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Hemodynamic and Catecholamine Responses to Laryngoscopy and Tracheal Intubation in Patients with Complete Spinal Cord Injuries

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Background: **Endotracheal intubation in patients undergoing general anesthesia often causes hypertension and tachycardia, which may be altered when the efferent sympathetic fiber to the cardiovascular system is interrupted. The aim of the current study was to investigate the effects of different levels of spinal cord injury on the cardiovascular responses to intubation.**

Methods: **Fifty-four patients with traumatic complete cord injuries requiring tracheal intubation were grouped into quadriplegics (above C7; n = 22), high paraplegics (T1–T4, n = 8), and low paraplegics (below T5, n = 24) according to the level of injury. Twenty patients without spinal injury served as controls. Arterial pressure, heart rate, and rhythm were recorded at intervals for up to 5 min after intubation. Plasma concentrations of catecholamines were also measured.**

Results: **The intubation increased the systolic blood pressure similarly in control, high-paraplegic, and low-paraplegic groups ($P < 0.05$), whereas it did not alter the blood pressure in the quadriplegic group. Heart rate was significantly increased in all groups; however, the magnitude was more pronounced in the high-paraplegic group (67%) than in the control (38%) and quadriplegic (33%) groups. Plasma concentrations of norepinephrine were significantly increased after intubation in all groups; however, values were lower in the quadriplegic group and higher in the low-paraplegic group compared with those in the control group. Incidence of arrhythmias did not differ among groups.**

Conclusions: **The cardiovascular and plasma catecholamine changes associated with endotracheal intubation may differ according to the affected level in patients with complete spinal cord injuries.**

LARYNGOSCOPY and tracheal intubation are often associated with tachycardia, hypertension, and arrhythmias.[1] The mechanisms underlying the hemodynamic responses are not completely understood, although they have been attributed to a reflex sympathetic discharge caused by stimulation of the upper respiratory tract. This speculation is supported by the previous observation that hemodynamic responses to tracheal intubation are associated with an increase in plasma catecholamine concentrations[2,3] and are attenuated by β-adrenergic blockade.[4]

The efferent sympathetic outflow to the heart originates from the spinal cord between T1 and T4, and that to the adrenal medulla from between T3 and L3.[5,6] The outflow may, in turn, be modulated by supraspinal centers. Therefore, the cardiovascular responses to laryngoscopy and tracheal intubation may be altered in cord injuries. Indeed, Wattwil *et al.*[7] observed that cardiovascular response to intubation is abolished in patients undergoing total thoracolumbar anesthesia. In contrast, the blockade of the sympathetic outflow by epidural anesthesia, either cervicothoracic anesthesia without blocking the adrenal gland or lumbar epidural anesthesia without blocking the heart, did not affect the cardiovascular response to tracheal intubation.[8]

Cardiovascular dysfunction associated with spinal cord injury or transection depends on the site, extent, and timing of the lesion.[9–11] However, the impact of different levels of injury on the cardiovascular response to endotracheal intubation has not been determined. The aim of the current study was to investigate the effects of different levels of spinal cord injury on the cardiovascular responses to laryngoscopy and endotracheal intubation.

Materials and Methods

The protocol of the study was approved by the Chonnam National University Hospital Ethics Committee, Gwangju, Korea. The study involved 54 patients with traumatic complete cord injuries scheduled for spinal or nonspinal surgery with general anesthesia. Patients were divided into three groups according to the most cephalic level of complete motor and sensory lesions: quadriplegics (level of injury above C7, n = 22), high-level paraplegics (HP; level of injury between T1 and T4, n = 8), and low-level paraplegics (LP; level of injury below T5, n = 24). Twenty age-matched, nondisabled patients served as controls.

