Cardiovascular responses and thermoregulation in individuals with spinal cord injury

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Introduction

A spinal cord injury (SCI) results in a complete or incomplete loss of somatic, sensory and autonomic functions below the lesion level. This result can be either temporary or permanent. With respect to the somatic impairment, lesions in the cervical region typically result in tetraplegia, whereas lesions in the thoracic and lumbar regions lead to paraplegia.

SCI of traumatic origin is frequently incurred at an early age. It represents one of the most disastrous tragedies in human life. Improved medical care and rehabilitation over the last decades have made it possible to extend the life expectancy of this population (Noreau and Shephard, 1995). However, SCI injured persons encounter a high risk for the development of secondary complications (urinary tract infections, pressure ulcers, coronary heart disease, obesity, type II diabetes etc.). These complications, together with a generally poor physical fitness status, restrict daily life activities and limit social opportunities, a fact which may seriously compromise the quality of life for those individuals who maintain a sedentary lifestyle.

A large proportion of studies conducted over the past 30 years have focused on the cardiovascular system of individuals with SCI and its adjustments at rest and during exercise. These are important aspects to consider from the point of view of rehabilitation, where the goal is the optimization of physical capacity and functional independence, as well as from the point of view of sport science, which is focused on the subject's physical performance. The present review focuses on the cardiovascular and thermoregulatory responses of individuals with SCI at rest and during dynamic upper body exercise.

Sympathetic vasoregulation at rest

Spinal cord injuries have commonly been considered in terms of the resultant loss of voluntary muscle function and sensitivity. The consequences of the deranged function of the autonomic nervous system, however, have received less attention. The cephalic parasympathetic nervous system (cephalic outflow of cranial nerves) is not affected by the spinal lesion, whereas the sacral parasympathetic outflow, which innervates the bladder, colon,
rectum and reproductive organs, is almost always involved. The sympathetic division, also called "thoracolumbar" nervous system, is disturbed to a variable degree, generally according to the level of the lesion and the extent of transverse damage. One of the properties of the sympathetic nervous system is tonic activity, i.e. a constant activity in many pre-and postganglionic neurons in the absence of external stimuli. Sympathetic tone is caused by input from the central nervous system (brain stem reticular formation, hypothalamus, cerebellum, etc.) and the periphery (skeletal muscle, skin, viscera, baroreceptors, chemoreceptors, temperature receptors, etc.), both sources of input being involved in tonic activity as excitatory or inhibitory influences. The various afferent impulses evoke reflex responses mediated by supraspinal as well as by spinal pathways.

Transection of the spinal cord causes a variety of cardiovascular sequelae (Cole, 1988). During the initial phase of spinal shock, resting blood pressure is drastically reduced in individuals with tetraplegia, and there is little significant cardiovascular reflex activity. After the initial phase of spinal shock, the loss of coordinated sympathetic activity results in an inability to raise blood pressure. This exposes the subject to postural hypotension due to accumulation of blood in the lower body parts. Postural hypotension is characterized by symptoms such as visual blurring, giddiness and often syncope, which occur during head-up tilt. This effect is observed mostly in persons with T5 lesions and above (Guttmann, 1976) and reflects the inability to activate the sympathetic efferent component of the baroreceptor reflex (Mathias and Frankel, 1988). However, recovery from this phenomenon occurs with time and training, and most patients can adapt to the sitting position and even to a standing-frame. There is some evidence of recovery from the mere effects of orthostatic hypotension. Indeed, individuals with tetraplegia tend to live at lower blood pressure, having perhaps shifted down their limits for cerebral blood-flow autoregulation (Cole, 1988, Kessler et al., 1986). On the other hand, some results suggest a likely reduction of blood accumulation in the lower body and thus a possible recovery from hypotension itself (Groomes and Huang, 1991, Hopman et al., 1994, Mathias and Frankel, 1988, Skagen et al., 1982, Theisen et al., 2000a).

Although isolated from the inhibitory or facilitatory impulses of higher control centres, the intact spinal cord remains capable of generating and mediating reflex actions of the autonomic nervous system. However, these responses are generally not well balanced or functionally appropriate. Autonomic dysreflexia represents such an uncoordinated, spinally-mediated reflex response, which mainly develops in patients with complete, high spinal lesions (above T6). This so-called 'mass reflex' (Frankel and Mathias, 1979) induces episodes of paroxysmal hypertension due to a massive, generalized peripheral and splanchnic vasoconstriction. It is triggered by a stimulus below the lesion level, such as urinary bladder or bowel distension (followed by micturition), stimulation of the skin, skeletal muscle spasms, etc. There is usually a fall in heart rate consistent with the integrity of baroreceptor afferent and vagal efferent pathways, with a coincident increase in cardiac stroke volume (SV) and cardiac output (Q) (Corbett et al., 1975). Personally distressing and socially embarrassing, autonomic dysreflexia also involves morbidity due to cerebrovascular events and a not inconsiderable mortality (Frankel and Mathias, 1979, Mathias et al., 1992). Strangely enough, plasma norepinephrine levels measured during such incidents do not show abnormally high values, and epinephrine levels do not increase, thus excluding adrenomedullary stimulation and a humoral basis for the hypertension at rest (Mathias, 1991). These episodes are probably largely contributed to by a vasoconstriction in the splanchnic bed, since dysreflexia is not observed in patients with lesions below the splanchnic sympathetic outflow (T5), who probably have adequate baroreflex control over this area (Lee et al., 1995, Mathias and Frankel, 1988). In more general terms, the lower the lesion level, the smaller the constricted vascular region and the more effective the compensatory mechanisms (vasodilation in innervated areas) (Bidart and Maury, 1973). This means that autonomic hyperreflexia may occur even in individuals with low-level paraplegia, although remaining inapparent. When intentionally provoked during exercise, autonomic dysreflexia leads to a higher release of catecholamines, thus
increasing peak exercise performance, heart rate, oxygen consumption and blood pressure (Schmid et al., 2001). This dangerous phenomenon known as “boosting” is considered a prohibited manipulation by the doping guidelines of the International Paralympic Committee.

