MEDICAL INTELLIGENCE

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Cardiovascular Complications during Anesthesia in Chronic Spinal Cord Injured Patients

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PATIENTS with chronic spinal cord injuries undergo many urological, plastic, orthopedic, and neurosurgical procedures during the course of their rehabilitation. We have reviewed the medical records of 93 such patients who underwent 219 surgical procedures during the period 1976–1979 in our institution, to determine the incidence of perioperative cardiovascular complications. We present several cases in detail in which the unexpected occurrence of severe hypertension associated with autonomic hyperreflexia, hypotension, and bradycardia illustrate some of the cardiovascular problems that may be encountered in the anesthetic management of these patients.

Spinal cord trauma is followed initially by a period of spinal shock, with total absence of spinal cord reflexes below the level of injury. During this period which lasts for one to several weeks after the injury, the major cause of morbidity and mortality is impaired alveolar ventilation combined with the inability to protect the airway and to clear bronchial secretions. Aspiration of gastric contents, bronchopneumonia, pulmonary edema, and pulmonary embolism occur frequently in this patient population and complicate the clinical situation. The anesthetic management of patients during spinal shock, and the problems associated with acute spinal cord injury have been reviewed in detail by Quimby.[1] Within several weeks of injury there is a gradual return of spinal cord reflexes and patients enter a chronic stage characterized by sympathetic overactivity and involuntary muscle spasm. The sequelae of this chronic stage of spinal cord injury that are of concern to the anesthesiologist include cardiovascular instability, impaired alveolar ventilation, chronic infection, anemia, decubitus ulceration, impaired thermoregulation, and an increased sensitivity to succinylcholine, which may result in hyperkalemia. In our

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review we focused on the cardiovascular problems that occurred during the management of chronic spinal cord injured patients. The manifestations of cardiovascular instability in our patient population may be classified into three categories: 1) hypertension, associated with autonomic hyperreflexia 2) arrhythmias, and 3) hypotension.

Hypertension and Autonomic Hyperreflexia

REPORT OF A CASE

Case 1. A 35-year-old man, admitted for transurethral sphincterotomy, sustained a fracture dislocation of the fifth cervical vertebra 13 years prior to admission which left him with complete motor and sensory loss below the sixth cervical segment. On physical examination the blood pressure was 100/70 torr, pulse rate was 78 beats per min, and auscultation of the heart and lungs was normal. Laboratory data were within normal limits. The patient was brought to the operating room unpremedicated, and spinal anesthesia was instituted at the L4-5 interspace with 75 mg lidocaine. The level of the block could not be determined, but there was loss of lower extremity spasticity. Sixty-five min after performance of the block, and with surgery still in progress, the patient complained of shortness of breath, facial tingling, and nausea. The surgeons noted increased bleeding in the operative field. The systolic blood pressure was 220 torr and the pulse rate was 30 beats per min; moments earlier the vital signs were at the preoperative level. The panendoscope was removed and the bladder emptied. General anesthesia was induced by mask with 2.5 per cent halothane in an equal mixture of nitrous oxide and oxygen, with prompt return of blood pressure and pulse rate to preoperative levels. The patient was maintained on 1 per cent halothane for another 30 min, until the termination of surgery, without further complications.

DISCUSSION

Autonomic hyperreflexia, or mass reflex, is a disorder of chronic spinal cord injured patients first described by Head and Riddoch in 1917.[2] This reflex response may be initiated by cutaneous or visceral stimuli below the level of spinal cord trauma. The symptoms of autonomic hyperreflexia include facial tingling, nasal obstruction, severe headache, shortness of breath, nausea, and blurred vision.[2-4] The signs include paroxysmal hypertension, bradycardia, arrhythmias,[5] sweating,[6] cutis anserina (goose flesh), cutaneous vasodilation above and vasoconstriction and pallor below the level of spinal cord injury, loss of consciousness, and seizures.[7] The precipitous increase in blood pressure may lead to retinal, cerebral, or subarachnoid hemorrhage[8] and increased operative