Neurologic examinations of all disabled patients were performed by Chonnam National University Hospital Spine Center according to 1996 American Spinal Injury Association standards.[12] Motor function was examined using key muscles for levels C5 through T1 and L2 through S1, and total paralysis of motor strength was regarded as complete lesion. Sensory level was examined by light touch and pin prick at each dermatome, and anesthesia and analgesia was regarded as complete lesion. All patients gave written informed consent to take part in the study. None had any cardiovascular, pulmonary, or metabolic diseases. Patients whose time lag from the injury to the operation was less than 4 weeks were excluded from the study, because acute severe injury to the cervical cord injury is regularly accompanied by arrhythmias and hemodynamic abnor-

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malities.[13] Patients who took medications that would influence autonomic or cardiovascular response to laryngoscopy and intubation were also excluded.

All patients were premedicated with 0.1 mg/kg midazolam orally 60 min before induction of anesthesia. Before arrival in the operating room, an intravenous catheter was placed to allow administration of intravenous fluids and medications. In addition, a 20-gauge catheter was inserted into a radial artery connected to a pressure transducer (Deltran; Utah Medical Products, Midvale, UT) to measure blood pressure and take blood samples. Heart rate (HR) was determined from electrocardiogram traces. For each patient, a rest period of at least 30 min was provided between the time of cannulation and the start of the study.

After recording baseline measurements, anesthesia was induced with 5–7 mg/kg intravenous thiopental, followed by 0.12 mg/kg intravenous vecuronium after full preoxygenation. Intubation was performed within 15 s when neuromuscular block was achieved, and anesthesia was maintained using 50% nitrous oxide and 1% isoflurane in oxygen. The lungs were mechanically ventilated to maintain an end-tidal carbon dioxide tension between 35 and 40 mmHg. Data from patients in whom intubation required more than 15 s were excluded.

Heart rate and arterial blood pressure were continuously monitored. Hypertension was defined as a systolic arterial blood pressure (SAP) more than 130% of the baseline value or more than 160 mmHg, whereas hypotension was defined as SAP less than 70% of the baseline value or less than 90 mmHg. Tachycardia and bradycardia were defined as HR greater than 120 beats/min and less than 60 beats/min, respectively. The incidences of hypertension, hypotension, tachycardia, and bradycardia were recorded throughout the study. A dysrhythmia was defined as any ventricular or supraventricular premature beat or any sustained rhythm other than sinus. The incidence of dysrhythmia after intubation was also compared among the groups.

Arterial blood samples were drawn before (baseline) and 1 min after the onset of intubation for measurements of plasma catecholamines. The samples were collected into prechilled tubes containing EDTA-Na and immediately centrifuged at 3,000 rpm for 10 min at 4°C. The plasma was stored at −70°C until assayed. Plasma concentrations of epinephrine and norepinephrine were measured in duplicate using high-pressure liquid chromatography.[14] The assay sensitivity was 10 pg/ml, and within-run precision coefficients of variation were 14.2 and 13.5% for epinephrine and norepinephrine, respectively.

Statistical Analysis

All results are expressed as mean ± SD. Statistical analyses of the data for each hemodynamic parameter and catecholamine were performed by a two-way analysis of variance with repeated measures. A Scheffé test was used for multiple pairwise comparisons when a significant difference was indicated with analysis of variance. Complication rates among the groups were analyzed using the chi-square test where appropriate. A P value < 0.05 was considered statistically significant.

Table 1. Demographic Data in Spinal Cord–injured and Control Patients

	Control (n = 20)	QP (n = 22)	HP (n = 8)	LP (n = 24)
Sex (M/F)	18/2	19/3	6/2	22/2
Age (yr)	38 ± 11	40 ± 13	42 ± 15	37 ± 10
Weight (kg)	57 ± 10	56 ± 17	60 ± 12	59 ± 11
Height (cm)	170 ± 7	169 ± 7	172 ± 8	167 ± 7
Hemoglobin (g/dl)	12 ± 1	11 ± 1	13 ± 1	12 ± 2
Postinjury (months)	—	26 ± 39	41 ± 52	55 ± 95

Values are mean ± SD.

Control = uninjured patients; QP = quadriplegic patients; HP = high-level paraplegic patients; LP = low-level paraplegic patients; n = number of patients.

Results

There were no significant differences among the groups with respect to sex ratio, age, weight, height, and hemoglobin concentrations (table 1). Baseline SAP and HR did not significantly differ among the four groups (figs. 1 and 2).

