusing, no one can logically prove or disaprove this statement).
4) Medical care is a provincial concern and thus it is up to the province to control its manpower situation.

With understanding based on this mythology, it is no wonder that the Minister of Health has suggested to the recently established Royal Commission that they examine manpower closely, and he has strongly suggested the outcome of that study before it even begins to work.

Unfortunately, in addition to the misconceptions noted above, there exists a real problem in manpower distribution in Canada, if not in this province. The President of the Medical Society of Nova Scotia has stated that our distribution of doctors in this province is good. This does not mean that all communities have the service they want and need. The Medical Society, Dalhousie and the Department of Health have not completely solved this problem for a small number of communities. It is unfortunate that government will use this undeniable fact, along with other misperceptions, to move to control numbers of doctors everywhere and in every situation.

A rapid solution to the real problem of distribution of doctors to needed areas would go a long way in undercutting the government's case for control of manpower. Mandatory service for a few years by new graduates might be that solution along with incentives as in Northern Ontario, and better support by the profession, for those physicians in outlying areas.

But after dealing or while dealing with this long neglected problem, it is equally important to deal with the myths surrounding the manpower issue.

Dalhousie's recent decision to cut back on its newly admitted class of medical students from 96 to 84 is not good news. Consider a recent quote from the Association of Canadian Medical College's Director of Research. "Fixing quotas of entrants to faculties of medicine in different parts of the country gives the illusion that future physician numbers are being controlled in line with some rationally determined policy; however, the number of physicians entering practice in Canada (and Nova Scotia) is determined not by the numbers graduating from Canada (or Nova Scotia) medical schools but by the numbers who achieve licensure."

Thus cutting back on Dalhousie graduates will not achieve anything if graduates from other parts of Canada or foreign medical schools move into the area. Past experiences have shown Nova Scotia or Dalhousie graduates to have a high out migration to the rest of Canada and that this area has a low attraction for graduates of other medical schools in Canada. The bottom line then is that native Nova Scotians will now find it more difficult to enter medical school since almost all medical schools favor natives of their own province when accepting applicants. Any underestimation of manpower needs will probably lead to emigration of physicians to Nova Scotia from other countries — many with different cultural backgrounds and not specifically trained for Canadian needs.

Even if we could and did control licences in this province, it would be done with no rational excuse other than limiting expense. There is no evidence to suggest that present needs, and even recommendations for health care service are even being met, let alone exceeded. When that evidence is available, then government should act, and not before. The present situation is a financial problem imposed on government due to its excessive promises that all services are free. The population should understand that this then is a problem of too little money and not too many services.

The medical profession, like other citizens, of course recognizes the limits of the provincial budget. A rational planning exercise to determine priorities of service might make sense, but this should not be done by suddenly limiting medical manpower and not by prejudging what priorities of service are necessary or desirable.

The manpower question is an important one and much more needs to be documented and researched about vital issues such as the impact of the increasing number of female graduates, changes in the way medicine is provided and the expectations about availability of specialist and other care.

Basic facts about how many physicians are actually practising in this province and what they do are still not available. At present the Canadian Medical Association, with the assistance of the Royal College of Physicians and Surgeons and the Collage of Family Physicians, are now for the first time documenting actual numbers of who does what in this country. This study will provide basic background, along with other studies, that would and should be used in any health planning manpower exercise.

The demographics of our aging population might possibly indicate increased needs and the promises of access by all, made by our government, requires wisdom and great caution before manipulating our manpower resources. □

J.F. O'C

"There is a persistent unwillingness of the human mind to accept persuasion that is in conflict with evidence. All who exercise power find this obstinacy by far the most annoying tendency with which they have to contend."

John Kenneth Galbraith (1908 -)

Guillain-Barré Syndrome

A 10 YEAR REVIEW FROM THE VICTORIA GENERAL HOSPITAL

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This disorder is an acutely or, more accurately, a subacutely evolving paralytic illness of unestablished etiology, occurring in all parts of the world and in persons of all ages. The condition is colloquially known as the Guillain-Barré syndrome (GBS). However initially, it was diagnosed as the Landry-Guillain-Barré-Strohl syndrome in recognition of the clear descriptions provided first by Landry in 1859[1] and again in 1916 by Guillain, Barré and Strohl.[2]

Many major facets of the disease remain imperfectly defined including its pathogenesis, although considerable evidence has been forthcoming in recent years to suggest that it represents an aberrant immune response, often triggered off by a precipitating insult most notably a viral illness.

The characteristic pathologic feature is a lymphocytic and macrophagic cellular infiltration which results primarily in the destruction of myelin. However, when severe, the underlying axon cylinders may be affected also leading to axonal degeneration. This distinction is of more than academic interest because the predominant pathophysiological insult, which can be determined by means of electromyography, dictates the prognosis. When due to myelin disruption, as it is in most instances, the outlook for functional recovery is excellent. Should there be a major component of axonal destruction, an infrequent occurence, then recovery depends upon axonal regeneration. This is a time consuming and often incomplete process which leaves the patient with residual dysfunction. The damage which does occur is inflicted in a diffuse fashion at both the proximal and/or distal portions of the peripheral nervous system, giving rise to the other common designation for this disorder, i.e., acute polyradiculoneuropathy.

The essence of management is supportive care — most particularly respiratory assistance when required. The role of steroids remains controversial. Plasmapharesis, based on a recent multi-centre study, appears to be of benefit primarily for patients who progress rapidly to marked paralysis and/or who require ventilatory assistance.

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We present in this article a review of 10 years experience with GBS at the Victoria General Hospital.

METHODS

Cases of acute GBS managed in the Victoria General Hospital, Halifax, Nova Scotia between the years 1973 and 1983 were analysed retrospectively. Cases were identified through a search of files in the Medical Records and EMG Departments. The criteria used for a diagnosis of GBS were those established by the Ad Hoc NINCDS Committee on GBS.[3] These include progressive motor weakness of multiple limbs and areflexia or distal areflexia with proximal hyporeflexia. Features used to strongly support the diagnosis included: 1) progression over a few days or a few weeks, usually complete by four weeks; 2) relative symmetry; 3) mild sensory symptoms or signs; 4) cranial nerve involvement; 5) recovery usually beginning 2-4 weeks after the disease has stopped progressing; 6) autonomic dysfunction; 7) absence of fever at the onset of the disease; 8) an elevated CSF protein with fewer than 10 WBC/mm^2; and 9) an abnormal electrodiagnostic study with evidence of conduction disturbance or slowing across proximal portions of the nerve (F-wave responses). Features that excluded the diagnosis of GBS included hexacarbon abuse, porphyria, diphtheria, lead intoxication and evidence of other paralytic diseases. Patients were not included if they had a pure sensory disorder. Recognized variants of GBS were included; most importantly the Miller Fisher syndrome which consists of ophthalmoplegia, areflexia and ataxia. Also included were some patients with GBS who progressed longer than four weeks. However, patients were excluded from this study if they progressed either gradually or in a step-wise fashion for longer than six months. Those patients were classified as chronic inflammatory demyelinating polyneuropathy which is likely a different disorder.[4]

Follow up information was obtained from office charts of the neurologist following the patient when available and hospital records from the Nova Scotia Rehabilitation Centre when patients were transferred from the VGH to that institution.

Grading disability of the patients was performed utilizing the disability scale described by Hughes *et al.*[5]

0. normal.
1. minor signs or symptoms but capable of work.
2. walking without support but unable to work.
3. walking with assistance or mechanical support.

4. bedridden or confined to a wheelchair.
5. requiring assisted ventilation.
6. dead.

This scale has been used in multiple studies including the Guillain-Barré syndrome Study Group analysis of the effect of plasmapheresis on this disorder.[6]

RESULTS

Sixty cases of GBS were admitted to the Victoria General Hospital over the 10 year period between 1973 and 1983. The average number was 6 per year ranging from 1 case in 1981 to 11 in 1977. There were 37 males and 26 females. The patients had a mean age of 42.2 years, ranging from 15 to 81 years old. As these patients were taken from the population of an adult care hospital, patients with GBS in the pediatric age range would have been excluded from this analysis. Twenty-six of the patients lived in Halifax County, 29 in other regions of Nova Scotia and 5 outside of the province. Statistical analysis of these epidemiological features of GBS in this group of patients was not performed, as patients with GBS during this period would have been admitted to other Nova Scotian hospitals. Information on patients admitted to other hospitals was not available.

The onset of the disease occurred during all months of the year with a somewhat greater incidence during the spring and summer months (63% between April 1 and September 30; 37% between October 1 and March 31).

Seventy-five percent of the patients recalled having a preceding illness in the eight weeks prior to the onset of their neurological symptoms. This was most often an upper respiratory tract infection (24 patients) and less commonly a gastrointestinal (7 patients) or nonspecific "flu-like" illness (5 patients). Other infections were seen in 7 patients, surgery in 2, pancreatitis in one and an upper G.I. hemorrhage with transfusion in one. Only one patient had a history of recent vaccination and this was with tetanus toxoid. Infectious pathogens were identified occasionally and were quite varied. They included cytomegalovirus, Epstein-Barr virus, varicella, staphylococcal aureus pneumonia, gram negative organism (septicemia), proteus (urinary tract infection) and mumps.

The standard criteria for GBS were satisfied in all patients with only two patients being identified as having a variant of the disease. Those two patients had the Miller Fisher syndrome.

Most often, the onset of the disease was with sensory symptoms, usually paresthesias in distal extremities. Pain, which was a significant symptom for several patients during the course of their disease, was the first neurological symptom in 13 patients. The pain was variable, consisting either of burning pain in the extremities, knife-like shooting pains, or deep aching pain in the extremities. Three patients were initially admitted with the suspicion of an acute surgical lesion, including acute cholecystitis, small bowel obstruction and an acute lumbar disk. The sensory symptoms were generally followed relatively quickly by motor weakness, 40% of the patients presenting with weakness as their initial symptom. An ascending pattern of weakness, thought to be typical of GBS, was seen only in 55% of patients. The other common motor presentation was simultaneous onset of weakness in the upper and lower extremities both distally and proximally.

The neurological manifestations at the height of the illness are noted in Table I. In addition to frequent weakness in the upper and lower extremities and trunk, many patients had cranial nerve involvement, most often with facial nerve weakness. Although two patients had classical features of the Miller Fisher syndrome with ophthalmoplegia, ataxia and areflexia, there were other patients with marked ophthalmoplegia who had the more common features of generalized extremity and facial weakness. Dysphagia due to bulbar weakness was a problem in some patients, requiring endotracheal intubation for protection of the airway in one patient despite reasonable repiratory function.

TABLE I

NEUROLOGICAL MANIFESTATIONS DURING COURSE OF GUILLAIN-BARRÉ SYNDROME IN 60 PATIENTS

Weakness		
Lower Extremity	58	96.7%
Upper Extremity	53	88.3%
Trunk	43	71.2%
Sensory Symptoms	46	76.7%
Pain	32	53.3%
Cranial Nerve Involvement	23	38 %
III, IV	5	8.3%
V	1	1.7%
VI	7	11.7%
VII	19	31.7%
IX, X	7	11.7%
XII	2	3.3%
Autonomic Involvement	17	28.3%
Urinary Sphincter or Bowel Disturbance	17	28.3%
Ventilatory Assistance	16	26.6%

Sensory symptoms, though commonly seen, tended to be milder than motor involvement in most patients. Autonomic nervous system involvement manifested either by hypertension, hypotension, cardiac arrhythmias, alterations in sweating, urinary sphincter disturbance or marked bowel involvement with severe constipation was seen in 28.3% of patients. Often, the cardiac manifestations were mild, with persistent sinus tachycardia. However, one patient died of a fatal arrhythmia. The blood pressure frequently fluctuated between episodes of hypertension and hypotension. One patient had marked persistent orthostatic hypotension with syncopal episodes upon standing. Hypertension was often managed with antihypertensives that usually were withdrawn by the end of the patient's disease course. Urinary retention was most often treated with catheterization. In two patients urecholine was given for persistent retention.