One explanation for the above-discussed observations could be the fact that cord section results in a certain “denervation sensitivity” (Koizumi and Brooks, 1984, Lee et al., 1995), in that receptor sensitivity develops at all autonomic synapses when they are deprived of presynaptic nerves and transmitters. Increased sensitivity of adrenergic receptors has been demonstrated by exaggerated pressor responses following intravenous infusion of norepinephrine, angiotensin II or other vasopressor agents (Mathias, 1991, Senard et al., 1991). This may indicate some sort of adaptation of the sympathetic system below the lesion and would explain low plasma catecholamine levels and the predisposition to a greater response to phasic inputs resulting in autonomic dysreflexia.

The restoration of some degree of blood redistribution in persons with SCI may be associated with long-term hormonal adaptations. During head-up tilt, renin plasma levels increase to higher values in individuals with tetraplegia than in able-bodied (AB) subjects. Since renin release by the kidneys seems to be independent of sympathetic activation (Cole, 1988), it probably results from the fall in renal perfusion pressure, with afferent arteriolar dilatation causing stimulation of juxtaglomerular renin-secreting cells. The subsequent formation of angiotensin-II with its direct effects on arteriolar vasoconstriction and the later elevation in plasma aldosterone levels are likely to play an important role in the preservation of blood pressure in SCI injured persons (Groomes and Huang, 1991, Mathias and Frankel, 1988). Vasopressin (antidiuretic hormone) may also act as an important adaptation mechanism, since levels rise substantially in persons with tetraplegia during orthostasis. Thus, these humoral responses, with their rapid influence on arteriolar vasmotor tone and their subsequent effects on salt and water retention and plasma volume expansion, may represent important compensatory reactions to acute and chronic hypotension, which help prevent excessive pooling of venous blood in the lower body.

Long-term histochemical changes in the paralyzed muscles of the lower limbs probably characterize another important adaptation which enhances recovery from venous stasis in SCI persons. Indeed, muscle immobilization induces muscle atrophy and results in drastic changes in the muscle structure, such as the reduction of fibre area, the increase of type IIb fibres with a loss of type I fibres (Aksnes et al., 1996, Martin et al., 1992, Scelsi et al., 1982), the proportional increase of intra-muscular connective tissue (Kannus et al., 1998, Scelsi et al., 1982) and the decrease of intra-muscular capillary supply (Kannus et al., 1998, Martin et al., 1992, Scelsi et al., 1982). Furthermore, Hopman et al. (1994) found that in individuals with long-standing paraplegia (> 3 years), venous volume variations and venous capacity were reduced to about 50% in comparison with AB control subjects. These results were attributed to vascular adaptations following muscle atrophy. Thus, it seems that muscle inactivity leads to muscle atrophy and structural changes that reduce the lower limb volume in persons with SCI and thus limit the space for blood accumulation. Excessive accumulation of venous blood in the legs is further prevented by concomitant and subsequent adaptations of the vascular system.

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Figure 1: Cutaneous vascular conductance (CVC) in dorsal foot (3-min averages after posture change, expressed as % of supine posture) during leg dependency and upright sitting in AB and individuals with SCI at levels T10-T12 and T5-T9; * significantly different from AB.

Circulatory reflexes which contribute to blood redistribution in persons with SCI can also occur independently of the spinal cord. Possible
determinants involve the veno-arteriolar reflex - venous distension leading to constriction of arterioles which supply them - mediated by a local sympathetic axon reflex which functions independently of the central nervous system (Henriksen, 1977). The veno-arteriolar reflex represents an important adjustment of the peripheral circulation by alleviating high vascular transmural fluid filtration under postural changes and venous stasis. Skagen et al. (1982) investigated subcutaneous blood flow during posture changes by the 133Xe washout technique in the lower leg of 7 patients with complete SCI at the cervical level (C4-C6). Blood flow in the leg decreased by 47% when the leg was lowered 40 cm below the reference level (supine position). Skagen et al. (1982) also noted a 47% decrease during 45° head-up tilt, which was not eliminated by proximal nervous blockade but only by local nervous blockade. The fact that proximal blockade did not influence the vasoconstrictor response in tetraplegics suggests that the adaptation was of mere local origin. Based on measurements of skin blood flow (SKBF) (laser Doppler flowmetry) and blood pressure, Theisen et al. (2000a) observed a significant reduction of cutaneous vascular conductance in the dorsal foot of individuals with longstanding complete paraplegia (T5-T12 lesions) during leg dependency and upright sitting. There was even a greater reduction in cutaneous vascular conductance in individuals with T5-T9 lesions compared to AB control subjects during leg dependency (Figure 1), while the central hemodynamic adaptations were basically the same in all groups. These findings support the existence of a veno-arteriolar reflex in the paralysed lower limbs, a peripheral reflex response that probably plays an important role in the cardiovascular stability of individuals with SCI.