Systolic arterial blood pressure significantly decreased after induction of anesthesia with thiopental in all groups. After tracheal intubation, SAP increased significantly in control (33%), HP (28%), and LP (39%) groups, whereas it remained unchanged in the quadriplegic

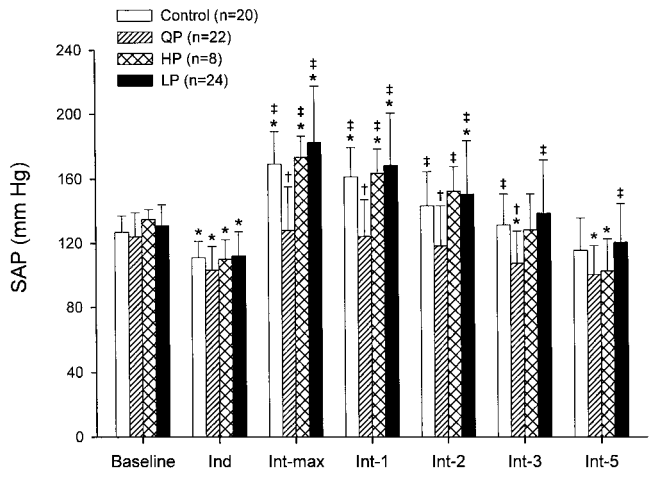

Fig. 1. Systolic arterial pressure (SAP) before and after endotracheal intubation in patients with spinal cord injury and in control patients. Values are mean ± SD (n = number of patients). QP = quadriplegic; HP = high-level paraplegic; LP = low-level paraplegic; Control = normal patients; Ind = 1 min after induction; Int-max = maximum response within 1 min after intubation; Int-1, 2, 3, and 5 = responses at 1, 2, 3, and 5 min after intubation. *P < 0.05 *versus* baseline; †P < 0.05 *versus* control group; ‡P < 0.05 *versus* the quadriplegic group.

group. The increase in SAP above baseline attained maximal level within 30 s and persisted until 2 min after intubation. SAP then decreased to the control level, even below baseline values, which was noted sooner in the quadriplegic and HP groups (from 3 and 5 min after intubation, respectively) than in the control or LP groups (fig. 1).

Heart rate increased significantly after anesthesia induction in all groups. In response to tracheal intubation, HR further increased; however, the magnitude was more pronounced in the HP group than in the control or quadriplegic groups (fig. 2).

Individual values of SAP exceeded 130% of the preinduction baseline value or 160 mmHg in 34 of the 72 patients studied. The incidence of hypertension was significantly lower and that of hypotension was significantly higher in quadriplegic group than in the control group. In addition, the incidence of tachycardia was significantly higher in the HP group than in the control group. There were no significant differences in the incidence of bradycardia among the groups. Premature ventricular contractions appeared immediately after tracheal intubation in 2 of 20 patients in the control group and in 4 of 21 patients in the quadriplegic group. The arrhythmias disappeared spontaneously without treatment (table 2).

Baseline norepinephrine and epinephrine concentrations did not differ among the groups. Endotracheal intubation caused an increase of plasma norepinephrine concentrations in all groups; compared with the control group, the magnitude of increase was attenuated in the quadriplegic group, exaggerated in the LP group, and did not differ in the HP group. No groups showed significant changes in plasma epinephrine concentrations after intubation (table 3).