Ventilatory assistance was required for 16 patients, of whom 12 eventually underwent tracheostomy. This was maintained for an average of 19 days, ranging from 2-55 days.

As noted above, pain was a frequent presenting complaint of patients. During the course of the illness pain, unexplained by procedures or complications of the disease, was experienced in over half the patients. Some patients required narcotic analgesics for control of pain. In one patient the introduction of carbamazepine (Tegretol®) and in two the introduction of steroids was accompanied by reduction in the pain.

The majority of patients (85%) had stopped progressing by four weeks following the onset of their symptoms, with 63% having no further progression after two weeks. A few patients continued worsening after 8 weeks but none for longer than 6 months. After reaching the point of maximum deficit a plateau phase of the illness occurred ranging from 2 days to 5 months. By 4 weeks 89% of patients had begun to improve. Two-thirds of patients reached a maximum disability grade of 4 or greater with 16 progressing to ventilatory assistance as noted above. Two patients died secondary to pneumonia, one from cardiac arrhythmia and one had a fatal hemorrhage at a tracheostomy site.

Follow up for at least one year or until the patients reached disability grade 2 was obtained in 45 of the patients. Of these 38 returned to grade 2 function or better with the majority grade 1 or better. By 4 months 26 of the patients were grade 2 or better. No patients were known to be on a ventilator at the end of the follow up. Only 3 patients were known to have persistent significant disability of grade 3 or greater for longer than the one year follow up. Increasing age affected adversely the outcome of patients. Three of the 4 patients who died were over 65 years of age. The mean age of patients who died or were left with severe disability was significantly greater than the mean age of the total group (55.4 ± 20.5 years, mean ± S.D. vs. 42.2 ± 18.9) ($p < .05$). This age difference was not seen between the total group and those that died or had severe disability at the height of their illness (grades 5 and 6), (42.2 ± 18.9 vs. 49.9 ± 19.0) (N.S.).

The time spent in the Victoria General Hospital for the entire group averaged 5.5 weeks ranging from 3 to 257 days. There was a marked difference in admission days between the ventilated and nonventilated group, 10.5 weeks versus 4 weeks respectively. Forty percent of patients were transferred to another hospital after discharge from the VGH, most commonly to the Nova Scotia Rehabilitation Centre. Patients who had been ventilated required transfer twice as often as those who did not.

There were several complications encountered during the management of these patients apart from those due to autonomic involvement noted above. Infection, seen in 19 patients, was the commonest complication. This was most often pulmonary. Two patients had pulmonary emboli and an additional one a deep venous thrombosis. Significant hyponatremia ($Na+ < 130$) was encountered in seven. A serious hemorrhagic complication leading to death occurred in one patient.

Malignancy has been reported as an accompanying feature in a small percentage of patients with GBS.[7] Associated neoplasm was found in four patients from this group. There were two cases of colon cancer, one adrenal tumour and one acute myelogenous leukemia. One patient developed nephrotic syndrome at the same time as her acute neuropathy which was persistent after several years of follow up. This association has been previously reported, though it is very uncommon.[8] The CSF protein was increased (>450mg/L) in 48 of the 53 patients in whom it was measured. Of the five with normal CSF protein only one had a measurement later than two weeks after the onset of neurological symptoms. Of interest, seven patients had normal CSF protein during the first week of their illness but increased levels when the CSF was sampled more than two weeks after the onset of the disease. Six patients had a mild to moderate increase in their CSF cell count, usually less than 10 mononuclear cells per mm^2, however, one patient had a cell count of 49.

Thirty-eight of the patients underwent EMG and abnormalities were seen in all. Most often, these consisted of conduction velocity slowing, prolongation of distal latencies and absent or reduced sensory nerve action potentials. Mild to moderate fibrillation on needle examination was seen frequently. Preserved distal motor conduction amplitudes and only mild fibrillation are indicators of a more favourable prognosis for early recovery. In our patients with persistent severe disability at one year the EMG early in the course of the disease showed markedly reduced distal motor amplitudes and florid fibrillation.

Oral steroids were given to approximately one-third of the patients. However, the variable time of introduction and dosage as well as selection biases for introducing steroids make any meaningful interpretation of their effect in this group impossible. Only one patient underwent plasmapheresis. He was a patient who required assisted ventilation and at 10 months was using leg braces and a cane.

DISCUSSION

Guillain-Barré syndrome has an annual incidence of approximately 1.7/100,000.[9] Using census data for Nova Scotia for the years between 1973 and 1983, there should have been approximately 104 cases in the population over 15 years old during that period. The 55 cases from Nova Scotia reported here would account for a little over half the cases in the Province. The results of this study should be representative of what occurred in GBS patients in Nova Scotia during that period.

The disorder is generally benign, with good or excellent recovery in the majority of patients. Management of GBS should include providing information to the patient. As they are generally very frightened of the implications of their problem during its acute stages, we feel it is important to provide reassurance that the majority who contract this illness do recover. In our group 6.6% died, 3.3% had recurrence of the disease and 5% were known to suffer severe residual disability. Older patients were more likely to die or have severe residual disability, but age did not predict how severely affected a patients would become before recovery began. Death was most commonly secondary to pulmonary infections, reinforcing the need for prompt and aggressive management of infectious complications. One patient died from pneumonia acquired near the end of a long hospital course (150 days). Long hospital stays are associated with increased likelihood of complications that can increase morbidity and mortality of the disease.

Autonomic nervous system involvement was frequent and often required intervention particularly with catheterization for urinary retention and with treatment of hypertension. Autonomic involvement can be more severe, as illustrated by the one case that died from a cardiac arrhythmia. Many aspects of the management of such patients may require the facilities of an Intensive Care Unit. Particular attention needs to be paid to the potential for rhythm disturbances and other cardiovascular difficulties. Careful management of respiratory difficulties is of course paramount in the care of patients with GBS. One of the major improvements in the outlook of these patients came with the evolution of modern methods of ventilatory assistance and this remains probably the single most important aspect of acute management of these patients.

This study illustrates the long hospital stays that characterize this disorder, particularly for patients who require ventilatory assistance during the course of their disease. As noted above, morbidity and mortality likely increases with requirements for longer hospital stay and more aggressive management. The complications of pneumonia, pulmonary emboli and bleeding from a tracheostomy site highlight this point. As the disease utimately has a good prognosis the goal for effective specific therapy for GBS should be to shorten the hospital stay and length of time for intensive management of patients.

There is a great deal of evidence that GBS is an immune mediated disorder. At autopsy, one of our fatal cases had marked inflammatory infiltrates around nerve roots. Serum from patients with GBS when passively transferred to laboratory animals has resulted in neuropathic changes.[10] Anecdotal reports had suggested that steroids and plasmapheresis may have a favourable effect on the course of GBS. Prednisolone does alter the course of experimental allergic neuritis, though the therapeutic effect was not marked.[11] A controlled study of the effect of steroids on GBS did not confirm expected benefits and in fact steroids may have been deleterious.[5] Currently it is being questioned whether the dosage of steroids may alter efficacy and plans to evaluate the effect of high dose steroids are underway in Great Britain and North America.

A large controlled multicentre trial of plasmapheresis on patients with GBS examined the effect on patients with grade 3 disease or greater when treated within 30 days of the onset of neurological symptoms.[6] This study did show benefit from plasmapheresis. The time to improve one grade was reduced by 21 days and the time for 50% of patients to reach grade 2 was reduced by 32 days in the entire group and 72 days in those requiring ventilatory assistance. The benefit from plasmapheresis appeared to be greatest when it was started early in the course of the disease. In an editorial of that study by Dyck and Kurtzky, they concluded that although they had concerns about some of the methodological aspects of the trial, they would treat patients with plasmapheresis, though not cases that were mild or had stopped progressing.[12] Further information from that trial that might affect how we manage patients with GBS may become available in the future as further analysis of the data is forthcoming.

The group of GBS patients reported here is very similar to those in other series. The disorder generally has a good outlook with serious complications or death in a small percentage of patients. Pain can be a promiment feature of the disease and lead to early misdiagnosis and problems with management. Careful attention must be paid to the respiratory, cardiac and general medical management of these patients. Current information suggests that plasmapheresis may benefit patients with severe disease when treated early. Such treatment may shorten the time required for ventilatory assistance and the overall hospital stay. This hopefully will decrease the morbidity and mortality in patients and reduce the cost to the medical care system of management of these patients. □

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Subarachnoid Hemorrhage:
DIAGNOSTIC CONSIDERATIONS

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Subarachnoid hemorrhage (SAH) is a major cause of stroke in North America, and its annual incidence is estimated at 12 per 100,000 population.[3 10] In Canada, this amounts to about 4,000 cases of SAH per year. Saccular or congenital berry aneurysms are responsible for 75% of cases of SAH; an additional 6% are caused by arteriovenous malformations; trauma, tumor, encephalitis, hypertension and blood disorders also account for some cases. In most of the remainder, a cause is never found in spite of extensive investigation.

It is estimated that 2-5% of the population will harbour congenital berry aneurysms and, of these, 2-3% per year will rupture. The median age of SAH due to aneurysm is 55 years, with 70% presenting between the ages of 40 and 70. SAH is a disease that is associated with a very high morbidity and mortality. The hemorrhage is fatal or disabling in up to 70% of patients; one-third will die from the initial insult within 72 hours; and an additional one-third will die or become disabled due to vasospasm, re-bleed, or other medical or surgical complications.[10]

Various studies have shown that the number of cases of SAH being operated on by neurosurgeons lies between 1.6-3.6 per 100,000 population per year. This means that neurosurgeons are treating only a fraction of the assumed cases of SAH (13-30%).[3] The reasons for this are unclear, but it may be partly due to a poor recognition of its clinical presentation. In this light, a review of the clinical features of SAH is pertinent.

CLINICAL PRESENTATION

The classical clinical presentation of SAH is a middle-aged individual who, during severe exertion, experiences the abrupt onset of a severe generalized headache, which is often described as being the worst headache ever experienced. This headache may be associated with nausea, vomiting and photophobia. The level of consciousness may be altered, if only temporarily.

There is usually no difficulty recognizing that an SAH has occurred when a patient presents with the above classical presentation. However in many patients, some elements of that classical picture will be missing and, therefore, a high index of suspicion is needed in order to avoid missing an SAH.

Headache is present in most patients who are conscious following an SAH. It is generally occipital in origin, but rapidly becomes generalized. It is generally responsive to mild narcotic analgesics such as codeine and tends to subside over several days.

Occasionally, patients will present with headaches that are less severe or last for shorter periods of time and these, probably, represent minor or warning leaks. This concept of warning leak was first brought forward by Gillingham[5] in 1967, although Richardson and Hyland[12] in 1941 had earlier recorded premonitory symptoms in patients with SAH. Fifty percent or more of patients [5 8] who present with a major hemorrhage gave a history of earlier headaches, always of sudden onset and without loss of consciousness. Recovery from these spells is usually rapid with some neck stiffness and generalized headache, and with the patient being able to return to work within approximately 48 hours.[5]

The site of the headaches is usually of no value in localizing the aneurysm. In one-third of patients, it begins during strenuous activity such as lifting, bending, straining or sexual intercourse. However, one-third of cases of SAH will occur during sleep and rest, and another third will occur during non-vigorous random activity.[13] Depending upon the degree of initial hemorrhage, the sudden violent headache may be followed by collapse, or brief confusion or unconsciousness. Some patients may rapidly progress to coma. In one-third of patients unconsciousness is the initial presentation. Nausea, vomiting, photophobia and neck stiffness may develop within one or two hours of the onset of headache. Occasionally, patients complain of back pain in the lumbar region secondary to blood tracking down the spinal subarachnoid space. The incidence and severity of these symptoms are a reflection of the severity of the initial hemorrhage.