Cardiovascular responses to dynamic exercise

Despite the common clinical observation that persons with SCI recover from orthostatic hypotension and the possible mechanisms involved in this phenomenon, some studies focussing more on exercise performance point out the possibility of blood pooling in the lower limbs of paraplegics (Davis, 1993, Dawson et al., 1994, Figoni, 1993, Glaser, 1989). During dynamic exercise, there is a redistribution of blood in AB subjects from metabolically inactive body areas to the exercising muscles. The normal cardiovascular reflex responses to exercise include vasoconstriction in more or less inactive regions such as non-exercising muscles, skin and the splanchnic region (Flamm et al., 1990). This results in an elevation of mean ventricular filling pressure and an increase in Q caused by a rise in both SV and heart rate (HR) (Hjeltnes, 1977, Miles et al., 1984). In SCI injured persons this redistribution of blood has been reported to be impaired (Figoni, 1993, Hopman, 1994, Kinzer and Convertino, 1989, Theisen et al., 2001a, Theisen et al., 2000b) and, therefore, less effective than in AB subjects. Peripheral vascular insufficiency and the absence of the skeletal muscle venous pump are believed to cause excessive venous blood pooling in the legs and abdomen (Davis, 1993, Davis et al., 1990, Figoni, 1993, Fitzgerald et al., 1990, Glaser, 1989, Hopman et al., 1992a, Hopman et al., 1993c), and would therefore reduce the circulating blood volume, thus decreasing venous return and cardiac preload. In accordance with the Frank-Starling mechanism, one consequence may be a limited SV, Q, and, possibly, exercise capacity. (Figure 2)

Central hemodynamics

At the same submaximal oxygen consumption (VO2) SCI subjects with a basically normal sympathetic regulation of cardiac function (lesion below T5) can reach Q levels similar to those of AP control subjects with matching characteristics (Hopman, 1994, Kinzer and Convertino, 1989). However, the SV-HR relationships are different between the two groups (Figure 2): individuals with a spinal cord lesion have lower SV and higher HR values at a given submaximal VO2. This holds for low (40% of maximum), moderate (60% of maximum) and high (80-90% of maximum) exercise intensities (Hopman et al., 1992a, Hopman et al., 1993c). The lower SV observed in the paraplegics with a complete lesion has been attributed to excessive blood pooling in the capacitance vessels of the legs and the abdomen. This may be the consequence of an impaired sympathetic vasomotor regulation (lack of sympathetic vasoconstriction) in the body areas.
Figure 2: Cardiac output (Q), stroke volume (SV) and heart rate (HR) as a function of oxygen uptake (VO2) in SCI and AB subjects during submaximal arm cranking (From "Circulatory responses during arm exercise in individuals with paraplegia," by M.T. Hopman, 1994, *International Journal of Sports Medicine*, 15, 126-131. Copyright 1994 by Georg Thieme Publishers. Reprinted with permission of the author).

Venous blood pooling will disturb blood redistribution by reducing cardiac filling pressure and end-diastolic ventricular volume. A reduced preload will cause the myocardium to contract in a less efficient part of the ventricular function curve and thus limit SV in individuals with paraplegia. In compensation for a lower SV, these individuals have to increase their HR to achieve the necessary Q at a given exercise level. Several authors (Davis, 1993, Davis et al., 1990, Figoni, 1993, Glaser, 1989, Hjeltnes, 1977, Kaprielian et al., 1998, Kinzer and Convertino, 1989) suggest that excessive venous pooling may even lead to a "hypokinetic" circulation (reduced Q for any given VO2 through lower SV and smaller than anticipated elevations in HR), a topic that is currently still being debated. Based on their findings from several studies, Hopman et al. (1994) claim that the circulation of persons with SCI with an intact cardiac sympathetic innervation (lesions < T6) can be considered to be isokinetic in comparison with the circulation of AB, even at high exercise intensities. On the other hand, Hjeltnes (1977) and Davis and Shephard (1988) found that at a given VO2, Q was lower even in individuals with low-level
paraplegia compared to the response to arm exercise reported for AB. Unfortunately, these studies did not include AB control groups. Kaprielan et al. (1998) reported lower Q values in individuals with T6-T12 lesions than in their AB subject group, regardless of the training status. However, a greater SV was found in trained versus untrained individuals with paraplegia. Taken together, these findings indicate that hypokinetic circulation in low-level paraplegics seems possible. This would imply a higher oxygen extraction in the working arm muscles, a point that is yet to be clarified in detail.

Decreased cardiac performance may be found predominantly in individuals with spinal cord lesions above T6 whose cardiac sympathetic innervation is likely to be affected so that vagal activity overrules. This is typically the case for individuals with complete tetraplegia who have no sympathetic innervation to the heart and thus a maximal HR limited to ~115-130 beats per minute (Lasko-McCarthey and Davis, 1991). Therefore, especially individuals with high spinal lesions may reach their maximum HR, Q and VO2 at lower exercise levels than paraplegics with injuries below T5/6 (Hopman et al., 1993b). Furthermore, a sedentary lifestyle in male paraplegics may be associated with a reduction in cardiac dimensions and left ventricular SV at rest (Huonker et al., 1998), thus further limiting cardiac performance.

