The Acute Abdomen in Spinal Cord Injury Patients*

George L. Juler, M.D. and Ibrahim M. Eltorai, M.D.

The Surgical and Spinal Cord Injury Services, Veterans Administration Medical Center, Long Beach, California and School of Medicine, Division of Surgery, University of California, Irvine, California

Summary

After return of the reflex arc below the level of anaesthesia, the spinal cord injury (SCI) patient will manifest an intra-abdominal emergency by the clinical signs of dysreflexia depending upon the level and completeness of the cord lesion. Thirty-six SCI patients are presented to correlate the autonomic response to visceral disease with the level of their cord lesion demonstrating that early recognition and diagnosis is possible in these patients.

Key words: *Spinal cord injury; Acute abdomen; Visceral pain; Dysreflexia.*

The profound neurological deficits in the spinal cord injury (SCI) patient makes early recognition and diagnosis of acute abdominal emergencies very difficult unless the physician is thoroughly familiar with the altered physiological responses to the visceral pathology (Charney *et al.*, 1975; Juler, 1979). The SCI patient's response to his/her visceral disease depends upon the level of the cord lesion, whether it is complete or incomplete, and whether the reflex arc below the level of the cord lesion is intact (Charney *et al.*, 1975; Juler, 1979). There are other factors which may complicate the management of these patients such as the presence of hyperreflexia, radicular pain, urological disease adynamic ileus, cardio-pulmonary disease, drug abuse and functional disorders (Charney *et al.*, 1975).

The impaired reflex, motor and sensory functions of the SCI patients with acute abdominal emergencies commonly results in delayed surgical management and is associated with a mortality rate of 10-15 per cent (Charney *et al.*, 1975; Tibbs *et al.*, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965). This

*Read at the Southwest Regional Medical Education Center Seminar 'Emergencies in Stable Spinal Cord Injury Patients', June 1, 1984, Long Beach, California.

unusually high mortality rate can be lowered by early recognition, diagnosis and appropriate surgical management which is based upon a thorough understanding of the pathophysiological changes in the SCI patient (Charney *et al.*, 1975).

A retrospective study of our SCI patients with acute abdominal emergencies was undertaken to determine the clinical responses to visceral disease as related to the type of disease and level of the cord lesion. This study is a continuation of our previously reported experiences (Charney *et al.*, 1975; Juler, 1979).

Methods

Forty-two spinal cord injury patients have been operated upon for acute visceral disease between 1966 and 1984 (Table 1). Three main groups of SCI patients with 'acute abdomens' required emergency surgery: acute appendicitis, acute perforation of a viscus, and acute bowel obstruction. Since only five patients with either acute cholecystitis, ruptured spleen or urinary bladder perforations required emergency surgery, these are not included in this study. Therefore, the three main categories of visceral disease provide 37 cases for review. The level of neurological deficit varied from C₃ to L₅ with only five patients manifesting incomplete lesions.

Table 1 Acute abdominal emergencies in SCI patients

Lesion	No. Patients
Acute cholecystitis	1
Ruptured spleen	1
Perforated urinary bladder	3
Acute appendicitis	10
Perforated viscus	11
Bowel obstruction	16
Total	42

The level and completeness of the cord lesions were correlated with the clinical response of the patient to the acute visceral disease. The character of the abdominal pain, its radiation, the ability to localize pain, the associated abdominal spasms, the reflex sweating response, temperature, pulse and blood pressure elevations, bladder spasms and early onset of anorexia, nausea and vomiting were the parameters used to evaluate each patient's response to the disease process.

Results

Ten patients were operated upon for acute appendicitis. Eight patients had cord lesions above the level of the splanchnic outflow tract (T₈) commonly referred to as 'high' cord lesions. All of these eight patients had appendiceal perforations with walled-off abscesses found at operation. They all had signs of dysreflexia from two days to three weeks preoperatively except the patient with a C₄ incomplete lesion. All had lesions higher than the T₆ level.

The two patients with lesions below the splanchnic outflow tract (T₁₁, T₁₂) did not have the clinical signs of hyperreflexia or dysreflexia and could localize their

abdominal pain to the right lower quadrant abdomen early in the disease, thus avoiding perforation and abscess formation. These patients with 'low' lesions below the splanchnic outflow tract responded to their disease much the same as a normal person.

