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POSTOCCLUSIVE REACTIVE HYPEREMIA AND THERMAL RESPONSE IN THE SKIN MICROCIRCULATION OF SUBJECTS WITH SPINAL CORD INJURY

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ABSTRACT. The response of skin blood cell flux (SBF) to locally applied pressure was evaluated by laser Doppler fluxmetry over the sacrum and the gluteus maximus muscle in twenty patients with spinal cord injuries (SCI)—ten with tetraplegia, ten with paraplegia—and ten healthy subjects. The SCI patients were further divided into two subgroups, one with sensation and the other without sensation over the sacrum area. The SBF over the sacrum, without applied pressure, showed somewhat higher values among the patients. The ten paraplegic patients ($p<0.05$) and the subgroup of patients without sensation over the sacrum ($p<0.05$) showed the highest values. Occlusion of the SBF was reached at a lower external skin pressure over the sacrum than over the gluteus maximus muscle in the group with spinal cord injuries ($p<0.01$). During the postocclusive reactive hyperemia we found a much shorter time to peak SBF over the gluteus muscle for the patients compared to the healthy subjects ($p<0.01$). In the subgroup of patients without sensation over the sacrum a prolonged time to peak SBF was found ($p<0.01$) over the sacrum compared to patients with sensation and to healthy subjects. The increase of the SBF during postocclusive hyperemia response was lower over both the sacrum and the gluteus maximus muscle areas in the patients with spinal cord injuries ($p<0.01$). The temperature response was weaker in the groups with spinal cord injuries compared to healthy subjects ($p<0.05$), and especially in the subgroup of patients without sensation over the sacrum ($p<0.05$) compared to the other subgroup and to healthy subjects. It is concluded that SBF and local skin temperature show different responses to local pressure in patients with spinal cord injuries and healthy subjects. The described technique seems to be of value for studying changes in the reactivity of the skin microcirculation in areas with a high or low risk of pressure sores.

Key words: decubitus ulcers, pressure sores, tetraplegics, paraplegics, skin microcirculation, skin temperature, laser Doppler fluxmetry.

INTRODUCTION

Individuals with spinal cord injuries (SCI) comprise a well-known risk group for developing pressure sores, mainly due to reduced motor function and loss of sensation. The paralysis and loss of sensation make the patients unable to feel pain and discomfort and to change the pressure areas, especially among the tetraplegics (13). The most susceptible area for pressure sores in the SCI is the sacrum area. Even if the risk is greatest immediately after the injury, the risk will still remain for the rest of their lives and seems to increase with age. The external pressure over bony prominences will decrease the skin blood flow, leading to ischemia and tissue damage. Our question is whether the impaired autonomic nerve function seen in the SCI may contribute to a disturbed local microvascular reactivity resulting in pressure sores in the sacrum area?

In the present study we have examined the reactivity of the skin microcirculation and temperature related to the external pressure in the sacrum area in patients with spinal cord injury. Similar data were obtained over the gluteus maximus muscle as a reference area.

MATERIAL AND METHODS

Twenty subjects with spinal cord injury were studied. Clinical data are shown in Table I. All were living in their own home and were regularly visiting a department of rehabilitation for training exercises. Most of them complained of problems with temperature regulation, such as difficulties in shivering, perspiration and in loss of sensation. However, some of them had sensation over the sacrum, therefore subgroups of patients with sensation (6 with cervical and 2 with thoracal lesion) and without sensation (4 with cervical and 8 with thoracal lesion) over the sacrum were evaluated separately (Table I). No actual sign of tissue damage or ulcers were present over the areas studied. All patients had received their injuries more than one year previous to the investigation. The