- **Techniques for enhancing venous return**

Some studies indirectly support the existence of blood pooling below the lesion of SCI subjects at rest and during arm exercise. Supine arm cranking exercise (ACE), compared to upright sitting exercise, elicited significantly higher SV and lower HR in subjects with tetra- and paraplegia (Figon et al., 1991, Hopman et al., 1998). The conclusion of these studies was that the supine posture promotes venous return, thus elevating end-diastolic ventricular volume and improving myocardial contraction. Hopman et al. (1992b) studied the use of an Air Force antigravity (anti-G) suit to apply external pressure on the legs and abdomen of individuals with paraplegia and AB control subjects during ACE. Their results showed no effect on AB subjects, whereas at 40% and 60% of individual maximal load, significantly lower HR (and slightly higher SV values) were recorded in individuals with SCI when lower body positive pressure was applied. Similar observations were made by Kaprielan et al. (1998), who reported significant decreases of HR in persons with paraplegia with lower body positive pressure, irrespective of training status, whereas no effects were found in AB. Pitetti et al. (1994) also reported beneficial hemodynamic effects of an anti-G suit during submaximal ACE. They found that the SV of SCI persons was significantly increased, Q was slightly higher and the HR was lower, with no influence on the parameters of AB controls. During maximal exercise, the influence of an anti-G suit both on subjects with paraplegia and on AB-subjects, was to significantly lower the maximal HR, with no effect being found for maximal power output and VO2 (Hopman et al., 1993a). Pitetti et al. (1994) showed that lower body positive pressure increased peak ventilation, VO2 and work capacity in SCI subjects during maximal ACE, with no significant change in peak HR. However, the subject group of this study was mainly composed of individuals with tetraplegia, who were not able to increase their peak HR to values higher than ~125 bpm and for whom the anti-G suit possibly had a greater influence, due to their limited work capacity. In contrast to the above-described studies, Kerk et al. (1995) found that a mere abdominal binder had no significant effect on either the physiological or the biomechanical exercise responses of paraplegic athletes. However, in subjects with tetraplegia, support stockings for the lower limbs combined with an abdominal binder resulted in significantly lower HR and in higher SV and Q, while no hemodynamic benefits were found for individuals with paraplegia (Hopman et al., 1998).

A recent and increasingly popular technique tested on SCI persons in clinical and experimental settings concerns functional neuromuscular stimulation (FNS) of the paralysed lower limbs. Some findings involving FNS have revealed enhanced cardiac response through better venous return, and would thus provide an indirect indication of blood pooling (Davis et al., 1988, Davis et al., 1990, Figoni et al., 1988, Raymond et al., 1999, Raymond et al., 2001, Raymond et al., 1997). Davis et al. (1988)
and Figoni et al. (1988) found that when passively tilting their SCI subjects from 0° to +70°, there was a significant reduction of resting and exercise cardiac SV, with a redistribution of blood to the lower extremities. When FNS was applied to induce rhythmic static contractions of the paralyzed legs, there was, in some instances a correction of the redistribution of blood to the lower limbs which resulted in increased SV and Q (Figure 3). However, this result could only be achieved in subjects classified as ‘responders’, i.e. those for whom stimulation intensities could be set high enough as to induce “vigorous pulsatile static contractions”. No such effects were found in more pain-sensitive ‘non-responders’ or in AB subjects (Burkett et al., 1997, Eijsbouts et al., 1997), probably because of their limited tolerance to electrical stimulation and the resulting poor muscle contraction (Burkett et al., 1997). Enhanced SV and Q values in ‘responders’ with SCI were also found by Davis et al. (1990) under some arm exercise conditions with FNS-induced leg contractions. Nevertheless, exercise Q was estimated on the basis of exercise steady-state HR and immediate post-exercise SV, which represents a drawback of that study. Raymond et al. (1997) showed that when ACE was performed immediately after a period of combined arm exercise and FNS-induced leg cycling exercise, VO2 was decreased in individuals with SCI, while HR was significantly higher, an observation attributed to blood pooling in the lower body. These results indicate better venous return of blood to the heart during FNS-induced leg cycling. Nevertheless, the authors point out that the effect was probably exaggerated because of the sequence used in their protocol. FNS-induced leg cycling combined with arm exercise preceded arm exercise alone and may have induced considerable leg muscle vasodilation and magnified blood pooling upon transition to ACE alone, thus eliciting increased cardiac stress. More direct measurements were done by Phillips et al. (1995) on eight paraplegics (C6-T12) at rest and during ACE at 60% and 80% of VO2 peak, both with and without FNS. Post-exercise peripheral blood flow, as determined by photoelectric plethysmography, was consistently reduced during FNS. However, exercise intensity in itself (without FNS) also reduced peripheral blood flow, which indicates some sort of autonomous adaptation to exercise.

Figure 3: Mean (±SE) cardiac output and stroke volume of paraplegics (n=7) during arm cranking exercise (ACE) with or without functional neuromuscular stimulation (FNS) at different tilt angles.

- Lower limb blood flow measurements

To date, there have been only a few studies in which procedures have been used to investigate more directly the blood flow adaptations in the lower limbs of persons with SCI. Bidart and Maury (1973) used invasive venous pressure measurements at constant volume to assess venous tone changes in the foot during and after supine muscular exercise (50W). Venous tone increased at the beginning of exercise in AB and in SCI subjects with a lesion level below T10. Individuals with lesions above this level showed irregular responses or no increase in venous tone. However, no quantitative differences were reported. The authors also studied arteriolar motricity with strain gauge plethysmography applied to the foot and found that during exercise, blood flow decreased in AB subjects whereas it increased in
SCI subjects. Unfortunately, the lesion levels of these subjects were not reported and the exercise conditions were not clearly defined. Kinzer and Convertino (1989) measured fluid accumulation and arterial pulse volume in the legs during 35 W ACE, using impedance plethysmography. They suggested that their T6-T11 paraplegics (n=5) had normal vasoconstriction, since the reduction in their leg arterial pulse volume was similar to that of the AB Controls. However, fluid accumulation in the legs, which decreased in the AB controls during exercise, increased significantly in the SCI subjects. The investigators concluded that these discrepancies were due to the absence of normal muscle pump action in the lower limbs, which consequently could act as a reservoir for “significant blood pooling” and thus limit venous return and cardiac performance of persons with SCI.