blood loss.[9] Eighty-five per cent of patients with chronic spinal cord injury at T6 or above, will exhibit this reflex at some time in their life, in response to stimuli resulting from routine daily activity.[8] Distention of a hollow viscus, such as the bladder or rectum, frequently precipitates its occurrence, and the response is proportional to that strength of the stimulus.[3] It is unclear why some patients (approximately 15 per cent) with levels of injury above T4 never experience this syndrome, while occasionally patients with levels below T8 do. However, surgery is a particularly potent stimulus to the development of autonomic hyperreflexia, and even patients who give no previous history of autonomic hyperreflexia are at risk during surgical procedures.

The neuroanatomic pathway of the syndrome was first described by Kurnick[8] in 1956. Afferent impulses enter the isolated spinal cord via the pelvic and pudendal nerves, and elicit reflex autonomic output over the splanchnic outflow that is not modulated by higher centers as in neurologically intact individuals. This leads to generalized vasoconstriction below the level of injury. The resulting hypertension stimulates the baroreceptors, which may cause bradycardia via intact pathways to the heart, and vasodilation via intact sympathetic pathways above the level of injury. Vasodilation in areas of the body above the level of injury is insufficient in patients with levels of injury above the splanchnic outflow, i.e., T4–T6, to significantly reduce the hypertension.

PREVENTION OF AUTONOMIC HYPERREFLEXIA

Many regimens have been proposed for the prevention or control of autonomic hyperreflexia occurring either during routine daily activity or during surgery. These include treatment with ganglionic blocking drugs, alpha-adrenergic blocking drugs, catecholamine depleters, direct acting vasodilators, and general or regional anesthesia (fig. 1). Therapeutic measures to lower the hypertension of autonomic hyperreflexia must act at sites below the level of injury. For example, in 1948 Thompson and Witham[9] reported that tetraethylammonium chloride (TEA), a ganglionic blocker, was successful in controlling autonomic hyperreflexia. Other ganglionic blocking drugs such as hexamethonium,[8,10] pentolinium,[11–13] and trimethaphan[14–16] have also been reported to be effective in the control of autonomic hyperreflexia. Although we would expect these agents to be of value, it is still somewhat difficult to judge their usefulness as the number of patients in these studies was either small or not mentioned, and other anesthetic agents were used concurrently with the ganglionic blockers. The use of alpha-adrenergic blocking drugs such as phentolamine have been advocated by Sizemore,[17] but doses as large as 40 mg intravenously may be required, and failures

with phentolamine have been reported by Kurnick.[8] Agents which lower blood pressure by a central action alone, either on the vasomotor center of the brain stem or on higher centers, are not effective in controlling the hypertension of autonomic hyperreflexia in spinal cord injured patients.

Brown et al.[18] recently reported that in 200 spinal cord injured patients with lesions above T5, 10 mg guanethidine sulfate, administered orally three times daily for 30 days prior to undergoing cystometrograms, successfully prevented the hypertensive crisis of autonomic hyperreflexia in all of their patients. The effectiveness of guanethidine in preventing autonomic hyperreflexia during surgical procedures, or following periods of administration of less than 30 days has not been evaluated. Patient acceptability of guanethidine, however, may be poor because of the side effects which include postural hypotension, generalized weakness, fluid retention, diarrhea, and inhibition of ejaculation.[9] Vandam and Rossier[20] suggested that sodium nitroprusside, a directly acting vasodilator, may be used for the rapid control of hypertension due to autonomic hyperreflexia, but they made no mention of their clinical experience with the drug for this purpose. While directly acting vasodilators may control the hypertension, they would not be expected to have any effect on visceral and somatic spasm which may occur as manifestations of autonomic hyperreflexia.