It should be noted that the patient with the C₄ incomplete lesion did not have the usual signs of dysreflexia and could localize his pain to the right lower quadrant abdomen. All but one of these eight patients could localize their pain to the right lower quadrant abdomen regardless of whether or not their lesions was complete or incomplete.

The 11 patients operated upon for a perforated viscus demonstrated the classical clinical signs of dysreflexia in the eight patients with 'high' cord lesions from C₃ to T₄ levels. Besides manifesting visceral reflex sensitivity they all experienced shoulder pain while only one patient (C₅-C₆) with a perforated duodenal ulcer could localize his pain to the upper abdomen.

The three patients with 'low' cord lesions (T₁₁, T₁₂ and L₄) and perforations of the duodenum and ileum could localize their pain fairly well and did not manifest the usual signs of dysreflexia. However, the patient with the T₁₁ level lesion did have a problem with increased spasms of the abdomen but no change from flexor to extensor spasm.

There were 16 patients who underwent emergency laparotomy for acute bowel obstruction, four of whom had large bowel obstructions. There are again noted to be two groups of patients according to the level of the lesions with eight patients in each group. Seven of eight patients with 'high' lesions manifested signs of dysreflexia. One patient (C₅-C₆ level) with a sigmoid volvulus had no signs of dysreflexia, probably due to the early diagnosis which avoided the advanced stage of the disease.

The patient (C₆ complete) with a jejunal volvulus could localize his pain to the left upper quadrant of the abdomen demonstrating a dermatome level of referred pain, while five other patients had only vague generalized abdominal pain. Two patients (T₆-T₇) with ileal obstruction experienced crampy lower abdominal pain.

The eight patients with 'low' cord lesions (T₁₀ to L₁) experienced crampy abdominal pain except the patient (T₁₂-L₁) with his transverse colon up in the left chest through a diaphragmatic hernia. The four patients with T₁₀ and T₁₁ level lesions had only temperature, pulse and blood pressure elevations as manifestations of dysreflexia. None of the patients with 'low' lesions had reflex visceral sweating. These patients responded to visceral disease much the same as does a normal (non-spinal paralysed) person.

Discussion

Early and accurate diagnosis of acute visceral disease in the stabilized SCI patient depends upon recognition of the early signs of autonomic dysreflexia (Charney *et al.*, 1975; Juler 1979; Wanebo *et al.*, 1965). These autonomic responses to intra-abdominal disease are dependent upon an intact reflex arc below the level of the cord lesion which allows visceral reflexes to be mediated by the sympathetic and parasympathetic nervous system (Charney *et al.*, 1975; Juler, 1979; Miller *et al.*, 1975). However, this is not so in SCI patients with spinal shock

immediately following injury when sensory, motor and reflex functions are absent (Tibbs *et al.*, 1979; Wanebo *et al.*, 1965). In this situation the usual clinical signs of the acute abdomen are absent so that one must depend upon laboratory, X-ray and technical procedures to make the diagnosis (Tibbs *et al.*, 1979; Wanebo *et al.*, 1965; Tibbs *et al.*, 1980).

If visceral sensitivity is intact below the level of the cord lesion, the site of the lesion determines the clinical response to the intra-abdominal disease in most cases (Charney *et al.*, 1975; Juler, 1979; Hoen and Cooper, 1948). This fact is clearly demonstrated by the three types of abdominal emergencies in our study (Juler, 1979; Tibbs *et al.*, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965). Patients with 'high' cord lesions demonstrated the classical signs of dysreflexia in most instances, while those with 'low' lesions manifested the usual signs of acute abdomen seen in the normal neuraxis patients. This phenomenon is described by Greenfield (1949) as well as other authors (Charney *et al.*, 1975; Wanebo *et al.*, 1965).

The classical signs of autonomic dysreflexia associated with cervical or high thoracic lesions are described by several authors (Charney *et al.*, 1975; Juler, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965; Greenfield, 1949; Dollfus *et al.*, 1974). Increasing abdominal pain associated with increasing spasticity are the most reliable signs of visceral disease (Miller *et al.*, 1975; Wanebo *et al.*, 1965). The early visceral pain is dull, heavy and oppressive, and is poorly localized in contrast to radicular pain (Juler, 1979; Tibbs *et al.*, 1979; Miller *et al.*, 1975).