Table I. *Clinical data*

+ feel sensation, – feel no sensation, over the sacrum area

Case no., sex	Level of lesion	Age (yrs)	BP (mmHg)	Skin sens. sacral area	Type of lesion
Tetraplegic patients (G 2)					
1 F	C 2–3	44	110/70	–	Complete
2 M	C 5–7	43	110/75	+	Incomplete
3 M	C 7	53	105/70	–	Complete
4 M	C 7	36	115/65	+	Clinically complete
5 M	C 6	30	110/70	–	Complete
6 M	C 7	21	120/70	–	Complete
7 M	C 4–6	23	115/65	+	Incomplete
8 M	C 7	45	115/70	+	Incomplete
9 M	C 5–6	45	90/55	+	Clinically complete
10 M	C 5–7	38	110/70	+	Incomplete
Paraplegic patients (G 3)					
1 F	Th 11–12	32	130/80	–	Complete
2 F	Th 12	53	125/75	–	Complete
3 M	Th 12	43	140/75	+	Clinically complete
4 M	Th 5–8	51	130/85	–	Complete
5 M	Th 10–11	23	110/70	–	Complete
6 M	Th 10–11	24	110/75	–	Complete
7 M	Th 6–7	45	140/90	+	Incomplete
8 M	Th 8–9	41	125/80	–	Complete
9 M	Th 10–11	43	130/85	–	Complete
10 F	Th 7–8	32	125/75	–	Complete

study was approved by the ethical committee of the hospital, and all subjects gave their prior informed consent.

Abbreviations

SBF: skin blood cell flux expressed in arbitrary units (AU).
occl P: (occlusion pressure) external pressure needed to stop SBF.
rSBF: SBF measured under resting conditions.
pSBF: peak value of SBF occurring after 3 min of arterial occlusion.
tpSBF: time from end of occlusion to peak SBF.
PRH: postocclusive reactive hyperemia response.
PRH %: percentage increase of SBF during the PRH.
ΔT: temperature changes measured during arterial occlusion and PRH.
W/H-index: weight to height index:

$$\frac{\text{individual weight (kg)}}{\text{reference weight (kg)}} \times 100\%.$$

The W/H-index was recorded for all subjects. The index is a relative measure of a person's weight. The individual reference weight is the height dependent average weight in the Swedish population and is calculated from the formulas: For males: 0.8 × individual length (cm) – 62 (kg). For females: 0.65 × individual length (cm) – 40.4 (kg). A W/H-index within the range 80–120% is considered as "normal" (1).

Group 1 (G 1) comprised 10 (5 F, 5 M) healthy subjects with a mean age of 36 years (SD ±4; range 22–54) and with a blood pressure of 128/70 ± 11/7 mmHg (mean ± SD).

Group 2 (G 2) comprised 10 (1 F, 9 M) tetraplegic subjects with a mean age of 38 years (SD ±10; range 21–53) and with a blood pressure of 110/68 ± 8/5 mmHg (mean ± SD). The spinal cord injury was at the C II–C VII level. All but one had had pressure sores over the sacrum early after injury, and two were treated for osteomyelitis.

Group 3 (G 3) comprised 10 (3 F, 7 M) paraplegic subjects with a mean age of 39 years (SD ±10; range 23–53) and with a blood pressure of 127/79 ± 10/6 mmHg (mean ± SD). The spinal cord injury was at the Th V–Th XII level. Three had previously developed pressure sores over the sacrum area, 4 over the buttocks and 1 over the trochanter area.

Skin blood cell flux measurements (SBF)

The local skin microcirculation was evaluated by the technique of laser Doppler fluxmetry (Periflux PF1d, Perimed, Stockholm, Sweden). The recorded signal is related to the flux of the blood cells, which is defined as the product of the number of blood cells and their velocities within the measuring volume (16). The measuring depth in the skin is not known, but the major part of the signal is generated by the movements of blood cells in the subpapillary, thermoregulatory vascular beds, and only a minute part from the nutritional capillaries (5). This technique shows both spatial and temporal variations in human skin blood flow (16).

The measurements were made at a bandwidth of 12 kHz, a

Fig. 1. Schematic picture of the two measuring points over the sacrum (1) and the gluteus maximus muscle (2) areas.

gain setting of ×10, and a time constant of 0.2 s. This setting was found to be optimal for recording the postocclusive reactive hyperemia response. The laser Doppler fluxmeter was connected to a printing device using a paper speed of 120 mm/min. The laser Doppler signal was recorded during 12 min, that includes the postocclusive reactive hyperemia response during 9 min. The whole investigation took about 20 min for each area, including equilibration time before the start.