AUTONOMIC HYPERREFLEXIA AND GENERAL ANESTHESIA

During surgery various general anesthetic agents have been used in an attempt to prevent or control autonomic hyperreflexia.[21] Little success was reported by Ciliberti et al.[10] who in 1954 noted an incidence of dysreflexia of 48 per cent in 27 patients with lesions above T5 undergoing surgery. Anesthetic agents mentioned in their review included pentothanitrous oxide, cyclopropane, ether, and trichloroethylene. Anesthesia was very light in this series of patients and the authors comment that depth of anesthesia played some part in the incidence and degree of reflex response. Drinker and Helrich[22] in 1963 were the first to report on the successful use of halothane to control autonomic hyperreflexia in a quadriplegic patient undergoing urological surgery. Based on this single case report, halothane has been recommended as the general anesthetic agent of choice for chronic spinal cord injured patients.[12] In 1975, Alderson and Thomas[23] reported that halothane prevented the development of autonomic hyperreflexia in 45 spinal cord injured patients undergoing urological surgery, 24 of whom had cervical levels of injury. The success of halothane in preventing autonomic hyperreflexia in this series was probably related to its potency and ease with which an adequate depth of anesthesia could be maintained. It could also, perhaps, be
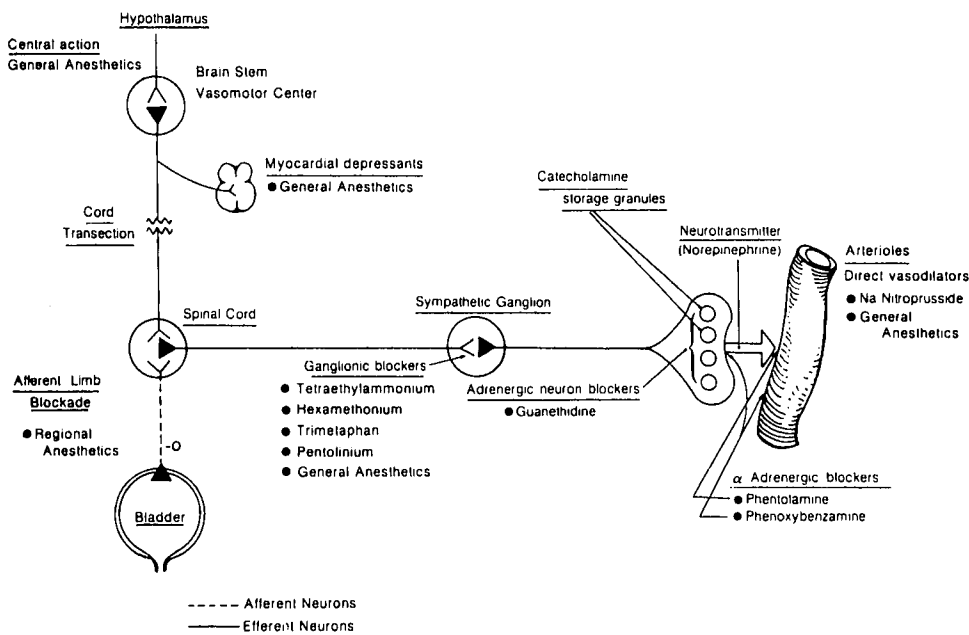
FIG. 1. The sites of action of the agents used for control of the hypertension of autonomic hyperreflexia are shown above.

related to an increased awareness of the reduced incidence of autonomic hyperreflexia associated with deeper planes of anesthesia noted by Ciliberti et al.[10] There was, however, a significant incidence of arrhythmias (27 per cent), sufficiently severe in seven patients to require treatment with an antiarrhythmic agent. Thus, while halothane is effective in preventing autonomic hyperreflexia, it could be inferred from this study that significant cardiac arrhythmias may limit its usefulness in these patients. Hassan[24] reported that pentothal, nitrous oxide, and curare successfully prevented autonomic hyperreflexia in 12 paraplegic patients. It is difficult to assess this report, however, as the type of surgery and the level of spinal cord injury were not clearly documented.

