THE NOVA SCOTIA MEDICAL JOURNAL

GUEST EDITORIAL

The Halifax Infirmary Hospital

ONE HUNDRED YEARS LATER

Carlos Del Campo,* MD, FRCS(C),

The Halifax Infirmary Hospital was founded by the Sisters of Charity in 1886. It was intended as a home for elderly Catholic ladies and its original name was "Our Lady of All Souls".

One hundred years later the Halifax Infirmary (HI) has evolved into one of the major referal centres in the Maritimes. The HI provides community, regional and referal health services to individuals over fourteen years of age and education for under and postgraduate medical and paramedical personnel.

The HI distinguishes itself in specific programs, namely: diagnostic imaging; endocrine diseases; gastroenterology; Nova Scotia Eye Centre; Urinary Stone Clinic; Ears, Nose and Throat; Non-Invasive Vascular Studies; and Arthroscopic Surgery. Two busy Intensive Care Units support medical and surgical management of our sickest patient population. A newly developed Palliative Care Service adds significantly in the management and support of our terminally ill patients. The Halifax Infirmary Auxilliary provides invaluable support and financing for some of these programs. A "Foundation" is being integrated for further support.

This current issue of *The Nova Scotia Medical Journal* is devoted to a representative overview of the work currently performed in our institution. It would be impossible to find enough space to publish articles related to all our activities in our different fields. Those published here have been selected not to represent specific departments but specific activities. Clinical evaluation, clinical research, results of therapy, complex surgical and investigational techniques and research are published here in a modest attempt to show who we are and what we do and to mark the end of one hundred successful years and the beginning of the next one hundred.

□

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Editors Note:

The majority of the articles published in this issue were written by the Medical Staff of the Halifax Infirmary.

NAME CHANGE

In the recently held Annual Meeting of The Nova Scotia Medical Society a resolution was passed changing the name of ***The Nova Scotia Medical Bulletin*** to ***The Nova Scotia Medical Journal*** as this will more adequately describe our present medical publication. It is intended to blend from old to new, following the fine tradition established by this publication over the years.

This change from *Bulletin* to *Journal* allows us to avoid connotations suggesting a newsletter rather than a scientific journal. It is hoped that both lay people and media outlets will easily recognize the title of *The Nova Scotia Medical Journal* which serves as our provincial professional publication.

Evacuation Proctography in Anorectal Disorders:
CASE REPORTS AND REVIEW OF THE LITERATURE

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Evacuation proctography is a new radiographic technique at the Halifax Infirmary used to study anorectal disorders, and five illustrative cases are presented in which it was used. A brief review of this technique and its use in studying abnormalities of anorectal function is included. This X-ray technique often provides more information than a barium enema because it evaluates the "function" of the anorectum. It is a useful adjunct to the clinical examination and barium enema examination of selected patients with constipation, solitary rectal ulcer syndrome, colitis cystica profunda and other disorders of anorectal function.

"Evacuation proctography" or "defecography" are terms applied to an X-ray technique to identify abnormalities in rectal emptying. The initial studies were often performed in the left lateral or prone positions with cine-radiography. Recent approaches have been to perform this technique in the sitting position to provide a more "physiologic" examination and the use of fluoroscopy with videocassette recording of the fluoroscopic image to reduce the radiation exposure of the examination.[1,2]

In this article, a description of the technique used, along with a description of individual case reports with illustrative examples, will be discussed along with the usefulness of this type of study in evaluating patients with anorectal disorders.

MATERIALS AND METHODS

The evacuation proctogram requires no bowel preparation, thus permitting a more physiologic examination of rectal emptying. A specially constructed radiolucent commode chair (Figure 1) is used. To compensate for differences in tissue density, a wedge-shaped copper and aluminum X-ray filter is attached to the X-ray tube.

With the patient in the sitting position on the commode chair, 100-200 ml of Microtrast esophageal barium sulfate paste 70% W/W is introduced per rectum. The patient experiences a mild sensation of rectal fullness (larger volumes reflux into the rectosigmoid and contribute nothing to the examination). Defecation is recorded with fluoroscopy and videocassette recording. Rectal emptying time is recorded on the videocassette recorder. "Hard-copy" laterals are obtained immediately pre- and post-evacuation. Further hard-copy is acquired from the video recording using a video-imager. The entire examination is usually completed in 15 minutes, and Figure 2 illustrates a normal examination.

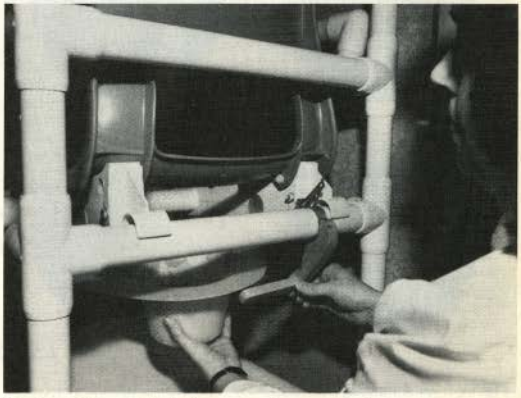

Fig. 1. View of chair demonstrating commode.

CASE REPORTS

Case I

A 64 year old female presented with over a year of rectal bleeding, initially diagnosed as hemorrhoids. She gave a long history of constipation and laxative abuse, but she had never seen rectal bleeding prior to the previous year. She also experienced, at all times, a "pressure" sensation localized to the rectum without abdominal pain, diarrhea or other GI complaints. Her past history revealed bilateral radical mastectomies in 1977 and 1979 for malignancy, followed by radiotherapy to the chest and axilla; recurrent kidney stones, requiring operations in 1976, 1979 and 1984, along with a cholecystectomy in 1981. She had a history of hyperthyroidism treated with surgery and thyroid replacement therapy. She also had chronic arthritis for which she took Motrin®.

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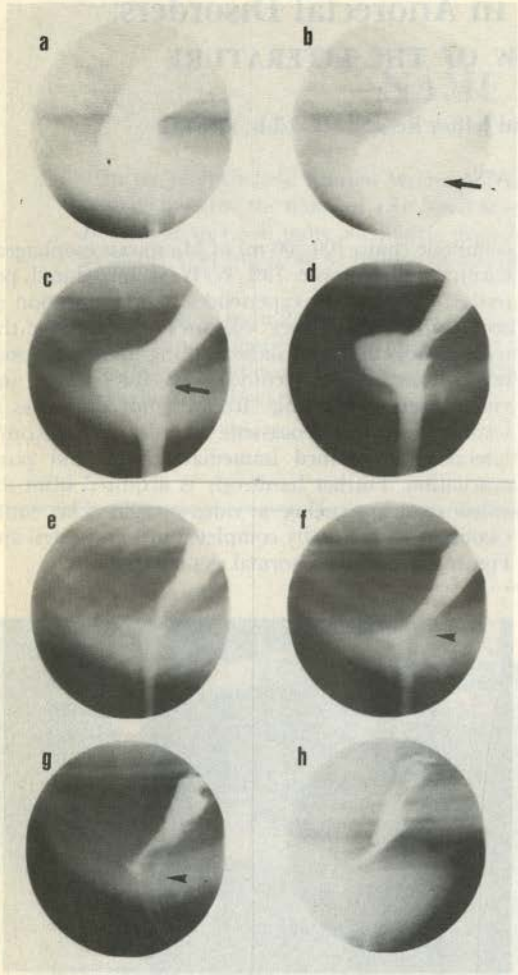

Fig. 2. Normal examination.

Note relaxation of puborectalis muscle with straightening and opening of anal canal — (b and c) (⟵). Passage of solid stool (f and g) (◄). Normal amount of retained barium (h).

General physical examination was unremarkable except for marked post-mastectomy lymphedema in the right arm. The abdomen was diffusely tender, particularly in the area of the cecum and sigmoid jcolon. A colonoscopy revealed friable mucosa limited to the distal 10 cm of the rectum. Rectal biopsies were consistent with a diagnosis of a solitary rectal ulcer syndrome.

Proctography results are illustrated in Figure 3.

Case II

A 30 year old male with a history of repeated episodes of rectal bleeding and episodic diarrhea for four years. There was a previous diagnosis of colitis cystica profunda made on rectal biopsy. He was accustomed to straining at stool for 4 to 6 hours a day. This was accompanied by passage of bloody stool 6 to 7 times daily. There was no nocturnal stool. The patient had been treated previously with Sulfasalazine®, steroid enemas, Metronidazole®, and 5-aminosalicylic acid enemas. Instruction in a high fibre diet and avoidance of straining had produced no clinical improvement.

The only physical finding was tenderness to palpation over the sigmoid colon. Sigmoidoscopy confirmed the presence of colitis cystic profunda with a suggestion of some anterior rectal mucosal intussusception but no external prolapse.

Proctography results are illustrated in Figure 4.

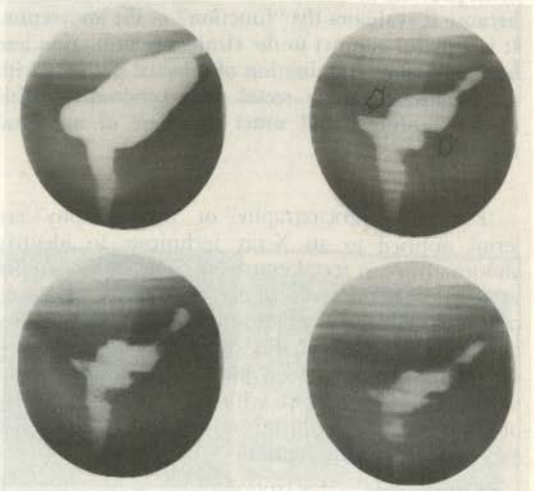

Fig. 3. Case I — Illustrates mucosal intussusception (⌂).

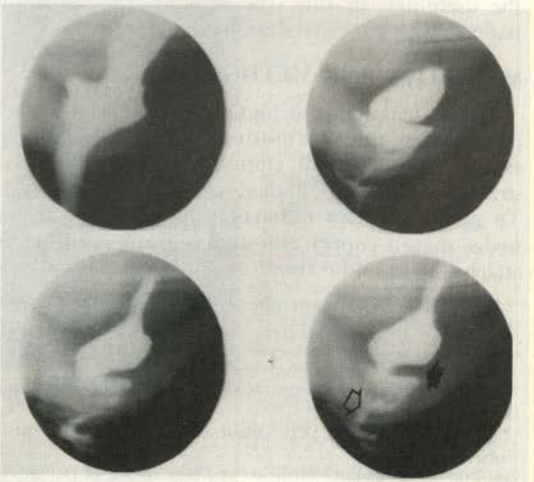

Fig. 4. Case II. Illustrates mucosal intussusception (▲) which progresses to full thickness rectal prolapse (⌂) with associated incomplete emptying.

Case III

A 34 year old female with a longstanding history of constipation and abdominal pain of 13 years duration, following a hysterectomy. She related episodes of constipation prior to this only during her pregnancies. Lumbar fusion had been performed 10 years previously and, in 1985, she had an exploratory laparotomy because of increasing abdominal pain. No abnormality was found but she became completely obstipated and unable to move her bowels with high dose laxatives and enemas, often requiring rectal disimpaction. She now has intractable abdominal pain, bloating and solid food intolerance, particularly high fibre food. Attempts at excessive straining to move her bowels were unsuccessful. The patient has lost about 80 pounds in weight since her surgery.

On physical examination, the patient was emaciated with pain and nausea on palpation over the left upper rectus abdominus muscle. Perianal examination, including sigmoidoscopic examination, was unremarkable.

Proctography results are illustrated in Figure 5.

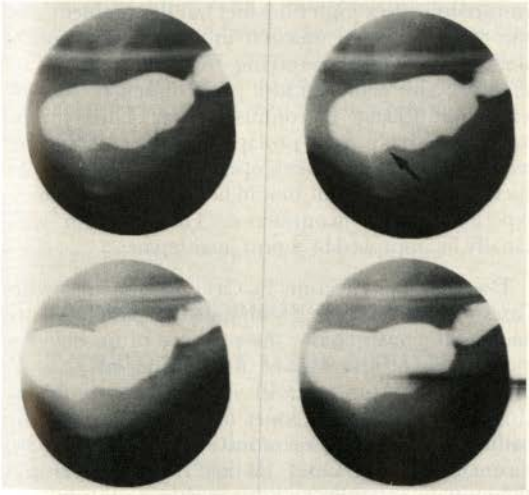

Fig. 5. Case III, Despite repeated straining, the rectum fails to empty. Note absence of relaxation of puborectalis (←). Over one minute of fluoroscopy.

Case IV

A 52 year old female was admitted with a history of fecal incontinence for seven months but, in addition, had constipation for over 30 years. She had had multiple operations, including an appendectomy in 1940, two succesful pregnancies in 1958 and 1959, and a hysterectomy for endometriosis in 1965. In addition, she had right kidney surgery in 1965, a colonic operation in Montreal with removal of adhesions in 1972 and following this, in 1973, removal of approximately 1.5 feet of transverse colon and a temporary colostomy tube. In 1984, she had had a bleeding cerebral aneurysm of the right carotid artery and bilateral carotid artery stenosis was diagnosed. This necessitated a craniotomy to clip the aneurysm but a right cerebro-vascular accident occured post-operatively. In 1985, a right endarterectomy was performed with evidence of another right hemisphere stroke.

On examination, there was mild weakness and reduced sensory awareness of the left side of her body. She was able to walk normally. Perianal examination was unremarkable.

Proctography is shown in Figure 6.

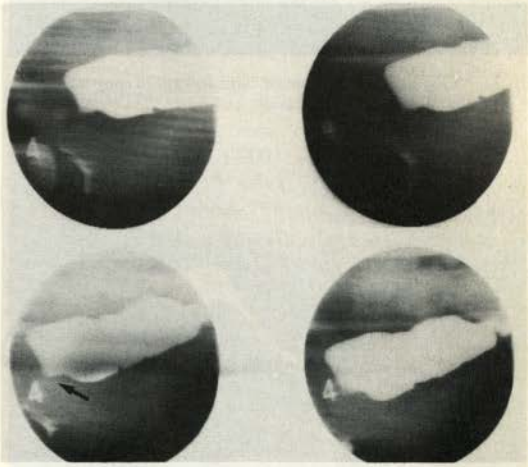

Fig. 6. Case IV, Failure of relaxation of puborectalis (←) with no rectal emptying. Over one minute of fluoroscopy.

Case V

A 34 year old female who developed sudden onset of constipation six months previously, requiring disimpaction of stool on an emergency basis. This was associated with severe abdominal pain and abdominal bloating. She was not helped by regular laxatives or enemas.

On physical examination, there was a marked perineal descent of 3 cm. Sigmoidoscopic and proctoscopic examinations demonstrated copious amounts of mucus and an anterior mucosal intussusception which did not prolapse into the anal canal.

The proctography result is shown in Figures 7 and 8.

DISCUSSION

Cases of solitary rectal ulcer syndrome (SRUS) and colitis cystica profunda (CCP) which show rectal prolapse of varying degrees are illustrated with Case I and II. The rectal prolapse was not recognized in Case I and initially, a diagnosis of ulcerative proctitis

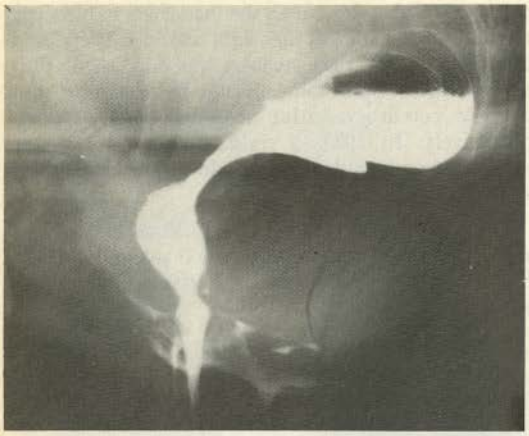

Fig. 7. Case V. Starting position. Note leakage of contrast despite complaint of constipation.