Focal signs or symptoms may occur with SAH. Some, such as hemiparesis, may be an immediate consequence of intracerebral extension of the SAH, while delayed hemiparesis can occur secondary to cerebral vasospasm. Cranial nerve palsies also constitute focal signs and may be a result of either SAH or expansion of the aneurysm in the absence of rupture.[2 6] The most common aneurysm to present with cranial nerve palsy is the posterior communicating artery aneurysm. In one study, 61% of patients with posterior communicating artery aneurysms

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presented with some degree of third nerve palsy, either as a result of aneurysm rupture or of aneurysm expansion.[9] In fact, aneurysm is the single most common cause of isolated third nerve palsy. It is classically taught that third nerve palsy, secondary to aneurysm, is invariably associated with a dilated and fixed pupil. However, up to 14% of patients who have aneurysmal third nerve palsies may have pupillary sparing.[9]

Findings on examination are often non-specific. Neck stiffness with or without a positive Kerning's sign is common. True meningismus represents a restriction of passive neck flexion without interference with neck movements in other directions. Meningismus may be absent in patients who are very young, very old or in deep coma.[4] It may also be absent or minimal in patients with a small warning leak. Elevations of blood pressure and mild increases in temperature are common. Fundiscopic examination may reveal the presence of pre-retinal or subhyaloid hemorrhages, but papilledema is uncommon.

Patients who present with less severe SAH are often misdiagnosed as having a systemic infectious illness, such as gastro-enteritis or the "the flu". Migraine headache and hypertensive encephalopathy are other common misdiagnoses.[1 7 11] It should not be forgotten that a previous history of migraine affords no immunity to SAH[4], and that intracerebral hemorrhage secondary to hypertention can cause SAH by direct extension into the subarachnoid space or by way of the ventricular pathways. Some patients, because of profound neck stiffness, are mistakenly felt to be suffering from primary neck pathology. Approximately 30-50% of patients who present with a major SAH will have had some warning signs or symptoms in the preceding two to three weeks. The most common of these is headache. Unfortunately, these symptoms are often attributed to aneurysms only in retrospect.[11] Such early mild symptoms require close clinical scrutiny, since their recognition offers the best opportunity to alter the natural history of SAH.

Adams *et al.* found that there was a delay in diagnosis in nearly 25% of all patients with SAH even though headache, vomiting and loss of consciousness were common presenting symptoms.[1] Patients are frequently dazed, confused or drowsy in the first few hours following a SAH. If the onset of the illness has not been observed, then SAH may not be a diagnostic consideration. It should be considered part of the differential diagnosis of stupor or coma. Although nausea, vomiting, fever and diffuse aches may lead to a diagnosis of viral illness, when these symptoms are of sudden onset a diagnosis of SAH must be considered.

INVESTIGATIONS

Lumbar puncture is almost always diagnostic, and uniformly blood-stained CSF can generally be obtained within minutes of a SAH. Xanthochromia takes somewhat longer to occur. It is almost never present in under two hours; it is usually present by 12 hours and always present by 24 hours.[13] Red cells disappear in the CSF over a period of 7-10 days. Xanthochromia persists for one week or longer.[12]

A diagnostic lumbar puncture should always be considered in every patient who presents with a history of sudden severe headache, provided that the patient is conscious and does not have focal neurologic signs or papilledema. All such patients who have a positive lumbar puncture should be referred for neurosurgical evaluation. All other patients who have papilledema, focal neurologic signs or a deteriorating level of consciousness should be referred for immediate neurological or neurosurgical evaluation prior to lumbar puncture. Most of these patients require CT scanning, although it should be borne in mind that 25-30% of lumbar puncture proven SAH have a normal CT scan. So, even if the CT scan is normal, the lumbar puncture may still be required to diagnose SAH.

SUMMARY

In summary, SAH is a devastating condition associated with significant morbidity and mortality. It is important that the various clinical presentations of SAH are recognized. The diagnosis of severe SAH usually poses no problems. Minor symptoms and signs of "early warning bleeds" or "sentinel hemorrhages" are of the utmost importance in diagnosis. In these particular patients, who are otherwise healthy but who present with sudden unexplained headache, lumbar puncture should be done to make an early diagnosis before a repeat, more serious hemorrhage occurs. □

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A Review of Sympathetically Mediated Pain:
REFLEX SYMPATHETIC DYSTROPHY AND CAUSALGIA

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The pathogenesis, common features and treatment modalities of sympathetically mediated pain syndromes are described. Early recognition and initiation of therapy are of vital importance in resolution of these syndromes. Delay in treatment may increase morbidity and socio-economic loss by prolonging rehabilitation.

The first clear description of sympathetically mediated pain following soft tissue or nerve injury was made in 1864 during the American Civil War by Weir Mitchell. He described a number of cases of soldiers who sustained peripheral nerve injuries and subsequently developed severe burning pain in the affected extremity.[1] Since that time a number of similar clinical situations have been described with an equal number of names, but they can collectively be referred to as "sympathetically mediated pain syndromes."[2]

These syndromes fall into two major categories: 1) Reflex Sympathetic Dystrophy; and 2) Causalgia. The International Association for the Study of Pain Subcommittee on Taxomony defines reflex sympathetic dystrophy as "continuous pain in a portion of an extremity after trauma which may include fracture but does not involve a major nerve, associated with sympathetic hyperactivity."[3] Causalgia is defined as "burning pain, allodynia, and hyperpathia, usually in the hand or foot, after partial injury of a nerve or one of its major branches."[3]

PATHOGENESIS

Various mechanisms have been postulated to explain the occurrence of these pain syndromes.

1. Peripheral Hypotheses

A. Livingston: Tissue damage produces chronic irritation of peripherial nerve endings that leads to reflex sympathetic efferent activity, decreased perfusion, muscle spasm and perpetuation of the pain process.[4]

B. Doupe: "Artificial synapse" theory; ephaptic (conduction of a nerve impulse between nerve fibres which are conducted directly through the nerve membrane from one fibre to the other) short circuit between sympathetic efferents and somatic afferents.[5]

2. Central Hypothesis

A. Melzack: A loss of normal sensory input from the periphery to the brainstem reticular formation reduces its usual inhibitory influence on the dorsal horn of the spinal cord. Thus, there is "central biasing dysfunction." This results in self-sustaining activity in closed neuron loops at all neural levels and can be triggered repeatedly by nocious impulses from the injured site.[6]

3. Combined Hypotheses

A. Bonica: Damage to A delta and C fibres make them hypersensitive to catecholamines. This results in a barrage of input to the central nervous system with disinhibition of dorsal horn circuits and development of self-sustaining loops with consequent hyperactivity.[7]

B. Roberts: Tonic activity in myelinated mechanoreceptor afferents is induced by sympathetic efferent action on sensory receptors. This afferent input produces tonic activity in Wide-Dynamic-Range dorsal horn neurons which have become sensitized by C fibre input following trauma. Roberts' recent review of sympathetically mediated pain documents considerable experimental evidence to support this theory.[2]

CLINICAL FEATURES

These syndromes exhibit a dynamic pathological process involving changes in the skin, muscles, vessels and bones in the extremities. The onset can occur immediately following an injury, which can vary from a high velocity missile injury to a trivial muscle sprain, or it can be remote from the time of injury. The common denominators are dysesthesia (an unpleasant abnormal sensation, whether spontaneous or evoked), hyperesthesia (increased sensitivity to stimulation, excluding the special senses) and hyperpathia (a painful syndrome, characterized by increased reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold).

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The clinical course can be arbitrarily divided into three phases:

1. **Acute or Denervation Phase:** (0-3 months).
   - hyperpathia and dysesthesia
   - increased blood flow leading to increased temperature
   - accelerated hair and nail growth
   - increased or decreased sweating
   - localized, soft, spongy edema
   - loss of venous architecture
   - dependent rubor
   - reduced range of movement

2. **Dystrophic or Hypersensitivity Phase:** (3-6+ months).
   - hyperpathia and dysesthesia, which may spread
   - decreased blood flow leading to decreased temperature
   - decreased hair growth and brittle nails
   - hyperhidrosis
   - spreading, brawny edema
   - pale cyanotic color
   - greatly limited range of motion, with decreased muscle use leading to greatly reduced extremity function
   - constant attention to protection of affected extremity, withdrawal, seeking seclusion, dramatic reactions to stimuli may occur

3. **Atrophic Stage:** (6+ months).
   - less hyperpathia and dysesthesia
   - less blood flow alteration, leading to less temperature difference
   - coarse hair and ridged nails
   - increased or decreased sweating
   - smooth, glossy and drawn skin with subcutaneous atrophy
   - wasted muscles with reduced strength
   - pericapsular fibrosis, tendon contracture, reduced range of motion
   - chronic pain personality

ASSESSMENT

Most of the information necessary for making the diagnosis of a sympathetically mediated pain syndrome is obtained from the history and physical examination. Laboratory aids are available to confirm diagnosis and may be useful for following the response to treatment.

1. Radiography. Patchy osteoporosis in the affected extremity may be seen as well as subepiphyseal and periarticular demineralization, especially in the distal joint areas. These finding, as a general rule, are late findings.
2. Thermograms, plethysmograms, bone scans and doppler techniques which can measure extremity blood flow and/or temperature differences between limbs may be useful, especially in atypical presentations.
3. Sympathetic blockade with local anaesthetics may be both diagnostic and therapeutic.

MANAGEMENT

1. Sympathetic Blockade

Sympathetic blockade has been and continues to be the classic and conventional treatment modality. The proposed mechanism of pain relief is the cessation of sympathetic stimulation of the "pain-afferent" fibres, be they A delta and C fibres as postulated by Bonica,[7] or mechanoreceptor afferents, as postulated by Roberts.[2] An interesting and valuable aspect of the sympathetic blockade is that the therapeutic effect usually outlasts the duration of the block. The usual course following each block is a progressive, step-like decrease in pain, and a decrease in residual signs and symptoms. Melzack believes that the block breaks the vicious cycle of the pain pathway and decreases the size of the self-sustaining neuron pool in the neuraxis.[6]

The longer the syndrome is allowed to persist without treatment, the more resistant it becomes. The dysfunction in the neuraxis increases with consequent spread of pain, hyperalgesia and other symptomatology beyond the distribution of the affected nerve. Eventually this central pool becomes self-sustaining and independent of peripheral input. At this stage, sympathetic blockade may have little effect; thus early intervention provides a better chance of successful therapy.

Sympathetic blockade of the upper body and limbs can be effected by stellate ganglion block; that of the lower body and limbs by lumbar sympathetic block utilizing a dilute solution of local anaesthetic.

Intravenous regional techniques utilizing guanethidine and reserpine have met with limited success. Hannington-Kiff first reported the treatment of sympathetically mediated pain with intravenous guanethidine in 1974.[8] Guanethidine produces sympathetic blockade by displacing and depleting norepinephrine from sympathetic nerve endings. In 1980 Benzon described a similar technique using reserpine.[9]. Wynn-Parry and Withrington are very aggressive with intravenous regional guanethidine, giving their patients 5-6 blocks over a two week period interspersed with intensive physiotherapy.[10].

2. Surgery

Surgical sympathectomy may be useful in cases in which a series of blocks produces complete but only temporary relief. However, because of variant anatomy, operative lumbar sympathectomy or stellate ganglionectomy may not provide complete sympathectomy.

3. Physical and/or Occupational Therapy

This is essential. Therapy is most effective if

performed whilst the patient is under the effect of the sympathetic blockade. Objectives are to increase the range of motion of the affected limb, re-establish muscle strength and to teach the patient to use the limb without precipitating pain. Continued therapy over a variable time course is almost always necessary.

4. Transcutaneous Electrical Nerve Stimulation (TENS)

This modality is helpful in some patients, especially in those in whom invasive therapies are not acceptable. It is also convenient as it can be used at home after a brief course of instruction. It does not have any systemic side effects. Unfortunately, in some situations, it has been found to increase sympathetic activity and thus pain.

5. Medications

Treatment with traditional analgesics, including narcotics, provides only variable or no relief of pain. Beta-blockers, alpha-blockers and calcium channel blockers have all been used with variable results.

