Obstetric Forum

Obstetrical anaesthesia and analgesia in chronic spinal cord-injured women

Edward Crosby BSc MD FRCPC, Bernard St-Jean MD, Dennis Reid MB ChB FRCPC, Robert D. Elliott MD FRCPC

Improved acute and rehabilitative care and emphasis on integrating patients into society after spinal cord injury is likely to result in increasing numbers of cord-injured women presenting for obstetrical care. Anaesthetists providing care to these women should be familiar with the complications resulting from chronic cord injury and aware that many may be aggravated by the physiological changes of normal pregnancy. These complications include reduced respiratory volumes and reserve, decreased blood pressure and an increased incidence of thromboembolic phenomena, anaemia and recurrent urinary tract infections. Patients with cord lesions above the T_5 spinal level are at risk for the life-threatening complication of autonomic hyperreflexia (AH) which results from the loss of central regulation of the sympathetic nervous system below the level of the lesion. Sympathetic hyperactivity and hypertension result in response to noxious stimuli entering the cord below the level of the lesion. Labour appears to be a particularly noxious stimulus and patients with injuries above T_5 are at risk for AH during labour even if they have not had previous AH episodes. Morbidity is related to the degree of hypertension and intracranial haemorrhage has been reported during labour and attributed to AH. We report our experience in providing care to three parturients with spinal cord injuries. Two patients had high cervical lesions, one of whom experienced AH during labour and was treated with an epidural block. The second was at risk for AH having had episodes in the past and received an epidural block to provide prophylaxis for AH. In both cases epidural blockade provided effective treatment and prophylaxis for AH. The third patient had a low thoracic cord lesion, a comfortable labour but required an urgent Caesarean section performed under general anaesthesia for placental abruption and antepartum haemorrhage. The outcome of all three maternal-neonatal pairs was excellent. We recommend antepartum anaesthesia consultation for all cord-injured mothers and an epidural block as prophylaxis against AH in all parturients with cord lesions at T_5 or above.

L'amélioration des soins aigus et de réadaptation après une lésion de la moelle épinière et l'emphase mise sur l'intégration de ces patients dans la société ont résulté en un nombre croissant de femmes avec atteinte médullaire qui se présentent pour des soins obstétricaux. Les anesthésistes prodiguant des soins à ces femmes devraient être familiers avec les complications résultant d'une lésion médullaire chronique et être conscients que plusieurs peuvent être aggravées par les changements physiologiques d'une grossesse normale. Ces complications incluent une réserve et des volumes respiratoires diminués, une tension artérielle diminuée et une incidence accrue de phénomènes thromboemboliques, de l'anémie et des infections urinaires récurrentes. Les patientes atteintes de lésions médullaires au-dessus du niveau T_5 sont à risque pour la complication potentiellement mortelle qu'est l'hyperréflexie autonomique (HA) résultant de la perte de régulation centrale du système nerveux sympathique sous le niveau de la lésion. Une hyperactivité sympathique et de l'hypertension sont observées en réponse à des stimuli nocifs entrant dans la moelle épinière sous le niveau de la lésion. Le travail semble être un stimulus particulièrement nocif et les patientes avec des lésions au-dessus de T_5 sont à risque pour l'HA durant le travail même si elles n'ont jamais présenté d'épisodes préalables de HA. La morbidité est reliée au degré d'hypertension et la possibilité d'hémorragie intracrânienne a été rapportée durant le travail et attribuée à l'HA. Nous rapportons notre expérience avec trois parturientes ayant des lésions médullaires. Deux patientes avaient des lésions cervicales hautes. Une de celles-ci a présenté de l'HA durant le travail et fut

Key words
ANAESTHESIA: obstetrical;
COMPLICATIONS: spinal cord injury, autonomic hyperreflexia.

From the Department of Anaesthesia, University of Ottawa, Ottawa General Hospital, Module A-11, 501 Smyth Road, Ottawa, Ontario K1H 8L6.

Address correspondence to: Dr. E. Crosby.

traitée avec un bloc épidural. La deuxième, étant à risque pour l'HA puisqu'elle avait déjà eu des épisodes, a reçu un bloc épidural afin de procurer une prophylaxie pour l'HA. Dans les deux cas, le bloc épidural a été un traitement et une prophylaxie efficaces pour l'HA. La troisième patiente avait une lésion médullaire thoracique basse mais, après un travail « en douceur », nécessita une Césarienne d'urgence sous anesthésie générale en raison d'un décollement placentaire et d'une hémorragie durant le travail. L'issue fut excellente pour les trois mères et leurs nouveaux-nés. Nous recommandons une consultation prénatale en anesthésie pour toutes les mères porteuses d'une lésion médullaire et un bloc épidural comme prophylaxie contre l'HA chez toutes les parturientes ayant des lésions médullaires au niveau de T_5 ou au-dessus.