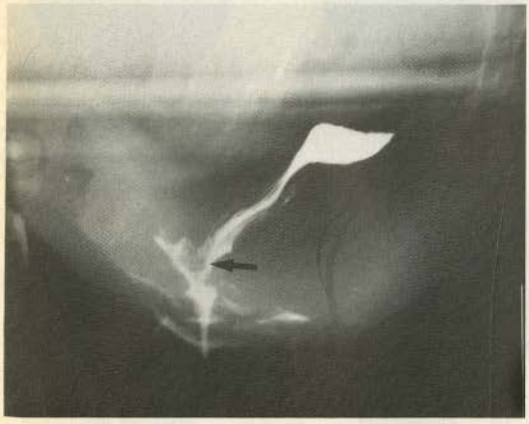

Fig. 8. Case V. Post-evacuation illustrating mucosal intussusception with occlusion of the anal canal (⟵).

had been made. The diagnosis of SRUS was confirmed by the typical histological findings on rectal biopsy. Case II had a previous diagnosis of CCP but the degree of rectal prolapse was not recognized until evacuation proctography was performed. It has been noted by several investigators that SRUS and CCP may have common etiologies in that rectal prolapse may be seen with both conditions.[3] These two patients both failed to improve on medical therapy including high fibre diets with avoidance of straining at stool, steroid enemas and 5-aminosalicylic acid enemas. Previous investigators have suggested that these patients often have a rectal prolapse and only respond to surgical correction of the prolapse. An underlying feature of the prolapse has been a suggestion of poor relaxation of the voluntary anal sphincter musculature.[4] However, Cases I and II did not show paradoxical contraction of the anal sphincter musculature on straining with concentric EMG needle testing. This suggests that these patients will do well following a rectal prolapse repair, if excessive straining at stool can be prevented post-operatively.

The last three cases illustrate how useful the evacuation proctogram can be in assessing patients with severe symptoms of constipation. All these patients had incapacitating symptoms of constipation unresponsive to a high fibre diet, often requiring large doses of laxatives or enemas. Cases III and IV are examples of constipation associated with paradoxical contraction of the anal sphincter on straining and, at proctography, neither patient could expel any of the barium paste despite repeated efforts of straining over 60 seconds each. Previous studies in severely constipated patients have also identified this problem of inability to empty the rectum and, as a result, they are very difficult patients to treat.[5,6] Ileorectal anastomosis for severe constipation may not work in these patients because of the inability to empty even liquid stool from the rectum, and this explains why these patients do not respond to the usual therapy of a high fibre diet or laxatives.[7]

Case V also presented with severe constipation, unresponsive to a high fibre diet but her problem was one of occult rectal mucosal intussusception which blocked solid stool emptying from the rectum. In addition, she demonstrated poor muscular control with some leakage of contrast at rest. This type of patient requires a rectal prolapse repair to prevent the rectal mucosal intussusception; however, post-operatively, if her stool should become loose, she may experience fecal incontinence. This problem will usually be improved by a post-anal repair.

The important feature in Case V compared with Cases III and IV is that although all these patients had severe constipation, the presence of an obvious mechanical obstruction of the rectum in Case V indicates that surgery will improve the problem. Unfortunately, at this time, the role of surgery in patients with paradoxic contraction of the pelvic floor is unclear.[8] Both Cases III and IV had multiple abdominal operations and this may be a contributing factor to the intractable constipation with abdominal pain and bloating.

As demonstrated by these case reports, evacuation proctography is a useful new radiologic procedure that can be used to evaluate complex problems of anorectal dysfunction. It can be performed quickly, with minimal patient discomfort and exposes the patient to approximately one-tenth the radiation dose of a standard barium enema.[2] In contrast to a barium enema, evacuation proctography provides a means of evaluating the "function" of the anorectum and, as illustrated above, can uncover abnormalities of anorectal function that may not be seen with standard clinical examination techniques. □

References

1. Mahieu P, Pringot J, Bodart P. Defecography: I. Description of a New Procedure and Results in Normal Patients. *Gastrointest Radiol* 1984; **9:** 247-251.
2. Bartram CI, Turnbull GK, Lennard-Jones JE. Evacuation Proctography — An Investigation of Rectal Expulsion in 20 Subjects Without Defaecatory Disturbance. *Gastrointest Radiol* 1987; (in press).
3. Levine DS. "Solitary" Rectal Ulcer Syndrome. *Gastroenterol* 1987; **92:** 243-253.
4. Rutter KRP. Electromyographic Changes in Certain Pelvic Floor Abnormalities. *Proc Roy Soc Med* 1974; **67:** 53-56.
5. Preston DM, Lennard-Jones JE. Anismus in Chronic Constipation. *Dig Dis Sci* 1985; **30:** 413-418.
6. Editorial. Constipation in Young Women. *Lancet* 1986; **i:** 778-779.
7. Turnbull GK, Lennard-Jones JE, Bartram CI. Failure of Rectal Expulsion as a Cause of Constipation: Why Fibre and Laxatives Sometimes Fail. *Lancet* 1986; **i:** 767-769.
8. Barnes PRH, Hawley PR, Preston DM, Lennard-Jones JE. Experience of posterior division of the puborectalis muscle in the management of chronic constipation. *Br J Surg* 1985; **72:** 475-477.

A specialist differs from a non-specialist in that the former learns more and more about less and less, until eventually he knows everything about nothing; whereas the latter learns less and less about more and more until eventually he knows nothing about everything.

— Anonymous

GROWTH HORMONE THERAPY and CREUTZFELDT-JAKOB DISEASE

There have now been seven confirmed deaths due to Creutzfeldt-Jakob Disease in young persons who received Human Pituitary Growth Hormone for treatment of growth hormone deficiency in the late 1960s and early 1970s. Four of these were diagnosed in 1985, three from the U.S.A. and one from Great Britain. Three new cases, two from the U.S.A. and one from New Zealand, have been confirmed in 1986. A direct causal relationship between Growth Hormone Therapy and Creutzfeldt-Jakob Disease in young persons has not been proven yet. All physicians in Canada are reminded of the importance of reporting any suspicious new unexplained neurologic symptoms or death in an individual who has received Human Pituitary Growth Hormone.

Please call Dr. Heather Dean at the Canadian Growth Hormone Registry office in Winnipeg, 204-787-4553 or 204-787-3011.

Diethylstilbestrol Fact Sheet for Health Professionals

DES, Diethylstilbestrol, is a synthetic female hormone (estrogen) that was administered to millions of pregnant women, from 1941 to 1971, primarily to prevent miscarriages. DES was given as pills, shots or suppositories and was sold under many different trade names.

Research findings indicate that the following health problems can be related to DES exposure. Research on the health hazards of DES is ongoing.

POTENTIAL HEALTH PROBLEMS

DES Daughters

- Risk of developing clear cell adenocarcinoma is approximately 1 in 1,000
- Approximately 45% have vaginal adenosis (may disappear over time)
- Cervical hoods, collars, or cockscombs (may disappear over time)
- Gytological alterations on cervical smears
- Vaginal septa
- Menstrual pattern changes
- Pregnancy problems (ectopic pregnancy, incompetent cervix, prematurity, first and mid-trimester abortions, T-shaped uterus)
- Need initial screening and special gynecologic examination at least once a year indefinitely

DES Mothers

- May be at higher risk for breast cancer
- Need to practise monthly breast self-exam
- Need yearly gynecologic examination

DES Sons

- Approximately 30% have testicular abnormalities such as benign epidiymal cysts, hypoplastic testes, or undescended testes
- Fertility problems associated with sperm and semen abnormalities
- Urethral stenosis, urinary tract infections, kidney and bladder pain, penile discharge, problems passing urine
- May be at higher risk for testicular cancer
- Need topractice monthly self-examination of the testes
- Need to have an examination by a Urologist

FOR MORE INFORMATION CONTACT:

DES ACTION/N.S., P.O. BOX 311, GREENWOOD, N.S., B0P 1N0, 765-3907

Percutaneous Gastrojejunostomy for Enteral Feeding

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Recent reports have described a non-surgical technique for introduction of percutaneous gastrostomy (PG) and percutaneous gastrojejunostomy (PGE) feeding tubes.[1,3] The traditional method of a surgically-created gastrostomy is associated with high mortality and morbidity, due mostly to the underlying disease which is often advanced, and to the fact that many patients are under-nourished.[4] PG and PGE tubes are indicated primarily in patients with persistent dysphagia and in control of aspiration.

MATERIAL AND METHODS

From January 1987, to date, three patients had PG tubes (all men), and four subsequent patients (3 men, 1 woman) had PGE tubes (Table I) performed at the Halifax Infirmary. Jejunal placement is now attempted as our procedure of choice.

TABLE I

UNDERLYING PATHOLOGICAL CONDITIONS
(7 patients, aged 47-70)

DYSPHAGIA	Recurrent carcinoma of the larynx	2
	Recurrent carcinoma of post cricoid	1
	Carcinoma lower esophagus	1
	Carcinoma floor of mouth	1
	Severe esophagitis, hypoxic brain damage	1
ASPIRATION	Recurrent pneumonitis, aperistaltic esophagus	1

Patients were fasted overnight, and were given a mild sedative as premedication. A naso-gastric tube was passed prior to the procedure, usually feasible even in the presence of advanced esophageal obstruction; occasionally, fluoroscopy and guidewire assistance were necessary. Ultrasound was utilized to delineate the liver margin, and the nasogastric tube was used to insufflate the stomach with air.

Under fluoroscopy, the distended stomach was easily seen separate from the transverse colon, and a trochar and cannula puncture of the anterior gastric wall was then made. A wire was passed, the tract dilated, and the wire and catheter were manipulated through the pylorus under fluoroscopy, and thence to the proximal jejunum. We have found the 10-French extended Cope nephrostomy chatheter (Cook) (Fig. 1) with a loop-forming distal segment, to be most suitable and to be well retained within the proximal jejunum (Fig. 2).

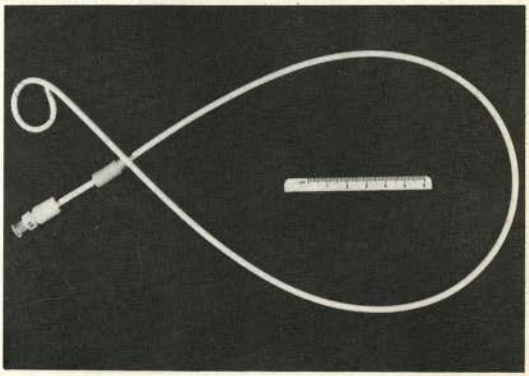

Fig. 1 The Cope nephrostomy catheter, with the drawstring which passes inside the catheter to maintain the loop when in the jejunum. The loop contains all the side holes.

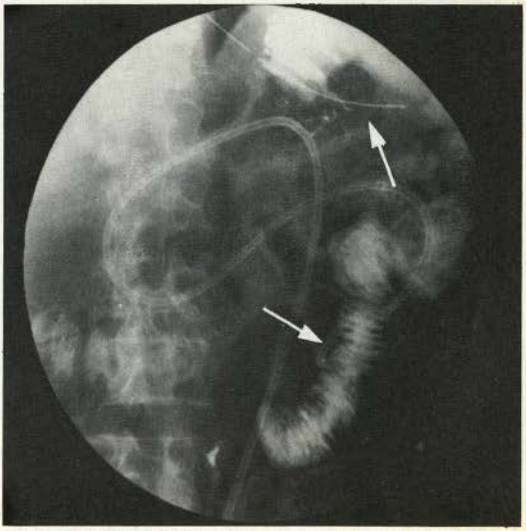

Fig. 2 Radiograph showing the catheter in place. The catheter passes into the stomach opposite the upper arrow and thence around the duodenal loop to the jejunum (lower arrow). Some contrast has been injected through the catheter and outlines the jejunal mucosa. The upper arrow points to a nasogastric tube in the proximal stomach which contains some fundal contrast medium.

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RESULTS

There were no deaths or major complications associated with the procedures. Three patients subsequently died of their disease and in one, the tube had become dislodged at two weeks and could not be replaced. Three patients were discharged, and one remains in hospital, all with the catheters functioning well.

DISCUSSION

PG and PGE tubes will provide adequate fluid and nutrition (medications must be in liquid form), on a short term basis, e.g. to cover a difficult post-operative course, or for longer term use especially for incurable disease in a patient who is not yet terminally ill. The procedure is relatively simple and, by placing the feeding tube through to the jejunum, the potential risk of gastroesophageal reflux and peritoneal-gastric stomal leakage are avoided. The catheter placed initially is 10-French (less than 4 mm diameter) and, after any subsequent exchanges, it never exceeds 16 French size. These small calibre catheters are readily accepted by the thick muscular gastric wall, and this together with rapid formation of adhesions and omentum gathering around the puncture site, produces a satisfactory gastrocutaneous tract in less than four weeks. Subsequent endoscopic examinations have shown that there is very little gastric mucosal reaction at the stoma site.

The cathers are easily managed by the patient or family in a home setting, requiring only domiciliary nursing care for dressing changes. Should the catheter become inadvertently removed, it is a simple matter to replace it.

PG and PGE tubes are well established procedures, offering an alternative to surgical or endoscopic catheter placement.

□

References

1. Ho CS. Percutaneous gastrostomy for jejunal feeding. *Radiol* 1983; **149:** 595-96.
2. Wills JS, Oglesby JT. Percutaneous gastrostomy. *Radiol* 1983; **149:** 449-453.
3. Ho CS, *et al.* Percutaneous gastrostomy for Enteral Feeding. *Radiol* 1985; **156:** 349-351.
4. Wasiljew BK, *et al.* Feeding Gastrostomy, Complications and Morbidity *Am J Surg* 1982; **143:** 194-195.

Transjugular Liver Biopsy:
EXPERIENCE WITH FIFTY-SEVEN CASES

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Conventional percutaneous liver biopsy is at least relatively contraindicated in patients with severe bleeding diathesis or moderate to large ascites. The transjugular approach to the liver has been performed since 1967 when Hanafee and Weiner utilized it for percutaneous cholangiography.[1] The first liver biopsy by this transvenous route was by Rosch *et al.* in 1973.[2]

We describe our experience with 57 attempted transjugular liver biopsies which in these patients we believe represents a safer and more informative procedure, and which has a good patient acceptance.

METHODS AND MATERIALS

Patients

Between October 1985 and June 1987, 57 transjugular liver biopsies were attempted at the Halifax Infirmary in 55 patients, ranging in age from 13 years to 84 years. There were 30 males and 25 females.

The indications for the transjugular route were, for the vast majority, bleeding diathesis, significant ascites, or both. In four other patients, previous percutaneous liver biopsy had been unsuccessful and the transjugular route subsequently yielded a diagnostic specimen.

Technique

All patients are monitored electrocardiographically while in the suite because of the risk of catheter induced arrhythmia. With the patient in slight Trendelenberg position, and under local anaesthesia, a sheath needle, guide wire, and subsequently a 9 French curved Teflon® cathether are introduced into the right jugular vein (in two patients, successful manometry and biopsy were obtained using the left internal jugular vein after the right jugular approach was unsuccessful). Under fluoroscopy, the catheter is advanced in sequence to the right atrium, to the low and high inferior vena cava, and finally to the hepatic vein (usually the right hepatic vein), Venous pressures in mmHg are recorded at all these sites. In the hepatic vein both wedged and free pressures are obtained and recorded in at least two different sites.

Following manometry, a modified Ross needle (Cook Canada Incorporated) is passed coaxially via the catheter and a biopsy specimen obtained with the catheter in a central non-wedged position. Some authors utilize a wedged position for the biopsy, but we believe this results in more frequent capsular perforation.[3] As many as 5 to 6 passes may be required for a satisfactory specimen, particularly in patients with hard, cirrhotic livers which are prone to fragmentation. Following each biopsy attempt, contrast medium is injected into the hepatic vein under fluoroscopy to exclude visible extravasation. Demonstrable extravasation should be controlled by selective embolization of the biopsy tract using Gelfoam or wire coils.[4] Following catheter withdrawal, the patient is nursed in the semi-erect position for four to six hours in order to minimize neck hematoma. Vital signs are monitored.

RESULTS

In 54 of 57 attempts a liver biopsy, adequate for histologic assessment, was obtained for an overall success rate of 94.7 percent. This figure compares favourably with reported success rates of from 64 percent[5] to 97 percent.[3] One specimen (a repeat study) was insufficient for adequate histologic assessment, containing no portal tracts. No specimen could be obtained in two patients, one in whom the jugular vein could not be entered; the other patient has had a subtotal hepatic resection resulting in the single remaining hepatic vein entering the vena cava at greater than 90 degrees, precluding entry of the rigid curved needle.

The histologic analysis by light microscoy of the specimens obtained is summarized in Table I.

TABLE I

SUMMARY OF HISTOLOGIC ANALYSIS

	Histologic Diagnosis	Number of Specimens	Percent
1.	Hepatitis or cirrhosis	39	70.9%
2.	Non-specific reactive changes	4	7.2%
3.	Large duct obstruction	3	5.4%
4.	Probable hepatoma	2	3.6%
5.	Metastatic carcinoma	2	3.6%
6.	Hepatic congestion (chronic cardiac failure)	1	1.8%
7.	Probable drug reaction	1	1.8%
8.	Extramedullary hematopoiesis	1	1.8%
9.	Alpha-1 Antitrypsin deficiency	1	1.8%
10.	Insufficient specimen	1	1.8%

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Pressures were recorded and a corrected sinusoidal pressure reported for all patients. This was determined by subtracting the free hepatic vein pressure from the wedged hepatic vein pressure. The degree of portal hypertension and classification of patients by severity of pressure elevation was then reported as summarized in Table II.