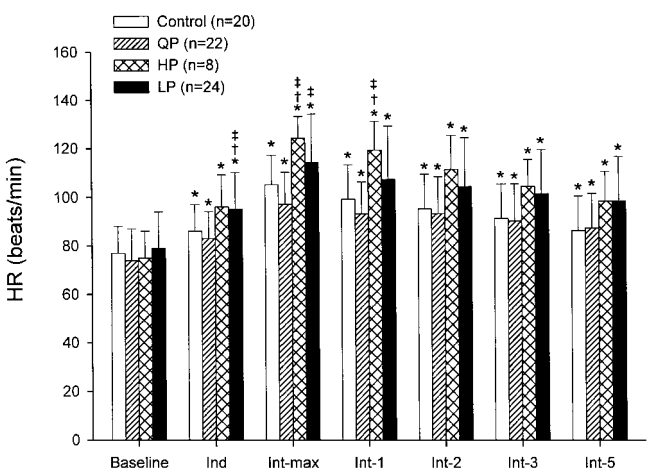

Fig. 2. Heart rate (HR) before and after endotracheal intubation in patients with spinal cord injury and in control patients. Values are mean ± SD (n = number of patients). QP = quadriplegic; HP = high-level paraplegic; LP = low-level paraplegic; Control = normal patients; Ind = 1 min after induction; Int-max = maximum response within 1 min after intubation; Int-1, 2, 3, and 5 = responses at 1, 2, 3, and 5 min after intubation. *$P < 0.05$ *versus* baseline; †$P < 0.05$ *versus* control group; ‡$P < 0.05$ *versus* the quadriplegic group.

Table 2. Catecholamine Data in Spinal Cord–injured and Control Patients

	Control (n = 20)	QP (n = 22)	HP (n = 8)	LP (n = 24)
Norepinephrine				
Baseline	196 ± 94	147 ± 53	180 ± 85	253 ± 130*
PI-1	358 ± 148*†	164 ± 44†‡	327 ± 137†	489 ± 193*†‡
Epinephrine				
Baseline	117 ± 84	71 ± 39	93 ± 36	167 ± 80*
PI-1	115 ± 67	81 ± 45	79 ± 39	148 ± 76*

Values are mean ± SD. Catecholamine (norepinephrine and epinephrine) measured in pg/ml plasma.

* $P < 0.05$ *versus* quadriplegic patient (QP) group. † $P < 0.05$ *versus* baseline. ‡ $P < 0.05$ *versus* control group.

Control = uninjured patients; HP = high-level paraplegic patients; LP = low-level paraplegic patients; n = number of patients; PI-1 = 1 min after the onset of intubation.

Discussion

The current study showed that the cardiovascular and catecholamine responses to tracheal intubation differed according to the level of injury. In particular, the pressor response, but not chronotropic response, was abolished along with diminished catecholamine responses in the quadriplegic group. On the contrary, the pressor response was not affected, but the tachycardiac response was augmented, in the HP group. The LP group showed similar cardiovascular responses, with catecholamine responses exaggerated (table 4).

A complete spinal cord injury or transection results in not only a loss of motor and sensory functions conducted *via* spinal pathways, but also an interruption of sympathetic outflow below the level of the injury.[15] Consequently, the pressor response to intubation was completely abolished and catecholamine response was minimal in the quadriplegic group. The interruption of the sympathetic fibers to the heart would also abolish a tachycardiac response to intubation, and the intact parasympathetic pathway would result in a bradycardiac

Table 3. Incidence of Adverse Effects in Spinal Cord–injured and Control Patients

	Control (n = 20)	QP (n = 22)	HP (n = 8)	LP (n = 24)
Hypertension	10	2*	5	19*
Hypotension	1	9*	2	1
Tachycardia (HR > 120 beats/min)	3	1	6*	8*
Bradycardia (HR < 60 beats/min)	0	1	2	0
Dysrhythmia	2	1	2	4

* $P < 0.05$ *versus* control group.

Control = uninjured patients; QP, quadriplegic patients; HP = high-level paraplegic patients; LP = low-level paraplegic patients; n = number of patients; HR = heart rate.