Spinal injuries occur with an incidence of 280 per million population and 10–30% result in cord damage.[1] Spinal cord injury is primarily a disease of the young and in Canada 20% of the victims are female.[2] Advances in both acute and rehabilitative care have led to improvements in outcome with victims able to attain higher levels of independent function after spinal cord injury than was previously possible. Rehabilitation emphasizes integration back into society and cord-damaged patients are encouraged to work, establish relationships and found or continue families. Pregnancy in cord-injured women is no longer rare and increasing numbers of anaesthetists will be providing care for these women. In order to provide optimum medical care, familiarity with the particular problems of pregnancy, labour and delivery in cord-damaged women is necessary. We present three cases of cord-injured parturients recently cared for at our institution, provide an overview of the recent literature with respect to the problems that have been encountered during labour and delivery in these patients and provide some recommendations for care.

Case reports

Case 1 was a 25-yr-old primigravida admitted at 41 wk gestation, in active labour. She had been paraplegic for ten years following a parachuting accident in which she had suffered a compression fracture of the thoracic spine and a cord injury at the T_5 level. She had stabilization of her spinal fracture by Harrington rod insertion and the Harrington rod was removed two years before this admission. She was not known to have had an episode of autonomic hyperreflexia previously. Her pregnancy had been uncomplicated although she had recurrent urinary tract infections. At the time of admission her blood pressure (BP) was 90/60 mmHg and a singleton fetus was in the vertex presentation at spines minus 1 cm, with the cervix dilated at 2–3 cm. An oxytocin infusion was started to augment labour and the subsequent course of labour was marked by a progressive increase in BP from 90/60 to 180/127 mmHg, associated with severe headache. The fetal heart rate was stable between 126 to 147 bpm during this period. The oxytocin infusion was discontinued and an anaesthetic consultation was requested.

When seen by the anaesthetist the patient was in severe distress complaining of the most severe headache she had ever experienced and her BP was 167/87 mmHg. Her pupils were equal and reactive to light and she had no neck rigidity. The skin on the lower part of her body was cool but she had warm, flushed skin on the upper part of the body. A diagnosis of autonomic hyperreflexia (AH) was made. Intravenous fentanyl, 100 μg in divided doses was administered for symptomatic relief of the headache. Both the condition and the implications of AH were explained to the patient and an epidural block was recommended. The patient agreed and following administration of 1.5 L of fluid *iv*, an epidural catheter was inserted through the L_{3-4} interspace. A number of attempts was made as there was difficulty in passing the catheter once the space had been entered. After a negative test dose of 3 ml lidocaine, 1.5%, with 15 μg epinephrine, a second aliquot of 3 ml lidocaine 1.5% with 15 μg of epinephrine was given. Fifteen minutes later 3 ml bupivacaine 0.25% were administered and this was repeated after a further 15 min. Some lability of the blood pressure was observed with onset of the epidural block with systolic blood pressure varying between 67 and 90 mmHg. Blood pressure was maintained over 90 mmHg systolic with repeated small doses of intravenous ephedrine. Thirty minutes after the block had been performed the blood pressure had stabilized at 120 mmHg systolic and the headache was considerably diminished. One hour later a 3410 g female infant was delivered with vacuum extraction. The infant was well with Apgar scores of 8 and 9 at one and five minutes respectively. The epidural block was permitted to wear off, the blood pressure remained stable, and the headache resolved spontaneously over the next 24 hr.

Case 2 was a 26-yr-old primiparous woman who was referred for anaesthetic consultation at 24 wk gestation. She became quadriparetic after a diving injury seven years previously, which had resulted in an anterior cord syndrome at the C_{6-7} spinal level. She had preserved sensation below the level of the lesion but discriminative abilities were poor. She had experienced a well-documented episode of autonomic hyperreflexia in the past during a cystourethrogram performed under local anaesthesia, during which her blood pressure was recorded at 180/120 mmHg. She reported facial flushing with bladder distention which was also felt to represent AH. Her perinatologists planned to allow the pregnancy to continue to term and, following confirmation of both a mature fetus and a

favourable cervix, to induce labour electively. It was our opinion that the patient was at high risk for an AH episode during labour and that this could best be prevented by establishment of an epidural block before labour induction. In addition, because of the nature of the cord injury (anterior cord syndrome), despite the level of the injury, it was considered that there was some potential for painful labour and the epidural would provide optimal analgesia.