Of the 219 cases in our series, 33 per cent were performed under general anesthesia (table 1). In patients with levels of injury at or above T5, there were no incidents of autonomic hyperreflexia during general anesthesia with halothane (37 cases) or enflurane (12 cases). In contrast to the report by Alderson and Thomas,[23] the use of halothane (and enflurane) in our series was not associated with arrhythmias; the electrocardiogram in all our patients was monitored with an oscilloscope. This difference may be because ventilation in our patients was assisted or controlled to maintain normocarbia, whereas Alderson and Thomas allowed their patients to breathe spontaneously. Nine patients with levels of injury above T5 had surgery performed with nitrous oxide–narcotic general anesthesia. Two of these patients developed intraoperative autonomic hyperreflexia (table 2). In one of these patients the administration of 1 per cent halothane was successful in controlling the hypertension, after 10 mg intravenous phentolamine produced no re-

sponse, suggesting that the depth of anesthesia was inadequate to prevent autonomic hyperreflexia. In Case 1, halothane also was successful in controlling the autonomic hyperreflexia that occurred during a spinal anesthetic with lidocaine. Thus, it appears that intraoperative autonomic hyperreflexia can be prevented or controlled by general anesthesia with the potent volatile anesthetic agents halothane and enflurane.

AUTONOMIC HYPERREFLEXIA AND LOCAL OR REGIONAL ANESTHESIA

Blockade of the afferent pathways (fig. 1) by local anesthetic agents is another means of preventing autonomic hyperreflexia. For cystoscopic procedures, this may be accomplished by the topical application of local anesthetic agents to the urethral and bladder mucosa. This technique, while simple to perform, is associated with many failures[25] because topical anesthesia does not block the bladder muscle proprioceptors which are stimulated by bladder distention.

Epidural anesthesia has been reported to prevent autonomic hyperreflexia in paraplegics during labor and delivery[26] and urologic surgery[27] but these reports represent only isolated case reports. In contrast, Johnson[7] and Texter[11] claim that epidural anesthesia is not predictable in preventing autonomic hyperreflexia, but they do not document their experience. Broecker[28] reports that of three patients undergoing urologic endoscopic procedures under epidural anesthesia with mepivacaine, two patients developed autonomic hyperreflexia requiring treatment with ganglionic blocking drugs. Our experi-

TABLE 1. Type of Operation and Anesthetic Technique Employed

Type of Operation	Total	General	Spinal	Epidural	Axillary Block	Intravenous Sedation	Local	Standby
Urological	100	14	76	3	—	1	4	2
Plastic	78	27	25	1	16	4	3	2
Orthopedic	27	18	5	—	—	2	—	2
Neurosurgery and General surgery	14	14	—	—	—	—	—	—
TOTAL	219	73	106	4	16	7	7	6

ence with epidural anesthesia in chronic spinal cord injured patients is limited to four cases. We have not used this technique more extensively because of the difficulty in evaluating the results of the test dose for inadvertant subarachnoid injection in patients with complete high levels of spinal cord injury. One patient in our series developed a total spinal anesthetic during an attempt at epidural anesthesia with bupivacaine. After a 3-ml "test dose" followed three minutes later with 14 ml via the epidural catheter, the patient became acutely apneic and required several hours of controlled ventilation.

Reports of the use of spinal anesthesia to prevent autonomic hyperreflexia are more frequent than those of epidural anesthesia. In 1954 Ciliberti et al.[10] reported on the use of spinal anesthesia in 13 patients, with levels of injury at T5 or above, undergoing urological procedures. There were no instances of hypertension or headache in their series. They did not mention any complications nor did they specify the local anesthetic agent that was used. Comarr and Woodard[29] in 1957 reported the use of spinal anesthesia with pontocaine in 47 spinal cord injured patients undergoing a variety of surgical procedures. However, they did not document the level of spinal cord injury, nor the reliability of spinal anesthesia in preventing autonomic hyperreflexia. Rocco and Vandam[14] administered spinal anesthesia for 33 transurethral procedures but they did not report the level of spinal cord injury, local anesthetic agent used, or the degree of success in preventing autonomic hyperreflexia. Broecker[28] used spinal anesthesia with 25–50 mg lidocaine in 25 patients with levels of injury above T7 undergoing urological endoscopic procedures. He reported no episodes of autonomic hyperreflexia or hypotension in his series.