Table 4. Hemodynamic and Catecholamine Responses to Intubation in Spinal Cord–injured and Control Patients

	Control (n = 20)	QP (n = 22)	HP (n = 8)	LP (n = 24)
Hemodynamic				
SAP	↑ ↑	—	↑ ↑	↑ ↑
HR	↑ ↑	↑ ↑	↑ ↑ ↑	↑ ↑
Catecholamine				
Norepinephrine	↑ ↑	↑	↑ ↑	↑ ↑ ↑
Epinephrine	—	—	—	—

Control = uninjured patients; QP = quadriplegic patients; HP = high-level paraplegic patients; LP = low-level paraplegic patients; n = number of patients; SAP = systolic arterial pressure; HR = heart rate; ↑ = minimal increase; ↑ ↑ = moderate increase; ↑ ↑ ↑ = marked increase; — = no change.

response in the quadriplegic group. However, an apparent tachycardiac response was observed after intubation in quadriplegic group. Schmid *et al.*[10,11] also demonstrated an increase in HR during physical exercise in long-term quadriplegic patients. These findings are contradictory to those in acute severely injured quadriplegic patients who frequently show reflex bradycardia and cardiac arrest during tracheal suction.[16] Lehmann *et al.*[13] also observed primary cardiac arrest in 5 of 31 individuals (17%) with acute severe cervical cord injury, although no episodes were observed in 40 patients with mild cervical and thoracolumbar injuries. It is likely that the chronotropic response in quadriplegic patients depends on the time elapsed after the injury, with improvement occurring over time.

Mechanisms underlying the chronotropic response may include autonomic adaptation of the heart where the vagus only is intact in the baroreflex pathway. Koh *et al.*[17] found that most quadriplegic patients have a low-frequency R-R interval, which is proportional to arterial pressure and abolished by atropine. Similarly, Grimm *et al.*[18] examined the effects of autonomic dysfunction on HR variability in individuals with quadriplegia and paraplegia and found that the resting levels of both sympathetic and parasympathetic outflows are lower when the injury is higher and more complete. These findings suggest that either the sympathetic or the parasympathetic pathway alone has the capability to maintain cardiovascular homeostasis when the other has been severely compromised. In this context, quadriplegic patients may modulate HR exclusively by changing vagal firing.

Alternatively, one may relate the tachycardiac response to partly preserved sympathetic outflow innervating the heart in quadriplegic patients. However, quadriplegic patients showed only a slight increase in norepinephrine (11.5%) compared with controls (82.7%), with no significant pressor response in the current study. In addition, epinephrine and norepinephrine were increased, albeit little, after physical exercise, even in quadriplegic patients.[10,11] It is unlikely that the partly remaining sympathetic pathway contributes to the tachycardiac response in the quadriplegic group.

On the other hand, HP patients showed an exaggerated tachycardiac and a normal pressor response to intubation, with a significant increase of norepinephrine, but not of epinephrine, concentrations. In accordance with our findings, Karlsson *et al.*[19] observed an exaggerated sympathetic response above the level of the lesion with normal pressor response in cord-injured patients (C7–T4); central sympathetic activation (mental stress induced by forced mental arithmetic testing) increased HR and norepinephrine spillover from the arm more pronouncedly than in controls. Schmid *et al.*[10] also demonstrated that paraplegic patients with high lesions display a higher increase of HR in relation to oxygen consumption during physical exercise. They speculated that HP patients had a smaller stroke volume caused by a decreased venous return resulting from damage of the sympathetic innervation to the vasculature and also to a loss of muscular pump.[10] Therefore, HP patients may show a more pronounced tachycardiac response to intubation because of enhanced activity of the cardiac sympathetic outflow to compensate for a large loss of sympathetic activity below the level of injury (T1–T4). In addition, thiopental used to induce anesthesia may have dilated the vasculature and thus elicited additional baroreflex, especially in patients with high-level cord injuries.

Nevertheless, the degree of tachycardiac response to intubation was smaller in the quadriplegic group than in the HP group. Coutts *et al.*[20] studied individuals with quadriplegic and HP injury with a continuous progressive loading exercise test to exhaustion on a wheelchair ergometer, and similarly found that the quadriplegic group had a significantly lower maximal HR than the HP group (119 *vs.* 175 beats/min). The tachycardiac response to intubation associated with a reduced vagal activity in the quadriplegic group may not be sufficient to compensate for the diminished sympathetic response.