In this study we found that anorexia is one of the earliest signs associated with the onset of visceral pain and is one of the first clues that something is wrong inside the abdomen (Charney *et al.*, 1975; Juler, 1979; O'Hare, 1966). However, it can occur after onset at which time it is usually associated with nausea and vomiting. Anorexia occurred early in all but six of the patients in this study.

As the intra-abdominal disease process advances, the increased visceral afferent activity via the dorsal root system may increase the activity of the somatic sensory neurons in the spinal cord, causing referred pain sensation (Miller *et al.*, 1975; Hoen and Cooper, 1948). Until this occurs, pain perception is vague and non-localizing.

Increasing abdominal spasticity associated with increased visceral sensory input is especially significant when there is a change in spasticity (Charney *et al.*, 1975; Juler, 1979; Miller *et al.*, 1975; Ingberg and Prust, 1968). All but one of our patients with a high cord lesion and appendicitis had increased abdominal spasms. His C₄ incomplete lesion allowed modification of his response to visceral pain and localization of his lesion. However, the C₅ incomplete patient could not localize his pain. It should be noted that most patients with 'high' lesions can localize visceral pain via the referred pain mechanisms.

Other early signs of autonomic dysreflexia in SCI patients with 'high' level lesions are early elevations of pulse and blood pressure which are often associated with headache (Charney *et al.*, 1975; Juler, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965; Dollfus *et al.*, 1974). This hyperreflexia is caused by either peritoneal inflammation, bowel distention or both. The sudden rise in blood pressure associated with headache is due to the intense vasoconstriction below the level of the cord lesion secondary to the autonomic hyperreflexia (Juler, 1979; Tibbs *et al.*, 1979; Miller *et al.*, 1975).

Temperature elevations above 39° centigrade are more representative of acute pyelonephritis than those associated with visceral disease (Juler, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965). While early temperature elevations are associated with 'high' cord lesions in patients manifesting signs of dysreflexia from visceral disease, Sugarman *et al.* (1982) have reported that only 5.1 per cent of 116 SCI patients studied had fevers of unknown origin. This would indicate that the thermoregulatory mechanisms of SCI patients are intact and that temperature elevations are related to the organic disease rather than hyperreflexia.

Spinal reflex sweating is frequently associated with visceral inflammation and bowel distention (Miller *et al.*, 1975; Greenfield, 1949). Because of sympathetic overlap, the level of sweating may be lower or higher than the level of anaesthesia (Miller *et al.*, 1975; Greenfield, 1949; Fast, 1977). In patients with cervical lesions, the sweating is usually above the level of anaesthesia (Greenfield, 1949). This was certainly true in our experience. However, there were two patients with high cord lesions associated with appendicitis and sigmoid volvulus who did not manifest reflex visceral sweating. It must be remembered that patients with incomplete cord lesions can modify their pain pathways and manifest altered responses to visceral pain (Charney *et al.*, 1975; Juler, 1979). None of our patients with lesions below the splanchnic outflow tract manifested reflex sweating. This phenomenon has been recognized by others and even bladder distention and powerful detrusor muscle activity will not elicit reflex sweating in these patients (Charney *et al.*, 1975; Wanebo *et al.*, 1965; Greenfield, 1949; O'Hare, 1966).

Abdominal distention is so common among SCI patients even in the absence of abdominal disease that it is difficult to attach significance to it unless it appears suddenly, is progressive and associated with air-fluid levels on roentgenograms of the abdomen (Charney *et al.*, 1975; Juler, 1979; Miller *et al.*, 1975; Hoen and Cooper, 1948).

Bowel sounds are rarely significant but when they become hyperactive and then suddenly disappear it is suggestive of bowel obstruction (Miller *et al.*, 1975; Wanebo *et al.*, 1965). In our patients with high cord lesions associated with bowel obstruction, all but one (C₅-C₆ sigmoid volvulus) had an initial increase in bowel activity followed by silence whereas those patients with lesions below the splanchnic outflow all had bowel activity until the time of operation.

Abdominal tenderness is not a common finding in the SCI patient. When it is found on physical examination it usually represents a perforated viscus (Charney *et al.*, 1975; Hoen and Cooper, 1948). This finding was present in all of our patients with a perforated viscus. It should be noted that shoulder pain associated with change in abdominal spasticity was present in all patients with a high cord lesion in this group of patients. This phenomenon has been reported by other authors (Miller *et al.*, 1975; Sugarman *et al.*, 1982).