Hopman et al. (1993d) employed strain gauge plethysmography to compare leg volume changes in C8-L1 SCI subjects (n=15, complete and incomplete lesions) to those of AB subjects. Electromyographic recordings from the calf muscles were used to verify that there was no muscle pump activity in the legs of the control subjects, so that sympathetic vasoconstriction could be considered the most important mechanism responsible for the changes that were noted. During exercise, AB subjects showed a decrease in leg volume whereas recovery was characterized by a leg volume increase. Leg volume changes in the paraplegic group had a much less abrupt pattern, the rate of change as well as the total volume decrease being significantly lower (Figure 4). Volume decrease at onset of exercise and total volume decrease were correlated to the lesion level, whereas the completeness of the lesion had no influence on any parameter. These results would indicate that SCI subjects lack vasomotor responses to exercise, probably due to an inappropriate sympathetic regulation below the lesion. Hopman et al. (1993d) deducted that the lack of sympathetic vasoconstrictive activity, which depends on the lesion level, is (at least partly) responsible for persistent blood pooling and a disturbed cardiovascular regulation during arm exercise. Theisen et al. (2001a) came to similar conclusions, based on SKBF measurements using laser Doppler flowmetry. Leg SKBF decreased in AB subjects during short-term arm cranking exercise, illustrating efficient blood redistribution through sympathetic vasoconstriction. By contrast, SKBF increased in individuals with paraplegia, reflecting the impaired vascular adaptations in the paralyzed lower limbs. However, the level of the spinal lesion in this group of T5-T12 paraplegics was not related to the SKBF response in terms of the maximum recorded values.

The above-discussed studies suggest that individuals with SCI have different cardiovascular responses in terms of HR-SV relationship, which can be attributed to a deficit in their blood redistribution capacities. Some results clearly show that external compression of the lower limbs or activation of the “peripheral muscle pump” by FNS may support venous return and thus enhance central hemodynamic responses in persons with SCI. Although these
effects cannot be questioned, they do not necessarily indicate that there is excessive blood pooling in the paralysed legs of SCI persons, as many authors conclude. Carefully analyzing the studies involving lower body positive pressure, one notices that statistically significant effects found in SCI persons are sometimes limited to a single parameter or specific condition (Hopman et al., 1993a, Kaprielian et al., 1998), and that the beneficial trend is also present in AB subjects (Hopman et al., 1993a, Muraki et al., 1996). The same reasoning holds for studies using FNS, an approach which in addition yields positive effects only in subjects classified as ‘responders’ who show vigorous static muscle contractions. Indeed, the notion of enhanced central hemodynamic responses through external compression or muscle venous pump activation in the lower limbs is not surprising. Furthermore, as already explained, the properties of the muscle tissue and the vascular system in the lower extremities of individuals with longstanding SCI are largely altered as a result of the paralysis and the ensuing atrophy of these tissues (Hopman et al., 1998, Hopman et al., 1994). Therefore, venous blood pooling in the legs of individuals with paraplegia has been considered “very unlikely” (Hopman et al., 1994). Recently, Raymond et al. (2001) showed that individuals with paraplegia had a smaller decrease in SV than AB individuals during arm cranking and lower body negative pressure. Thus, despite a similar orthostatic challenge, the volume of blood "pooled" in the lower body is probably less in these subjects, a fact which can at least partly be attributed to the reduced compliance and capacity of the lower limb vasculature. When referring to the impaired blood flow adaptations of individuals with SCI during dynamic upper body exercise, it would seem more appropriate to describe this phenomenon as a reduced ability or inability to redistribute blood from inactive body areas below the spinal lesion to the heart, a notion which is supported by direct observations of blood flow in the lower limbs (Hopman et al., 1993d, Theisen et al., 2001a). Nevertheless the splanchnic region, which so far remains unexplored, represents an area where significant venous blood pooling as a result of spinal lesion may persist.

Temperature regulation at rest and during exercise

Complications in the cardiovascular adaptation and sympathetic vasomotor function have an influence on thermal homeostasis and will thus be reflected in thermoregulatory responses. The damage to the autonomic nervous system in SCI subjects seems to affect both sweating and the ability to vasoregulate the peripheral vasculature (Normell, 1974).

Core temperature during passive heat exposure

Skin and body temperatures of resting SCI subjects are influenced much more by environmental temperatures than those of AB control subjects. Guttmann et al. (1958) found that when patients with complete transverse lesions of the mid-cervical cord were exposed naked to low or high temperatures, they could not adequately regulate body temperature as do individuals with SCI below T5. When exposed to a cold (18-20°C) environment, these patients lacked shivering, which occurred only in the few remaining muscles above the lesion level, and they showed no sweating in hot (35-37°C) conditions. However, when exposed to high degrees of heat, patients with clinically complete lesions of the cervical and upper thoracic cord demonstrated sweating over some areas below the level of the lesion (Guttmann, 1976, Silver et al., 1991). This could indicate thermoregulatory sudomotor responses of the isolated cord to intensive heat stimulation.