It has been observed that the degree of sympathetic dysfunction becomes greater as the level of injury is higher. With injuries below the T5 level, the sympathetic innervation to the vasculature is generally maintained to elicit normal baroreflexes, particularly in the splanchnic bed. In the current study, the paraplegic patients with a lesion below T5 showed normal cardiovascular responses to intubation along with a greater catecholamine response, suggesting a compensatory autonomic response to preserve the circulatory homeostasis. The augmented catecholamine response may also be attributed to an altered end-organ responsiveness, since the degree of hemodynamic responses was similar between the LP and control groups despite the different catecholamine concentrations.

Interestingly, a correlation is lacking between norepinephrine concentrations and HR. The greatest increase in HR was observed in the HP group, whereas the big-

gest increase of norepinephrine was in the LP group. The exaggerated HR response in the HP group may be related to a compensatory increase of sympathetic activity to the heart.[19] Although a greater increase of HR would then be expected in the quadriplegic group compared with the HP group, a total loss of cardiac sympathetic activity in the former may have resulted in a less pronounced HR response. On the contrary, the exaggerated catecholamine response in the LP group may be attributed to a more widespread sympathetic activation, with resultant increase of norepinephrine spillover, possibly from the cardiovascular system, including the splanchnic and renal vascular beds as well as the adrenal gland. In fact, the plasma norepinephrine concentrations may only partially reflect the overall sympathetic nerve activity, since norepinephrine is subject to neuronal as well as extraneuronal uptake before reaching the circulation once released from the neuron. The discrepancy between the catecholamine and hemodynamic responses in different groups needs to be further explained.

On the other hand, the cord-injured patients showed an increase in HR either alone or concomitant with SAP after intubation. In healthy patients, this transient hypertension and tachycardia are of little consequence; however, they may be hazardous in patients with hypertension, limited coronary or myocardial reserve, or cerebrovascular diseases.[21] Individuals with cord injuries are particularly at an increased cardiovascular risk.[22] In fact, coronary heart disease is one of the most important causes of death in patients with spinal cord injuries.[23] Therefore, we would recommend a routine use of pharmacologic methods modifying the hemodynamic response to laryngoscopy and tracheal intubation in patients with long-term cord injuries. In particular, HP patients showed an exaggerated chronotropic response compared with controls. HR is a major determinant of myocardial oxygen consumption, and tachycardia is poorly tolerated during the perioperative period in patients with coronary heart disease. Several recent studies have shown that there is an increased evidence of myocardial ischemia when intraoperative HR exceeds 110 beats/min.[24] Therefore, modulation of HR should be considered when a tachycardiac response to tracheal intubation would be more likely, as in the HP group.

However, most drugs used to attenuate cardiovascular responses to intubation, including narcotics, calcium channel blockers, and β-adrenoceptor blockers, have been associated with hypotension. In the current study, 3 of 22 quadriplegic patients and 1 of 8 HP patients developed hypotension immediately after intravenous administration of thiopental. In addition, the incidence of hypotension after intubation was significantly greater in the quadriplegic group than in the control group. Caution should be exercised to use any pharmacologic tools in alleviating the circulatory response to an intubation with careful titration of induction and maintenance agents, as well as close monitoring of cardiovascular parameters in patients with high-level cord injury.

In conclusion, the current study showed that the circulatory and catecholamine responses to laryngoscopy and tracheal intubation differ with the level of the lesion in patients with complete cord injury. The pressor but not tachycardiac response to tracheal intubation was abolished in the quadriplegic group, whereas in the HP group, the tachycardiac response was augmented. In addition, the catecholamine response was attenuated in the quadriplegic group, whereas it was more pronounced in the LP group.