The operative management of acute abdominal emergencies in the SCI patient is the same as the patient with a normal neuraxis (Charney *et al.*, 1975; Juler, 1979).

Two of 42 patients (4.7%) operated upon as emergencies died post-operatively from sepsis associated with an infarcted jejunal volvulus and multiple small bowel fistulae in a patient with radiation enteritis and ileal obstruction. This mortality rate is lower than the reported 10 to 15 per cent (Charney *et al.*, 1975; Tibbs *et al.*, 1979; Miller *et al.*, 1975; Wanebo *et al.*, 1965).

In summary, early recognition of the clinical manifestations of visceral dysreflexia in the SCI patient will allow the clinician to determine the aetiology of the intra-abdominal disease and institute early appropriate surgical treatment.

Résumé

Après la reprise de l'activité de l'arc reflexe sous le niveau anesthésique les malades atteints de trauma médullaire peuvent se présenter avec l'hyperréflexie autonome comme la manifestation d'un abdomen aigu dépendant de niveau et de degré de lésion. Trente-six malades sont présentés au but corrélatif entre la réponse autonome due à la maladie viscérale avec le niveau de trauma médullaire en même-temps démontrer que le diagnostique avancé est possible chez tels malades.

Zusammenfassung

Nach der wiederkehr des Reflexbogens Tätigkeit unten des Niveau der Anästhesie die Patienten mit Rückenmarksverletzung haben die Möglichkeit der Entwicklung Autonomische Hypertonie als Bekanntmachung einen Akut Bauchanfall. Diese Hypertonie Kommt darauf an das Niveau und den Grad der Verletzung. Ein Bericht über Sechs-und-dreizig Patienten wird gegeben in den die autonome Reaktionen durch Baueingeweidestörungen hängt ab das Niveau des Rückenmarks Trauma. Resultate zeigen das Frühe Diagnose zur erkennung von Akute Chirurgische Bauchanfalle bei dieser Patienten ist möglich.

References

- CHARNEY KF, JULER GL, COMARR AE (1975) General surgery problems in patients with spinal cord injury. *Archives of Surgery* 110:1083-1088.
- DOLLFUS P, HOLDERBACH GL, HUSSER JM, JACOB-CHIA D 1974 Must appendicitis be still considered a rare complication in paraplegia? *Paraplegia* 11:306-309.
- FAST A (1977) Reflex sweating in patients with spinal cord injury; A Review. *Archives of Physical Medicine and Rehabilitation* 58:435-437.
- GREENFIELD J (1949) Abdominal operations on patients with chronic paraplegia. *Archives of Surgery* 107:1087.
- HOEN TI, COOPER IS 1948 Acute abdominal emergencies in paraplegics. *American Journal of Surgery* 75:19-24.
- INGBERG HO, PRUST FW 1968 The diagnosis of abdominal emergencies in patients with spinal cord lesions. *Archives of Physical Medicine and Rehabilitation* 49:343-349.
- JULER GL (1979) Acute abdominal emergencies in spinal cord injury patients. *Journal of American Paraplegia Society* 2:1-5.
- MILLER LS, STAAS WE, JR, HERBISON GJ 1975 Abdominal problems in patients with spinal cord lesions. *Archives Physical Medicine and Rehabilitation* 56:405-408.
- O'HARE JM 1966 The acute abdomen in spinal cord injury patients. *Proceedings, Annual Clinical and Spinal Cord Injury Conference* 15:113-117.
- SUGARMAN B, BROWN D, MUSER D 1982 Fever and infection in spinal cord injury patients. *Journal of American Medical Association* 248(1):66-70.
- TIBBS PA, BIVINS BA, YOUNG AB 1979 The problem of acute abdominal disease during spinal shock. *American Surgeon* 45:366-368.
- TIBBS PA, YOUNG AB, BIVINS BA 1980 Diagnosis of acute abdominal injuries in patients with spinal shock: Value of diagnostic peritoneal lavage. *Journal of Trauma* 20:55-57.
- WANEBO HJ, WEBB E, COMBS R 1965 Diagnosis of acute abdominal disease in paraplegic patients. *Californian Medicine* 103:193-197.