Freund et al. (1984) showed that T1-T11 paraplegics (n=5), when submitted to passive heating of the insensate skin, develop only mild sweating on the upper body, hardly any at all on the lower body and little or no forearm blood flow elevation, even at core temperatures close to the limits of subjective tolerance. This was in contrast to the vigorous thermoregulatory responses of an AB subject who exhibited grossly observable sweating and forearm blood flow elevation. When both sensate and insensate skin was heated, abundant sweating occurred above, and in some individuals also below the spinal lesion. Forearm blood flow increased in all SCI subjects but stayed well below levels reported for AB subjects (Figure 5). The authors concluded that the reduced effector activity observed might be partly due to a deficit of afferent information from extracerebral sites of thermosensitivity within the spinal cord or other deep tissues below the lesion. Diminished thermoregulatory responses were also found by Petrofsky (1992), who noted overall lower sweat production than in AB controls and no increase in regional sweat rates below the spinal lesion level.

Thermoregulatory responses seem to depend on the spinal lesion level. Gerner et al. (1992) reported that a 15-minutes sauna session induced higher rectal and oral temperatures, lower sweat rates and reduced losses of body weight in individuals with tetraplegia than in those with paraplegia. The vasomotor and sudomotor responses of persons with SCI are likely to depend on the lower-most intact part of the sympathetic chain (Normell, 1974). Consequently, it can be concluded that the thermoregulatory disadvantage encountered by individuals with SCI is probably dependent on the lesion level and is the result of (1) a deficit of vasomotor and sudomotor function over sympathetically uncontrolled areas and (2) a reduced thermoregulatory effector drive for a given core temperature, which is probably due to a lack of afferent input into hypothalamic control centres (Sawka et al., 1989).

**Core temperature during dynamic exercise**

When performing arm exercise, the challenge imposed by thermoregulation on the cardiovascular system may become critical in persons with SCI. In AB subjects there is an initial cutaneous vasoconstriction with the onset of dynamic exercise, after which core temperature and SKBF begin to rise, eventually exceeding pre-exercise levels (Kenney and Johnson, 1992). The core temperature threshold for active cutaneous vasodilation is increased to a higher value during dynamic exercise, when the central blood volume is reduced by the upright exercise posture (in comparison to the supine posture), or by the application of lower body negative pressure (blood pooling) or by diuretic administration. These conditions reduce the SKBF at any given core temperature. When exercise is continued after the threshold is passed, SKBF increases in parallel with the increase in core temperature, up to about 38°C. This seems to be an upper limit for SKBF and is the reflection of a competition between the skin and the working muscle for a limited Q. Thus, vasodilation has not reached its maximum (the upper limit for SKBF represents 50-75% of maximal SKBF), but this limit is presumably caused by a decreased central filling pressure that is sensed by cardiopulmonary baroreceptors, which would have control over the active vasodilator system.

During exercise and thermal strain, different regulatory control mechanisms can generate opposite reflex responses with respect to SKBF (exercise-induced vasoconstriction and temperature-induced vasodilation), which places a serious burden upon the cardiovascular system. Prolonged submaximal exercise and thermal stress may induce a so-called 'cardiovascular drift' (Davis, 1993, Dawson et al., 1994, Gass and Camp, 1987, Gass et al., 1988). This phenomenon is most marked during exercise above a certain intensity or under strenuous thermal conditions (Hopman et al., 1993b: 35°C and 70% of relative humidity, 40% VO2 peak workload) and may be unobservable in a thermoneutral environment (Gass et al., 1981: 23.5°C and 50% of relative humidity, 50% VO2 peak workload). It consists in a decrease in SV with an increase in HR, to keep Q at a more or less constant level throughout the exercise bout. In persons with SCI, this situation may become a potential physiological problem, since a lack of sympathetic vasomotor adjustments and reduced sweating capacity below the lesion level may hamper appropriate blood redistribution and limit cooling efficiency. Hopman et al. (1993b)
found that during sustained exercise (45 minutes) at a constant intensity (40% VO2 peak), VO2 and Q remained unchanged in AB and SCI subjects with lesions below T6, whereas high-level paraplegics (T2-T6) showed a significant decrease in Q. All the subjects demonstrated a significant decrease in SV, but only the AB and the low-level paraplegics could compensate for this drop by increasing their HR throughout the exercise. Nevertheless, these two groups still differed in terms of the HR-SV relationships. The authors speculated that this cardiovascular drift was caused by an increase in SKBF to disperse metabolic heat and a decrease in circulating blood volume due to fluid loss, thus imposing an additional load on the circulation. Unfortunately, SKBF was not measured in this investigation, so that the occurrence of active vasodilation could not be verified.