Measuring procedure

The external pressure was applied to the skin over the sacrum and the gluteus maximus muscle with a specially designed device which has been described previously (14). The device consisted of a pivoted arm equipped with a pressure cup in its front end. The pressure cup was made of plexiglass and had a loose bottom disc ($r=1.0$ cm) which could be fastened to the skin with double-sided adhesive tape. The laser Doppler probe was placed in a probe holder fastened to the center of the bottom disc. In order to study the postocclusive reactive hyperemia response, the pressure could quickly and easily be removed from the skin by lifting the pressure cup leaving the bottom disc and the laser Doppler probe on the skin.

During maximal external pressure the laser Doppler signal fell to a level just above the zero value of the instrument. This value is considered to be partly due to a motion of blood cells in the skin microcirculation and/or optical noise produced by the laser Doppler probe (16) and is named the biological zero for the tissue (3).

The subjects were studied, lying in prone position. The measurements were made over the sacrum, about 10–12 cm below a line between the crista iliaca (Fig. 1). The reference point over the gluteus maximus muscle was situated on a line from the sacrum to the trochanter major, and in an area about 10–12 cm from the midpoint of the sacrum.

The measurements were started by recording the SBF. The pressure was then successively increased until the SBF signal leveled off for a few seconds, indicating no blood flow under the probe. This pressure was recorded and called the occlusion pressure for the area. The highest individual occlusion pressure recorded was 380 mmHg (50.5 kPa).

In order to abolish blood flow in the area and cause ischemia in all subjects, a local pressure of 400 mmHg (53.3 kPa) was then momentarily applied for three minutes. The mean baseline SBF during this period was recorded and represents the biological zero for the skin in that area (3).

When the pressure was released after 3 min, the laser Doppler signal started to increase immediately and reached a peak value after a certain time. During the decreasing phase of the postocclusive hyperemia response, a rhythmic variation in the laser Doppler signal started.

The skin temperature was monitored with a thermoelement probe which was positioned under the plexiglass disc and connected to an electronic thermometer with an accuracy of 0.1 % (Anritsu Meter Co., Ltd, Tokyo, Japan). The temperature was recorded prior to, and then every 30 s during the whole investigation of 12 min.

Statistical analysis

The SBF values were analysed in terms of mean value, standard deviation (SD) and standard error of the mean (SE). The data were compared using the Student's *t*-test. A probability level of 0.05 or less was considered significant. The reproducibility of certain SBF parameters was analysed in one subject from Group 1. The variability was evaluated by the coefficient of variation: $C=(\mathrm{SD/mean})\times 100\,\%$. Delta temperature ($\Delta T$) is calculated as the measured temperature subtracted by the temperature value at the end of the arterial occlusion period.

Reproducibility

The reproducibility of the SBF parameters was analysed in one subject from Group 1 on five separate occasions during a period of two weeks. As no significant differences between the values over the sacrum and gluteus were seen, the variability of the parameters was evaluated for the two areas jointly. The resulting coefficients of variation were 22 % for rSBF, 21 % for pSBF, 22 % for tpSBF and 24 % for PRH %. To determine whether a systematic difference existed between the first and the last test, we used Student's *t*-test. There were no significant differences between these measurements.

RESULTS

The individual clinical data for the SCI patients are shown in Table I and the different SBF values obtained are shown in Table II. All individuals studied had a weight to height index that was within the "normal" range. When measuring the arterial blood pressure we found that the tetraplegic patients had a significantly lower systolic and diastolic blood pressure both compared to the paraplegic patients ($p<0.001$) and the healthy subjects ($p<0.001$). There was also a lower systolic blood pressure in the subgroup without sensation ($p<0.05$) compared to the healthy subjects. However, no significant difference was seen between the two subgroups.