TABLE II

CORRECTED SINUSOIDAL PRESSURES AS A REFLECTION OF PORTAL HYPERTENSION

	No. of Patients*	Percent of Patients*
< 5 mmHg — Normal	4	7.3%
6-10 mmHg — Mild elevation	8	14.6%
11-19 mmHg — Moderate elevation	28	51.0%
> 20 mmHg — Severe elevation	14	25.5%

* There was pressure monitor failure during one case

COMPLICATIONS

There were no deaths directly attributable to the procedure and no transcapsular bleeding significant enough to necessitate laparotomy for operative closure of a perforation. Two subcapsular hematomas were observed, determined by subcapsular extravasation and collection of contrast introduced into the hepatic vein following biopsy. This extravasation resolved in both cases in less than five minutes as embolization of the bleeding tract was being prepared, and there was no later alteration of vital signs.

Subclinical and undetectable intraperitoneal bleeding does occur, as in one patient undergoing continuous intraperitoneal dialysis who experienced blood-tinged dialysate for one week following biopsy. One biopsy on a 13 year old male demonstrated chronic active hepatitis and normal renal cortex, again confirming undetectable capsular transgression. This was followed by pyrexia to 39.5°C overnight and less than 24 hours of hematuria which resolved spontaneously. Post biopsy pyrexia was otherwise not reported as with other authors who also noted decrease in frequency once ultrasonic cleaning of the biopsy needle had been instituted.[3,6]

A moderate neck hematoma was seen in four patients, invariably in those in whom the common carotid artery was inadvertently punctured while attempting to enter the internal jugular vein. There were no neurologic symptoms or sequelae. One patient experienced supraventricular tachycardia lasting approximately 10 minutes which reverted spontaneously to normal rhythm, allowing completion of manometry and biopsy.

DISCUSSION

Any liver biopsy is attended by the risk of significant life threatening intraperitoneal bleeding and death, with mortality rates of 0.014% to 0.25% reported for percutaneous biopsy.[7] In patients with ascites and impared bleeding mechanisms, the rate is greater; in a review of 2271 transvenous biopsies a mortality rate of 0.13% was reported.[8] In these higher risk patients, it has been advocated that embolization of the tract in the liver be performed with a wire coil following percutaneous biopsy.[9] However, this route does not allow for selective pressure determinations which are of additional value in assessing severity of disease and in planning patient management, e.g. portocaval shunting in patients with variceal bleeding, and moderate or severe portal hypertension. Manometry and biopsy are also useful in follow-up of patients to determine efficacy of treatment.

Percutaneous biopsies sample primarily the outer portions of the liver, areas where extensive fibrosis and architectural distorition may preclude satisfactory histologic examination.[10] The transjugular route samples more central portions where the underlying etiologic process may still be active and definable.

The transvenous approach does not lend itself readily to biopsy of specific mass lesions within the liver. In our series, one patient with diffuse metastatic foci demonstrated sonographically and by nuclear liver scan, was biopsied from multiple sites but revealed only nonspecific changes.

The procedure is tolerated well by most patients with or without prior sedation or analgesia; the major complaint is brief visceral pain during the actual needle insertion. Four patients had undergone previous percutaneous liver biopsy as well, of whom three (75%) preferred the transjugular route.

Transjugular liver biopsy is not intended to replace conventional percutaneous biopsy. The former requires fluoroscopic control, ideally with a C-arm unit, experienced personnel, and greater operator time. It is however a useful addition to the armamentarium of a gastrointestinal investigative and treatment centre, for assessment of a number of patients in whom histologic diagnosis would otherwise be obtainable only by high risk percutaneous biopsy or laparotomy. □

References

1. Hanafee W, Weiner M: Transjugular percutaneous cholangiography. *Radiology* 1967; **88:** 35-39.
2. Rosch J, Lakin PC, Antonovic R, et al: Transjugular approach to liver biopsy and transhepatic cholangiography. *New Engl J Med* 1973; **289:** 227-231.
3. Bull HJM, Gilmore IT, Bradley RD, et al: Experience with transjugular liver biopsy. *Gut* 1983; **24:** 1057-1060.
4. Braillon A, Revert R, Rémond A, et al. Transcatheter embolization of liver capsule perforation during transvenous liver biopsy. *Gastrointest Radiol* 1986; **11:** 277-279.

References continued on page 28.

An Approach to the Esophagus as a Cause of Chest Pain:
THE HALIFAX INFIRMARY GI UNIT

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Of patients who present to their physicians for symptoms of chest pain suggestive of angina pectoris, between 10 and 30 percent are subsequently found to have normal coronary arteries at the time of coronary angiography.[1] Other causes of chest pain mimicking coronary pain must then be considered. These include musculoskeletal, esophageal, and (rarely) pulmonary causes. It is estimated that up to 50 percent of these patients may have an esophageal cause for their pain.[2]

The patients who are referred to the Halifax Infirmary GI Unit for investigation of their chest pain are broadly divided into two groups. The first includes patients with known coronary artery disease in whom the physician feels that the particular pain that the patient is complaining about is not cardiac in origin. The second group are patients who have a typical chest pain, or pain very suggestive of angina pectoris with negative cardiac investigations.

In patients with known coronary artery disease, Mellow has demonstrated in 37 patients — 25 of whom had known coronary artery disease — that the ischemic threshold, defined as a product of the heart rate and systolic blood pressure, rose in many patients with nasogastric intubation and with acid perfusion.[3] Several of these patients developed EKG changes with this stimulation. Others have demonstrated that acid infusion into the distal esophagus may decrease the exertional threshold to angina during stress testing. Thus, nasogastric intubation and the acid perfusion test may serve as a type of cardiac stress test. In patients with coronary artery disease and a history of angina who have pain during acid perfusion of the distal esophagus, greater than 50 percent in one series thought they were having their typical angina during acid perfusion. Thus, patients with known heart disease frequently are unable to tell the difference between angina pectoris and symptomatic acid reflux in the distal esophagus.

In patients without cardiac disease, esophageal causes of chest pain include gastroesophageal reflux, esophageal motility disturbances, and a newly-defined condition "the sensitive esophagus". Also to be considered are other gastrointestinal conditions which can cause chest pain such as acid peptic disease of the stomach and duodenum or, less commonly, biliary or other gastrointestinal disorders.

A clinical history is absolutely essential for the consideration of a gastrointestinal cause for chest pain. In particular one should note the relationship of the chest pain to gastrointestinal function. A patient with gastroesophageal reflux most commonly will complain of symptoms immediately after eating, in particular after a large, heavy meal with fatty foods, or in association with excessive consumption of alcohol or cigarettes. The symptoms may, of course, be associated with regurgitation of gastric contents, retrosternal pyrosis, epigastric fullness, or belching. Symptoms are often worse when lying down and may be associated with nocturnal aspiration. Esophageal motility disturbances are noted with either solids or liquids. They may be associated with a sensation of odynophagia or dysphagia and may be noted particularly on drinking very hot or very cold liquids. A history of exertional chest pain leads one away from an esophageal cause as an explanation of chest pain. Relief with antacids supports an esophageal cause of pain while nitrates may relieve the pain of angina pectoris or esophageal spasm, thus adding confusion to the differentiation from the diagnosis of coronary artery disease.

The second diagnostic tool, the physical examination, is less useful in differentiating cardiac from esophageal causes for chest pain; however, it is essential that the chest wall be carefully examined to exclude musculoskeletal causes of pain.

The next step is often the most difficult as there are multiple investigations that one could order to try and sort out the cause of the pain. In most instances if there is any doubt as to whether or not the pain might be related to the heart, I would recommend that cardiac disease be excluded as completely as possible before proceeding with esophageal investigations. Depending on the clinical situation, this might include stress testing, radionuclide studies or coronary angiography.

As far as the gastrointestinal tract is concerned, the first diagnostic test would ordinarily be an upper gastrointestinal endoscopy. While this is not a sensitive test for gastroesophageal reflux nor a good way of assessing esophageal motility, it does rule out peptic ulcer disease, gastritis, or duodenitis. Of course, it will also demonstrate inflammation in some cases of esophagitis as well as ruling out strictures, rings, or webs as a cause of dysphagia.

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The question is frequently asked whether or not patients should have a barium swallow and upper GI series prior to endoscopic examination. It is rare for the barium study to add useful diagnostic information in a patient presenting with chest pain that is not detected with a gastroscopy. The presence of tertiary waves, a hiatus hernia, or free reflux often depends on the vigor of the procedure. The barium swallow does help in the diagnosis of achalasia or minor strictures, but these conditions do not usually present with chest pain. Gastroscopy has the advantage of being more sensitive in detecting mild inflammatory changes in the esophagus, stomach, or duodenum. Thus, I think it is reasonably justified to proceed directly to upper gastrointestinal endoscopy without upper GI radiology in the investigation of of the possible gastrointestinal causes of chest pain.

The next series of investigations is done to try and answer very specific questions. It is simply not enough to know that there is something abnormal with the esophagus such as gastroesophageal reflux or esophageal spasm. One would like to be able to say that when the patient has reflux they have pain, or when they have esophageal spasm they have pain and, when they do not have either of these conditions they do not have pain. There are two approaches generally used. The first is to do continuous esophageal monitoring. Within the next year the Halifax Infirmary will have the ability to continuously monitor intra-esophageal pH for 24 hours. This is done with an indwelling naso-esophageal pH electrode connected to the recording device. The pH is continuously monitored, and the patient is able to record episodes of chest pain or any other event by simply pressing a button. It is then a matter of comparing the intra-esophageal pH with the chest pain to see whether it is associated with a drop intra-esophageal pH. In the future it will be possible to monitor motility similiarly.

The other approach relies on provocative testing. Historically, these tests have included recording baseline esophageal motility and then monitoring

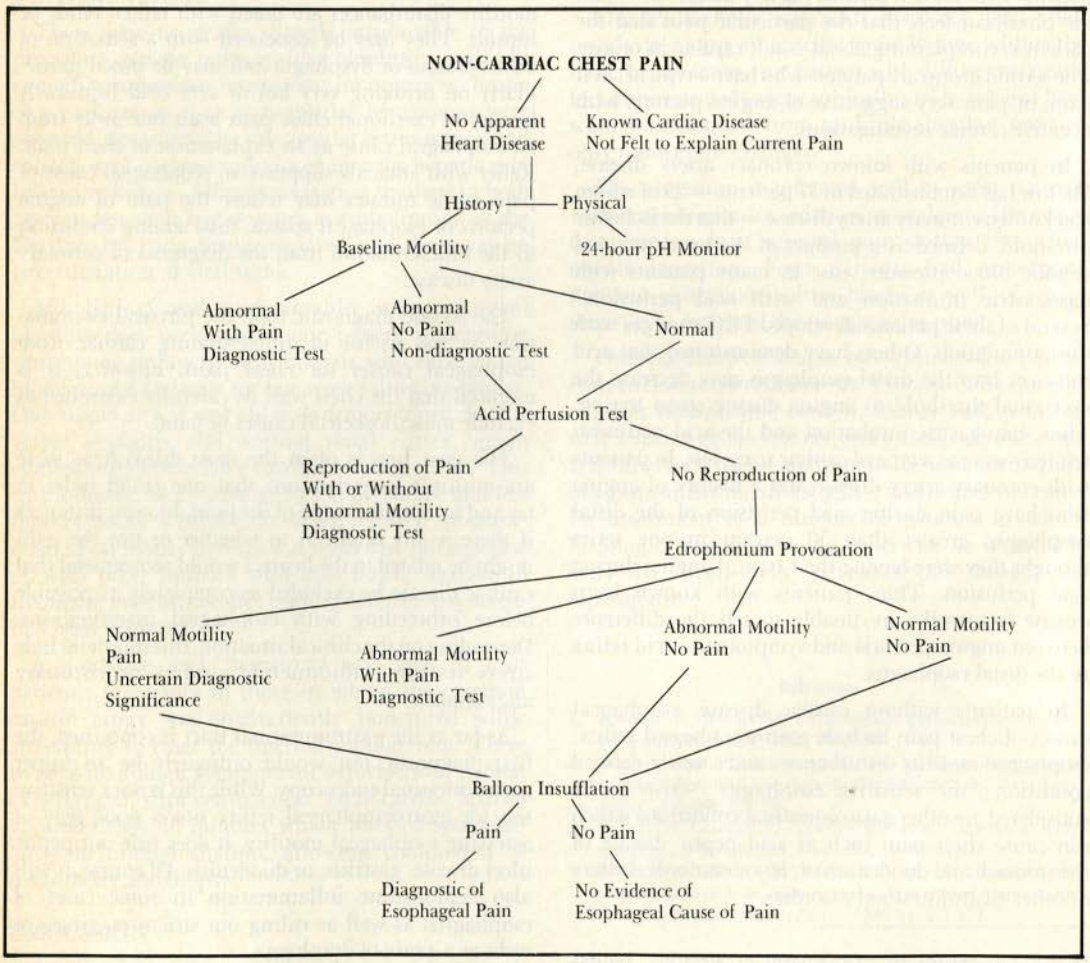

Figure 1

esophageal motility during various provocations. The tests have included stimulation of the esophagus with acid (the acid perfusion test or Bernstein test), subcutaneous injections of pentagastrin, bethanecol, intravenous infusion of ergonovine or the cholinesterase inhibitor edrophonium. In various papers each of these has its proponents. Having reviewed the literature and considering the ability of each to produce a positive diagnostic test with minimal side-effects, we have concluded that the acid perfusion test followed by an edrophonium stimulation is the optimal protocol.

The baseline motility recording tells us if the patient has important abnormalities such as a nutcracker esophagus with high amplitude prolonged contractions, diffuse esophageal spasm, non-specific esophageal dysmotility disturbances, or achalasia. The acid perfusion test asks two questions. "Does acid in the esophagus reproduce the patient's chest pain?" "Does acid in the esophagus cause esophageal dysmotility?" The edrophonium test is an attempt to provoke chest pain and dysmotility. In one series this reproduced chest pain in 30 percent of non-coronary artery disease chest pain patients while it did not produce any pain in controls.[5]

Recently Joel Richter, working with Donald Castell, has demonstrated that some patients with non-cardiac chest pain appear to have abnormal sensory perception of esophageal distension.[4] In 30 patients with non-cardiac chest pain, insufflation of an intra-esophageal balloon reproduced chest pain in 60 percent of patients compared with no chest pain when the balloon was insufflated with similar volumes of air in normal patients. This seemed to be unassociated with esophageal dysmotility, and the authors concluded that patients with non-cardiac chest pain may have a lower pain threshold to intra-esophageal distension.

Therefore, (Figure 1) in the GI Unit at the Halifax Infirmary, our protocol for the investigation of non-cardiac chest pain includes a complete history and physical examination followed by upper gastrointestinal endoscopy. We will then do a baseline esophageal motility study followed by an acid perfusion test. In selected patients we will, at the same time, monitor heart rate, systolic blood pressure and electrocardiogram. If these tests are negative, we then attempt to provoke chest pain and dysmotility with ergonovine and balloon insuflation.

Patients with undiagnosed chest pain frequently are significantly incapacitated by their pain, concerned that perhaps they do have cardiac disease. Proving that the problem is esophageal may serve to alleviate their fears and assist in our management of their pain. □

References

1. Proudfit WL, Shirley EK, Sones FM. Selective cine coronary arteriography: correlation with clinical findings in 1,000 patients. *Circulation*. 1966; **33:** 901-10.
2. Richter JE, Castell DO. Diffuse Esophageal spasm: a reappraisal. *Ann Int Med*. 1984; **100:** 242-5.
3. Mellow MH, Simpson AG, Watt L, Schoolmeester L, Haye LH. Esophageal acid perfusion in coronary artery disease. *Gastroenterol*. 1983: **85:** 306-312.
4. Richter JE, Barisit CF, Castell DO. Abnormal sensory perception in patients with esophageal chest pain. *Gastroenterol*. 1986; **91:** 845-52.
5. Richter JE, Hackshaw BT, Wu WC, Castell DO. Edrophonium: a useful provocative test for esophageal chest pain. *Ann Int Med*. 1985; **103:** 14-21.

Total Esophagectomy Without Thoracotomy

A HIGH PRIORITY FOR TREATMENT OF ESOPHAGEAL CARCINOMA

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Carcinoma of the esophagus still carries a poor prognosis. Overall five year survival ranges from 5 to 20 percent despite aggressive treatment.[1-3] Resection with reconstruction, alone or in association with radiotherapy, provides the best results. The stage and biological behavior of the tumor is the most significant single factor determining survival. This is exemplified by a greater than 80 percent survival after esophagectomy in patients with Stage I tumors in China. These tumors were detected at a very early stage due to an extensive screening program.[4] Once the diagnosis has been made, operation may be undertaken for two different reasons according to the stage of the tumor. A "curative" operation should be performed for those tumors detected in early stages, while a palliative operation is done to alleviate symptoms in the remainder.