References

1. Prys-Roberts C, Greene L, Miloche R, Foex P: Studies of anaesthesia in relation to hypertension. II. Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1984; 43:531–47
2. Shribman AJ, Smith G, Achola KJ: Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth 1987; 59:295–9
3. Derbyshire DR, Chmielewski A, Fell D, Vater M, Achola K, Smith G: Plasma catecholamine responses to tracheal intubation. Br J Anaesth 1983; 55:855–60
4. Safwat AM, Reitan JA, Misle GR, Hurley EJ: Use of propranonol to control rate-pressure product during cardiac anesthesia. Anesth Analg 1981; 60:732–5
5. Bonica JJ: Autonomic innervation of the viscera in relation to nerve block. ANESTHESIOLOGY 1968; 29:793–813
6. Landsberg L, Young JB: Catecholamines and the adrenal medulla, Williams Textbook of Endocrinology. Edited by Wilson JD. Philadelphia, Saunders, 1996, pp 621–705
7. Wattwil M, Sundberg A, Olsson J, Nordstrom S: Thoracolumabar epidural anaesthesia blocks the circulatory response to laryngoscopy and intubation. Acta Anaesthesiol Scand 1987; 31:529–31
8. Dohi S, Nishikawa T, Ujike Y, Mayumi T: Circulatory responses to airway stimulation and cervical epidural blockade. ANESTHESIOLOGY 1982; 57:359–63
9. Kessler KM, Pina I, Green B, Burnett B, Laighold M, Bilsker M, Palomo AR, Myerburg RJ: Cardiovascular findings in quadriplegic and paraplegic patients and in normal subjects. Am J Cardiol 1986; 58:525–30
10. Schmid A, Huonker M, Barturen J-M, Stahl F, Schmidt-Trucksass A, Konig D, Grathwohl D, Lehmann M, Keul J: Catecholamines, heart rate, and oxygen uptake during exercise in persons with spinal cord injury. J Appl Physiol 1998; 85:635–41
11. Schmid A, Huonker M, Stahl F, Barturen JM, Konig D, Heim M, Lehmann M, Keul J: Free plasma catecholamines in spinal cord injured persons with different injury levels at rest and during exercise. J Auton Nerv Syst 1998; 68:96–100
12. American Spinal Injury Association: International Standards for Neurological and Functional Classification of Spinal Cord Injury, 5th edition. Chicago, American Spinal Injury Association, 1996, pp 1–24
13. Lehmann KG, Lane JG, Piepmeier JM, Batsford WP: Cardiovascular abnormalities accompanying acute spinal cord injuries in humans: Incidence, time course and severity. J Am Coll Cardiol 1987; 10:46–52
14. Holly JMP, Makin HLJ: The estimation of catecholamines in human plasma: A review. Anal Biochem 1983; 128:257–74
15. Teasell RW, Arnold MO, Krassioukov A, Delaney GA: Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury. Arch Phys Med Rehabil 2000; 81:506–16
16. Frankel HL, Mathias CJ, Spalding JMK: Mechanisms of reflex cardiac arrest in tetraplegic patients. Lancet 1975; 2:1183–95
17. Koh J, Brown TE, Beightol LA, Ha CY, Eckberg DL: Human autonomic rhythms: Vagal cardial mechanisms in tetraplegic patients. J Physiol 1994; 474:483–95
18. Grimm DR, De Meersman RE, Almenoff PL, Spungen AM, Bauman WA: Sympathovagal balance of the heart in subjects with spinal cord injury. Am J Physiol 1997; 272:835–42
19. Karlsson A-K, Friberg P, Lonnroth P, Sullivan L, Elam M: Regional sympathetic function in high spinal cord injury during mental stress and autonomic dysreflexia. Brain 1998; 121:1711–9
20. Coutts KD, Rhodes EC, McKenzie DC: Maximal exercise responses of tetraplegics and paraplegics. J Appl Physiol 1983; 55:479–82
21. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD: Complications related to the pressor response to endotracheal intubation. ANESTHESIOLOGY 1977; 47:524–5
22. Yekutiel M, Brooks ME, Ohry A, Yarom J, Carel R: The prevalence of hypertension, ischemic heart disease and diabetes in traumatic spinal cord injured patients and amputees. Paraplegia 1989; 27:58–62
23. Geisler WO, Jousse AT, Wynne-Jones M, Breithaupt D: Survival in traumatic spinal cord injury. Paraplegia 1983; 21:364–73
24. Stone JG, Foex P, Sear JW, Johnson LL, Khambatta HJ, Triner L: Myocardial ischemia in untreated hypertensive patients: Effect of a single small oral dose of a beta-adrenergic blocking agent. ANESTHESIOLOGY 1988; 68:495–500