6. Psychological Therapy

It is well known that in some patients emotional status can aggravate or alleviate pain in sympathetically mediated pain. This probably occurs via action at the level of the reticular formation. Biofeedback, relaxation therapy, development of coping strategies and hypnosis have all been used. □

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The noblest service comes from nameless hands, and the best servant does his work unseen.

Oliver Wendell Holmes (1809 - 1894)

Somatosensory Evoked Potentials

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Somatosensory evoked potential studies have become fairly routine in clinical medicine over the last five years, in most major medical centres in North America. The test has become easy to do because of the advances in electronics and computer technology. Basically, the technique involves measuring conduction time in the peripheral as well as the central nervous system, in order to detect conduction delays or blocks in the somatosensory pathways not only in the peripheral nerves but also in the spinal cord, brain stem and cortex. In the past few decades we have been able to do peripheral nerve conduction studies and electromyography in order to detect neurogenic dysfunction in the anterior horn cell, the nerve axon, the myoneural junction and the muscle itself. Now we are able to go one step ahead and are able to measure conduction time in the central nervous system.

METHOD

The equipment used is very similar to that used for nerve conduction studies and electromyography but has greater sensitivity and selectivity in the electronic sense of the term. In addition, it has 'on board' a computer averager which records extremely small evoked resposes generated in the spinal cord, brain stem and the cortex, in response to stimulation of a peripheral nerve. The special filter in the equipment helps in 'weeding out', extraneous electrical signals generated from structures like the muscles and the heart.

The peripheral nerve is stimulated by surface skin electrodes. The intensity of the stimulation is high enough to stimulate the large myelinated fibres in the peripheral nerves.[1] The duration of each stimulus is 0.2 to 2 msec at a rate of about five per second, and this is fairly comfortable to most patients. The nerves stimulated are the median nerve at the wrist, the tibial nerve at the ankle and the peroneal nerve at the knee. Other peripheral nerves can be stimulated also including cranial nerves such as the trigeminal nerve.

The short-latency somatosensory evoked potentials are picked up by surface electrodes placed close to the generator source along the somatosensory pathways. For example, in stimulation of the median nerve at the wrist, recording electrodes are placed in the supraclavicular area over the brachial plexus, over the posterior aspect of the neck at the second cervical vertebra which is close to the graciles and cuneate nuclei, and over the scalp on the contralateral side overlying the sensory cortex.

In the case of the tibial nerve, it is stimulated at the ankle and recording electrodes are placed over the popliteal fossa overlying the tibial nerve, spineous process of T12 overlying the sacral root-entry zone and at the vertex of the head 2 cm posterior to CZ (EEG 10-20 system). Recording electrodes can also be placed over the cervical and thoracic spine in order to detect any prolongation of conduction time or latencies. The evoked responses thus picked up with recording electrodes are amplified and filtered with a midfrontal or noncephalic electrodes used as references. The evoked responses are averaged about 500 to 1000 times and the test is repeated at least once in order to verify the wave forms thus obtained.

The evoked potentials latencies can be affected by peripheral neuropathy or limb temperatures. Often, clinical interpretation would have to be based on time intervals between evoked responses recorded at different locations along the neuroaxis. Side-to-side differences in latencies are also found to be sensitive and reliable measurements. One cannot always depend on the amplitude of evoked responses, as they can be extremely variable.

There is still some controversy about the generators source of certain evoked responses recorded. For example N19 latency recorded on the contralateral side of the head over the parietal area was thought to originate in the primary sensory cortex.[1,2] However, later clinicopathological studies with human clinical material suggests that N19 is generated in the thalmus.[3]

CLINICAL APPLICATIONS

The short-latency somatosensory evoked response study can be helpful in the diagnosis of proximal lesions in the peripheral nerves such as plexopathies and radiculopathies, in conjunction with the conventional nerve conduction studies and electromyography. These studies have also been found to be useful in studying conditions such as multiple sclerosis, compressive myelopathies, transverse myelitis and trauma to the spinal cord. In some centres intraoperative monitoring of the somatosensory evoked response studies are done frequently during surgery for scoliosis and for monitoring comatose patients.[1]

Somatosensory evoked response studies have been found to be equally sensitive as visual evoked potential studies (where a visual stimulus to the retina evokes a visual cortical response which can be recorded over the occiptal area on the scalp) in multiple sclerosis.[4]

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Also, somatosensory evoked potentials have been found to be much more sensitive than auditory brain stem evoked potentials (where an auditory stimuli evokes an auditory cortical response that can be recorded over the ear lobe using CZ on the scalp as a reference) in this condition.

CONCLUSIONS

The somatosensory evoked response study is another accurate, objective and almost completely reproducible neurophysiologic study, which can be an effective extension of the clinical neurologic examination of the sensory system. □

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Palliative Care for the Alzheimer's Patient

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Patients in the final stage of Alzheimer's disease deserve palliative care. Technology competes with compassion, making treatment decisions difficult when the patient's wishes are not known. Careful weighing of the benefits and burdens of each treatment option can prevent unwarranted treatment and help avoid subjective "quality of life" decisions. The physician must deal with such issues as the admission of final stage Alzheimer's patients to acute care institutions, antibiotic use and artificial feeding. Technology can be used in a palliative care approach that focuses on the patient's comfort.

As our population ages, physicians are responsible for an increasing number of patients in the final stage of Alzheimer's disease. What kind of medical care is most appropriate for these patients? A physician's training and medical oath emphasize actions to sustain and prolong life. However, in our sincere efforts to provide thorough treatment for patients with late state Alzheimer's do we sometimes impose discomfort and interfere with dying?

"The process of dying has been institutionalized and surrounded by technology. By denying death the medical profession can be accused of forgetting an ancient duty — to comfort always."[1]

In our medical management of these hopelessly ill patients, technology competes with compassion.[2]

THE PATIENT

The patient in the terminal phase of Alzheimer's has lost the ability to speak or communicate. Bowel and bladder control have gone. Seizures may have developed. There may be an almost complete absence of any spontaneous movement or reaction to people or other stimuli. A general stiffening of muscles has resulted in marked immobility. This severely and irreversably demented patient passively accepts what we do for (and to) him.

Most physicians treating a patient in the final stage of Alzheimer's disease would not provide emergency recuscitation or artificial life-support measures. However, once accepted that such heroic treatment will not be offered, what kind of medical care should be undertaken?[3]

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PALLIATIVE CARE

The concept of palliative care (comfort in place of cure) has become accepted for patients with terminal cancer. The concept applies equally for the final stage of Alzheimer's disease as the patient's comfort should be the accepted treatment goal.

Palliative care means excellent symptom control, especially pain relief. It should also mean a judicious use of investigations and no unwarranted treatment.[4] If a treatment is not beneficial to a particular patient, we are under no moral obligation to use it, since it is useless and interferes with the natural course of the disease. If it is marginally effective, we could use it provided it is not burdensome to the patient.[5]

TREATMENT ISSUES

Treatment requirements in the final stage of Alzheimer's Disease will vary to meet individual patient needs. The current literature can be of help to the physician who identifies comfort as his/her management goal and who assesses the effectiveness versus the burden of each treatment option.[2-9] Several medical issues in managing the terminal phase of Alzheimer's disease can be particularly difficult.

One issue is the transfer of final stage Alzheimer's patients from nursing homes to emergency departments, and their admission to acute care hospitals. Appropriate care for the patient with end-stage Alzheimer's disease can and should be provided in facilities for the elderly. In addition, the relocation of elderly patients during terminal illness, away from a familiar environment and familiar staff is contrary to good management.[8]

A second issue is the use of antibiotics for pneumonia and other infections. A group of experienced Boston physicians, representing a variety of specialties, have suggested that severely and irreversably demented patients need only be given care to make them comfortable. The physicians agree that it may be ethically appropriate not to treat intercurrent illness, except with measures required for comfort.[2]

They state that when a patient in the final stage of Alzheimer's rejects food and water, it is ethically permissable to refrain from artificial feeding by vein or gastric tube, while continuing spoon feeding for comfort.[2]

This raises the third issue of feeding and hydration by intravenous lines, nasogastric tubes and jejunostomies. Insofar as the physical comfort of the patient guides management, fluid depletion in dying patients should be regarded as a disorder with relatively benign

symptoms. Successful treatment of the discomfort of thirst and a dry mouth generally does not require rehydration. When the goal of management is to promote comfort or relieve suffering, treatment generally can be confined to simple measures in the form of mouth care.[9] If artificial hydration and nutrition are not given, the physician must be sensitive to the symbolic meaning of this step. Family, friends and staff need to understand that many patients in a terminal situation are not aware of thirst or hunger.[2]

THE PATIENT'S WISHES

Everything done for the patient should meet the test of not only whether it will make him more comfortable, but also whether it will honor his wishes.[2] Competent patients have the legal right to refuse treatment, even if the refusal would eventually lead to death. However, severely and irreversibly demented patients are not competent and the physician must do what he feels is best for such patients. Included among the patient's rights described in the *Canadian Law Reform Report* is the right which states that in the advent of a patient's incompetence, the law should not impose on the physician a legal duty to provide aggressive treatment under all circumstances.[10]

When the severely demented patient has previously made his wishes known through a living will, or through a proxy, the physician's treatment can be guided by the patient's wishes. In other cases, family members can indicate what the patient would have wanted.

Often, though, the physician is responsible for the philosophy of the management program, and he must be comfortable with his decisions. This requires clarification of any values that are at issue and definite personal soul-searching.[6]

QUALITY OF LIFE

Currently there is much discussion on quality of life. This term is subjective and dependent upon each person's values about the meaning of his own life. It is a presumptuous and risky business for a physician to assess the quality of life of another human being. Instead, the careful weighing of the effectiveness and burdens of each proposed treatment measure provides the physician with a more objective approach in decision-making. Only those treatment measures which would be beneficial to the patient (i.e. effective and not burdensome) are used, and inappropriate measures rejected.

CONCLUSION

In the care of late-stage Alzheimer's patients treatment is never stopped. Rather, it is aggressively redirected from inappropriate cure measures to appropriate supportive care.[4] The physician must carefully use technology in a palliative care approach which stresses comfort and the patient's wishes.

Physicians must examine their personal feelings about the care of patients in the final stage of Alzheimer's disease and come to terms with the ethical issues involved. "Now that death is no longer regarded as an opponent whose comings and goings remain strictly in the hands of other powers, the doctor must mix his responsibilities for the life of his patient with an intelligent and sympathetic concern for his death."[7] □

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The Medical References in "The Count of Monte Cristo"*

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The Count of Monte Cristo is more than a novel — it is an idealized personal fantasy of the author, Alexandre Dumas, père. To have limitless wealth and power, to be the instrument of destiny and controller of events, to have suffered and to conquer, to take revenge against enemies, to have wealth and power to repay friends — these represent a common dream fantasy. Because *The Count of Monte Cristo* captures this dream-ideal, the novel was at one time the most popular novel in the world.

ALEXANDRE DUMAS, PÈRE

Dumas was born in 1802, the grandson of a French nobleman and a black woman from the West Indies. His father was a gigantic mulatto who was a great heroic figure in the French Army. His mother was a black woman who raised him when the father died and left them penniless.

He had only the rudiments of an education from a village priest in Villers-Cotterets, near Soissons, before he moved to Paris to find work. Because of his beautiful handwriting he was employed as a clerk for the Duc d'Orléans, later King Louis Philippe. Dumas lived with a dressmaker, Katherine Lebay, eight years his senior, and she bore him a son. After seeing theatre he decided to write plays. These were an immediate success and he had an amazing career as a prolific writer of dramas, historical novels, romantic novels, adventure novels, newspaper stories, books on cooking and travel — over 600 books in all.

I noted he knew a medical student, Bixio, who acted as a second in a number of his duels and he often wrote on medical subjects for his newspaper. He visited the mental hospital in Aversia and gave a description of a very early attempt to use occupational therapy and theatre to treat mental illness. He also had many collaborators in his writing projects, but none seemed to have a medical background. From his early autobiography it seems clear the medical information came from his acquaintance with the young doctor Thibaud.