In our series, spinal anesthesia was the single most frequently employed anesthetic technique for the prevetion of autonomic hyperreflexia in patients with levels of injury at T5 or above undergoing a variety of surgical procedures (table 3). In 97 cases of spinal anesthesia with tetracaine, there were no incidents of intraoperative autonomic hyperreflexia. Urological surgery has been reported previously to be a potent stimulus for triggering autonomic hyperreflexia. Intraoperative autonomic hy-

perreflexia during urological surgery performed with spinal anesthesia occurred only once in our series, when lidocaine was used as the local anesthetic agent (Case 1). In this case it is likely that recovery from spinal anesthesia occurred prior to the end of surgery. Therefore, we believe that with the appropriate local anesthetic agent, spinal anesthesia offers an effective means of preventing intraoperative autonomic hyperreflexia.

Perhaps the technical difficulty anticipated in performing subarachnoid block in chronic spinal cord injured patients, especially in those patients with levels of injury above T5, has been overstated. In 90 spinal anesthetics performed in patients with levels of injury above T5, administered by residents in training under faculty supervision, only two of our patients required three or more attempts for successful lumbar puncture. However, in patients with low levels of injury, three of 19 attempted lumbar punctures were abandoned and an additional three patients required three or more attempts for successful lumbar puncture. The most likely explanation for difficulty or failure in these cases was the presence of an anatomical deformity secondary to previous laminectomy and/or spinal fusion in the lumbar area. None of our patients developed symptomatic postlumbar puncture headache or, to date, exacerbation of neurologic symptoms following spinal anesthesia.

AUTONOMIC HYPERREFLEXIA IN THE POSTOPERATIVE PERIOD

It is important to note that whatever the anesthetic technique, autonomic hyperreflexia may occur during recovery from anesthesia (table 2). Of 11 recorded episodes of autonomic hyperreflexia in our series, four occurred in the recovery room. Two cases followed recovery from spinal anesthesia with tetracaine (Patients 10 and 11). The other two cases occurred following recovery from general anesthesia with halothane (Patient 8) and enflurane (Patient 9). The site of surgery in Patient 8 was above the site of spinal cord injury, yet he still developed autonomic hyperreflexia postoperatively. We do not know for certain what the etiology of this was. Certainly, distention of the bladder and rectum may occur

TABLE 2. Episodes of Hypertension* Intraoperatively and in the Recovery Room in 219 Operative Procedures

Patient	Level	Procedure	Anesthetic Technique	Anesthetic Agents	Preoperative Blood Pressure (torr)	Maximum Blood Pressure (torr)	Comment
Operating Room							
1	T4	Debridement sacral ulcer	Standby	—	110/70	195/-	Headache. Responded to phentolamine, 5 mg, iv, with return of blood pressure to 110 torr systolic.
2	C5	Excision suprapubic scar	Standby	—	110/70	160/110	Headache.
3	T12	Transurethral sphincterotomy	Topical	Lidocaine	100/60	160/110	Headache.
4	T3	Cystoscopy	Intravenous Sedation	Fentanyl	100/70	170/-	Headache.
5	C5	Transurethral sphincterotomy	General	Nitrous oxide–demerol	100/70	170/110	In recovery room, blood pressure increased to 200 torr, and the patient complained of headache.
6	C6	Deltoid-triceps tendon transfer	General	Nitrous oxide–fentanyl	110/70	180/-	Sudden rise in blood pressure after stable anesthesia. No response to phentolamine, 10 mg, iv. Controlled with 1 per cent halothane.
7	C5	Transurethral sphincterotomy	Spinal	Lidocaine	100/70	220/-	See Case 1.
Recovery Room (8-11)							
8	T5	Carpal tunnel release	General	Nitrous oxide–halothane	120/80	170/100	Headache, diaphoresis, lower extremity spasm one hour after arrival in recovery room with patient awake.
9	C5	Deltoid-triceps tendon transfer	General	Nitrous oxide–enflurane	110/60	170/120	Headache forty-five minutes after arrival in recovery room with patient awake.
10	T4	Transurethral sphincterotomy	Spinal	Tetracaine	110/80	180/120	Headache. Hypertension occurred 3.5 hours after spinal anesthesia was administered.
11	T11	Cystoscopy Transurethral sphincterotomy	Spinal	Tetracaine	100/60	150/120	Headache. Hypertension occurred 3 hours after spinal anesthesia was administered.