The patient was admitted electively at term and the following morning was taken to the labour and delivery suite. A large gauge intravenous catheter was placed in a forearm vein and a fluid bolus of 750 ml of lactated Ringer's solution was administered. An automated blood pressure cuff was placed on the opposite arm. With the patient supported in the sitting position, the skin in the L_{3-4} interspace was infiltrated with local anaesthetic. Prominent local reflexes resulted in uncontrolled paravertebral muscle spasms and so a field block was established. This resulted in excellent conditions and the catheter was inserted into the epidural space without further difficulty. Following a negative test dose of 3 ml lidocaine 1.5% with 15 μg epinephrine, 75 mg of preservative-free meperidine in ten ml of normal saline were administered through the catheter and an infusion of bupivacaine 0.125% and 4 $\mu g \cdot ml^{-1}$ fentanyl was started at 6 $ml \cdot hr^{-1}$. Artificial rupture of the membranes followed and a fetal scalp clip was placed for continuous fetal heart rate monitoring. Oxytocin augmentation was commenced after six hours and with the increased intensity of the contractions the patient experienced some breakthrough pain. This was managed by increasing the infusion rate to 8 $ml \cdot hr^{-1}$ with good effect. Eight hours later the patient delivered a healthy female infant, assisted by vacuum extraction. Maternal blood pressure had ranged between 120/60 and 140/80 mmHg throughout labour. The postpartum course was notable only for transient neonatal hyperbilirubinaemia and mother and infant were discharged home four days postpartum.

Case 3 was a 36-yr-old primiparous woman who became paraplegic after a motor vehicle accident 21 yr previously which had resulted in a complete cord lesion at T_{11-12}. She was initially seen in consultation at 14 wk gestation for assessment of AH potential and a discussion of labour analgesia options. She was considered to be at low risk for AH and, because of the level of her injury, analgesia would not likely be required. She was admitted electively at 38 wk gestation and, following confirmation of fetal maturity, labour was induced. Labour progressed slowly and oxytocin augmentation was employed. There was little, if any, maternal awareness of contractions and no discomfort. After 12 hr of labour she began passing blood clots and these increased in frequency and volume over the next several hours and were followed by a gush of liquid blood. A tentative diagnosis of placental abruption was made and she underwent urgent Caesarean section under general anaesthesia. Placental abruption was confirmed at Caesarean section and a healthy male infant was delivered. The early postpartum period was uneventful for both mother and child and both were discharged home on the fifth day after delivery.

TABLE Medical complications in spinal cord-injured women aggravated by pregnancy

Pulmonary
Decreased respiratory reserve
Atelectasis and pneumonia
Impaired cough
Haematological
Anaemia
Thromboembolic phenomena
– Deep vein thrombosis
Urogenital
Chronic urinary tract infections
Urinary tract calculi
Proteinuria
Renal insufficiency
Dermatological
Decubitus ulcers
Cardiovascular
Hypotension
Autonomic hyperreflexia

Discussion

Pregnancy in chronic spinal cord-injured women is becoming more common because of improved acute medical care and rehabilitation. Physicians providing care to parturients should be aware of the medical complications of spinal cord injury since many may be aggravated by pregnancy (Table). Decreased respiratory reserve and less forceful cough may result from impaired or absent intercostal and abdominal muscle function due to cord injury and may lead to atelectasis and pneumonia. These changes are aggravated in pregnancy by the further loss of both functional residual capacity and expiratory reserve volume and by the requirement for an increased minute ventilation to support the increased oxygen consumption occurring in the pregnant state. *Muscle atrophy and osteoporosis* result from persistent disuse following cord injury and may result in pathological fractures. Patient transfers must be carried out with care. Self-transfers will also be more difficult with the weight gain as the pregnancy progresses and all three of our patients experienced this. *Anaemia* is common in cord-injured patients and the haematocrit will be further lowered by the physiological

anaemia of pregnancy. Although oral iron supplementation may be useful, it may further compromise bowel function already inhibited by both the cord injury and the pregnancy. *Thromboembolic phenomena*, especially deep vein thrombosis (DVT), are common in both cord-injured patients and in pregnancy and there is a clinical impression that DVT is more common when the two are associated. Two of our patients had experienced DVT in the past. *Chronic bacteruria and urinary tract infections* are common and may result in urinary tract calculi, renal insufficiency and proteinuria. Albuminuria is an early sign of renal compromise. With intermittent catheterization replacing indwelling catheters, urinary tract problems may be less than in the past. *Decubitus ulcers* are common and may result from as little as two hours of sustained pressure on a site. Skin care must be meticulous, especially in pregnancy, as the pressure on the skin will be augmented by weight gain. Cord-injured patients are poikilothermic below the level of the lesion as they are unable to regulate skin blood flow or shivering and care must be taken to avoid exposing the patients to overheated or cooled environments. *Hypotension* is common in cord-injured patients and reflects impaired autoregulatory function. It is most prominent in patients with high cord injuries as they have lost both the ability to vasoconstrict the splanchnic bed (T_5-L_2) to prevent orthostatic decreases in blood pressure as well as the ability to increase heart rate reflexly via the cardioaccelerator fibres (T_{1-4}). The tendency towards orthostatic decreases in blood pressure may be further aggravated in pregnancy by the hormonally induced decrease in systemic vascular resistance.