Patients with terminal disease often receive palliative treatment with a minor operation (i.e. celestin tube) with the sole purpose of improving the quality of life and dignity. Esophageal obstruction with inability to swallow even their own saliva is devastating for patients and family.

For those patients with resectable lesions, two different techniques are usually advocated by surgeons: transthoracic or transhiatal esophagectomy.[5-9] The scope of this paper is a comparison of the advantages and disadvantages between the techniques.

Transthoracic esophagectomy is usually performed through two separate incisions — an abdominal midline incision followed by a thoracotomy. Through the abdominal incision the abdominal cavity is explored for staging. Disection of the hiatus is initiated and the organ to replace the esophagus is selected and prepared. The stomach is used most commonly followed by colon and jejunum. A right thoracotomy is then utilized to complete the resection and to establish continuity with an esophagogastric anastomosis that lies inside the chest. Left thoracotomies are usually reserved for patients with lesions of the gastroesophageal junction when part of the stomach has to be removed, thereby restricting its ability to reach the upper third of the esophagus.

Transhiatal esophagectomy, also known as esophagectomy without thoracotomy, is also initiated with a midline abdominal incision with the same procedures for staging. Assessment of operability and organ preparation are done as for the previous procedure, except for a more extensive and meticulous dissection of the stomach (or alternative organ), because the proximal anastomosis will be completed in the neck instead of the chest. The chest is not open and the esophageal dissection is accomplished through a manually dilated hiatus and the neck incision. Thus this is a more demanding operation, with very small margin for error.

COMPARISON BETWEEN TRANSTHORACIC (TT) AND TRANSHIATAL (TH) ESOPHAGECTOMY:

1. ***Anastomotic Leak.*** This is the most feared technical complication, especially if the anastomosis is placed inside the chest. Anastomotic leaks in this location produce mediastinitis and empyema with respiratory insufficiency, and could evolve into sepsis and death. Surgical correction is often difficult, and mortality of up to 30 percent has been reported.[1-3]

If the anastomosis is placed in the neck (TH) and leakage occurs, only wound infection usually results and all other complications are avoided. Operative mortality is usually below 10 percent.[10]

2. ***Reflux Esophagitis.*** Since resection of the lower esophageal sphincter (GE junction) is performed in both operations, the incidence of reflux will depend on where the anastomosis is located. The transthoracic approach, with anastomosis inside the chest (thoracic esophagus), exposes esophageal squamous epithelium to reflux with incidences of esophagitis as high as 40 to 60 percent. When the anastomosis is done in the neck (cervical esophagus), the upper esophageal sphincter is preserved. Practically no squamous epithelium is exposed to reflux, and thus reflux is an insignificant 3 percent.[10] We must remember that these patients' presenting symptom is dysphagia. An intrathoracic anastomosis fails to eliminate the symptom for which the operation was intended.

3. ***Resectability.*** Advocates of the transthoracic approach maintain that if the chest is not opened, then the resection may be incomplete since visualization of the tumor during removal is not always possible. Orringer has pointed out that by the time an esophageal tumor has invaded adjacent nodes, the disease has become systemic and this will be the

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primary determinant of their survival, not the lymph node disease *per se*.

I perform computerized tomography of the chest routinely in all my patients to anticipate the problem. On the other hand, we must remember that a unique characteristic of esophageal carcinoma is the early spread through the submucosa. Thus it is this tumor, more than any other in the GI tract, that requires the widest margins of resection away from the tumor in both directions (Fig. 1). Thus "curative" resection is made more likely with TH mainly in smaller tumors. For larger tumors, it will always be beneficial not to have recurrence in the chest regardless of distant metastasis.

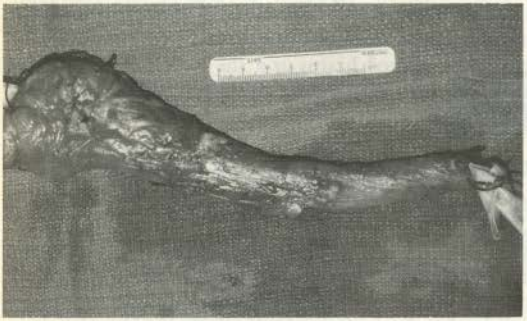

Fig. 1 Resected specimen from patient number one. Tumor is located in lower third of esophagus, several centimetres above the gastric level of resection. Notice that a cuff of stomach has been resected to provide as safe a margin as possible.

4. ***Survival.*** It is tempting to assume that TH will provide better survival in the long term as this technique already provides lower operative mortality. Even if this does not prove to be true, my personal preference is to resect the esophagus without opening the chest to avoid the potential complications that will prolong hospital stay, thus depriving these patients of precious symptom-free days. The following cases illustrate this benefit with three different tumors.

CASE SUMMARIES

Patient #1

A.M. — 63 year old male admitted to the Halifax Infirmary with dysphagia for one year. Previous history revealed hypertension; peripheral vascular disease, with intermittent claudication; and chronic obstructive pulmonary disease. An upper GI series revealed a fungating lesion at the level of the lower third of the esophagus, and areas of ulceration were present. Gastroscopy revealed a tumor in the distal esophagus located 33 to 36 centimetres from the incisors. Liver/spleen scan and CT scan of the chest and abdomen showed absence of metastatic disease. The tumor was confined to the esophageal wall.

The patient was then taken to the operating room where a bronchoscopy revealed no involvement of the posterior wall of the trachea. A total esophagectomy without thoracotomy was performed through the abdomen and stretched hiatus. The fundus of the stomach was brought up into the neck.[1,2] A pylormyotomy was added to prevent gastric stasis and a feeding jejunostomy provided for post-operative nutritional care in the early stages.

The patient had an uneventful recovery. On the sixth post-operative day, a barium swallow revealed no evidence of leakage at the level of the cervical anastomosis. He was fed by mouth and supplemented by the feeding jejunostomy for a week, and this reversed his catabolic stage satisfactorily. A week later, he was discharged from hospital asymptomatic and on a regular diet.

Histology of his tumor revealed a poorly differentiated adenocarcinoma invading the inner and outer muscular layers but not entirely through the muscle layers of the esophagus. Five lymph nodes showed reactive changes but no evidence of malignancy.

At 20 months follow-up he remains well. There is no evidence of recurrence. He is having a regular diet and there is no dysphagia or evidence of reflux.

Fig. 2 Stomach has been completely mobilized and can be seen on top of the chest and easily reaching the patient's neck. Once the stomach is placed inside the chest, relative length is even better since the distance between hiatus and neck is shorter. Notice the pylorus has been well mobilized and the stomach shows excellent color throughout its entire length without signs of ischemia.

Patient #2

O.B. — 70 year old male, originally admitted to the ENT service with dysphagia, odynophagia and weight loss.

Previous history revealed extensive alcohol abuse, heavy smoking and dyspepsia, and reflux for years. Endoscopy revealed a small lesion in the mid-esophagus and biopsies reported an invasive squamous cell carcinoma. A small hiatal hernia was also visualized. CT scan of thorax and abdomen showed thickening of the mid-portion of the esophagus at the level of the carina, consistent with carcinoma, but no

extension outside the tumor was detected. Bronchoscopy failed to reveal involvement of the tumor into the membraneous portion of the trachea.

A total esophagectomy through a transhiatal approach was performed. The stomach was brought up to the neck and anastomosed to the esophagus. A plyormyotomy was performed as well as a feeding jejunostomy.

Post-operatively the patient developed a right pleural effusion that was drained. Ten days later he was discharged from hospital asymptomatic and having a regular diet.

Tumor histology revealed highly malignant squamous cells through the full thickness of the muscularis of the esophagus and into the periesophageal tissue. Malignant cells were also seen in the esophageal lymphatics and small veins. Radiation therapy was used in view of this tumor being a squamous cell carcinoma and involving the periesophageal tissues. Three-thousand cGy were delivered in fifteen fractions over a three week period.

At four months the patient remains asymptomatic, and is gaining weight on a regular diet.

Patient #3

S.O. — 62 year old female referred to our service with diagnosis of an occupying lesion of the middle-third of the esophagus, found during an upper GI series for investigation for dysphagia of three month duration. Previous history revealed a partial gastrectomy performed several years earlier for peptic ulcer disease.

A CT scan of the abdomen and chest revealed the tumor to be located in the mid-esophagus and little involvement of periesophageal tissues. Preoperative biopsy revealed squamous cell carcinoma.

In view of the patient's previous partial gastrectomy, the colon was prepared prior to surgery. Bronchoscopy was done for staging but no abnormalities were found. At surgery a Billroth type II gastrectomy was observed. As planned, the right colon was used for esophageal replacement since the stomach could not be used. The terminal ileum was included with the right colon and the anastomosis in the neck was performed between the terminal ileum and the cervical esophagus, on an end-to-end fashion.

Histology revealed a very poorly differentiated squamous cell carcinoma extending through the full thickness of the esophagus. Only one node contained carcinoma and it was located in the perinodal lymphatics. The node itself was free of malignancy.

The patient was discharged from hospital asymptomatic on her ninth post-operative day on a regular diet. She was referred to the cancer clinic for a 5000 cGy course of radiation, and she remains asymptomatic.

DISCUSSION

Several steps are crucial prior to resection. First, as evidenced by case number three, whenever there is doubt concerning the availability of the stomach for replacement, a bowel preparation should be done. The surgeon should be prepared to change his/her mind at the time of surgery and use an alternative approach when indicated. The stomach is the organ of choice for esophageal replacement because it has a better "dual" blood supply, requires only one anastomosis and avoids manipulation of the lower gut. When the stomach is not available, colon — either right or left — at the surgeon's preference is a satisfactory alternative. A loop of jejunum should be used only as a last resort, since it does not reach up to the neck in the average patient.

Bronchoscopy is essential mainly in mid-esophageal lesions to detect possible tracheal invasion. CT scan should be done in all cases to assess periesophageal infiltrate and to decide the best route for resection.

Radiotherapy is used as adjuvant therapy in all cases of squamous cell carcinoma, mainly if invasion of periesophageal tissues or nodes is present.

For all the above reasons, the author's approach to all esophageal carcinoma is transhiatal with anastomosis in the neck.

□

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References

1. Earlam R, Cunha-Melo JR. Oesophageal squamous cell carcinoma. I. A critical review of surgery. *Br J Surg* 1980; **67:** 381.
2. Giuli R, Gignoux M. Treatment of carcinoma of the esophagus — retrospective study of 2400 patients. *Ann Surg* 1980; **192:** 44.
3. Postlethwait RW. Complications and deaths after operations for esophageal carcinoma. *J Thorac Cardiovasc Surg* 1983; **85:** 827.
4. Huang KC. Diagnosis and surgical treatment of early esophageal carcinomas. In Stipa S, Belsey RHR, Moraldi A (eds): *Medical and Surgical Problems of the Esophagus.* London: Academic Press, 1981, pp 296-299.
5. Akiyama H, Tsurumaru M, Kawamura T, Ono Y. Principles of surgical treatment for carcinoma of the esophagus: analysis of lymph node involvement. *Ann Surg* 1981; **194:** 438.
6. Ellis FH Jr, Gibb SP. Esophagogastrectomy for carcinoma: current hospital mortality and morbidity rates. *Ann Surg* 1979; **190:** 699.
7. Piccone VA, LeVeen HH, Ahmed N, Groberg S. Reappraisal of esophagogastrectomy for esophageal malignancy. *Am J Surg* 1979; **137:** 32.
8. Orringer MB, Sloan H. Esophagectomy without thoracotomy. *J Thorac Cardiovasc Surg* 1978; **76:** 643.
9. Garvin PJ, Kaminski DL. Extrathoracic esophagectomy in the treatment of esophageal cancer. *Am J Surg* 1980; **140:** 772.
10. Orringer MB. Technical aids in performing transhiatal esophagectomy without thoracotomy. *Ann Thorac Surg* 1984; **38:** 128-132.

Dysarthria as the Initial Presentation of Motor Neuron Disease

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Recently, three elderly female patients with clinical histories and physical findings consistent with amyotrophic lateral sclerosis have been seen on the consultative neurologic service at the Halifax Infirmary Hospital. The clinical scenarios of all three patients were quite similar and, most interestingly, all were referred initially to the Otolaryngology Clinic in the Halifax Infirmary Hospital with complaints of dysarthria, dysphonia, and dysphagia, interpreted as being due to isolated pharyngeal pathology. Following assessment by the consultant otolaryngologists, neurologic opinion was requested. The case summaries are presented.

CASE SUMMARIES

Patient #1

Mrs. S.C., a 72-year-old white female from Digby, Nova Scotia, presented with a three-year history of slowly progressive extremity weakness with associated muscle wasting. Eight months prior to presentation, she developed dysphonia, dysarthria and subsequently a 20 pound weight loss. Just prior to presentation, she had further exacerbation of her bulbar symptoms and began to note marked nasality of speech and regurgitation of liquids.

Cranial nerve examination revealed wasting of facial musculature associated with fasciculations of the right facial muscles and as well, there was prominant tongue wasting with fasciculations. Motor system examination revealed diffuse wasting and weakness in muscle groups of all extremities and fasciculations were evident globally. There was spasticity in both lower extremities. Pathologically brisk reflexes were present symmetrically in the upper and lower extremities; however, plantar responses were flexor bilaterally. The remainder of the neurologic examination and, in particular, the sensory system examination was within normal limits. Electrodiagnostic studies revealed findings in keeping with motor neuron disease. The patient expired secondary to respiratory complications three months following the diagnosis of motor neuron disease.

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Patient #2

This 70-year-old white female from Stewiacke, Nova Scotia, presented with a one-year history of progressive dysarthria. Over the same time period, she had noted a 30 pound weight loss and although she denied weakness, she was aware that her extremity muscle bulk was diminishing and, as well, she had noted intermittent cramps in the right calf area.

Cranial nerve examination revealed asymmetric tongue wasting with diffuse prominant tongue fasciculations. Motor system testing revealed generalized decrease in muscle bulk in all extremities with fasciculations being present globally. Muscular weakness was present in all extremities with this being somewhat more pronounced proximally. Deep tendon reflexes tended to be pathologically brisk and plantar responses were flexor bilaterally. The remainder of the neurologic examination and, most importantly, the sensory system examination was normal for her age. Electrodiagnostic studies revealed findings consistent with motor neuron disease. Interestingly, in subsequent follow-up, she developed vertical diplopia and was found to have clinical and echographic evidence of Grave's (dysthyroid) orbitopathy as well.

Patient #3

This 70-year-old white female from Truro, Nova Scotia, presented with a 6-month history of progressive dysarthria. She had noted an increasing nasality in her voice along with increasing difficulty with swallowing. Progressive weakness of the upper extremities and wasting of the musculature in the hands was evident to her. Periodic cramping in the lower extremities was noted as well. There was a 25 pound weight loss over the two months prior to presentation.

Cranial nerve examination revealed asymmetric atrophy of the tongue with evident fasciculations. Motor system testing revealed global weakness in all extremities along with wasting of the intrinsic musculature of the hands. Deep tendon were pathologically brisk generally and the right plantar response was extensor. The remainder of neurologic examination and, in particular, the sensory system examination was within normal limits for the patient's age. Once again, the clinical clustering of upper and lower motor neuron signs accompanying bulbar dysfunction were felt to indicate a clinical diagnosis of motor neuron

disease. Electrodiagnostic studies are pending.

The similarities among these case presentations are immediately obvious. The patients were all elderly Caucasian females who were referred initially to the Otolaryngology Clinic with prominant bulbar symptoms (i.e. dysarthria, dysphonia, and dysphagia). Otolaryngologic assessment revealed the presence of tongue wasting and fasciculations; however, there was no evidence of other concomitant otolaryngologic assessment revealed the presence of tongue wasting and fasciculations; however, there was no evidence of other concomitant otolaryngologic disease to account for the patients' symptoms. Interestingly, all patients were able to document historically the progressive nature of their symptoms and, as well, over a short time frame (3 to 12 months) all noted an unexplained significant weight loss.

Upon subsequent neurologic clinical assessment, all displayed the classic feature of amyotrophic lateral sclerosis in that there was a mixture of bulbar signs (tongue wasting and fasciculations), upper motor neuron dysfunction (pathologic hyperreflexia, spasticity, extensor plantar responses), and lower motor neuron dysfunction (muscle weakness in association with wasting and fasciculations). As well, the initial failure to recongnize the nonbulbar neurologic symptoms and signs led to a significant delay in arriving at the final clinical diagnosis in each case.