Dumas wrote "I had meanwhile made the acquaintance of a young doctor named Thibaud. Though he had no practice at this time he was not without ability. In 1823 and 1824 it was fashionable to suffer from chest complaints. Everybody was consumptive, especially poets. It was considered good form to spit blood after each emotional upset and die young, preferably before 30. Of course Adolphe and I, being tall and very thin, considered we were entitled to indulge ourselves too." Dumas and his friend decided as a lark to visit the young doctor and pretend to be consumptive. Thibaud recognized their game easily and they later became good friends.

"Thibaud knew everything of which I was ignorant and undertook to educate me. We spent nearly all our evenings in his tiny room. In the mornings I often accompanied him to the hospital, where I picked up a little knowledge of physiology and anatomy, although I have never been able to overcome my

Fig. 1. Alexandre Dumas wrote over 600 books of adventure, history, travel and cooking. His friendship with Dr. Thibaud gave him medical information for his writing.

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aversion to operations and corpses. From these visits I acquired a certain amount of medical and surgical knowledge which has come in handy in my writings."

Thibaud taught Dumas in his small room in the rue de Pelican which overlooked the passage Bero-Goddat. There they studied physics and chemistry and Dumas first learned about the poisons. A coquettish young milliner named Mme. Walker sometimes took part in these physiological studies.

He went on to give examples of how he used his medical information in his novel *Amaury* when he described the various stages of lung disease in his heroine so accurately that the publisher ceased publishing the serial because his daughter and son-in-law were passing through the stages of consumption at that time. The material was not resumed until they both died.

Thirty years later he wrote "I owe much to Thibaud for teaching me method in working as well as actual knowledge."

Dumas made medical references in some of his other novels, but not to the extent that he incorporated them in *The Count of Monte Cristo*. In his numerous novels there are occasional references to medical illnesses and accurate descriptions of trauma. In *The Black Tulip* he described carefully how an arm fractures and how it would be appropriately splinted and set.

In 1833 be contracted cholera. He quickly decided to treat himself with sugar dipped in ether. His mistress misunderstood and brought him a wine glass full of ether. When he awoke the cholera had disappeared and he felt that he had unintentionally discovered a great cure for cholera.

Following a career of amazing and dramatic variety, Dumas died famous but depressed by the fear that his writings were of little value. It has been suggested that he died of syphilis, contracted from Lola Montez, and was said to die with "paralysis of his brain and limbs."

THE COUNT OF MONTE CRISTO

Set in the Napoleonic era, *The Count of Monte Cristo* relates the story of Edmund Dantes, a man sentenced to life imprisonment on the famous fortress, the Château d'If, for a crime he did not commit. After his amazing escape from the fortress, the story tells of his complex plotting for terrible vengeance on the enemies who put him there. The novel was a *roman de moeurs*, a novel of manners, mixing reality and fantasy, particularly the fantasy of a man with extraordinary power, knowledge and talent, and with the wealth to reward friends and crush enemies.

We first meet the hero, Edmund Dantes, as he sails into Marseilles as the acting captain of the three-masted *Pharaon*. He took over command when the captain died of brain fever, a term indicating inflammation of the nervous system encompassing both encephalitis and meningitis. Brain fever was an accepted medical term in the 19th Century, defined by James Copland in *A Dictionary of Medical Practice* (1858) as "acute pain in the head, with intolerance of light and sound; watchfulness, delirium; flushed countenance, and redness of the conjunctiva, or a heavy suffused state of the eyes; quick pulse; frequently spasmodic twitchings or convulsions, passing into somnolency, coma, and complete relaxation of the limbs."*

When Captain Leclere fell ill with brain fever he recognized that he was about to die and gave Dantes a letter to deliver for him. Two hours later he became delirious and the next day he was dead. The resentful purser of the ship, Danglers, and the young man Fernand, who was competing with Dantes for the hand of a pretty young lady, Mercedes, concocted a plot to suggest that the letter was meant for conspirators against the King.

Fig. 2. Edmund Dantes returns as the powerful, rich Count of Monte Cristo. His knowledge, skills and actions are similar to an idealized view of physicians in France in the mid-19th Century.

* Other literary figures who had brain fever were Catherine Linton, in Emily Bronte's *Wuthering Heights*, Emma Bovary, in Gustave Flaubert's *Madame Bovary*, and Lucy Feverel, who died of brain fever in the *Ordeal of Richard Feverel*. Pip, in Charles Dicken's *Great Expectations*, had a similar disorder, although the term is not used. Two of Dostoyeveky's characters — Ivan, in the *Brothers Karamazov*, and Raskolnikov's mother, in *Crime and Punishment* — had brain fever. It was a commonly used diagnosis by authors to create a dramatic state with impending death, delirium, and fear, often with complete recovery.

Faria's Disease

Dantes was thrown in prison and suffered isolation, starvation and fear of the unknown. Initially he refused the repulsive, rotting food he was fed and began to experience the symptoms of starvation. He expected he would die, and began to feel numbness, a mild euphoria, vertigo, weakness, visual flashes, cloudy thinking, and eventually a loss of his hunger pains. He slowly learned to survive and struggled through eight years of imprisonment.

He eventually made contact with the other prisoner, Faria, and they began to plot an escape together. Faria knew the secret of a buried treasure and they had plans to escape and find the treasure to establish a new life.

While digging their way out Faria is suddenly attacked by a severe pain which he said could only be treated by a bottle of red liquid he kept in his cell. The illness he experiences resembles an aura prior to an epileptic seizure. He began to have shivering and said: "The attack is coming. I am going to have a cataleptic fit. I may not make a movement or utter a sound, but on the other hand I may foam at the mouth, stiffen convulsively and shriek. If I do, try not to let them hear me, because they'd put me into another cell and we would be separated forever. When you see me motionless, cold and apparently dead, then, and only then, pry apart my teeth with the knife and pour eight to ten drops of this liquid into my mouth. Perhaps it will revive me."

In the 19th Century opium was imported as solid opium, a brown bitter granular powder, but much of it was sold in tinctures, particularly as laudanum which was a reddish-brown liquid varying in colour according to its strength. Perhaps this was the red medicine used by Faria.

The attack then came suddenly and violently and he was unable to complete his next sentence. "His eyes dilated, his mouth became twisted, his cheeks turned purple and he writhed, foamed and shrieked." Dantes smothered his cries with a blanket as Faria had instructed him, and the attack lasted for two hours, "then he stiffened in one last convulsion and turned as cold, as pale and as inert as marble." Dantes then put the drops of red liquid into his mouth, prying the teeth apart with a knife. After an hour Faria began to awaken, looked anxious, but was unable to speak.

Later, Faria said that a previous attack had lasted for an hour, "after which I was hungry and got up unaided. This time I am unable to move either my right leg or my right arm. The third attack will either kill me or leave me completely paralyzed." It becomes apparent that in this latest attack he has sustained a right hemiparesis, without aphasia, and Faria recognizes that the deficit is permanent.

"I'll never be able to swim again. The arm is paralyzed, not just for the moment, but forever. Believe me, I have been reflecting on this illness ever since I had my first attack of it. I was expecting it, for it runs in my family. My father died from the third attack of it, and so did my grandfather. The physician who gave me this medicine, and he was none other than the famous Cabanis, predicted the same fate for me."

Dantes tried to reassure Faria, but the next day he found him "sitting calmly with a piece of paper in his left hand, the only hand which he could now use." The paper indicated the directions to the buried fortune. Later that day the old man tried to drag his paralyzed arm and leg through the passageway to tell Dantes the story of how the fortune came to be buried.

Later, Faria felt the cold shivering that indicated the beginning of his dreaded third attack. He said, "I'm growing cold, I feel the blood rushing to my brain, and that horrible trembling is beginning to shake my whole body". Dantes decided to prevent the problem rather than treat it after the attack and poured twelve drops down his throat, later giving him the rest of the medication. The attack was terrible. His limbs twisted and his eyelids became swollen. He had bloody foam from his mouth and became motionless. Despite the medication the old man shook violently in every limb, his eyes opened, he heaved a sign which sounded like a shriek, and his trembling body became rigid. His heart ceased, his face grew livid, and the light faded entirely from his open eyes. The old man was dead.

The jailers put the body into a canvas sack with a weight at each end. Dantes crawled through the passageway, slit open the sack, put the corpse in his cell and sewed himself into the sack. When the body was thrown into the sea he slit open the sack with a knife and swam to safety.

Edmund Dantes was now thirty-three years old, and he suffered through fourteen years in prison. His appearance had dramatically changed because of the effects of imprisonment and he was now more aged and thin, but hardened by the experience. He signed on a ship and was eventually able to make his way to the island of Monte Cristo where the treasure was buried. He later reappears in the south of France as the mysterious rich nobleman, the Count of Monte Cristo.

The Chronic Illness of Madame Caderousse

When he arrived back he visits an old friend, Gaspard Caderousse. Of greater interest to us is his pale sickly, thin wife, who is described as being constantly in the grip of fever which kept her in her bedroom on the second floor while her husband went about his daily tasks. She continually complained about her many problems and hysterical symptoms. At one point she "mumbled a few unintelligible words, let her head drop back to her knees and continued to tremble with fever." Caderousse had obviously learned to steel himself against the constant nattering of his sickly wife and would answer "be quiet! it's God's will".

Although bedridden much of the time she would sneak to the head of the stairs to listen to conversations occurring below.

Another emotional reaction is noted in the swooning of Mercedes, his former fiancé. In this erea, and up to the Victorian era, it was a socially acceptable response for women to faint and swoon at times of emotion. Dumas describes Mercedes as fainting whenever she passed the tavern where she had celebrated her betrothal to Dantes, prior to his imprisonment.

Death and Dying

Caderousse gave Dantes the disturbing news that his father had died during his imprisonment, of what the doctors called gastroenteritis, but Caderousse recognized that it was starvation from poverty and neglect. The old man had become more and more isolated and withdrawn. As he was dying the doctor misdiagnosed his appearance as gastroenteritis and prescribed a fast. The old man smiled when he heard that prescription. After nine days of despair and fasting he died.

Later, when talking to friends Dantes talks about death and dying. "Death is the only really serious preoccupation in life. Isn't it worthwhile, therefore, to study the different ways in which the soul can take leave of the body, and how, according to their character, their temperament and even the customs of their country, different individuals undergo this passage from being to nothingness? As for myself, I can tell you one thing: The more one sees others die, the easier it becomes to face death oneself. Thus, in my opinion, death is perhaps an ordeal, but it is not an expiation."

Later the Count comments "I have only three adversaries. The first two are distance and time, but with persistence I am able to overcome them. The third one is the most terrible one: the fact that I am mortal."

He then makes an oblique reference to his need for vengeance. He asks that if someone has killed your father, your mother or your sweetheart, "do you think society has given you sufficient reparation because the blade of the guillotine has passed between the murderer's trapezius and his occipital bone, because the man who made you undergo long years of mental and emotional suffering has undergone a few seconds of physical pain?" In this accurate use of anatomical terms, we again see Dumas's careful observation of medical matters.

Next the Count's friend, Caderousse, is attacked and stabbed. The Count administers three or four drops of his mysterious red medicine but refuses to give the man more even when he begs for it, indicating that it would kill him. He holds the flask under his nose and lets him inhale the odour. However, he dies of blood loss.

The Count's Sleeping Potion

At one point a friend notes that the Count of Monte Cristo has not eaten in twenty-four hours and that he can apparently sleep at will. When asked, the Count mentions that he has an infallible recipe for that. "I make no secret of it, it is a mixture of some excellent opium for which I made a special trip to Canton in order to get the purest quality, and of the best hashish grown in the Orient. You mix these two ingredients in equal proportions and make them into pills, which you swallow when you need them. The effect is produced within ten minutes." The Count indicates that he carries these pills with him at all times and he displayed a small emerald case containing five or six greenish little balls, about the size of a pea, which gave off an acrid, penetrating odour. Opium and hashish were both known to physicians and to literary circles in the 19th Century, and were felt to be stimulants to artistic inspiration. Dumas is known to have used hashish.