Autonomic hyperreflexia (AH) is a life-threatening complication in high cord-injured patients that may present during labour and delivery. It is very common in patients with cord injuries at T_5 or above, is less common in lesions from T_{5-8} and is uncommon when the lesion is below T_{8-10}.[3] Patients with incomplete lesions that preserve ascending pathways while obliterating descending pathways may remain at risk despite lower levels of injury. Patients whose injuries have resulted in cord infarction below the level of injury are at little risk despite the level of injury. Autonomic hyperreflexic episodes occur as a result of loss of the modulating influences of the central nuclei on that portion of the cord lying below the level of the lesion. In an episode of AH, noxious sensory stimuli are transmitted via peripheral nerves to the dorsal horn of the spinal cord (Figure). These afferent neurons synapse, either directly or via interneurons, with sympathetic neurons in the intermediolateral columns of the lateral horns which then project via the anterior roots to the paraspinal sympathetic chain. The sympathetic impulse is both propagated peripherally at that spinal level and, as well, travels both cephalad and caudad in the sympathetic chain and exits at multiple thoracic and lumbar levels. The resultant sympathetic hyperactivity leads to vasoconstriction, and visceral spasm. If the cord lesion is below the midthoracic level then sufficient centrally mediated vasodilator reserve may be present to compensate for the vasoconstriction. With higher cord injuries there is insufficient centrally mediated vasodilator reserve and the vasoconstriction results in severe systemic hypertension. The hypertension is sensed in the baroreceptors of the aortic and carotid bodies and reflex arcs projecting through supraspinal nuclei result in bradycardia and vasodilatation above the level of the spinal lesion. These phenomena are not usually sufficient to compensate for the vasoconstriction and the result is an episode of AH characterized by hypertension and bradycardia. Symptoms resulting from the centrally mediated vasodilatation include profuse sweating, blurred vision, increased cutaneous temperature, facial flushing and nasal congestion. These associated symptoms may be mild and the diagnosis missed.

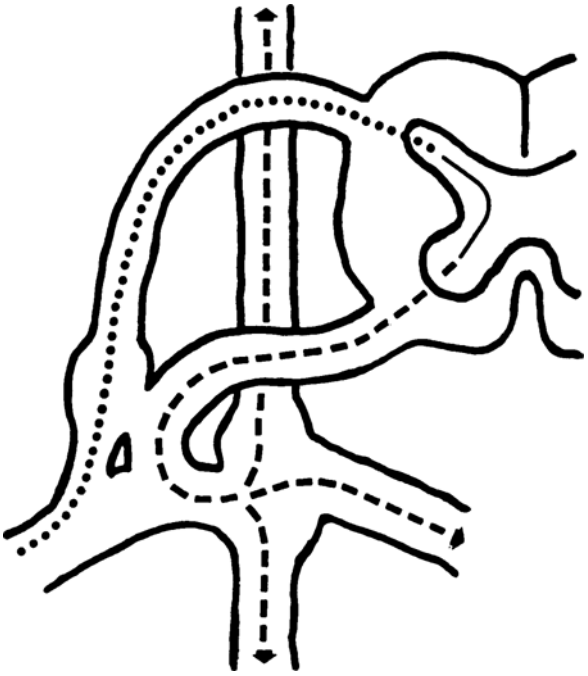

FIGURE Noxious stimuli enter the dorsal horn of spinal cord via the dorsal spinal root (dotted line). These afferent neurons synapse, either directly or via interneurons (solid line), with sympathetic neurons in the intermediolateral columns of the lateral horns which then project via the anterior roots to the paraspinal sympathetic chain (dashed line). The impulse is both propagated peripherally at that spinal level and, as well, travels both cephalad and caudad in the sympathetic chain exiting at mutiple thoracic and lumbar levels (dashed line) and resulting in sympathetic hyperactivity.