DISCUSSION

Amyotrophic lateral sclerosis (ALS) is a subtype of motor neuron disease and is known to the French as Charcot's disease but, as well, is popularly known in North America as Lou Gehrig's disease. Motor neuron disease (MND) is really a group of progressive, noninflammatory, and ultimately fatal disorders of the central nervous system in which the motor neurons in the spinal cord, brain stem and, to a lesser extent the motor cortex, are affected.

The MNDs can be broken into several subtypes, depending on which area of the neuraxis is primarily affected. When the cortical motor neurons are involved, the predominant signs are those of upper motor neuron (UMN) dysfunction (weakness, spasticity, hyperreflexia, Babinski response), whereas when the motor neurons in the brain stem (cranial nerve motor nuclei) or spinal cord (anterior horn cell) are involved, lower motor neuron (LMN) dysfunction predominates (weakness, muscle wasting, fasciculations).

Classically four divisions of MND are described:

1. *Progressive Muscular Atrophy:* In this condition the anterior horn cells of the spinal cord are primarily affected and thus, clinically, lower motor neuron signs as described above predominate.
2. *Progressive Bulbar Palsy:* In this entity the motor nuclei of the brain stem degenerate and lead to lower motor neuron signs in the head and neck region. Classic features include facial muscle wasting in association with weakness and fasciculations but, most commonly, there is wasting and fasciculations in the tongue with subsequent dysarthria, dysphonia and dysphagia. Interestingly, the three ocular motor cranial nerve nuclei (CN 3, 4, and 6) are generally always spared; however, one case report in the recent medical literature has documented clinical and postmortem pathologic findings in keeping with involvement of the ocular motor nerve nuclei.[1]
3. *Primary Lateral Sclerosis:* The lesion here affects primarily the pyramidal tracts descending from the cortical upper motor neurons to the spinal lower motor neurons, thus producing essentially isolated upper motor neuron findings as outlined above.
4. *Amyotrophic Lateral Sclerosis (ALS):* This is the largest subgroup of MNDs in which there is involvement of the pyramidal tract along with the lower motor neurons in the brain stem and spinal cord. (See Figures 1 and 2.) This leads to a combination of upper and lower motor neuron findings, in the absence of sensory findings, which is the hall mark clinical picture of ALS. Bulbar dysfunction is frequently noted also. Approximately 75% of all patients affected with motor neuron disease will eventually develop this clinical picture.[2] A review of the aforementioned three case presentations indicates such a mixture of clinical findings indicating that all patients fell within this diagnostic category.

ALS is a sporadically occurring disease with a worldwide incidence of 1 or 2 per 100,000 and a prevalence of 5 per 100,000 thus accounting for 1 in every 1,000 deaths.[3] Interestingly, there are several locales with exceeding high incidence such as on Guam among the Chamorros, and the Kii Peninsula in Japan. Murray *et al.* found the incidence of ALS in Nova Scotia to be near 2 per 100,000.[4] In fact, Nova Scotia had one of the highest incidences of ALS in the world; however, this may have been artifactual due to the thoroughness with which the study population was reviewed. At this rate it would mean a Nova Scotian general practitioner would see one patient with ALS every 10 or 15 years. The male to female ratio is generally reported to be 2 to 1, a figure confirmed for Nova Scotia.[3,4] The mean age of onset has been found to be the mid-sixth decade and again this has been confirmed for Nova Scotia.[5] It is decidely rare to see motor neuron disease under the age of 35.

An Israeli study by Gubbay *et al.*[2] found that their series of patients with ALS presented with the following findings at diagnosis:

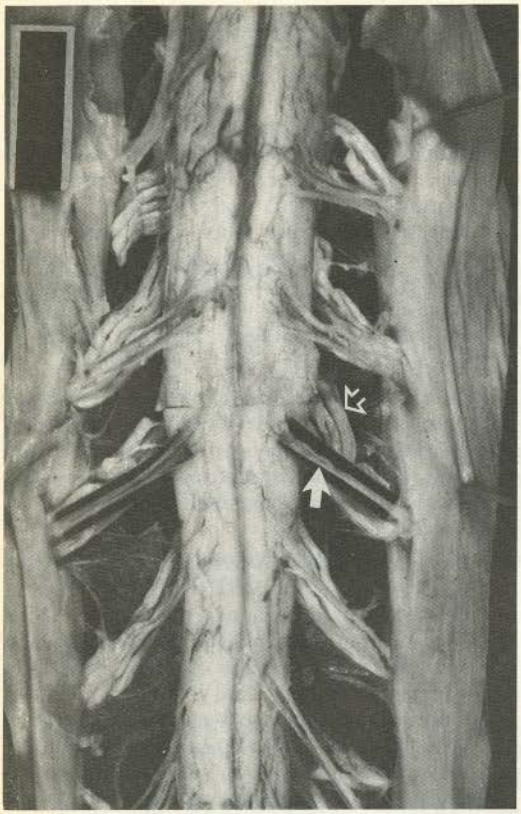

Fig. 1 Spinal cord specimen demonstrating ventral root atrophy in amyotrophic lateral sclerosis. The ventral (motor) root (closed arrow) is seen to be abnormally thin in comparison to the normally sized dorsal (sensory) root (open arrow).

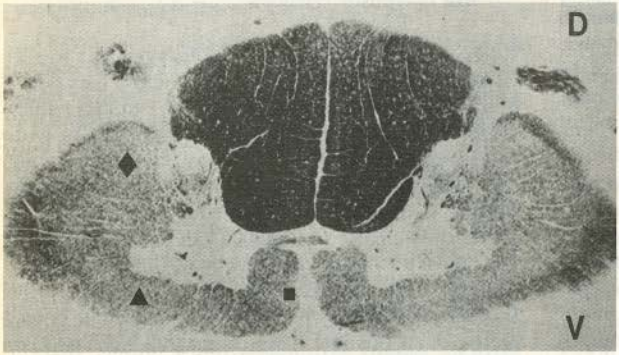

Fig. 2 Transverse section of spinal cord specimen demonstrating the pathologic features of amyotrophic lateral sclerosis. The specimen is prepared with Weigert stain which makes myelin appear dark. Note the atrophy of the descending motor tracts — lateral corticospinal tract (◆) and ventral corticospinal tract (■). As well there is ventral flattening of the cord due to the loss of the myelinated motor roots (▲) originating from the anterior horn cells.

(D = Dorsal, V = Ventral)

Weakness — 67%

Cranial Nerve Findings — 22%

Atrophy — 10%

Alone, these clinical findings are essentially nondiagnostic. However, if their presence arouses clinical suspicions, a more intensive physical assessment of the patient may reveal the presence of other neurologic signs which would enable one to find the clustering of features diagnostic of motor neuron disease. The subtlety, however, of some of these signs accounts for the difficulty in making an early diagnosis and thus accounts for the average 29 month delay before diagnosis in a recent British study.[6] Thus it is not surprising that in the cases which we have presented there was such a delay prior to confirmatory diagnosis.

Interestingly, Gubbay *et al.*[2] found an increase in the incidence of elderly women presenting with ALS and this was confirmed in the study by Murray *et al.*[4] for Nova Scotia as well. Again, this bears relevance to our recent experience at the Halifax Infirmary Hospital where for cases diagnosed by the Neurologic Consultative Service over the last nine months, all have involved elderly females.

ALS is an irreversible disease which has ramifications that are devastating for the patient and terrifying for both the patient and their family members. In the aforementioned British study by Newrick and Langton-Hewer[6], the following frequency of symptoms in a cohort of patients previously diagnosed with ALS was found:

Bulbar		
	Slurring	67%
	Choking	57%
	Drooling	50%
Limb		
	Fatigueability	50%
	Clumsy Hand	45%
	Severe falls	40%
Miscellaneous		
	Frustrations and Boredom	95%
	Pain	64%
	Weight Loss — Greater than 6 kg	43%

The frequency of these very distressing and incapacitating symptoms reflects the devastation caused by motor neuron disease. By the time of death 98% of patients with ALS had developed clinically significant atrophy and 95% had fasciculations.[6]

The diagnosis of ALS is clinical confirmed by typical electrodiagnostic (EMG) findings. The clinical hallmark is the combination of upper and lower motor neuron findings, frequently in the same extremities,

with a normal sensory examination. EMG evidence of a reduced number of motor units, along with evidence of acute and chronic denervation, is seen. In the differential diagnosis of ALS, one must rule out entities such as cervical cord disorders (syringomyelia, cord tumor, cervical spondylosis) and, as well but more rarely, inflammatory polyradiculoneuropathy, multiple sclerosis, thyrotoxicosis, and central nervous system syphilis may masquerade as ALS. Generally, these entities can be separated on the basis of clinical signs; however, investigative studies such as EMG and myelography may be required.

The pathogensis of ALS is still obscure; however, etiologic factors such as aging, heavy metal exposure, toxin exposure, and autoimmune mechanisms are among some of the proposed mechanisms. Worldwide, 5% of ALS is said to be inherited.[3,7] Murray *et al.* have demonstrated that in Nova Scotia 6% of cases of ALS are familial.[5]

Just as there have been many proposed causes of ALS so are there many proposed cures and indeed, in a recent review article, 24 different therapeutic modalities were documented as having been failures, including such diverse items as vitamin supplementation, neurotransmitter replacement (L-dopa), neurotoxins, and thyroid releasing hormone (TRH).[8] At this time, there is no proven therapy.

Once the diagnosis is confirmed, what then has the physician to offer the patient? Certainly the patient with ALS faces a very difficult time with many debilitating symptoms and the effort should be to give support, comfort and alleviation of the pain and complications of this disease. Suggestions follow as to the management approach to some of the possible complications seen in ALS:

1. *Choking, Drooling, and Weight Loss:* Anticholinergic medications can decrease the salivation and subsequent drooling and, should this fail, a transtympanic neurectomy has been shown to reduce secretions by 95%.[9] Special food preparations may help in the management of the dysphagia, and advice from a dietician may aid the patient in the management of this problem.
2. *Slurring and Communication Difficulties:* Speech therapy and finger-type communicators were found to be the most gratifying of any management employed by a physician in the management of ALS in a recent British review.[6]
3. *Limb Spasms and Cramps:* Phenytoin (Dilantin®) and Carbamazepine (Tegretol®) have been reported to be of benefit.
4. *Severe Falls:* A recent reveiw of ALS has noted that 40% of patients subsequently had severe falls which lead to significant injury.[6] The main reason for this seemed to be an effort to keep the patient independently mobile for the maximum amount of time. Earlier wheelchair placement may be indicated to prevent such sequelae even though it would mean earlier loss of some ambulatory independence.
5. *Insomnia:* This is often due to inability to turn in bed due to the attendant muscle weakness and so waterbeds and electric turning beds have been found to be of benefit.
6. *Frustration and Boredom:* This is almost universal among ALS patients and may be alleviated by organization of family and friends to offer support and entertainment.
7. *Mobility and Posture:* Physiotherapy and occupational therapy specialists can aid the patient greatly in these areas and should be consulted early in the patient's management.
8. *Constipation:* Due to the inactivity attendant upon the muscle weakness, this can become a very difficult problem and can be treated with a variety of intestinal motility agents including laxatives and enemas.
9. *Sexual Frustration:* Advice on positioning can be of great help to the patients.
10. *Pneumonia:* This requires medical therapy as in any patient; however, the patient with ALS may require earlier and more agressive chest physiotherapy due to muscle weakness complicating the expectoration of secretions. It must not be forgotten that respiratory failure and penumonia is the most frequent cause of death in patients with ALS.[9]
11. *Fear:* For the patient with ALS, there can be a real fear of the future given the complications which can arise and the imminent facing of death. The attending physician, by being supportive and empathetic to the patient, may help the patient through their ordeal. If hospice services are available in the patient's area, help from consultants affiliated with the service should be requested at an early juncture.

The average survival following the diagnosis of ALS is three years, and onset of bulbar signs is more ominous when the average survival drops to 2.2 years.[2] Overall however, 29% of patients have been reported to survive over 5 years and 16% have lived as long as 10 years after the diagnosis. Thus in some cases, the physician will be involved in very long term care of patients with this diagnosis. Most doctors suggest a team management approach to ALS involving physicians, therapists, family, friends, clergy and various support groups.

In Canada, there is a support association called the Amyotrophic Lateral Sclerosis Society of Canada (ALSSOC). This society has a Nova Scotia Chapter

which can be reached at 5 Beaumont Drive, Lower Sackville, Nova Scotia B4C 1V5; (902) 865-5346. The ALSSOC has been of great help to patients and their families as often patients find clues from their fellow sufferers in how to deal with certain problems including some aspects which physicians may not have even addressed.

In summary, ALS is a disease that the family physician can recognize, diagnose, and subsequently arrange the appropriate specific referrals. Knowledge of the disease and its sequelae can help in the management of these patients and their families. Although eventually leading to devastation of the motor system and a profound array of neurologic symptoms and signs, the initial manifestation of ALS, as exemplified by our patients, may be as subtle as the progressive bulbar symptoms of dysarthria and dysphagia. Thus the general physician should certainly extend his examination beyond the head and neck region when assessing patients with these complaints, to enable him to make an early diagnosis of this condition. Continued clinical suspicion of this disease will lead to earlier diagnosis and hopefully earlier referral and better management of patients with this entity. □

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References

1. Harvey DG, Torack RM, Rosenbaum HE. Amyotrophic lateral sclerosis with opthalmoplegia: A clinicopathologic study. *Arch Neurol* 1979; **36:** 615-617.
2. Gubbay SS, Kahana E, Zilber N, Cooper G, Pintou S, Leibowitz Y. Amyotrophic lateral sclerosis. A study of its presentation and prognosis. *J Neurol* 1985; **232:** 295-300.
3. Kurland LT, Mulder DW. Epidemiological investigations of amyotrophic lateral sclerosis. *Neurology* (Minneap.) 1955; **5:** 182-196.
4. Murray TJ, Cameron J, Heffernan LP *et al.* Amyotrophic lateral sclerosis in Nova Scotia. In: Cosi V, Kato AC, Parlette W, Penelli P, Poloni M, eds. *Amyotrophic Lateral Sclerosis.* New York: Plenum, 1987; 345-349.
5. Murray TJ, Pride S, Haley G. Motor neuron disease in Nova Scotia. *Can Med Assoc J* 1974; **110:** 814-818.
6. Newrick PG, Langton-Hewer R. Motor neuron disease: Can we do better? A study of 42 patients. *Brit Med J* 1984; **289:** 539-542.
7. Tandan R, Bradley WG. Amyotrophic lateral sclerosis. Part 2 — Etiopathogenesis. *Ann Neurol* 1985; **18:** 419-431.
8. Tandan R, Bradley WG. Amyotrophic lateral sclerosis. Part 1 — Clinical features, pathology, and ethical issues in management. *Ann Neurol* 1985; **10:** 271-280.
9. Campbell MJ. Management of patients with motor neuron disease. *Int Rehab Med* 1980; **2:** 111-115.

Non-Invasive Evaluation of the Vascular Patient:
THE HALIFAX INFIRMARY

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Non-invasive vascular technology has now become widely accepted as the first step in investigation for the management of patients with suspected peripheral vascular disease. During the late 1960s several types of plethysmographs were available to clinicians. Their use was limited mainly because, as an isolated test, they failed to provide enough information for further action. The introduction of A and B mode ultrasound a few years later allowed physicians to make more precise diagnoses. Imaging and Doppler effect modalities, oculoplethysmography, strain gauge and impedance plethysmography, and volume pulse recorder became standard tests in most large vascular centres. Currently, it is difficult to concieve a centre performing vascular surgery in which these techniques are not available.

The following sections are descriptions of the non-invasive peripheral vascular studies we conduct at the Halifax Infirmary which are standard and indispensable tests in the non-invasive field.

ARTERIAL DISEASE

Waveform Analysis

Ultrasonic and Doppler velocity detectors are as useful for a vascular surgeon as a stethoscope is for a cardiologist. Small hand held cylindrical probes are used to insonate the vessels with frequencies of either 5 Mhz or 8 Mhz. The returning signals are shifted upwards in frequency (Doppler shift) by arterial flow towards the probe or downwards by receding venous flow. The frequency shift of the transmitted Doppler signal is in proportion to the velocity of blood flow within the artery being studied. The simplest way to use the Doppler frequency shift is to amplify it through a loudspeaker or a headphone set. The audible Doppler signal may be interpreted by an experienced examiner who is able to distinguish normal signals from those that have been changed by disease processes. We record Doppler pulse velocity waveforms on a strip recorder for visual analysis. This transforms a subjective into an objective test.

On the strip recorder, normal arterial flow velocity waveforms have a triphasic pattern corresponding to the audible sounds. Under normal circumstances, the first major deflection represents forward flow during systole. The second deflection is caused by reversed low frequency flow during diastole, and the third waveform is indicative of normal flow proximal to the site of examination.