The group of Gass et al. (1988) was one of the first to investigate thermoregulatory responses to prolonged (wheelchair) exercise in SCI individuals (80 minutes at 63% of VO2 peak). Basically, they found that rectal and insensate skin temperature increased, whereas sensate skin temperature either did not vary or else it decreased in paraplegics. However, their subjects were wearing nylon tracksuit pants and the covering of the lower limbs by these pants may have amplified temperature increases in insensate skin. Surprisingly small absolute increases were recorded in rectal temperature (0.7 °C) as opposed to esophageal temperature, which questions the validity of rectal temperature as a measurement of core temperature in exercising paraplegics. Indeed, rectal temperature reacts slowly and may thus largely underestimate the thermal strain encountered, whereas the faster reacting
esophageal temperature is probably more representative (cf. Figure 6). The first investigation carried out on both SCI (T9-L4, n=5) and AB subjects (women) with respect to thermal adjustments during prolonged upper body (wheelchair) exercise is the one reported by Fitzgerald et al. (1990) (90 min at 50% VO2 peak). Oral temperature showed practically no change in the AB group, whereas it increased progressively in the wheelchair-dependent subjects, thus indicating less efficient thermoregulation. Mean skin temperature, as obtained from measurements on the trunk, forearm and calf had a decreasing trend in the AB subjects, suggesting effective evaporation cooling, by way of contrast to the increase found in the SCI individuals. According to the authors, the latter fact is indicative of heat transfer from the core to the cutaneous vasculature by the blood and inefficient heat dissipation through little sweat evaporation.

Hopman et al. (1993b) found that during prolonged arm work in a hot environment, weight loss and mean sweat rate of SCI subjects were related to the level of the lesion and thus to the area of sensate skin. Sweating below the lesion was minimal, and, accordingly, hemoglobin concentrations increased significantly only in AB and in subjects with lesions below T6. Paraplegics above this level had no significant changes in hemoglobin concentration. Although this could be viewed as an advantage for the circulatory system in these subjects, they appeared to be at an exposed risk of hyperthermia, as evidenced by the significant increase of their rectal temperature. Surprisingly, the latter parameter did not rise significantly in AB subjects or in low-level paraplegics, thus confirming that rectal temperature underestimates the thermal burden encountered during arm exercise. Similarly, Dawson et al. (1994) detected little increases in this parameter under thermoneutral conditions, with no differences between their T12-L1 paraplegics and the AB subjects. As an alternative, Petrofsky (1992) studied aural temperature by placing a thermistor temperature probe near the eardrum of his subjects and found rather more pronounced differences between AB and SCI subjects during heat stress combined with arm cranking or FNS-induced leg cycling. The investigation carried out by Price and Campbell (1997) revealed no differences for this parameter, probably because their ten paraplegics exercised at a lower absolute VO2 than the AB subjects (1.34 versus 1.92 l×min-1), and because they were very heterogeneous with respect to the level and nature of their SCI.

**SKBF and thermoregulation**

![Figure 7: Changes in leg skin blood flow (SKBF) and leg skin temperature (Tskin) in AB subjects (circles, n = 6) and in individuals with T5-T12 paraplegia (squares, n = 12) during a maximal arm cranking test (similar maximal exercise performance); * significant difference between the two groups (From "Cutaneous vasomotor adjustments during arm cranking in individuals with paraplegia," by D. Theisen, Y. Vanlandewijck, X. Sturbois, M. Francaux, 2000b, European Journal of Applied Physiology, 83, 539-544. Copyright 1994 by Springer-Verlag. Adapted with permission of the author).](image-url)
adequate, temperature gradient must exist between core and periphery and between ambient temperature and the skin, which are optimised by a higher core temperature and a greater shunting of blood to the skin, respectively. This reasoning has been referred to in an effort to explain the cardiovascular adaptations (lower SV and higher HR values) in individuals with SCI on the basis of a hypothetical greater shunting of blood to the cutaneous vasculature (Fitzgerald et al., 1990). This hypothesis has gained additional credibility through observations of higher skin temperatures in individuals with SCI as compared to AB subjects (Dawson et al., 1994, Fitzgerald et al., 1990). However, skin temperature changes should not be interpreted in terms of SKBF since blood flow is likely to increase through active vasodilation, while temperature may decrease due to sweating and evaporative cooling (depending on environmental conditions). Indeed, during maximal (Theisen et al., 2000b) or prolonged submaximal arm cranking exercise (Theisen et al., 2001b) in a thermoneutral environment, leg SKBF increased in AB subjects to ~350% of pre-exercise rest values while leg skin temperature decreased by 1-2 °C. In contrast, no active vasodilation was observed in either T5-T9 or T10-T12 paraplegics who increased leg SKBF only to ~150% of pre-exercise levels. These observations suggest that there is no excessive shunting of blood to the skin to favour dry heat exchange in the paralysed body regions. Leg skin temperature did not decrease in individuals with SCI, thus implying a lack of evaporative cooling in the legs. As a consequence, increases in esophageal temperature were significantly higher than in the AB group. Similarly, Muraki et al. (1995) found that sympathetic vasodilation in the anterior thigh occurred only in AB subjects and in individuals with lesions below L1 (n=4), not in persons with lesions above T12 (n=6). This would suggest that dry heat exchange through active vasodilation can only be enhanced if sympathetic innervation of the area is preserved (the lower limb is innervated by sympathetic nerve fibres emerging from levels T10-L2).