The morbidity of AH appears to be related primarily to the degree of hypertension. The elevation in blood pressure may be extreme and intracranial haemorrhage has been reported recently in two labouring cord-injured women and attributed to episodes of AH.[4,5] Rossier *et al.*

noted that when labour is the provoking stimulus for AH, considerable increase in blood pressure may occur concurrent with contractions and be followed by a rapid decrease as the contraction abates.[6] Electrocardiographic changes associated with AH during labour reflect increased vagal activity and include AV conduction abnormalities with first and second degree heart block and sinus arrest.[7] Fetal heart rate monitoring has revealed bursts of tachycardia coincident with episodes of maternal AH but these heart rate changes have not been associated with fetal acidosis.[8]

It is estimated that 85–90% of parturients with cord injuries at T_6 or above are at risk for AH during labour and delivery.[3] Labour is a particularly potent stimulus to the development of AH and even patients who give no previous history of AH may be at risk during labour, as evidenced by our first patient. Although labour, in general, is a potent stimulus for AH, amniotomy, augmentation with oxytocin infusion, perineal distention and vaginal instrumentation may be particularly noxious stimuli.[2,5,9,10] The lower the spinal segment receiving the afferent stimulation, the more potent the stimulus, the greater the autonomic response and the more severe the hypertension.[11] Maximum reaction occurs with the anogenital area innervated by S_{2-4}.[11] Thus, it is possible that AH could present in the late first stage or early second stage as stretching of the perineum produces additional stimulation of the pudendal nerve entering the cord at S_{2-4}, despite an uneventful course of labour to that point in time. McGregor and Meeuswen reported intracranial haemorrhage in a C_{6-7}-injured parturient, without a known history of AH.[5] She appeared to be tolerating labour well until full cervical dilatation when she suffered severe neurological deterioration with coma. Her blood pressure was noted to be between 170/110 and 210/100 mmHg at the time. She was left with extensive central neurological dysfunction in addition to her preexistent C_{6-7} quadriplegia.

Reported treatments for episodes of AH during labour and delivery vary, reflecting the experience and practice of the physician managing the patient, and have emphasized the use of anxiolytics, arteriolar vasodilators and epidural anaesthesia.[12] Young reported four pregnancies in three women with cord injuries, all of whom experienced AH during labour and delivery.[8] The patients were treated with anxiolytics and intravenous hydralazine and although the outcome was good in all four cases, BP control was poor with recorded pressures as high as 240/140 mmHg. Ravindran reported on the use of sodium nitroprusside to treat AH in a quadriplegic parturient during labour.[9] Despite good BP control during contractions and alleviation of the symptoms of AH, vasopressors were required following cessation of the contractions to treat hypotension. The nitroprusside therapy was abandoned and an epidural block was instituted with good effect.

There seems to be a consensus developing that optimum propylaxis and management of AH during labour includes an epidural block.[2] Watson and Stirt have reported onset of signs and symptoms of AH in parturients in whom epidural catheters were placed but were not to be activated until evidence of AH developed.[13,14] The potential for maternal morbidity with this management schema should be considered. With respect to the use of epidural blockade in these patients, some issues that have been raised by authors include: (1) appropriate levels of monitoring and the use of invasive haemodynamic monitors; (2) the use of regional block in patients with neurological impairment; (3) the use of regional block in patients with previous back surgery (stabilization procedures following traumatic injury); (4) the institution of an epidural block in patients with low baseline systolic pressures; (5) how best to monitor and control the level of the block; and (6) uncertainty as to the most appropriate agent for establishment of epidural analgesia.

Invasive haemodynamic monitoring including radial artery and central venous catheters has been recommended by some authors as routine monitors.[2,9,10,15] MacGregor and others appear satisfied with non-invasive haemodynamic monitoring reserving the invasive modalities to situations where they are specifically indicated.[5] This is the approach to which we subscribe.

It is our impression that there is no contraindication to the use of regional blocks in patients with preexisting neurological impairment. A discussion of risks and benefits of epidural analgesia should take place with the patient during an antepartum anaesthetic consultation.[16] There is no contraindication to the use of epidural analgesia in patients who have had previous back surgery including those who have spinal instrumentation *in situ*.[17,18] The block may be more difficult to perform and may be associated with a higher incidence of incomplete block, failed block, and perhaps dural puncture. This information should be related to the patient during antepartum consultation. Despite the low baseline blood pressures recorded in some patients, epidural blockade appears to be haemodynamically well tolerated if it is induced after appropriate intravascular volume expansion. Slow establishment of epidural blockade using volumes of anaesthetic agents appropriate for the desired level of block should result in a low incidence of hypotension. Use of higher volumes of more dilute agents, may serve to reduce the incidence of hypotensive events during establishment of the block.

Intraspinal meperidine has analgesic and anaesthetic effects in humans[19] and epidural meperidine has been used to treat AH in labour with good effect.[20] Fentanyl, although an analogue of demerol, lacks its local anaesthetic properties, and has been demonstrated to be an ineffective prophylactic agent for AH, when used alone in the epidural space.[21] Initial administration of epidural

meperidine followed by a continuous infusion of a combination of fentanyl and dilute bupivacaine (0.125%) provided effective prophylaxis against AH in one of our patients with a history of previous AH episodes. The use of intraspinal narcotics and continuous infusion of dilute anaesthetic-narcotic combinations may provide adequate prophylaxis against AH with a low incidence of hypotension. Obviously, application of this technique to a larger number of at-risk patients is required before a definitive conclusion is reached.