* Hypertension is defined as a rise in systolic pressure > 30 torr above the preoperative level, or a systolic pressure > 160 torr.

TABLE 3. Distribution of Patients, Operative Procedures, and Anesthetic Techniques by the Level of Spinal Cord Injury

Level of Injury	Number of Patients (Percentage of Total)	Number of Operative Procedures (Percentage of Total)	Anesthetic Technique							
			General		Spinal†	Epidural	Axillary Block	Intravenous Sedation	Local	Standby
			Volatile Agents*	Nitrous Oxide–Narcotic						
C1-C7	50 (53.8)	131 (59.8)	40	6	60	2	16	1	4	2
T1-T5	22 (23.7)	46 (21.0)	9	3	30	2	—	1	—	1
T5-T12	14 (15.0)	28 (12.8)	6	1	13	—	—	3	2	3
L1-L5	7 (7.5)	14 (6.4)	7	1	3	—	—	2	1	—

* Halothane in 48 procedures and enflurane in 14 procedures.
† Spinal anesthetic agents were tetracaine 1 per cent in doses ranging from 8 mg to 14 mg in 97 procedures, and lidocaine 5 per cent in doses ranging from 50 mg to 75 mg in nine procedures.

during surgery and precipitate the onset of autonomic hyperreflexia during recovery from anesthesia. Evacuation of these organs may be sufficient to alleviate the problem. If these measures are not effective, prompt use of pharmacological agents to control hypertension is indicated. Of these agents, sodium nitroprusside will probably be most effective in producing a rapid reduction in blood pressure.

SUMMARY

In our series of 219 surgical procedures, intra-operative autonomic hyperreflexia occurred during standby (i.e., physiological monitoring without the use of sedative drugs), topical anesthesia, intravenous sedation, lidocaine spinal anesthesia, and with nitrous oxide–narcotic general anesthesia (table 2), but also occurred postoperatively following both tetracaine spinal anesthesia and general anesthesia with halothane and enflurane. It occurred in nine of 72 patients with levels of injury at T5 or above, but also in two of 21 patients with levels of injury below T10. Treatment during surgery consisted of producing an adequate depth of anesthesia with a potent volatile anesthetic agent. Following surgery, treatment should be directed to removing the cause, if possible, or by the appropriate use of pharmacological agents to control the hypertension.

Arrhythmias

REPORT OF A CASE

Case 2. A 39-year-old man, scheduled for a transurethral sphincterotomy, sustained a cervical injury which rendered him quadriplegic five years prior to admission. On physical examination the blood pressure was 120/80 torr, pulse rate was 68 beats per min, and auscultation of the heart and lungs was normal. There was complete motor and sensory loss below the fifth cervical segment. Laboratory data and an electrocardiogram were within normal limits.

Spinal anesthesia was instituted at the L4-5 interspace with 8 mg tetracaine. Following the block, blood pressure was 100 torr systolic, and the pulse rate was 68 beats per min. Forty-five min after the induction of anesthesia, the patient, while talking to the anesthesiologist, suddenly became bradycardic, then asystolic and unresponsive. Blood pressure was not recordable. The patient responded rapidly to a precordial thump; 0.4 mg, intravenous atropine and intravenous fluids were administered, and the patient was placed in the Trendelenberg position. No further problems were reported and there were no postoperative sequelae.