**Conclusion**

The development from the acute phase to longstanding SCI involves a number of physiological adaptations that contribute to a generally appropriate cardiovascular stability in these individuals at rest. Possible determinants involve spinal or peripheral sympathetic reflexes, long-term hormonal adjustments, histochemical changes in the paralyzed lower limb muscles and reductions in the capacity of the vascular tree. Episodes of autonomic dysreflexia, triggered by a noiceptive stimulus below the spinal lesion, represent a distressing and serious problem. The mechanisms involved are increased sensitivity to adrenergic receptors and limited compensatory mechanisms, especially in individuals with SCI above T5. Intentional stimulation of this reflex to enhance exercise performance is dangerous and prohibited. During arm exercise, individuals with SCI generally have higher HR and lower SV than AB control subjects. This results from a reduced or lost ability to redistribute blood from the paralysed body parts through sympathetic vasoconstriction. The lower limbs do not represent an area of significant blood pooling, due to the atrophy of the muscular and vascular tissues. However, the splanchnic area, which so far remains unexplored, represents a possible candidate for blood pooling as a result of spinal lesion. The regulation of body temperature at rest and during exercise is disturbed in individuals with SCI, and the degree of disturbance is directly related to the level of the lesion. Body core temperatures tend to be higher compare to those of AB subjects, due to a deficient sweat response and limited cutaneous vasodilation in the insensate body areas. Sweat responses and skin blood flow above the lesion are limited during passive heat exposure - and probably also during exercise - due to a lack of sensitive input.
References


Leistungsentwicklung im freistilschwimmen bei Paralympics-Teilnehmern

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Abstract

Evolution des performances chez les nageurs de nage libre paralympiques

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Abstract

Une série d’informations ont été réunies et chiffrées afin de quantifier l’évolution des performances en nage libre entre les Jeux paralympiques d’Atlanta en 1996 et ceux de Sydney en 2000. Le nombre total de participants n’a pas augmenté, mais ceux-ci ont été plus uniformément répartis dans les différentes catégories en compétition. L’écart entre le nombre de femmes et d’hommes est resté inchangé. Les courses ont été plus rapides. Les meilleurs chronos ont été améliorés, mais les temps des 8e places (de qualification) ont progressé davantage. Par conséquent, l’éventail des performances s’est réduit. Les performances des courses ont diminué dans tous les cas par rapport à la catégorie des handicaps fonctionnels, bien que ces écarts n’aient pas toujours été significatifs. Les performances des catégories n’ont pas accusé des différences plus marquées après Sydney, sauf dans les catégories supérieures du 100 mètres. Les résultats confirment l’opinion que le sport évolue mais qu’il y a encore des possibilités d’amélioration, notamment dans quelques catégories féminines.
Cardiovascular responses and thermoregulation in individuals with spinal cord injury

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Abstract

The cardiovascular system of individuals with spinal cord injury (SCI) has been the focus of many studies over the last decades. The findings of these studies have provided insight into the acute and long-term cardiovascular adjustments at rest and during exercise following SCI. They provide a basis for efficient intervention in rehabilitation and sports science.

During the initial phase of SCI, the individual may suffer from orthostatic hypotension, especially if the lesion level is in the cervical region. Recovery from this phenomenon occurs with time and training. The cardiovascular stability of individuals with longstanding spinal cord injury (SCI) is restored by a series of long-term adaptations. These involve spinal and peripheral sympathetic reflexes (veno-arteriolar reflex), hormonal adaptations (enhanced renin and vasopressin responses) and atrophy of the lower limb muscles and vascular tree. Individuals with high SCI, especially those with lesions in the cervical region, may be subject to autonomic dysreflexia. This so-called “mass reflex” is characterized by a generalized peripheral and splanchnic vasoconstriction leading to hypertension and is generated by nociceptive input from below the spinal lesion. The triggering of this potentially harmful reflex during exercise is known as “boosting” and is prohibited according to the doping guidelines of the International Paralympic Committee.

During exercise, subjects with SCI tend to have a higher heart rate and a lower stroke volume than able-bodied subjects as a result of an inability to redirect blood from the paralysed body areas. This may limit the maximal cardiac output, the oxygen consumption and thus the exercise capacity compared to the able-bodied, especially in high-level SCI. Individuals with tetraplegia have a limited ability to increase their heart rate (maximal heart rate is usually less than 130 beats per minute) due to a lack of sympathetic innervation to the heart. In exercising individuals with SCI, venous return can be enhanced by lower body positive pressure or by activation of the lower limb muscle pump via electrical stimulation of the paralysed muscles. These techniques tend to lower heart rate at a given exercise intensity and may increase peak exercise performance in persons with high spinal lesions. Results from studies using such an approach have been interpreted as an indirect indication of excessive venous blood
pooling in the lower limbs. However, recent studies suggest that the lower limbs do not represent an area of significant blood pooling in subjects with long-term paralysis, due to the dramatic atrophy of the leg muscles and vascular tree. It is therefore more appropriate to describe the impaired blood flow adaptations in individuals with SCI as a reduced ability to redirect blood from inactive body areas below the spinal lesion to the heart.

Temperature regulation is impaired in individuals with SCI, its efficiency being related to the level of the spinal lesion. Environmental conditions have a stronger influence on skin and body core temperatures than in able-bodied subjects. During passive heat exposure or exercise, the reduced capacity to sweat and to actively increase skin blood flow in the insensate body regions, as well as the likely reduced thermoregulatory effector drive in sensate body parts, exposes individuals with SCI to higher core temperatures compared to able-bodied subjects. Prolonged sub-maximal exercise under strenuous thermal conditions provokes a decrease in cardiac stroke volume and a compensatory increase in heart rate, which may be insufficient in individuals with high lesion levels (> T5). This cardiovascular drift has been interpreted in terms of a hypothetical greater shunting of blood to the cutaneous vasculature to favour dry heat exchange and to compensate for the reduced sweating ability. However, direct measurements have shown that increases in skin blood flow in the lower limbs of individuals with SCI are small, as opposed to the large increases through active vasodilation observed in able-bodied subjects.