Recommendations for care

All spinal cord-injured parturients should be seen for antepartum anaesthetic consultation. Patients with lesions below the level of T_{10} are likely to have pain during labour. Those with complete lesions above T_{10} but below T_5 are likely to experience painless labour but the patient may be able to detect uterine contractions because of increased spasticity, flexor spasms or clonus. These patients should be closely questioned to determine if previous episodes of AH have occurred. If so, they should be treated as being at risk during labour. Patients with incomplete lessons from T_{5-10} may experience pain with labour if ascending spinal tracts are preserved. Finally, in patients with lesions above T_5, there is a high risk of AH during labour even if the patient has not previously experienced an episode of AH. Depending on the anticipated course of labour, the mother should be counselled on the available methods of analgesia as well as prophylaxis for AH. It is our opinion that epidural analgesia should be recommended to women at risk for AH during labour and that a discussion of the benefits and risks of epidural block should occur during the antepartum visit. An evaluation of pulmonary function can be made during antepartum consultation. Good pulmonary function and reserves should be anticipated in most of these patients. In patients whose pulmonary function is thought to be compromised, arterial blood gas analysis, spirometric pulmonary function tests and respirology consultation are recommended. Supplemental oxygenation and pulse oximetry during labour may be indicated in this latter group.

Current obstetrical opinion recommends weekly cervical examinations in cord-injured patients beginning at 28 wk, with admission to hospital once cervical dilatation or effacement is detected. Caesarean delivery is appropriately restricted to circumstances in which an obstetrical indication exists. Should an abdominal delivery be required in a patient with an epidural catheter *in situ*, 14-16 ml carbonated lidocaine with 5 $\mu g \cdot ml^{-1}$ – epinephrine should provide a safe level of surgical anaesthesia. Patients with high level injuries often undergo induction of labour and frequently receive augmentation of labour with oxytocin infusions. In patients at risk for AH it is our opinion that, before induction or augmentation or as soon as the patient presents in labour, the patient should have an epidural catheter inserted, tested and activated as prophylaxis for AH. Following appropriate intravenous prehydration (20 $ml \cdot kg^{-1}$), epidural block may be instituted with either meperidine (1 $mg \cdot kg^{-1}$ in 10 ml preservative-free saline), dilute bupivacaine (8–10 ml, 0.125%) or a combination of fentanyl and bupivacaine (6–8 ml, 0.125% bupivacaine with 2–5 $\mu g \cdot ml^{-1}$ fentanyl). A continuous infusion of dilute bupivacaine or a fentanyl-bupivacaine combination at 7–8 $ml \cdot hr^{-1}$ should then be started and continued until delivery. The best concentration is the lowest that is required to prevent AH. Increasing rates of infusion and the use of more concentrated solutions of local anaesthetic (bupivacaine 0.25%) may be employed, if necessary, to treat AH, augment AH prophylaxis or provide more intense analgesia if the more dilute concentrations are insufficient. This protocol can be applied, as well, to the patient with lower level injury experiencing pain with contractions. Invasive haemodynamic monitoring is only required when there are specific indications for either arterial or central venous catheters but should not represent routine intrapartum management of cord-injured parturients. However, frequent blood pressure measurement is essential.

With respect to the nursing management, the nurse attending the at-risk patient should be informed of the syndrome and its presenting signs and symptoms. Frequent turning should be emphasized to minimize pressure points on the skin. Intermittent catheterization should be carried out to prevent bladder distension and the bladder should be assessed after delivery to detect latent distension masked by residual neural blockade.

Careful antepartum assessment and knowledgeable intrapartum management of cord-injured patients and cooperation between perinatal, anaesthetic and nursing personnel will result in good maternal-neonatal outcomes in cord-injured patients. Preparations for care must be initiated long before the crisis of autonomic hyperreflexia supervenes.

Acknowledgment

The authors would like to acknowledge the assistance of Sylvie Paquette in the preparation of this manuscript.

COMMENTARY

A.A.S. Kamani MD FRCPC
Department of Anaesthesia, Grace Hospital and University of British Columbia

Chronic spinal cord-injured parturients present many challenges to the anaesthetist caring for them. Crosby *et al.*

have produced an excellent summary of the management of such parturients.