The patient underwent insertion of a penile prosthesis one month later under spinal anesthesia with 12 mg tetracaine. After 30 min of stable anesthesia, the pulse rate decreased from 68 to 50 beats per min, with a decline in systolic pressure from 110 torr to 90 torr. No treatment was reported and there were no sequelae.

DISCUSSION

Electrocardiographic abnormalities are common during the acute phase of spinal cord injury, and include nodal escape beats, atrial fibrillation, premature ventricular beats, ventricular tachycardia, and ST-T wave changes consistent with subendocardial ischemia.[1] Welphy et al.[31] reported that tracheal suction in recently injured quadriplegics may result in reflex bradycardia and cardiac arrest. In chronic spinal cord injured patients during episodes of autonomic hyperreflexia, Kendrich[5] reported the occurrence of arrhythmias in 75 per cent of patients. Ectopic beats and sinus bradycardia were the disturbances most commonly seen. Supraventricular and ventricular ectopic beats have also been noted during halothane anesthesia of chronic spinal cord injured patients.[23]

Of 11 patients in our series who developed autonomic hyperreflexia, only one developed an arrhythmia (Case 1). As mentioned above, cardiac arrhythias were not observed in our patients receiving halothane or enflurane.

TABLE 4. Episodes of Bradycardia* Associated with Hypotension in 106 Spinal Anesthetics

Patient	Level	Procedure	Spinal Anesthetic Agent and Dose	Preoperative Pulse Rate (beats/min)	Pulse Rate during Bradycardia (beats/min)	Preoperative Blood Pressure (torr)†	Blood Pressure during Bradycardia (torr)†	Occurrence in Minutes after Induction of Anesthesia
12	C6	Transurethral sphincterotomy	Tetracaine, 10 mg	72	48	110	80	30
13	C6	Transurethral sphincterotomy	Tetracaine, 10 mg	80	50	100	75	40
14	C5	Transurethral sphincterotomy	Lidocaine, 75 mg	78	46	110	80	30
15	C5	Transurethral sphincterotomy	Tetracaine, 8 mg	68	Asystole	120	Not Recordable	45 (See Case 2)
15	C5	Insertion of penile prosthesis	Tetracaine, 12 mg	68	50	110	90	30 (See Case 2)
16	C4	Insertion of penile prosthesis	Tetracaine, 12 mg	68	52	120	80	35
17	T3	Transurethral sphincterotomy	Lidocaine, 50 mg	70	48	100	80	30
18	T10	Transurethral sphincterotomy	Tetracaine, 12 mg	85	42	110	60	45

* Bradycardia is defined as a pulse rate < 60 beats/min. All cases were sinus bradycardia.

† Systolic blood pressure.

We noted an 8 per cent incidence of bradycardia during spinal anesthesia which were followed by hypotension. They occurred with the administration of either lidocaine or tetracaine (table 4). All episodes of bradycardia occurred during urological procedures, 30 minutes or more after the induction of spinal anesthesia, and the onset of bradycardia in all cases was sudden, unpredictable, and followed a period of stable anesthesia. The most serious of these episodes is reported in Case 2, where bradycardia was followed by asystole. There was no change in surgical stimulus, patient position, or introduction of adjuvant anesthetic agents in any of our cases which might have accounted for the occurrence of bradycardia.

Bradycardia is a characteristic finding during spinal anesthesia in normal patients. To some extent, especially during high spinal anesthesia, bradycardia may be due to block of the cardiac accelerator fibers, but primarily it is caused by a decrease in central venous and right atrial pressures.[32] This has been called the Bainbridge reflex. Pulse rate during spinal anesthesia decreases proportionately to right atrial pressure, as does arterial blood pressure, which is dependent on venous return. From our series, we believe that chronic spinal cord injured patients are also susceptible to the Bainbridge reflex. The suddennesss and unpredictability of bradycardia during spinal anesthesia made constant monitoring very impor-

tant in this group of patients and early treatment of this arrhythmia is indicated.