Travel as Medicine

The Count's young friend, Albert, complains of headache. The Count indicates that there is an infallible remedy which has been successful for him every time he met with some annoyance. Albert asks what his remedy might be. "Travel". We'll go where the air is pure, where all sounds are soothing, where no matter how proud one may be, one feels humble and finds oneself small — in short, we will go to the sea. I love the sea as one loves a mistress and I long for her when I haven't seen her for some time."

Antispasmodics for Syncope

The Count treats the son of Villefort, using one drop of liquid that brought him back from an episode of unconsciousness, but cautions that three drops would have forced blood to his lungs in such a way as to give him palpitations of the heart, and ten would have killed him. The boy had fainted during a runaway of their horse and carriage and the horses were soothed by "aromatic vinegar".

He comments that the medicine is not a poison "since, in medicine, the most violent poisons become health-giving remedies when they are used properly." He explains that the medicine is an excellent antispasmodic, which he occasionally used with all possible caution himself. Madame de Villefort indicates that she is extremely nervous and inclined to fainting, but because she doesn't have an excellent medication like his, she is obliged to go on using Monsieur Planche's antispasmodic." He offers her some of the medication cautioning "one drop of it restores life, as you've seen; five or six drops would kill infallibly. It is all the more dangerous because if those five or six drops were mixed in a glass of wine they wouldn't change the taste of it at all." With that buildup, one is not surprised when she later goes on a rampage of poisoning those around her.

The Stroke of Monsieur Noirtier de Villefort

"There are other things to fear besides death, old age and madness", says his friend Villefort. "There is, for example, apoplexy, that thunderbolt that strikes you without destroying you, but after which everything is finished. Come to my house some day, Count, and I will show you my father, Monsieur Noirtier de Villefort." He describes his father as a fiery Jacobin of the French Revolution. "Yet the rupture of a blood vessel in his brain changed all that, not in a day, not in an hour, but in a second." He comments that his father was now "a paralyzed old man, a mute, frozen corpse, awaiting its final decomposition." He felt that his father must have committed one of those wrongs which escape human justice, but no divine justice and that God had struck him down with a stroke because of this. The idea that illness was the reprimand of God was a common belief in the 19th Century.

Locked-in Syndrome

When we meet Monsieur Noirtier he is seated in a wheelchair where he is placed in the morning and lifted out at night. "Sight and hearing were the only senses, which, like two sparks still animated that physical body already so close to the grave. As often happens when one organ is used to the exclusion of the others, in his eyes were concentrated all the energy, strength and intelligence which had formerly been distributed throughout his body and mind. He commanded with his eyes, he thanked with his eyes; and it was almost frightening to see them flashing with anger or sparkling with joy in that otherwise stony face."

Three individuals were able to understand his method of communication. They began to learn the expression in his eyes, but he also communicated by fluttering his eyelids, expressing assent by closing his eyes, refusal by blinking them several times, and a desire for something by looking up at the ceiling. If he wanted to see his granddaughter Valentine, he would close his right eye only; if he wanted Barrois, his servant, he would close his left eye. They also received messages when he was upset or angry because of the change of colour in his face. Next, if she wanted to understand an answer to a question, she would go through the letters of the alphabet and he would blink yes when she came to the first letter. She would then repeat that letter with each vowel until he again indicated the correct one, and she would build up words in that manner. His family recognized that he was mentally alert in this state, and they were eventually able to convince a lawyer that he was also mentally capable of altering his will through this means of communication.

This condition has been well described only in recent years as the locked-in syndrome. Patients in this locked-in syndrome are maintained by good nutrition, often by nasal gastric feeding, and by good bladder care, usually by continuous catheterization. There is no indication of how he was fed, although he may have been able to reflexly swallow, and catheters were well known in that era.*

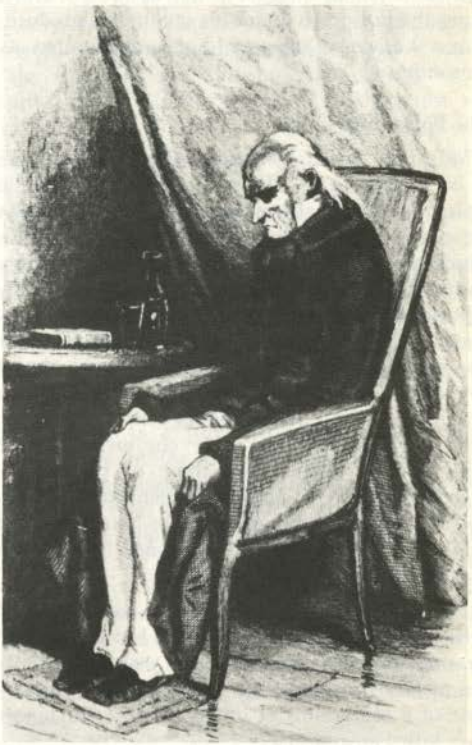

Fig. 3. M. Nortier, locked in by a severe stroke, was able to communicate only by eye blinking. The "Locked-in Syndrome" was not described in the medical literature until a century later.

Tolerance to Poisons

At one point the Count was mistaken for a physician because he cured the valet of a fever, and the hotel keeper of jaundice. He explains to Mademoiselle de Villefort that although he was not a doctor he had made a thorough study of chemistry and the natural sciences. He further states that when he was intending to live in the Orient he followed the example of King Mithdridates, who used to take a cup of poison each morning with cream at breakfast, to develop an increasing resistance to poisoning. The Count says that he took similar precautions to being poisoned in Naples, Palermo and Symra, and this saved his life in all three places. He indicates that this method would work with, for example, brucine, which comes from *Brucea ferruginea.* He explains "well, then, suppose the poison is brucine. The first day you take 1 mg of it, the second day 2 mg each day until on the twentieth day you are taking 30 mg, a dose which

* In fact, French catheters were regarded as some of the best available. Lafayette promised to get some French catheters for Thomas Jefferson when he was having urinary difficulties late in his life.

does you no harm but which would be extremely dangerous to someone who hasn't taken the same precautions." He explains that one can use the same type of method as a dagger as well as a suit of armour, using the poison to attack an organ and produce an illness known to doctors, but unrecognized as a poisoning.

The Poisonings

Monsieur de Saint-Meran died suddenly, thought to be a fatal stroke. His wife thought that she saw a phantom that night, representing his ghost, and the ghost moved the pitcher of orangeade by her bedside, which she had because of recent and persisting fever. She later developed nervous excitation and restlessness followed by coma and death. Although some thought that she died of sorrow over the death of her husband; Dr. d'Avrigny, who was called, thought that the circumstances were suspicious. "Sorrow may kill in certain rare cases, but it doesn't kill in a day, in an hour, in ten minutes!" He comments that the symptoms of tetanus and those of poisoning by vegetable matter are absolutely identical and he had observed them three-quarters of an hour prior to her death and felt that she was poisoned by large doses of either brucine or strychnine.

He hypothesizes that the poison may really have been meant for Monsieur Noirtier, but he had begun treating him with brucine three years before, using the method of increasing tolerance, increasing to a dose of 6 centigrams of brucine daily, which would have killed anyone else.

Next the old servant, Barrois, develops a fever. He began to tremble violently with twitching of his facial muscles and he cried out that he was in pain, that his eyes were going dim and his head felt as if it were on fire. His eyes took on a wild expression and his head fell backwards while the rest of his body stiffened. He turned around, staggered backwards, stumbled and fell at the feet of his master, Monsier Noirtier. Lying there his face twitching, his eyes bloodshot and his neck bent backwards he thrashed about with his hands, his legs so stiff that they looked as if they would break before they would bend. Flecks of foam began to appear on his lips as he panted painfully.

The attack ceased and he began to return to his senses. He indicated he didn't want anyone to touch him because he felt that if he were touched, even with the tip of a finger, the attack would come back. The attack had seized him like lightning, he said, and he had no prior illness or warning. He indicated that he did not eat anything that day but had a drink of Monsier Noirtier's lemonade, which tasted a little bitter to him. The doctor then rinsed his own mouth with some and recognized that it was poisoned. He had asked for someone to bring him water and ether, some oil of turpentine, and some tartar emetic. Barrois began to go into another attack and the doctor called for the emetic, but it was too late to administer anything. The doctor then asked for them to bring some syrup of violets, but Barrois gave out a loud cry "as though he had been struck by lightning" and died.

The doctor gravely indicated that there was a poison being used which kills without leaving a trace, but he felt that there was a way to determine the presence of that poison; to see if it turns red litmus paper blue and turns syrup of violets green. He poured a few drops of lemonade into a cup containing the syrup of violets and it took on a bluish colour, then changed to green. He stated "poor Barrois was poisoned with brucine". Monsier Noirtier survived because none but the doctor knew that he was treated with increasing doses of brucine which the doctor had been using to treat his paralysis.

The motive became clear. The two Saint-Merans were dead so the inheritance was doubled. Because Monsieur Noirtier was given the poison, it was suspected that Valentine, the granddaughter, was poisoning them to receive all of the inheritance.

Valentine later begins on her own to take increasing doses of the medicine the doctor gives to her grandfather. Her grandfather felt that this medicine was a panacea. She indicates that it is very bitter and gives a bitter taste to everything she drinks. Then one day she drank some sugared water and it had the same bitter taste. She then began to feel weak and unsteady and had the appearance of a bright light in her vision. Others noted that she was alternatively flushed and then pale. She began to leave the room but fell down the last few steps. Her grandfather recognized what was happening. She then made some convulsive movements while still speaking, burst into a strident laughter, her arms stiffened and twisted, her head fell back and she remained motionless. The doctor was called and he felt he might save her, and ordered a prescription from the pharmacist. Noirtier then communicates to the doctor that he has been giving her increasing doses of the medication to protect her. Villefort returns with the medicine and the doctor tests it by tasting a few drops put into the hollow of his hand, and treats her successfully. She survives because she already had some tolerance to the poison brucine.

Valentine survived but continued to be ill for many days. She had nightly fever and was often in a state of delirium. Figures in the room were a hazy apparition. She learned not to be afraid of the hazy images of people that would come into her room and would just take a small amount of the doctor's medicine to make them go away by sleeping. The Count of Monte Cristo appears at one point and tastes her medicine to make sure it is safe. She asks "are you a doctor?" and he answers, "yes, and the best one you can possibly have at this time, believe me". He offers her some of his medicine which she recognizes as the medicine that brings down her fever and calms her brain. The author comments that people later abandoned the house by the unexpected appearance

of the police "as though one of the guests had suddenly developed a cause of Asiatic cholera or the plague."

At another point Dumas makes an interesting reference to the young women who are planning to travel together, one disguised as a man. The young women took out masculine clothes from a trunk and dressed quickly "with a swiftness that indicated that this was probably not the first time that she had abused herself by dressing in the garments of the opposite sex."*

Madame de Villefort tries repeatedly to poison Valentine again and later switches from brucine to a simple narcotic, but the Count recognizes the alcohol in which it is dissolved. He then carries out a plot to make it seem that Valentine is dead. He gives her a pill containing a narcotic and she falls into a deep sleep. Those who later see her find that she has no respiration and no heart beat. Her arm was hanging over the edge of the bed and her wrist was already stiff and the fingernails blue. The doctor is called and recognizes that there is a different poison in the glass, which is not brucine. He went to a cabinet which had been transformed into a miniature pharmacy and took out a vial of nitric acid and poured a few drops into a glass. The liquid instantly changed into a blood red colour. "Aha!" he explained with the horror of a judge to whom the truth is revealed, mingled with the joy of the scientist who had solved the problem.

The grandfather, locked in by his stroke, realizes what has happened and "at this moment the old man's whole soul seemed to rush into his eyes; the veins of his neck swelled and his cheeks and temples turned purple, as though he had been strickened with an epileptic fit."