There are several important points to remember in attempting epidural anaesthesia in these patients. Technically, the insertion of the epidural catheter may be difficult. Positioning of the parturient may be impeded by muscle spasm and inability to maintain a certain position. Assistance is therefore necessary. As well, many of these patients have had spinal stabilization procedures which increase the technical difficulty. Alternative techniques such as the use of intrathecal narcotics or intrathecal local anaesthetics may need to be considered. Better fixation of epidural catheters is also important as tape may become loose if the parturient develops autonomic hyperreflexia (AH). Tincture of benzoin improves the adhesiveness of tape. Some patients will have haemodynamic instability and local anaesthetics should be administered incrementally.

Both pregnancy and chronic spinal cord injury are associated with an increased incidence of urinary tract infection (UTI). A recent report suggests that UTI is associated with the onset of preterm labour,[1,2] which may occur silently in these women.

Autonomic hyperreflexia could be a presenting symptom of the onset of labour. It is the most serious potential complication in these patients. We have had two patients who developed AH within the first 24 hr postpartum. The stimulus for AH was assumed to be uterine involution in one (vaginal delivery) and gastrointestinal dilatation in the other (Caesarean section). In our institution we now maintain the epidural catheters for 24–48 hr postpartum and closely monitor these patients in a high-risk setting.

REFERENCES (Kamani)

1 *McGregor JA.* Prevention of preterm birth: new initiatives based on microbial-host interactions. Obstet Gynecol Surv 1988, 43: 1–14.

2 *Hillier SL, Martius J, Krohn M, Kiviat N, Holmeskk, Eschenbach DA.* A case-control study of chorioamniotic infection and histologic chorioamnionitis in prematurity. N Engl J Med 1988; 13: 972–8.

COMMENTARY

Bill Y. Ong MD FRCPC
Department of Anaesthesia, Women's Hospital and University of Manitoba

Patients with spinal cord injury can be divided into two groups: acutely and chronically injured. The major problems for patients with acute spinal cord injuries are ventilatory insufficiency, hypotension, and associated injuries. Most of the stable compression fractures of the spine can be managed with bed rest until the patient is delivered. For patients with unstable fractures, surgical interventions might be needed. Semi-elective surgery can be performed safely during the second and third trimesters which introduces the additional considerations of the anaesthetic management during pregnancy for non-obstetrical surgery. In acutely quadriplegic or paraplegic parturients, neurological changes due to primary disease may be mistaken as anaesthetic complications. In this situation, I employ regional anaesthesia only if there is a strong indication and only after full discussion with the patient. Patients with acute spinal injuries with a duration of 24 hr to six months are at high risk for marked elevation of serum potassium concentration after succinylcholine administration. Patients with cervical spine injuries need special attention in airway and ventilatory management. Some are hypoxaemic and require increased F_IO_2 to maintain adequate arterial oxygen saturation.

In patients with chronic spinal cord injuries, the management of regional anaesthesia can be arduous. Anatomical changes due to spinal injuries can make placement of needles and catheters difficult. The loss of sensation makes it impossible to determine whether the medication has been administered into the correct space until blockade has extended into the uninjured area.

There is no evidence to suggest that regional anaesthesia will cause further injuries in patients with stable neurological function. It must be pointed out to the patients in antepartum consultation that neurological injuries involving peripheral nerves can still occur with pregnancy, with obstetrical interventions, and with herniated lumbar discs which are not related to anaesthetic care.[1]

In terms of patients with other types of spinal cord pathology, the literature is more controversial. Patients with a history of multiple sclerosis may experience postpartum relapses. Bader *et al.* questioned whether bupivacaine at concentrations greater than 0.25% over a longer period of time can lead to more relapses in patients with multiple sclerosis.[2] It is best to discuss the matter with patients before the onset of labour, and to make a choice in each case based on possible benefits and risks.

Continuous epidural anaesthesia is an effective method for the prevention of autonomic hyperreflexia during labour and delivery. Although meperidine has been reported to be efficacious for this purpose, fentanyl was not. At this time, it appears that agents with local anaesthetic effects are the most appropriate. Further experience is needed to compare dilute concentrations of local anaesthetic combined with fentanyl against local anaesthetic only. Since AH is caused by distension of uterus, bladder, or rectum, the epidural should be maintained until functions in all these organs have returned or distension can be prevented.

Since severe hypertension with AH can lead to cerebrovascular accidents and deaths, prevention of this response is critical. Although patients with lesions above T_6 are known to be of high risk, patients with lesions between T_{10} and T_6 also should be considered to be potentially at risk. All patients who have had a history of AH should be managed like patients with lesions above T_5. Since it is difficult to determine the exact level of epidural anaesthesia, intravenous vasodilators should also be immediately available for treatment of hypertension should that occur.