Using acoustic transmission gel, pulse recordings of the common femoral, popliteal and pedal arteries are made by angling the Doppler at 45° to 60° and pointed cephalad. The probe must be angled and moved to obtain the best signal. Distal to lesions or occlusions, the waveforms become monophasic with decreasing amplitude.

Segmental Blood Pressures

Non-invasive measurement of systolic limb blood pressure is an easily reproducible test and a sensitive indicator of arterial disease. This method uses pneumatic cuffs, which are applied around the extremity, and pressures may be measured anywhere the cuffs can be applied. After inflating the cuffs to sufficient pressure to stop distal blood flow, they are slowly deflated and some method is used to detect the cuff pressures at which flow into the distal part of the limb resumes. We employ an ultrasonic flow detection for this purpose. We record bilateral brachial, thigh, below knee and ankle pressures. Abnormal differences between the leg segments provide information about the number and site of occlusive lesions. Despite differences in cuff sizes and techniques used, most workers agree that a gradient of less than 20 mm Hg between two sites is normal. Gradients 20 to 30 mm Hg would be considered borderline and greater than 30 mm Hg abnormal.[1]

Wide thighs may produce artifactually high pressures and abnormal gradients may appear without the presence of a lesion. Calcification of vessels, often associated with diabetes, will produce abnormally high and inacurrate blood pressures — approximately 10 percent higher than normal. Differences of more than 15 to 20 mm Hg between pressures in the two lower limbs, measured at the same level, are also indicative of arterial disease.

Exercise Testing

As part of a non-invasive arterial examination, patients who are sufficiently ambulatory, are exercised on a treadmill. We have selected 1.5 and 2.25 MPH walking speeds, combined with a 10 percent slope, as our standards. This reproducible study is used to

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evaluate the functional impairment caused by disease. Treadmill exercise produces the symptoms and provides information such as onset time of pain, severity, location and walking distance. Post exercise ankle pressures and pedal pulse waveforms distinguish true claudication from neuro-spinal conditions. These results produce objective data regarding the significance of the disease, the degree of disability, and help determine the extent of collateral circulation. Exercise testing is a sensitive method of assessing disease progression or improvements and the effects of therapy.

VENOUS DISEASE

Non-Invasive Venous Assessment

During the past 10 to 15 years, several non-invasive techniques have been developed to accurately diagnose and document venous disease. These methods are safe and relatively inexpensive.

Doppler ultrasound is potentially the simplest and fastest method to evaluate venous disease, particularly deep venous thrombosis. A diagnosis is made from an experienced listener's interpretation of venous signals. The audible signals of venous flow are low pitched and normally vary with respiration. Blood flow through veins has been described as sounding like a phasic windstorm.

The examination technique most commonly used places the patient supine, with the head of the examing table or bed slightly elevated to permit pooling of blood in the leg veins. For examination of the popliteal veins, the patient is placed on his side or assumes a prone position with the feet elevated on a pillow. At the common femoral, popliteal and posterior tibial vein sites, we assess four qualities of venous signals.

When the foot is warm, the presence of an audible signal at all these sites, including the posterior tibial, is a normal quality referred to as *spontaneity*.

Phasicity refers to the waxing and waning of the velocity signal with respiration. A deep breath or Valsalva maneuver by the patient normally diminishes or interrupts the pattern by increasing intraabdominal pressure. In the presence of proximal venous obstruction, distal venous flow velocity becomes more continuous and less affected by respiration.

Augmentation of the velocity signals can be achieved by distal limb compression. In the presence of deep vein thrombosis (DVT) such velocity increases, are diminished or absent.

Compression of a limb segment proximal to a listening site prevents vein flow and obliterates the signal if the valves are competent. Reverse flow or reflux may be heard in the presence of valvular damage, possibly secondary to a previous DVT. Upon release of compression venous blood surges forward with an increased signal.

A fifth quality, *pulsatility*, may be heard in the presence of elevated venous pressure as in congestive heart disease. Compression or Valsalva maneuvers are routinely used to diminish these signals and distinguish them from arterial pulsations.

Plethysmographic methods can be used to add in the diagnoses of acute DVT. Of the various types available, we employ a strain-gauge plethysmograph (SPG). The SPG offers easy application and calibration, is reliable and provides a high degree of sensitivity and specificity. Basically, the test we apply includes measurement of calf volume expansion in response to a standardized venous congesting pressure and measurement of the rate at which blood flows out of the leg after pressure has been released.

For this study, the patient is positioned supine with calves elevated 35 cm and heels resting on a foam block or pillow. The knees are bent 30° to 45° to avoid pressure on the popliteal vein. To prevent an increase in central venous pressure the patients head rests only on a low pillow. The thighs may be supported with foam wedges. Nothing should impinge upon the calves but the strain-gauges which are double strands attached snugly around the calves with velcro tape. Large contoured cuffs are placed around each thigh and connected with tubing to the equipment.

Three conditioning cycles are run with alternate inflations and deflations, after which the measurement cycle(s) is carried out consisting of two minutes of cuff inflation before deflation. Computed values of venous capacitance (VC) are taken at the moment of cuff inflation and exactly two minutes later. Values of venous outflow (VO) are calculated from measurements taken at exactly one-half and two seconds after vacuum cuff deflation. These values are plotted on a discriminate line graph chart with VO as the ordinate and VC as the abscissa. The chart, divided into normal, equivocal, and abnormal ranges, is the result of a seven-hospital, 200-patient study (490 VO/VC measurements), which yielded 95 percent sensitivity and 99 percent specificity. Other studies have produced similar accuracy when compared with venography.

Whenever possible, we always use a combination of Doppler ultrasound and VO/VC testing in the diagnosis of DVT. Results from one test support the other and add confidence to the diagnosis.

Venous Reflux Testing

During the past decade, there has been increasing application of various non-invasive techniques for studying altered venous physiology in chronic venous insufficiency. We conduct a venous reflux test using a photoplethysmograph (PPG). This equipment uses sensitive photoelectric transducers that detect changes in blood content of the skin on the basis of varying amounts of light reflected from red blood cells. The PPG is connected to a dual-channel strip recorder and

photocells emitting infrared light are placed approximately 5 cm above the medial malleoli using double-faced transparent tape. Stable baselines are obtained by mechanically zero-setting the PPG.

In a sitting position on the edge of an examining table, the patient is asked to contract calf muscles by dorsi and plantar flexion of the feet, five or more times, to empty the calf of venous blood. Manual compressions of the calf are sometimes required to achieve sufficient emptying. Insufficient emptying (i.e. relying on 5 contractions only) may produce false positive results. Refilling times are measured in seconds, as the tracings return to baseline or plateau below baseline. Results below 23 seconds (the dividing line) are considered abnormal and the test is repeated with thigh cuffs inflated to 50 mm Hg to occlude the

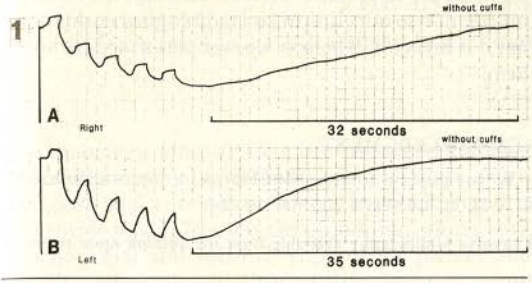

Fig. 1 SPG (strain-gauge plethysmograph)
Normal test compatible with normal venous circulation in deep and superficial systems.

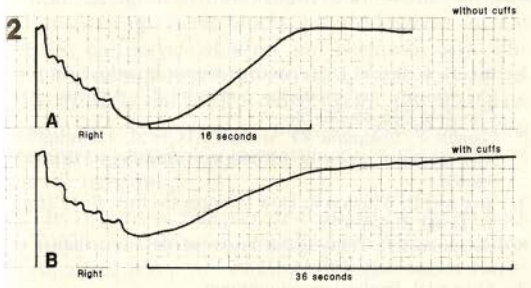

Fig. 2 SPG — Note difference in venous filling time without cuffs as compared with normal time with cuffs. Abnormal test — superficial venous insufficiency.

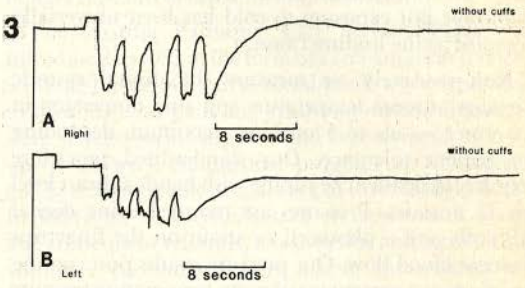

Fig. 3 SPG — Abnormally accelerated venous filling times with and without cuffs indicative of deep venous valvular insufficiency.

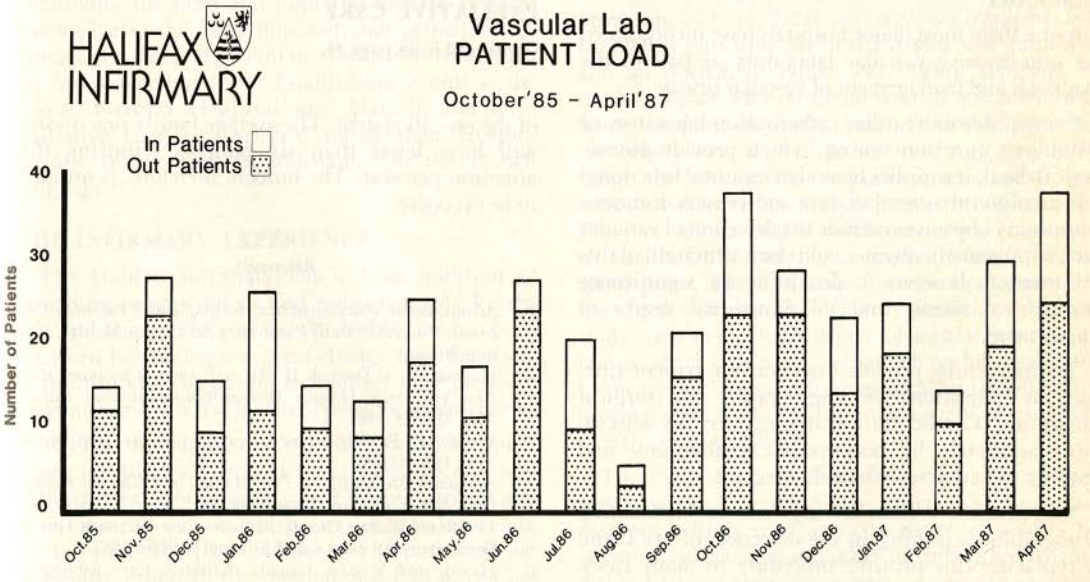

Fig. 4 Patient load at the vascular laboratory H.I. Note the upward trend in the number of tests performed. Decrease of number of tests during the summer months is due to overall decreased activity (i.e. bed closure, etc.) Number of patients does not represent the number of total tests since some patients require up to three or four evaluations during a single session.

superficial venous system. Abnormal refilling times recorded with the cuff inflated implicate the deep venous system as being incompetent. Normal results with cuffs indicate superficial insufficiency only. These tests are a 'must' for those surgeons performing reconstructive venous procedures.

Vasospastic Evaluations

The constriction and/or closure of the digital circulation resulting from emotional stress or from exposure to cold was first described by Raynaud in 1862. This disorder is divided into two types. Primary Raynaud's phenomenon is the closure of arteries that are otherwise normal. Secondary Raynaud's phenomenon results from occlusive disease in palmar or digital arteries that are constricting normally, or from impairment due to high blood viscosity. Many factors have been implicated in triggering Raynaud's syndrome but exposure to cold has been universally accepted as the leading cause.

Non-invasively we measure the digital systolic pressure at room temperature and after immersion in ice water — up to 5 minutes maximum depending on patient tolerance. Our standardized procedure requires the patient be supine with hands at heart level for 15 minutes. Pressures are recorded using digital BP cuffs and a photocell or strain on the fingertips to sense blood flow. Our pressure results post cooling are given as a percentage of room temperature pressure and interpreted as follows: Normal — 100% to 70%; cold hands but not Raynaud's — 69% to 25%; Raynaud's phenomenon — near 0% (25% to 0%).

SUMMARY

In the 1980s most major hospitals have incorporated the non-invasive vascular laboratory as part of the diagnosis and management of vascular disease.

Comparable to a cardiac catherization laboratory or pulmonary function testing (which provide physiological data), it supplies hemodynamic and functional information on arterial disease and venous disorders. Its primary objectives include the detection of vascular disease presence or absence, and documentation of this information. It serves to determine the significance and site of disease and the functional degree of impairment.

Testing results provide direction for patient care, such as indications for angiography and surgical procedures. Candidates for angiography are selected more accurately by non-invasive evaluations, and angiograms are reserved until necessary.

Non-invasive venous testing aids in eliminating venographic screening in the detection of DVT and is replacing this invasive procedure in many cases.

The follow-up of surgical and medical therapy is facilitated by non-invasive procedures which document flow improvement or deterioration. These easily applied diagnostic methods are ideal for research applications of medical or operative therapy.

The above mentioned studies have added so much information to our clinical and radiological evaluation that it is difficult to believe we ever performed without them.

□

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Extensive bibliography available from the authors upon request.

TRANSJUGULAR LIVER BIOPSY

References continued from page 12.

5. Bataille C, Bercoff E. La ponction biopsie hepatique par voie transveineuse: une experience portantsur 213 examens. *Acta Clin Belg* 1983; **38:** 222-227.
6. Gamble P, Colapinto RF, stronell RD, et al. Transjugular liver biopsy: a review of 461 biopsies. *Radiology* 1985; **157:** 589-592.
7. Westerby D, Williams R. How to biopsy the liver. *Brit J Hosp Med* 1980; **23:** 527-529.
8. Colapinto RF. Transjugular biopsy of the liver: *Clinics in Gastroenterology* 1985; **14:** 451-467
9. Allison DJ. Personal communication
10. Scheuer PJ. *Liver Biopsy Interpretation*, Second Edition; 1973: Williams and Wilkins, Baltimore.

PALLIATIVE CARE

Continued from page 31.

of the on-call system. The average family practitioner will have fewer than six patients requiring this attention per year. The burden, therefore, is unlikely to be excessive.

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References

1. Adams, Brian. *Assessment of a Hospital-Based Palliative Care Service From the Family Physician's Perspective.* Halifax, 1987; unpublished.
2. Hannan EL, O'Donnell JF. An evaluation of hospices in the New York State Hospice Demonstration Program. *Inquiry XXI,* 338-348, 1984.
3. Webster R. Palliative care for cancer patients. *Med Austral* 1985; **143:** 547-550.
4. Ajemian I, Mount BM. *R.V.H. Manual on Palliative/Hospice Care.* New York: Arno Press, 1980.
5. Health and Welfare Canada, *Palliative Care in Canada.* Ottawa: Development of National Health and Welfare, 1982.
6. Health and Welfare Canada *Palliative Care Services in Hospitals.* Ottawa: Health Services Directorate, 1981.
7. Nova Scotia Department of Health: *Vital Statistics.*
8. Provincial Cancer Registry of Nova Scotia, *Annual Report,* Halifax Cancer Treatment and Research Foundation, 1985.

Palliative Care:
THE INFIRMARY EXPERIENCE

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Palliative or Hospice Care involves active compassionate therapy for patients with terminal illness when diagnosis and cure are no longer issues. The aim is to provide a pain-free but alert condition, along with psycho-social and spiritual support for patient and family, when the quality of life becomes more important than the duration. Satisfactory pain control can be provided in more than 90% of patients with carefully adjusted, individualized programs utilizing simple analgesics, intermediate and strong opioids, adjuvant drugs, nerve blocks, physical agents such as TENS, palliative radiation and chemotherapy. The aim is to eliminate pain and to erase its memory, rather than treat it on a PRN basis. With physical pain eliminated, other issues may be addressed, often leading to remarkable improvement in the quality of the life remaining.

The origins of Hospice or Palliative Care go back to antiquity. During the middle ages, religious orders maintained rest centres along the main travel routes throughout Europe. Therein travellers were given shelter and nourishment and care was provided for the sick and for the dying. The word Hospice in its current medical connotation emerged in the 1960s and Dr. Cicely Saunders of London deserves credit for identifying the need and applying modern medical knowledge to the basic humanitarian principles. In Canada, Dr. Balfour Mount of Montreal is recognized for his pioneering work. Establishing a unit at the Royal Victoria Hospital and McGill, the term Palliative Care was selected rather than Hospice because of accuracy in translation to the French language.

THE INFIRMARY EXPERIENCE

The Halifax Infirmary has a long tradition of providing compassionate and competent care for the sick and dying. The way in which this care has been delivered has undergone great change over the years, as medical knowledge and technology have provided new and more effective means of meeting patient needs.