Hypotension

REPORT OF A CASE

Case 3. A 57-year-old man with a C7 spinal cord transection, sustained two years prior to admission, was scheduled for percutaneous rhizotomy to relieve severe spasticity. On physical examination the blood pressure was 100/70 torr, pulse rate was 68 beats per min, and auscultation of the heart and lungs was normal. Severe spasm of the lower extremities and trunk occurred with mild cutaneous stimuli. Laboratory results were within normal limits. The patient was brought to the operating room unpremedicated. Anesthesia was induced with 300 mg sodium thiopental, 100 mg succinylcholine was administered and an endotracheal tube introduced into the trachea. Anesthesia was maintained with 2 per cent enflurane, in an equal mixture of nitrous oxide and oxygen, and ventilation was controlled to maintain normocarbia. After five minutes the patient was turned prone and the systolic blood pressure decreased almost immediately from 100 torr to 50 torr with no change in pulse rate. Rapid intravenous infusion of fluids and 10 mg ephedrine, and decreasing the depth of anesthesia, resulted in rapid recovery. There were no further problems during the procedure and no postoperative sequelae.

DISCUSSION

Patients with high levels of spinal cord injury experience postural hypotension during routine activity be-

TABLE 5. Episodes of Hypotension* Not Associated
with Bradycardia

	General Anesthesia	Spinal Anesthesia
Total Number of Cases	73	106
Number of Episodes of Hypotension	8	3

* Systolic blood pressure < 80 torr.

cause of instability of their vascular tone, increased venous capacitance, hypovolemia, and decreased venous return.[33] Hypotension not associated with bradycardia may occur during induction of anesthesia in spinal cord injured patients. The cause for this may be alterations in vascular tone, or the administration of a relative overdose of anesthetic agents. We observed hypotension (a systolic blood pressure equal to or less than 80 torr) on induction of general anesthesia in 11 per cent of our patients (table 5). The incidence of hypotension on induction of regional anesthesia was 3 per cent. All episodes of hypotension were transient, and most responded rapidly to intravenous fluids alone, except for two patients who required intravenous ephedrine to maintain their blood pressure. As illustrated by Case 3, controlled ventilation and abrupt changes in position in an anesthetized patient can cause further decreases in venous return, resulting in severe hypotension. Adequate hydration of these patients will help to avoid such problems. Alterations of posture should be minimized and patients observed carefully after such changes are made, so that hypotension may be recognized and treated.

Conclusions

Chronic spinal cord injured patients have disorders of many organ systems and present a unique challenge to the anesthesiologist. Autonomic hyperreflexia is a potentially life-threatening complication which may develop during surgery in these patients. The administration of general anesthesia with a potent volatile agent or spinal anesthesia with tetracaine were equally successful in preventing autonomic hyperreflexia in our patients. Hypotension on induction of anesthesia may occur with either of these techniques and is best avoided by adequate preoperative hydration, and avoidance of abrupt changes in position of overdosage of anesthetic agents. Bradycardia may occur suddenly during spinal anesthesia and may lead to profound hypotension. Treatment consists of the administration of atropine and increasing venous return by placing the patient in the Trendelenberg position and rapidly infusing intravenous fluids. Despite these complications we believe that the prevention of intraoperative autonomic hyperreflexia by

the anesthetic techniques described in preferable to the initiation of therapy after the syndrome develops. Increased awareness that autonomic hyperreflexia can occur during recovery from anesthesia should lead to the early diagnosis of this syndrome in the recovery room and the initiation of appropriate therapy. Cardiovascular complications during surgery in chronic spinal cord injured patients are common and potentially fatal. With increased awareness of their occurrence, their incidence can be minimized and anesthesia administered with increased safety.

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