In terms of monitoring, an arterial catheter is probably not needed as a routine. Patients with concomitant illnesses, such as preeclampsia, may require more invasive monitoring. There are alternatives for continuous non-invasive blood pressure monitoring such as the Finapres™ monitor. Often, the patient is knowledgable about the hyperreflexic response and can recognize the symptoms very quickly. If she is not obtunded by other medications, she can assist in monitoring herself. Continuous oxygen saturation and heart rate monitoring may be beneficial for quadriplegic patients. Some of these patients have pulmonary dysfunction, and decreased arterial oxygen saturation even before pregnancy. In the postpartum period, the paraplegic patients are at increased risk for urinary tract infection, thrombophlebitis, pulmonary embolism, and decubitus ulcer. Anaesthetic interventions must not delay mobilization after delivery.

REFERENCES (Ong)

1 *Ong BY, Cohen MM, Esmail A, et al.* Parathesias and motor dysfunction after labor and delivery. Anaesth Analg 1987; 66: 18–22.

2 *Bader AM, Hunt CO, Datta S, et al.* Anesthesia for the obstetric patient with multiple sclerosis. Journal of Clinical Anesthesia 1988; 1: 21–4.

References

1 *Kalsbeek WD, McLaurin RL, Harris BSH, Miller JD.* National head and spinal cord injury survey: major findings. J Neurosurg 1980; 53: S19–31.

2 *Boucher M, Santerre L, Menard L, Sabbah R.* Epidural and labor in paraplegics. Can J Obstet Gynecol 1991; 3: 130–2.

3 *Schonwald G, Fish KJ, Perkash I.* Cardiovascular complications during anesthesia in chronic spinal cord injured patients. Anesthesiology 1981; 55: 550–8.

4 *Abouleish E.* Hypertension in a paraplegic parturient. Anesthesiology 1980; 53: 348–9.

5 *McGregor JA, Meeuwsen J.* Autonomic hyperreflexia: a mortal danger for spinal cord-damaged women in labour. Am J Obstet Gynecol 1985; 151: 330–2.

6 *Rossier AB, Ruffieux M, Ziegler WH.* Pregnancy and labour in high traumatic spinal cord lesions. Paraplegia 1969; 7: 210–6.

7 *Erickson RP.* Autonomic hyperreflexia: pathophysiology and medical management. Arch Phys Med Rehabil 1980; 61: 431–40.

8 *Young BK, Katz M, Klein SA.* Pregnancy after spinal cord injury: altered maternal and fetal response to labour. Obstet Gynecol 1983; 62: 59–63.

9 *Ravindran RS, Cummins DF, Smith IE.* Experience with the use of nitroprusside and subsequent epidural analgesia in a pregnant quadriplegic patient. Anesth Analg 1981; 60: 61–3.

10 *Tabsh KMA, Brinkman CR III, Reff RA.* Autonomic dysreflexia in pregnancy. Obstet Gynecol 1982; 60: 119–21.

11 *Bors E, French JD.* Management of paroxysmal hypertension following injuries to the cervical and upper thoracic segments of spinal cord. Arch Surg 1952; 64: 803–12.

12 *Verduyn WH.* Spinal cord injured women, pregnancy and delivery. Paraplegia 1986; 24: 231–40.

13 *Watson D, Downey GO.* Epidural anesthesia for labour and delivery of twins of a paraplegic mother. Anesthesiology 1980; 52: 259–61.

14 *Stirt JA, Marco A, Conklin KA.* Obstetric anaesthesia for a quadriplegic patient with autonomic hyperreflexia. Anesthesiology 1979; 51: 560–2.

15 *Greespoon JS, Paul RH.* Paraplegia and quadriplegia: special considerations during pregnancy, labour and delivery. Am J Obstet Gynecol 1986; 155: 738–41.

16 *Cheek TG, Banner RN.* Orthopaedic/neurologic diseases. Problems in Anesthesia 1989; 3: 112–29.

17 *Crosby ET, Halpern SH.* Obstetric epidural anaesthesia in patients with Harrington instrumentation. Can J Anaesth 1989; 36: 693–6.

18 *Daley MD, Relbin SH, Hew EM, Morningstar BA, Stewart JA.* Epidural anesthesia for obstetrics after spinal surgery. Regional Anesthesia 1990; 75: 280–4.

19 *Naguib M, Famewo CE, Absood A.* Pharmacokinetics of meperidine in spinal anaesthesia. Can Anaesth 1986; 33: 162–6.

20 *Baraka A.* Epidural meperidine for control of autonomic hyperreflexia in a paraplegic patient. Anesthesiology 1985; 62: 688–90.

21 *Abouleish EI, Hanley ES, Palmer SM.* Can epidural fentanyl control autonomic hyperreflexia in a quadriplegic patient? Anesth Analg 1989; 68: 523–6.