Madame de Villefort recognizes that she is caught and faints. During the later court scene and trial a number of revelations are made and the shocked individuals often respond by fainting. The public prosecutor faints when he finds that the thief and forger is his son. Another woman faints in the courtroom at the same time. Madame Danglers gives out a shrill cry, sobs and succumbs to "a violent fit of hysteria". Later Madame de Villefort commits suicide, poisoning her son as well. The Count tries unsuccessfully to revive the son. De Villefort becomes psychotic at the terrifying spectre of his dead wife and son.

Brucine

The poison brucine plays a central part in the story, The alkaloid brucine is found in nux vomica. The principle alkaloid in nux vomica is strychnine. Nux vomica is a term for the seeds of a tree native to India, *Strychnos nux vomica.* The term 'nux vomica' has been incorrectly translated as an emetic nut, and purveys the idea that it would induce vomiting. Actually, strychnine and brucine are not emetics, and the word vomica means depression or cavity, a feature of the strychnos seed, attributed by legend to the imprint of the Creator's finger. Nux vomica was introduced into Germany in the 16th Century as a poison for rats and other pests, and is still used as a rat poison today.

Strychnine was first used as a medicine in 1540 but did not gain wide usage until the 18th Century. The alkaloid brucine is a less potent alkaloid in nux vomica but is closely related chemically and pharmacologically and poisons in a similar fashion. Strychnine and brucine produce excitation of the nervous system, not from direct synaptic excitation, but by selectively blocking inhibition.

Strychnine is a powerful convulsant with a characteristic motor pattern. Because it reduced inhibition, the seizure involves antagonistic muscles, but the pattern is determined by the most powerful muscles. The convulsion is characterized by tonic extension of the body and of all limbs. Another characteristic of all drugs that block inhibition is that the phasic movements are symmetrical extensor thrusts. These can be initiated by a sensory stimulus, as indicated by the servant Barrois, usually when the dose is lower than that to produce a continuous convulsion.

All voluntary muscles, including those of the face are in full contraction. Repiration ceases because of contraction of the diaphragm and thoracic and abdominal muscles. Death results from hypoxia. In the early stages the patient is not only conscious but acutely perceptive to all stimuli. Muscle contractions are painful and the patient is extremely apprehensive and fearful of impending death as he awaits the next tetanic spasm.

Revenge and the Happy Ending

In the end the Count kidnaps Danglers with a number of henchmen who remove his fortune bit by bit as he starves in his imprisonment and tries to bid his fortune for food. His hair turns white during the ordeal. The Count then savors the last moments when he reveals that he is Edmund Dantes, the man that he sent to prison for life.

Maximilien, who believes Valentine to be dead, decides to commit suicide. "You know I am not given to histrionics," he says, "but I swear to you that my soul no longer belongs to me". Monte Cristo has told him that he would have assisted him earlier and goes to a cabinet and comes back with a small quantity of a substance on a spoon for him to take. In a melodramatic scene the young man takes the medicine that makes him confused and disoriented and then

* Dumas himself was known to dress in unusual attire, of questionable gender. A female visitor once barged in on the author to find his obese body adorned only with crimson tights, while three naked girls surrounded his chair. He also toured Italy in the company of Emilie Cordier, whom he called "the admiral". By day he kept her dressed as a sailor boy, but fooling no one. It was a particularly poor disguise when she became pregnant.

the Count opens the door and Valentine arrives. The narcotic had its effect over a few hours and then he revived to find his love at his side.

CONCLUSION

The Count of Monte Cristo, once the most popular novel in print, has numerous references to medical diseases, pharmacology and medical therapy. In this paper I have outlined some of the medical references used by Dumas, and indicated that his knowledge of medicine, toxicology and anatomy came from his early friendship with the physician Dr. Thibaud, who gave him private lessons when both were just beginning their careers and had time to spare. Throughout his 600 novels the influence of the young physician can be noted, but nowhere clearer than in *The Count of Monte Cristo.* □

TABLE I

Medical References of The Count of Monte Cristo

Encephalitis	Hysteria
Starvation	Gastroenteritis
Grand mal epilepsy	Attitudes towards dying
Familial hemiplegic epilepsy	Decapitation
Curare poisoning	Opium and hashish benefits
Mushroom poisoning	Heart palpitations
Opium treatment	Stroke from cerebral hemorrhage
Fever treatment	Locked-in syndrome
Jaundice	Brucine poisoning
Emotional syncope	Drug tolerance
Antispasmodic medication	Emetic drugs
Asiatic cholera	Pill making
Chemical tests for drugs	Plague

References may be obtained from the author.

Boxing

F.W. Woodruff,* M.B., B.S.

One of the first experiences a boy has to deal with in relation to his peers is to establish acceptance by the group.

This entails taking part in various activities based on curiosity and a desire to sample life's experiences. Inevitably to some degree this also leads to finding one's level in the pecking order of the group. By the time the boy is eight or nine years old, I think, he knows how well he performs in relation to the others in ball games, tree climing, behaving appropriately in the face of authority (parent, school or "other") and to some extent, how important these are to him. He may well realise that he feels that non-competitive activites are more important to him or just that he can cope better with these than most people and thereby focus his priorities.

Many of us consider it very important to expose our children to potentially life-long activities such as cross-crountry skiing, running, camping-associated skills and so on — but there is still a high proportion of children who will, for various reasons (lack of opportunity, parental direction or personal inclination) continue to develop the competitive activities and remain personally directed to what may by some be regarded as aggressive, combative recreational sports.

Inevitably in some of these young people there will be physical aggression and as long as boys grow to be men, fighting will be significantly important to some. I think this is irrefutable.

In some cities and in deprived areas, with very high unemployment, it is very easy for frustration and anger to be channelled inappropriately.

Amateur boxing if introduced into this type of environment and if run by well-motivated personnel will often develop a more responsible youth who will accept that he has special training and must not take advantage of this by aggression out in the street. This is a personal view but has been described by many ex-professional boxers such as Floyd Patterson (ex-world champion) who have moved into a social-worker type of activity once their boxing days are over.

The object in amateur boxing is *not* to knock out the opponent, though some of the commentators on the frequently televised boxing shows seem to give that impression — and indeed some boxers, especially those blessed (or cursed?) with an exceptionally hard punch, do develop along these lines.

The object is to establish supremacy in the Art of self-defence, using only the knuckle part of the glove to deliver blows within the target area which does not include the back of the neck nor the kidneys. In the vast majority of amateur bouts, a points decision is given and no one is hurt.

It is indeed tragic to see signs of "punch-drunkenness" with Parkinsonism and Alzheimer's Syndrome and everything possible should be done to prevent this — and even with well-controlled matches and early stoppages, some injuries will occur. However, if boxing is an expression of a basic instinct, let us control it properly and support it as a sport.

A well run Amateur Boxing Club is disciplined and its activities are well supervised by approved officials; closer involvement with medical advice at the ringside is always welcomed; for the participant who is well trained and well schooled there can be few activities which are as exhilarating and as demanding of self-discipline.

I think amateur boxing has a part to play in the life of a proportion of your youth; closer involvement by young physicians can only help to make it safer and less objectionable to those who, with justification, take the opposite view. □

EDITOR'S NOTE: The above is a response to an article on the subject of Boxing. The first article was published in the December 1983 issue of the Bulletin under the heading "One Physician's View" Boxing, written by Dr. T.J. Murray. Dr. Murray was asked to comment, his response follows.

Response from the Author*

I must disagree with Dr. Woodruff's arguments.

I disagree that a "high proportion of children" continue as adults to remain personally directed to aggressive, combative, recreational sports. Amateur and professional boxing involves a very small group of individuals, and as a brutal and potentially dangerous "sport" of one person bashing another, it should not be encouraged, particularly by physicians. Because some adult males continue to show aggressive behaviour does not logically lead to the conclusion that we should condone, encourage, and help organize such primitive behaviour.

If boxing develops the positive attributes in our youth suggested by Dr. Woodruff, I wonder why he limits it to the poor, the unemployed, the uneducated and those from deprived homes. Surely this argument

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should apply to everyone. In addition, why does he limit it to males? Surely, if there are beneficial personal qualities developed by boxing, these would accrue to our daughters if they were encouraged to get in the ring and punch and batter, and accept the bloody noses, swollen eyes and traumatized brains.

The "sport" of boxing does not teach control of aggression. It institutionalizes and acts on the principle of winning through beating up another person. Boxing is not self-defense, as Dr. Woodruff suggests — it is personal aggression with some rules.

Dr. Woodruff points out that they do not allow punches to the back of the neck or kidneys. I would prefer that they would exclude a much more important organ, the brain. There is no justification for repeatedly traumatizing the brain of a person, as part of the object of a "sport". What kind of thinking protects the kidney over the brain?

Finally, I object to having physicians participate in the supervision of boxing, as they lend a lie to many of the principles of medicine, and to the suggestion that their presence prevents the serious and long term injuries that result from boxing. At best, the physician can call a fight after a patient has been beaten, and provides solace after he has watched the combatant bruise, bleed and lie unconscious.

Boxing is often discussed as an inevitable part of our social fabric. But we no longer throw people to the lions as public entertainment, and we don't even permit dog fights or cock fights. Surely our civilization is now progressed to the point where we can discontinue a public spectacle of one person attempting to beat up another.

I think that our children would benefit if we fostered and encouraged the other expressions of self-discipline outlined by Dr. Woodruff. □

You can't take it with you.
Life is a terminal illness.

(Maureen Forrester (1931 -)

Invasive cancer of the cervix lends itself to being prevented by means of cytology screening programs. Cytology registries have been established to record pap smear data; to ensure follow-up for all patients having an abnormal pap smear; to provide epidemiological statistics; and to create a system for its quality assurance. Individually and collectively, if physicians promote and provide pap smear screening with accurate documentation, the Cytology Registry will serve the medical community of Nova Scotia as a most meaningful resource. A successful Cytology Registry in Nova Scotia will serve to stimulate and be an example to other provincial medical societies.

***Recommendations concerning the frequency of gynaecologic Pap Smears:**

Initial smear age 18-20,
or at onset of first intercourse.

Screen annually to age 35.

With *all* previous paps normal,
at age 35, screen q 3 years.

With *all* previous paps normal,
at age 60, screening may cease.

At any age, if the frequency of
previous pap smear tests is
non-existant or sporatic, advise
patient of need for regular screening.

Vaginal vault smears are
recommended post hysterectomy.

These *recommendations apply *only* when a Registry has been established and has an appropriate Quality Control component (such as is currently being developed in Nova Scotia). *Until then,* ALL women — 18 years of age and older, or any age if sexually active, should have annual cytologic examinations. □

ACKNOWLEDGEMENT

Special Acknowledgement to the Members of the Cytology Subcommittee of the Nova Scotia Medical Society Cancer Committee. Subcommittee members represent the Government of Nova Scotia Department of Health, the Departments of Management Engineering and Gynaecology, and Section of Cytology, Victoria General Hospital, Halifax, N.S.