In 1979, the Standards Committee of the Hospital struck an Ad Hoc Committee to review the type and appropriateness of hospital care for dying patients and to recommend ways in which care could be improved. At that time, Dr. J.E.H. Miller, the Deputy Minister of Health, proposed the creation of a Palliative Care Unit (PCU) at the Infirmary. In May 1980, the Committee presented its findings and recommended the establishment of a Palliative Care Service. A subcommittee was established to define more precisely the nature of this service. In December 1980, the subcommittee recommended the formation of consultation and home care services and the creation of a 12-bed inpatient unit. Although this concept was favourably received by the Department of Health, budget restraint delayed its implementation.

The existing Palliative Care Service (PCS) was introduced in 1982 in the form of a consultation service, with the hiring of a nurse-coordinator who responded to referrals with pain and symptom control information, psycho-social support and bereavement follow-up. A ten-week educational program for volunteers and health care professionals began three months later. In 1983, due to increased staff recognition of needs and consequent referrals, a counsellor and a part-time volunteer coordinator were added, introducing an inter-disciplinary approach toward meeting those needs.

In 1984, a home support nurse was funded by the Department of Health, enabling patients to remain at home for longer periods of time. An on-call service providing 24-hour, 7-day coverage was implemented. In 1986, a part-time Medical Director was employed and an outpatient clinic was begun, resulting in improved pain and symptom control and additional home support. Recently, two additional part-time physicians have joined the service. From the beginning, the service has responded to clinical and educational referrals and requests throughout the Province. A great deal of collaboration with community health, VON and other community resources has taken place. The Palliative Care Service appears to have been well accepted by family physicians. A recent study indicated a high degree of satisfaction among family physicians whose patients have been cared for by the service.[1] Support from medical and allied health hospital staff is evident from the variety and number of referrals to the serice.

The Palliative Care Service has evolved to provide better patient care for dying patients, by providing services which complement those provided by attending physicians and other hospital departments. Table I summarizes the volume of services provided for the period 1983 to 1987. Tables II and III indicate that the percentage of older people in Nova Scotia will continue to rise, placing considerable additional

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strain on health care resources, particularly hospital beds. A breakdown of the age distribution of current referrals to the Palliative Care Service is shown in Table IV. Seventy-one percent of these referrals are patients over the age of 60. In view of projected demographic changes, it is anticipated that the demand for Palliative Care Service will increase. Evidence of the increasing demand for health care resources is suggested in Tables V and VI which indicate increases in the incidence of Cancer in Nova Scotia.

TABLE I

VOLUME OF SERVICE — PCS, 1983-1987

	1983-4	1984-5	1985-6	1986-7
Referrals	148	170	249	228
Monthly Average	12	14	21	19
In-Pt. Days	3,619	3,907	5,078	5,311
Daily Average	10	11	14	15
Home-Metro (40 mi.) Days	5,205	6,441	6,481	10,097
Daily Average	14	18	18	28
Long Distance Days	7,320	6,205	8,052	13,803
Daily Average	20	17	22	38
Home Visits Staff	17	79	319	225*
Volunteer Hours	819	535	249	726
Deaths	106	142	175	202
Staff (FTEs)	1.5-2.5	2.5-3.5	3.5-3.8	4.1

* A decrease in the number of home visits has resulted from more effective use of phone consultations and the introduction of the Palliative Care Clinic.

TABLE II

PROJECTED POPULATION BY AGE, 1981-2001

Year	% of Total Population 65+ Canada	% of Total Population 65+ Nova Scotia	% of Total Population 75+ Canada	% of Total Population 75+ Nova Scotia
1981	9.7	10.9	3.6	4.1
1986	10.0	11.2	3.9	4.4
1991	10.7	11.6	4.3	4.9
1996	11.0	11.5	4.6	5.2
2001	11.2	11.4	5.0	5.4

Source: Statistics Canada

TABLE III

PROJECTED POPULATION BY AGE, 1981-2001

Year	Age 75+ As a Percentage of Age 65+ Canada	Nova Scotia
1981	37.4	37.6
1986	38.8	39.2
1991	40.1	45.5
1996	41.4	45.4
2001	44.4	47.1

Source: Statistics Canada

From January to December 1986, there were 17,554 inpatient days at the Halifax Infirmary devoted to patients whose primary diagnosis was malignant neoplasms, accounting for 15.9% of all inpatient days. The average length of stay for these patients was 14.9

TABLE IV

AGE DISTRIBUTION OF REFERRALS TO PCS
April, 1986 TO March 31, 1987

Age Group	Number	(rounded) Percent
Teens	2	1
20 -	0	0
30 -	8	4
40 -	15	8
50 -	36	16
60 -	81	37
70 -	57	26
80 -	17	8
90 -	0	0
TOTAL	216	100%

TABLE V

INCIDENCE OF CANCER WITHIN AGE GROUPS, PER 1,000 POPULATION (FEMALES)

Age Group	1984 Actual No. of Cases	1984 Incidence Per 1000	1985 Actual No. of Cases	1985 Incidence Per 1000
0-9	9	.1	7	.1
10-19	12	.2	5	.1
20-29	145	1.8	165	2.1
30-39	198	3.0	188	2.8
40-49	180	4.0	202	4.4
50-59	308	7.8	366	9.3
60-69	472	12.5	505	13.3
70-79	478	19.0	500	19.2
80-89	230	21.8	250	23.4
90+	49	106.3	47	89.9
Unavailable	17		23	
TOTAL	2,098	4.8	2,258	5.2

Rate of increase 1984-5 = 8.3%

Source: Provincial Cancer Registry of Nova Scotia

TABLE VI

INCIDENCE OF CANCER WITHIN AGE GROUPS, PER 1,000 POPULATION (MALES)

Age Group	1984 Actual No. of Cases	1984 Incidence Per 1000	1985 Actual No. of Cases	1985 Incidence Per 1000
0-9	8	.1	3	.0
10-19	14	.2	7	.1
20-29	30	.4	35	.4
30-39	43	.7	63	.9
40-49	119	2.6	129	2.8
50-59	349	9.3	366	9.8
60-69	675	20.5	661	20.1
70-79	664	32.9	694	34.0
80-89	234	41.9	252	43.9
90+	32	191.6	43	286.7
Unavailable	13		29	
TOTAL	2,181	5.1	2,282	5.4

Rate of increase 1984-5 = 5.9%

Source: Provincial Cancer Registry of Nova Scotia

days, significantly higher than for all patients. These data support our belief that the provision of coordinated home support services and the establishment of hospital units designed to provide appropriate care for dying patients, are necessary to meet these health care needs. Active treatment wards, oriented to prompt and efficient investigation and treatment are inappropriate for the care of patients for whom diagnosis and cure are no longer issues. This often results in inefficient and inappropriate utilization of resources, including the following:

1. Inappropriate admissions and admission procedures;

2. Less than satisfactory control of pain and management of other symptoms such as nausea, vomiting, and dypsnoea;

3. Inappropriate diagnostic tests and other procedures, excessive use of intravenous solutions and nasogastric suction;

4. Inadequate response to the psycho-social and spiritual needs of patients and their families resulting in reduced quality of life and increased morbidity among survivors;

5. Inadequate discharge planning and home support, resulting in poor and uncoordinated home care and unnecessary readmission; and

6. Lack of appropriate teaching facilities where health professionals, including medical students, can increase their knowledge regarding pain and symptom control, and the appropriate use of resources to meet the needs of patients and their families.

The potential for Palliative Care Units to meet patient needs more effectively is well recognized. In Britain, the Hospice movement is credited for the reduction in the demand for acute care beds and with significant improvement in the quality of life of a patient facing death. In Edinburgh, the average length of stay at St. Columbia's Hospice is 11 days and one-third of patients are discharged to their homes. In the United States, a 1984 study of 12 New York Hospice programs, including hospital-based autonomous unit programs, reported that these programs were less costly than those providing conventional terminal care and provided high levels of patient satisfaction.[2] In Australia, the establishment of regionalized hospital-based Palliative Care Units with Medical Directors has been recommended as a partial solution to high health care costs.[3] Evidence from the Royal Victoria Hospital indicates that the unit has resulted in a significant shortening of terminal care admissions. Increased costs resulting from the higher nurse-patient ratio have been more than offset by savings accrued through the curtailment of irrelevant investigations.[4]

At the Halifax Infirmary, the impact of the existing Palliative Care Service on resource utilization is readily evident. A study of 53 cancer patients receiving home support services during July 1985, to June 1986, showed that these patients were able to spend 83% of their time at home. The average length of hospital stay for patients receiving consultation from the PCS during this period was 15 days, compared with 17 days for a control group of 73 terminally ill cancer patients. The length of stay fell to 12 days after the addition of the Medical Director, presumably due to better pain/symptom management. It is anticipated that the length of stay could be further reduced to approximately 10 to 11 days with the creation of a Palliative Care Unit where more complete control over patient care would be exerted.

It is important to recognize that Palliative Care Services and Units are not simply places for terminally ill patients to die. Care is based on sound medical principles. Management of pain and other symptoms is emphasized to enable patients to return to their homes and remain at home for as long as possible with home support services. When patients are discharged to home, readmission is assured if required. With this reassurance, patients and families are more likely to cope effectively in the home. Readmission is arranged as necessary if pain and symptom control require medication adjustment in hospital, when the family requires a respite and when the patient and family prefer to have death occur in the hospital.

DISCUSSION

The pioneers in Palliative Care have brought new expertise in the area of therapeutics, pain control and nursing techniques, along with a philosophy of care based on a more spiritual plane, but the primary care of the patient with terminal illness should remain the responsibility of the family physician. The Palliative Care Service should, therefore, be regarded as consultative and advisory. Team work, co-operation and communication are essential.

Discharge planning must be meticulous and advice concerning drug administration explicit and clearly understood by patient and/or family. Continuing care by the family physician must be equally attentive and availability assured. "Sign-out" to a colleague, who is unfamiliar with the patient and who does not have access to the clinical record, frequently leads to confusion and distress.

The Palliative Care service, providing 24 hour availability, is prepared to respond to inquiries and offer assistance on request to patients and family physicians at any time. To ensure continuity of care however, it is suggested that the family physician should arrange accessibility to these patients outside

Continued on page 28.

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The Role of *Blastocystis hominis* in Enteric Disease

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Blastocystis hominis has been generally ignored, despite early suggestions it was a cause of diarrhea. There has been renewed interest in this organism as an enteric pathogen, and we now report it when present in high numbers in stool specimens. As many physicians may be unfamiliar with *Blastocystis hominis*, this review will discuss the role of the organism in disease.

For many years after its original description by Brumpt in 1912, *B. hominis* was considered a fungus. More recently, however, electromicroscopic investigation has shown the organism to be protozoan. This change in classification is based on structural and physiological characteristics. Evidence against fungal classification includes failure to form mycelia, absence of a cell wall or budding either *in vivo* or *in vitro*, and no growth on fungal media. Evidence for protozoan affiliation include pseudopod formation, ingestion of particulate matter, requirement for bacteria for *in vivo* growth, optimal growth at 37°C in neutral or alkaline conditions, and reproduction by binary fission or sporulation.[1] In addition, the subcellular organelles are more characteristic of protozoan than yeast morphology.[1,2] Difficulties in classification have arisen as a result of the variable morphology of the parasite.

CLINICAL SIGNIFICANCE

B. Hominis is frequently seen in stool specimens in low numbers (less than 5 per high power field). Zierdt identified it in 18% of stool specimens.[1] In these numbers, the organism is commensal and there is no evidence for a pathogenic role. It is suggested the organism may be significant when present in high numbers (5/hpf).[1] Ninety-one of 2,232 specimens (4.1%) received at the Victoria General Hospital Parasitology section in 1987 contained high numbers. There is, however, geographic variation; Casemore *et al.* reported only one of 2,000 stools contained high numbers.[3]

Evidence for pathogenicity is derived from both animal and human reports. Phillips and Zierdt inoculated gnotobiotic guinea pigs orally or intracaecally with *B. hominis* in axenic or monoxenic culture or with enteric flora. Only those animals inoculated with enteric flora and *B. hominis* developed infection, which correlated with high numbers of the organism on stool microscopy. Histologically, there was epithelial cell invasion only, with little inflammatory response. A control group inoculated only with enteric flora was not possible, because of difficulties in selectively removing *B. hominis* from stool but no infections occurred in the absence of large numbers of *B.* hominis.[4]

The failure of monoxenic or axenic cultures to initiate disease is in keeping with protozoan requirements for a complex bacterial flora *in vivo*. McClure *et al.* reported an infection in a pig tailed Macaque.[5] Diarrhea was unresponsive to trimethoprim-sulfamethoxazole or chloramphenicol but resolved rapidly with diiodohydroxyquin. *B. hominis* was identified on initial stool examination, but was seen rarely following treatment. Although very low numbers of *Balantidium coli* and *Yersinia enterocolitica* were detected, the prompt clinical and microscopic response to antiprotozoal therapy supported the authors' suggestion *B. hominis* was the causative agent.

The human evidence for pathogenicity remains incomplete. Case reports suggest *B. hominis* is an enteric pathogen, but no extensive studies are readily available. Gallagher *et al.* reported two patients who were febrile with abdominal pain, nausea, vomiting, and diarrhea.[6] Extensive investigation excluded virological, bacteriological or other parasitological causes, and the patients responded to treatment with metronidazole. Follow up examinations showed the stool was cleared of *B. hominis*. Lebar *et al.* reported a similar case, who had suffered a week of diarrhea with cramping.[7] An extensive search for enteric pathogens revealed only "numerous" *B. hominis*. Symptoms resolved following a seven day course of metronidazole, and *B. hominis* was not detected in follow up stool specimens.

Other case reports similarly report symptomatic patients with large numbers of *B. hominis* seen on stool examination, with both clinical and microbiological resolution following metronidazole.[8,9] The extent of investigation for other enteric pathogens is not described, however, and a causative association cannot be demonstrated. Although most reported cases have been mild, Zierdt and Tan reported a severe cholera like illness which lasted three months and did not respond to metronidazole.[10] The patient died of aspiration pneumonia. In a retrospective chart review, Garcia *et al.* reported diarrhea, and abdominal pain

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to be the most common symptoms, with cramps, nausea, fever, and vomiting less commonly seen.[11]

The evidence for pathogenicity is incomplete. An association is demonstrated between diarrhea and high numbers of *B. hominis* in stool. Response to appropriate antimicrobials suggests there may be a causal relationship in the continuation, if not the initiation, of symptoms. It is not clear if the organism is transmitted, with subsequent proliferation, or if overgrowth of endogenous organisms occurs after a disruption of the normal flora. Serological evidence would be useful to distinguish between infection and secondary overgrowth, but it is not available. More extensive work is required to establish the importance of this organism in the causation of enteric disease.

IDENTIFICATION

B. hominis is identified in stool specimens for ova and parasites. In our laboratory we use formalin ether concentration with iron hematoxylin staining. Other stains used include Giemsa and trichrome.[6 9 11] Although the organism may be recognized on wet preparation, or when stained with iodine, confusion with other intestinal protozoa may occur. Three morphological forms have been identified. The most commonly seen in clinical specimens is the spherical or cyst form, the amoeboid form is seen infrequently and the granular form is seen rarely.

Although culture methods have been described for the organism, these are not available for diagnostic use, and the significance of a positive culture would be uncertain. Like other intestinal protozoa, microscopic examination of stained slides is the method of choice for diagnosis.

TREATMENT

Blastocystis hominis responds to many of the same drugs used for other enteric protozoal infections. Metronidazole (adult dosage 750 mg tid x 10 days) or iodoquinol (adult dose 650 mg tid x 20 days) have been recommended.[12] Both have been found effective *in vitro*, and metronidazole has appeared to be effective in many of the case reports.[6 7 8 9] Other drugs that have been found effective *in vitro* include emetine, furazolidone, trimethoprim-sulfamethoxazole, pentamidine, and chloroquine.[1] These drugs action *in vivo*, however, has not been assessed and none of these could be considered as therapy of choice.

Therapy is generally reserved for symptomatic cases where other infective causes of diarrhea have been excluded.[8] This can be achieved by submitting stool for culture and at least three stool specimens for parasitological examination. If symptoms persist, a virological cause is unlikely. Treatment to eradicate asymptomatic carriage is not recommended.