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INVESTIGATIONAL THERAPIES FOR CANCER PATIENTS OFFERED BY THE CANCER TREATMENT AND RESEARCH FOUNDATION OF NOVA SCOTIA

The Cancer Treatment and Research Foundation of Nova Scotia (CTRF OF NS) has been established with a mandate to provide clinical care as well as to conduct research in the Province of Nova Scotia. With respect to the latter, the Clinical Trials Unit has been established to facilitate clinical trials research within the Foundation under the auspices of several cooperative clinical oncology groups. The following is a description of current areas in which we are now or will shortly be active:

Breast Cancer
- — Adjuvant therapy for postmenopausal females
- — Hormonal or chemotheraphy for pre- and postmenopausal females

Lung Cancer
- — Small cell lung cancer
- — Non-small cell lung cancer
- — Mesothelioma

GI Malignancies
- — Advanced gastric carcinoma
- — Advanced colorectal carcinoma
- — Adjuvant study for colorectal carcinoma (activation Spring 1987)

Sarcomas
- — Advanced soft tissue sarcomas
- — Adjuvant therapy for osteogenic sarcoma

Lymphomas
- — Advanced or recurrent Hodgkin's disease
- — Relapsed or refractory non-Hodgkin's lymphoma of unfavourable histology

Genito-Urinary
- — Advanced incurable bladder carcinoma
- — Advanced renal cell carcinoma (activation Spring 1987)

Head and Neck
- — Locally advanced newly diagnosed carcinoma of the head and neck (pending)

If there are any questions about any of these studies enquiries should be directed to Mrs. G. Hirsch, Clinical Trials Nurse at 428-4223. □

Current Topics in Community Health

Prepared by: Dr. Frank M.M. White
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POST POLIO SYNDROME: U.S. Report

Post-Polio Syndrome, also called Post-Polio Sequelae and Post-Polio Muscular Atrophy (PPMA), refers to a group of disorders experienced by many poliomyelitis survivors, typically starting 25-35 years after recovery. Symptoms include renewed, *usually gradual* progression of muscle weakness, increased fatiguability, joint pain, muscle cramps, intolerance to cold, and sometimes increased difficulty breathing (when repiratory muscles are involved). Post-Polio Syndrome appears to be more frequent and severe in persons who had a more severe poliomyelitis illness initially.

No single examination procedure or laboratory test can definitely diagnose this condition. Pathophysiologically, there appear to be at least two subgroups of problems:

1. Deformities of joints, limbs or trunk (resulting from long-standing muscle weakness) that produce nerve entrapment, pain, degenerative arthritis, or mechanical difficulties.
2. Increasing muscle weakness of unclear cause but which may represent progressive loss of axons from motor neurons that survived the initial poliomyelitis episode and had sprouted extra axons to innervate muscles whose original motor neurons were killed.

The incidence of this disorder appears to have increased markedly in the past decade, as the cohorts of patients who survived polio during the peak incidence years of the late 1940s and early 1950s have come to the end of the 25-35 years "incubation period" of Post-Polio Syndrome. Many of the patients are senior citizens, but many others are in the 4th to 6th decades of life. Estimates indicate that there are currently 200,000-300,000 polio survivors in the U.S. and that 25 percent of them are now suffering from the Post-Polio Syndrome.

The reappearance of symptoms and deterioration of functional capacity after years of apparent stability present the patients with a variety of medical, financial, occupational and psychosocial problems. For this reason, several regional support groups have been formed. A major difficulty for patients has been lack of medical community understanding of Post-Polio Syndrome, leading to:

a. Misdiagnosis, with patients sometimes being told that they are simply growing old, depressed, and/or hypochondriacal.
b. Mistreatment: e.g., offering no treatment at all, prescribing too vigorous an exercise program which can lead to increased weakness and/or further joint/tendon/ligament damage, failing to employ support devices (e.g., braces, wheelchairs) when indicated, failing to immediately institute mild passive exercises of involved muscles when a patient is put to bed in a hospital, failing to reduce dosage of curare-like drugs used in surgery, etc.

While researchers and clinical workers do not fully agree on therapies for this disorder, most recommend the use of individually tailored, limited exercise programs, support devices where appropriate, and stretching out or surgical release of contractures that may have developed.

An occasional problem has been the inappropriate denial of influenza vaccine to these patients, based on the misunderstanding that a variety of chronic neurologic conditions, including Post-Polio Syndrome, are contraindications to influenza immunization. On the contrary, persons with these conditions, particularly with Post-Polio Syndrome involving intercostal muscle weakness that affects repiratory function, are especially in need of the protection provided by influenza vaccine. Such patients are at no known increased risk of influenza vaccine side effects. Both the medical and public health communities need to become informed about Post-Polio Syndrome.

Source: Adapted from *California Morbidity, March 27, 1987.*

VACCINE FAILURE: Haemophilus Influenzae Type B Polysaccharide Vaccine

Case I

In November 1986, M.F., a 28 month old adopted mulatto male from Dartmouth, Nova Scotia was given *Haemophilus influenzae* type b (Hib) vaccine by his family doctor. He was well until forty days later when he was noted by his mother to be lethargic. He subsequently developed a sore throat, low grade fever, drooling, increasing respiratory difficulty with stridor, and a muffled voice with a hoarse cough. He had no complaints of headache or neck stiffness but did have three episodes of vomiting before his initial assessment.

The patient had no history of frequent infections, airway problems or foreign body aspiration. He was on no medications, had no allergies or previous significant illnesses. He attended a day care centre two days per week, the last day being four days prior to onset of symptoms.

On admission to hospital, he was not swallowing his secretions, and he appeared ill with respiratory distress and stridor. His respiratory rate was 45 and temperature was 38.5. His chest was clear with good air entry bilaterally. The remainder of his physical examination was normal. His epiglottis was visualized under general anaesthesia and was grossly inflamed. Blood and epiglottic swabs grew *H. influenzae* type b. His white blood cell count was 30.2 x 10^9/L with 65% neutrophils, 19% bands, and 5% lymphocytes. His hemoglobin was 111g/L and the platelet count was 310 x 10^9/L.

Following intubation, cefuroxime (100mg/kg) was started. He was successfully extubated 48 hours later. He received Rifampin® as did his five month old sibling, his parents and his day care contacts. He was discharged four days after admission in good condition to complete his ten day course of antibioltic.

In view of the failure of the vaccination to prevent this episode of *H. influenzae* infection, a immunological assessment was done. His serum immunoglobulins were normal with the exception of IgA which was minimally elevated (IgA 0.84g/L; Normal 0.16-0.75). Hemoglobin electrophoresis, tetanus antibodies, *H. influenzae* serology, and allotyping are pending.

Case II

A 29 month old Caucasian male from St. John's, Newfoundland received *Haemophilus Influenza* type b vaccine on November 7, 1986. Prior to this immunization, he had no history of undue susceptibility to infection. Following immunization, he remained well, except for an uncomplicated nonfebrile respiratory illness in the subsequent month. On January 8, 1987, he developed fever, irritability and cough. He was examined in the hospital emergency room, where a chest Xray revealed a left lower lobe pulmonary infiltrate. White blood cell count showed a leucocytosis. A blood culture taken at that time grew a beta lactamase positive *H. Influenzae* type b. The child made a rapid uneventful recovery following institution of antibiotic therapy. Serum immunoglobulins G, A, M and IgG2 and C3, C4 were in the normal range. Further immunologic studies are underway.

Discussion

H. influenzae type b systemic disease after Hib immunization was first noted in the Finnish field trial (1974-1976) involving 49,000 children.[1] Vaccine failures occurred after immunization in only two children vaccinated at 24 and 27 months of age. In the United States, Hib vaccine was licensed in April 1985. Recently, Granoff in St. Louis identified 55 vaccine failures of whom 4 (7%) were black.[2]

Genetic factors which might account for poor immune response to the vaccine have been identified.[3] Among blacks, those lacking the Km allotype have a higher incidence of disease and a poorer immune response to Hib polysaccharide. In Caucasians, the presence of the Gm phenotype lacking G2m.23 was associated with a significantly higher relative risk for vaccine failure.

To our knowledge, the cases presented here are the first Hib vaccine failures reported in Canada. Considering the small number of vaccine failures in blacks noted in Granoff's paper, it is surprising that one of the first reported Hib vaccine failures in Canada occurred in a black child.

The use of the Hib vaccine became routine throughout Nova Scotia after December 1, 1986 when the provincial Department of Health began to provide Hib to physicians for two year old children. Currently in the province of Nova Scotia, approximately 10,000 units of vaccine have been distributed but it is not possible to determine easily the number of doses administered.

Vaccine efficacy has been reported to be approximately 80 to 90%.[1] However, prospective studies have not as yet evaluated efficacy of vaccines used in Canada.

It is imperative that physicians are aware that Hib vaccine, like other immunizing agents, is not 100% effective and that *H. influenzae* invasive disease cannot be prevented with certainty. One should continue to monitor both the incidence of *H.influenzae* type b invasive disease and vaccine failures and report them immediately. Vaccine manufacturer and lot number should be recorded at the time of immunization in order to aid in subsequent investigation if vaccine failure occurs. With this information, reasons for failures can be identified and improvements in efficacy can be made.

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Source: Dr. C. Nijssen-Jordan in collaboration with Dr. R. Bortolussi, Dr. R. Ozere, and Dr. W. Sprague. Izaak Walton Killam Hospital for Children, Halifax, Nova Scotia and The Dr. Charles A. Janeway Child Health Centre, St. John's Newfoundland. Also published in *Can Dis Wkly Rep* 1987; **13-7:** 75-76. □

Personal Interest Notes

A grant of $62,500.00 from The Cancer Research Society Inc., Montreal was presented by Governor General Jeanne Sauvé to **Dr. D. Howard Dickson,** Associate Dean of Medicine, research, Dalhousie University in a recent ceremony. The money will go directly to support an expanding cancer research thrust.

Three Nova Scotia doctors have received the Stephen Fonyo Training Fellowship for Family Physicians designed to share new knowledge for better treatment of their cancer patients. The recipients are **Dr. Paul F. McIntyre** and **Dr. Neils H. Hanson** of Halifax, and **Dr. Daniel S. Reid** of Pictou. The awards have been made possible by money raised during Stephen Fonyo's cross-Canada run and by a grant from the Government of Canada. They are administered by the Canadian Cancer Society.

Dr. Michael W. Gray, Professor of Biochemistry in Dalhousie Medical School, is to add another honor to the list in his curriculum vitae. Recently, he was awarded the Boehringer Mannheim Canada Prize by the Canadian Biochemical Society, which he will receive at the annual meeting of the Canadian Federation of Biological Societies in Winnipeg in June. This prize is awarded every other year in recognition of outstanding achievement in research in biochemistry or molecular biology in Canada.

Dr. Michael Gross is the new director of the Victoria General Hospital's Bone Bank. Dr. Gross, a native of England, attended school in York and graduated from Newcastle-upon-Tyne University Medical School in 1975. In addition to his duties as director of the VG's Bone Bank, Dr. Gross is Assistant Professor, Department of Surgery, Dalhousie, and a consultant in musculoskeletal oncology for the Cancer Treatment and Research Foundation of Nova Scotia.

The founder and first director of the VG's Bone Bank, **Dr. Pat McDermott,** died last December. A memorial plaque in his honour was unveiled earlier this year in a ceremony in which Dr. R. Yabsley and Dr. P. Belitsky both paid tribute to Dr. McDermott as a friend and colleague of unusual stature.

The Dalhousie Medical Research Foundation has awarded a fellowship to a physician-researcher exploring the use of computers and telecommunications in health services. **Dr. Truls Østbye,** 32 of Bergen, Norway graduated from medical school in his native city in 1979 and was awarded a Master's degree in Public Health from Harvard University in 1983. He is affiliated with the Department of Community Health and Epidemiology and will co-operate with members in the Departments of Family Medicine, Nursing, Continuing Medical Education, Physiology and Biophysics, and the Committee on Undergraduate Medical Education. The fellowship awarded to Dr. Ostbye is the eighteenth made by the Dalhousie Medical Research Foundation. □

OBITUARIES

Dr. Marie Jean Whittier, (89) of Windsor, N.S. died on April 27, 1987. Born in Upper Rawdon she graduated from Dalhousie Medical School in 1929. For three years she was a medical supervisor and teacher at the Maritime Girls School in Truro. Then in 1934 she was appointed medical missionary to India and in 1979 she was given by Dalhousie University an honoary Doctor of Laws for her outstanding and courageous work in India. She is survived by her three nieces. The *Bulletin* extends sincere sympathy to her family.

Dr. Donald R. Webster, (85) of Pictou County, N.S. died on May 25, 1987. Born in Pictou, he graduated from Dalhousie University and McGill University. He served with the Royal Canadian Navy as Surgeon Captain during the Second World War. He was a member of The Canadian Medical Association and the Royal College of Surgeons. He is survived by his wife, a daughter, and a son. The *Bulletin* extends sincere sympathy to his wife and family.

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