□

References

1. Zierdt CH. *Blastocystis hominis*, a Protozoan Parasite and Intestinal Pathogen of Human Beings *Clin Mircobiol Newsletter*, 1983; **5:** (9 May 1) 57-59.
2. Zierdt CH, Rude WS, Bull BS. Protozoan Characteristics of *Blastocystis hominis*. *Am J Clin Pathol* 1967; **48:** 495-501.
3. Casemore DP, Armstrong M, Jackson FB. Clinical Relevance of *Blastocystis hominis*. *Lancet*, 1984; **1:** 966.
4. Phillips BP, Zierdt Ch. *Blastocystis hominis*. Pathogenic Potential in Human Patients and in Gnotobiotes. *Exp Parasitol* 1976; **39:** 358-364.
5. McClure HM, Strobert EA, Healy GR. *Blastocystis hominis* in a Pig Tailed Macaque: A Potential Enteric Pathogen for Nonhuman Primates, *Lab Anim Sci* 1980; **30:** 890-894.
6. Gallagher PG, Venglarcik JS. *Blastocystis hominis* Enteritis, *Pediatr Infect Dis* 1986; **4:** 556-7.
7. Lebar WD, Larsen EC, Patel K. Afebrile Diarrhea and *Blastocystis hominis*. *Ann Intern Med* 1985; **103:** 306.
8. Vannatta JB. Adamson D, Mullican K. *Blastocystis hominis* Infection Presenting as Recurrent Diarrhea, *Ann Intern Med* 1985; **102:** 495-496.
9. Ricci N, Toma P, Furlani M, Caselli M, Gullini S. *Blastocystis hominis:* a neglected cause of diarrhea? *Lancet*, 1984; **1:** 966.
10. Zierdt Ch, Tan KH. Ultrastructure and Light Microscopic Appearance of *Blastocystis hominis* in a Patient with Enteric Disease. *Z Parasitenkd* 1976; **50:** 277-283.
11. Garcia LS, Bruckner DA, Clancy MN. Clinical Relevance of *Blastocystis hominis*.[1] *Lancet*, 1984; **1:** 1233-4.
12. Drugs for Parasitic Infection, *Med Lett Drugs Ther 1986;* **28:** 706.

Current Topics in Community Health

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SUICIDE IN CANADA
Report of a Task Force

There are many unanswered questions about suicide and a multitude of conflicting theories. The role of environmental influences and mental disorder, the existence and nature of predisposing genetic or biochemical factors, and the parallel issues of proper and productive treatment and prevention — the questions are complex. Suicide is an action; it is not an illness. Identifying the chain of causal and triggering factors — which may in any case be highly individual — and deriving from this an overall prevention and treatment strategy is perhaps one of the most vexing problems facing professionals in the health sciences.

Given the breadth of research in this area, a comprehensive review of the state of current knowledge was necessary and was undertaken by the Task Force on suicide in Canada commissioned by Health and Welfare Canada. It was asked to report on the nature and extent of suicide and suicide-related problems, to discuss demographic and sociological parameters, and to identify Canadian groups at greatest risk. It was also asked to summarize our knowledge of etiological processes and to gather information on programs of suicide prevention, intervention and postvention.

The Task Force makes a series of recommendations. The scope and nature of the recommendations reflect the expertise of the Task Force members. The mental health priorities in any jurisdiction will determine the saliency and urgency of the recommendation. Suicide, however tragic, is a low frequency event having a more limited societal impact than other pressing mental health issues such as the care and treatment of the chronically mentally ill, the mental health needs of victims of violence including battered women and abused or neglected children, and the mental health problems associated with aging. In addition, not all the recommendations will apply across Canada. Some may be incorporated already in provincial and local mental health services, or the intent may have been met with service arrangements not anticipated by the Task Force.

Introduction

In the introduction the breadth of the problem is immediately realized. "Suicides do not form, as may be thought, a wholly distinct group." Behaviours which fall under this rubric include: chronic substance abuse, hyperobesity, habitual high-risk-taking behaviour, the willful self-neglect of the elderly and non-compliance with the treatment of serious physical illness. The degree to which such behaviours share a common basis with suicide in cause and treatment is a matter of debate. Nevertheless, it is clear that a recognition of the self-destructive and suicidal aspects of these behaviours is crucial if these disorders are to be effectively diagnosed and treated and, even more importantly, prevented.

In 1983, the rate of suicide per 100,000 Canadians was 15.1 and a recent study has calculated that between 1963 and 1976, a total of more than 2,000,000 years of life were prematurely lost to suicide in Canada (Peters and Termansen, 1982). What makes these figures even more alarming is the probability that under-reporting results in a significant underestimation of the true magnitude of the suicide phenomenon.

Epidemiology

Epidemiological data and comparisons are presented in several tables and figures. Suicide rates in Europe are compared with those in Canada. Overall annual rates per 100,000 per annum ranged from a low of 2.9 in Greece to a high of 44.9 in Hungary, with a median of 10.0. Canada's rate of 15.1 per 100,000 in 1983 is in the upper half of countries listed. Moreover, Canada's rate now consistently exceeds that of the USA. This section of the report details differences for different age levels in Canada, differences between males and females, and interprovincial differences. For example: "During 1969-73, a striking interprovincial difference was apparent: a gradient of steady increase in male suicide from the east to the west coast." Suicide has also increased nationally since World War II, particularly in males, and evidence from Alberta suggests that the rising incidence is cumulative. Within age groups national figures pin-point the increases among young males aged 20 to 29, and among those aged 10 to 19. This section of the report also shows a correlation of suicide with many other factors: lack of strong family and social networks, economic declines and unemployment.

Etiology

Some thirty pages of the report review and summarize current literature on many causal factors. Knowledge concerning cause comes from many professional disciplines and perspectives.

Studies of common personal characteristics and traits are reported, as are studies of family relationships and structure. There are job-related factors as well as findings in terms of social disorganization. Physical illness can be a factor. Of major importance in many suicides is mental disorder. Drug and alcohol abuse are also common factors. Findings concerning the role of stress are discussed, as well as such biological factors as seasonal variation, the menstrual cycle and socio-biochemical and genetic determinants. Some psycho-analytic contributions are briefly noted. The necessity for a 'multi-dimensional approach' in understanding the causal chain is clearly illustrated and this section ends with discussion of that approach.

Identification of "High-risk" Populations

This chapter overviews Canadian data and literature revealing that several groups are at 'high risk'. Problems such as under-reporting and the effect of certain laws and common attitudes are illustrated. Contributing factors are discussed. The 'high-risk' populations identified are:

- those suffering from certain mental disorders
- alcoholics
- young people
- the elderly
- native peoples
- persons in custody
- the bereaved

Prevention, Intervention and Postvention

These three headings are used to discuss strategies and programs for the reduction and prevention of suicide. Prevention deals with measures which might reduce the prevalence or probability of suicidal behaviour. Areas discussed include: public education (through the media and other means), reduction in the availability and lethality of means, and specific education and training programs for health care professionals and gate-keepers.

The category of intervention includes sets of procedures to be used in managing suicidal crises. This involves the development and utilization of specialized techniques in assessment and counselling, and in treatment.

The section on postvention delineates measures to be taken following a suicide. The objective here is twofold: to provide follow-up support and counselling services for the bereaved and to carry out psychological autopsies of the victims of suicide to obtain information on pre-suicidal states and activities. The latter process is a valuable contribution to the understanding of the event for the family, as well as health care workers and researchers.

Prevention

The media have a central role in shaping public attitudes and in public education. There will be a continuing need for mental health professionals knowledgeable about suicide to consult with media representatives in an attempt to mitigate the negative effects of media coverage of suicides. Most members of the media are genuinely concerned about how best to balance the responsibility to keep the public informed and the danger of 'contagion', i.e., describing ways and means to potential suicide victims. One recent report indicated that the decision of Canadian newspaper editors to publicize a suicide is governed by the following criteria: occurrence in a public place; prominence of the victim; effect on other people; and/or the unusual nature of the method involved.

There have long been public education programs aimed at reducing the stigma attached to seeking treatment for states of depression. Education programs could also aim at such things as discouraging the accumulation of lethal amounts of drugs in household medicine cabinets.

The lethality and availability of instruments of suicide is another issue discussed in the context of public education (for example, more stringent control of the distribution of medications, and wherever possible, limitations on the accessibility of "attractive hazards").

In discussing the education of professionals and gate-keepers, the report notes that surveys have revealed a limited level of education about suicide across all disciplines, including undergraduate medicine.

In the Report's discussion of professional education, particular attention is given to physicians, clergy, teachers, and personnel of correctional facilities, police and others, as well as the mental health professions. Examples of several approaches are given. Recommendations are made.

Intervention

Very few Canadian suicide prevention/intervention programs have been carefully evaluated. Here the Task Force found few hard research data but considerable informed opinion and many descriptions of programs. The Appendix gives details of several excellent hospital- or community-based programs.

A general hospital is perhaps the best facility for treatment of self-injuries because it can provide medical and surgical services, as well as the psychiatric skills needed to conduct a thorough assessment and management of the patient.

The Task Force found that Canadian hospitals lack established standards of care for suicidal patients in emergency wards; that there are only a few preliminary protocols in existence and that research in the area is practically non-existent. Suicidal patients in general hospital emergency wards are frequently treated exclusively in terms of medical aspects of their

condition, especially if the self-injury is not considered to be even potentially lethal or, worse still, a mere "gesture". Skilled in surgery or resuscitation, the emergency medical staff may not be skilled in dealing with the personal crisis, family problems, or other factors precipitating the suicidal attempt.

The importance of the interdisciplinary team is illustrated, as are principles in assessing and managing the psychiatric emergency. Immediacy is essential. The person's problems and strengths, the life situation, the potential sources of help must all be assessed. Discussions and negotiation of referral should take place during the initial interview. Specificity of appointment and a minimal waiting period should be imperative. These and other principles are outlined. Recommendations are made.

Postvention

The first aim is reduction of the trauma to the bereaved, who constitute one of the high risk groups noted earlier.

The second aim for staff is to reconstruct the events leading to the suicide, to provide clarification regarding the nature of the death and the socio-psychological factors within the victim's life, immediately prior to the act. This is the 'psychological autopsy', described in some detail. Data are gathered from "significant others", preferably in the home setting. Typically the individuals interviewed are distraught.

The Task Force found the procedures of the psychological autopsy much less threatening than the quasi-judicial procedures of an inquest which often aggravate the distress. The psychological autopsy is both less intrusive and also an avenue for therapeutic intervention. Moreover it can supply researchers with valuable information. Task Force members are aware, however, that some of the objectives of a formal inquest cannot be met by a psychological autopsy.

Programs for 'High-risk: Groups

This section considers existing programs for certain "high-risk" groups. There is considerable discussion of programs for those suffering mental disorder, particularly severe depression and certain types of schizophrenia where the risk is high.

In general, an increasing incidence of alcoholism has been noted among individuals who commit suicide. This association warrants the inclusion of alcoholics in the high-risk category. Strategies of prevention and intervention acknowledge the complexity of the relationship, particularly with regard to the overlap with depression and other mental disorders.

In its discussion of the problems for young people and the avenues for prevention and intervention, considerable attention is given to the schools and education as well as counselling and psychiatric services.

In outlining the problems of the elderly and prevention of suicide in this group, the report stresses the importance of comprehensive programs: retirement programs, self-help groups, education of family physicians and public education about typical personality changes in the suicidal elderly person.

Native peoples are also a high-risk group. Here the importance of a culturally oriented approach is stressed. Some of the most compelling descriptions in this report involve native populations. These descriptions are found in the appendices.

Persons in custody are at high risk also. Self-help, peer group assistance, an inmate watch and many other components of programs are described. Importance is given to educational programs for custodial and police personnel and for improved inter-facility communication.

Contact with the bereaved as soon as possible following a suicide is critical. In programs described, volunteers often play a major role. Again the needs for education and program evaluation are stressed.

Suicide and the Law

This section discusses aspects of the Criminal Code, problems associated with committal under existing Mental Health Acts, and problems concerning confidentiality.

Data Gathering and Research

The report highlights the pressing need for more information, especially concerning specific groups of people and specific programs. The Report contains many recommendations about data gathering, common classification systems, multidisciplinary research, intersectoral collaboration, and so forth.

The Chairperson's Concluding Remarks

Fifteen of the most central conclusions are summarized by the Chairperson. The Report ends on this positive note: "Obviously, members of the Task Force hope that serious consideration will be given to the ideas expressed and suggestions and recommendations put forth in this Report. If even some of these are acted upon, it is felt that the long-range result will be a reduction in both the incidence and impact of suicide in Canada."

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Source: Executive Summary: *Report of the Task Force on Suicide in Canada.* Ottawa: Health and Welfare Canada, 1987. ISBN 0-662-15341-3.

An Appreciation

DR. ERNEST I. GLENISTER

The medical profession and ophthalmology in particular lost an eminent member in the passing of "Ernie" as he was known to his many friends.

He received his pre-medical education from St. Mary's University and graduated from Dalhousie University in 1925 with his M.D.C.M.

He then did general practice in Dartmouth until 1943 when he went to Toronto for post-graduate training in Ophthalmology. He practised ophthalmology in Halifax from 1945 until his retirement in 1974. He was associated with the Glaucoma Clinic at the Victoria General Hospital from its inception in 1962, and with both undergraduate and postgraduate teaching at Dalhousie University.

He obtained his F.R.C.S.(C) and was a Fellow of the American Academy of Ophthalmogy whose meetings he attended regularly. For the latter I can vouch as my wife and I usually accompanied Mary and him, and we were grateful for the many gourmet eating places they introduced and shared with us.

He was a member of the Halifax Medical Society, The Medical Society of Nova Scotia and the Canadian Medical Association. He was the first secretary of the now A.P.O.S. and a councillor for five years of the Canadian Ophthalmological Society. He was past president of the International Association of Secretaries of Ophthalmological Societies.

He was very active in his church and was a Fourth Degree member of the Halifax Knights of Columbus.

He is survived by his wife, the former Mary Burke; two sons, Paul and Peter, both of Halifax; a sister May, Montreal; a brother Joseph, Dartmouth; two grand-daughters and two great-grandchildren.

He will be remembered for his skill, honesty, compassion and above all he was a true gentleman.

C.F. Keays, M.D.

Consultations

When thou arte callde at anye time,
A patient to see;
And dost perceave the cure too grate,
And ponderous for thee:

See that thou laye disdeyne aside,
And pride of thyne owne skyll:
And thinke no shame counsell to take,
But rather wyth good wyll.

Gette one or two of experte men,
To help thee in that nede:
And make them partakers wyth thee,
In that work to procede.

. . . . But one thing note, when two or moe
Together joyned be;
Aboute the paynfull patient,
See that ye doe agree.

See that no discorde doe arise,
Nor be at no debate;
For that shall discomforte hym,
That is in sycke estate.

For noughte can more discomforte him,
That lies in griefe and peyne,
Then heare that one of you dothe beare
To other such disdeine.

Wherefore what so ye have to saye,
In things about your arte;
Let it be done among yourselves,
In secrete and a parte.

With one consent uniformlye,
Comforte the ailing man;
But unto some good friende of hys
Express all that ye can. . . . . .

— John Halle, M.D. (1529-1566)
from *Goodlye Doctrine and Instruction*

Submitted by Dr. Douglas L. Roy, Halifax, N.S. (and still pertinent).

Personal Interest Notes

Dr. Stuart C. Robinson, of Halifax has been invited to spend a year as visiting Professor at the Aga Khan University, Karachi, Pakistan. He will be teaching Obstetrics and Gynaecology and Medical Education.

This new medical university founded by his Highness the Aga Khan, has a modern campus, 750 bed hospital and a faculty of Nursing affiliated with MacMaster University.

Upon his return Dr. Robinson will return to his practice in Gynaecology and Geriatric Gynaecology and teaching at Dalhousie.

Dr. John H. Quigley, of Halifax, assumed the vice-presidency of the Joint Commission on Allied Health Personnel in Ophthalmology starting January.

The joint Commission is the National certifying agency for Opthalmic Assistants, and is committed to the improvement of eye care by the implementation of better educational and training programs.

No one ever claimed our personal interest notes were up to date. Somehow. . . ., the following came into the possession of the staff of the *Journal*. We thought it might be of interest to many members.

By the KING'S Order the name of
Captain (T Major) C.E. van Rooyen
Royal Army Medical Corps,
was published in the London Gazette on
6 April, 1944,
as mentioned in a Despatch for distinguished service.
I am charged to record
His Majesty's high appreciation.

Secretary of State for War

OBITUARIES

Dr. James J. Carroll, (86) of Antigonish, N.S. died on December 15, 1987. Born in Massachusetts, he graduated from Dalhousie Medical School in 1924. He was President of The Medical Society of Nova Scotia, as well as President of St. Martha's Hospital medical staff. He was made a senior member of the Medical Society in 1972, received the Doctor of the Year Award from the Canadian Medical Association in 1973, and a senior citizen's complex was named for him in 1975. He is survived by a brother and a sister. The *Journal* extends sincere sympathy to his family.

Dr. John O. McNeil, (66) of Glace Bay, N.S. died January 2, 1988. Born in Glace Bay he graduated from Dalhousie Medical School in 1945 and then went on to do post graduate studies in general surgery. He opened his general practice and surgery in Glace Bay in 1951 and was still active until his death. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by his wife, one daughter and four sons. The *Journal* extends sincere sympathy to his family.

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