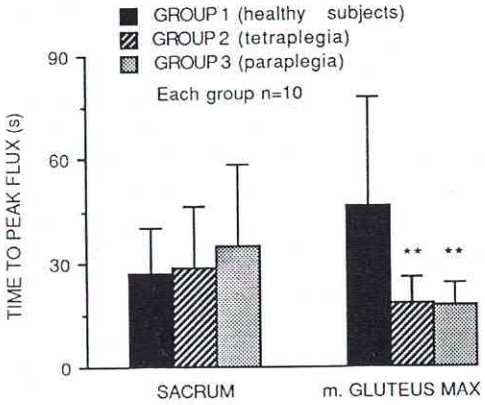

Fig. 2. Time to peak skin blood cell flux (tpSBF, mean ± SD) during reactive hyperemia after 3 min of arterial occlusion measured over the sacrum and the gluteus muscle. $^{**}p<0.01$.

Resting skin blood cell flux was measured over each area before the tests started. There were no significant differences in rest SBF between the sacrum and the gluteus areas within each group (Table II). The rest SBF over the sacrum showed nearly identical values in the two groups with spinal cord injuries (G 2 and G 3). The rest SBF in G 3 was significantly higher than in G 1 ($p<0.05$), and also tended to be higher in G 2 ($p<0.07$). There were no significant differences between the two subgroups, but the rest SBF in the subgroup without sensation was significantly higher ($p<0.05$) compared to the healthy subjects.

The rest SBF over the gluteus muscle showed no significant differences between the three groups or the two subgroups.

Arterial occlusion pressure limit value

In order to achieve arterial occlusion, a higher external pressure was needed over the gluteus muscle than over the sacrum in both G 2 ($p<0.01$) and G 3 ($p<0.01$). In G 1 there was no significant difference between the two areas.

When comparing the groups with each other (Table II) we found a significantly higher occlusion pressure value over the sacrum for G 1 compared both to G 2 ($p<0.001$) and G 3 ($p<0.01$).

No significant difference was seen over the sacrum between the SCI groups, or over the gluteus area between any of the groups or subgroups. In the subgroup with skin sensation the external pressure needed to stop the SBF over the sacrum was significantly lower (113±26 mmHg) compared to the subgroup without sensation (170±48 mmHg, $p<0.01$) and compared to the healthy subjects (259±97 mmHg, $p<0.01$).

Postocclusive reactive hyperemia response

The peak SBF did not show any significant difference between the SCI groups or between the two areas for any group (Table II). The only difference seen was a lower peak SBF over the gluteus maximus muscle in G 2 ($p<0.01$) compared to G 1. In the subgroup with preserved sensation a significantly lower peak SBF (31±11 AU, $p<0.05$) was seen over the sacrum area compared to G 1. The peak SBF over the gluteus muscle was significantly lower in both subgroups (with sensation: 34±5 AU, $p<0.01$; without sensation: 45±9 AU, $p<0.05$) compared to G 1 and the peak SBF over the gluteus muscle in the subgroup with sensation was significantly ($p<0.01$) lower compared to the other subgroup.

The time to peak SBF (Table II, Fig. 2) over the sacrum increased successively from G 1 to G 3, but there were no significant differences between the groups. The time to peak SBF over the gluteus was significantly ($p<0.01$) shorter for the two SCI groups compared to G 1. No significant difference was found between the tetraplegic and the paraplegic patients.

When evaluating the two subgroups, there was a

Table II. *Different values of skin blood cell flux (mean ± SD) in healthy subjects (Group 1) compared to patients with spinal cord injuries (Group 2=tetraplegic; Group 3=paraplegic patients) over the sacrum and the gluteus muscle*

For abbreviations, see text

	Group 1 ($n=10$)	Group 2 ($n=10$)	Group 3 ($n=10$)
Sacrum			
rSBF (AU)	4.9±2.2	7.4±3.9	7.8±3.9*
occl P (mmHg)	259±97	133±42***	161±52**
tpSBF (s)	27±14	28±18	35±23
pSBF (AU)	50±24	37±16	38±13
PRH %	1 022±394	493±315**	497±354**
Gluteus			
rSBF (AU)	4.6±1.5	5.0±1.6	6.4±4.0
occl P (mmHg)	262±77	222±75	230±55
tpSBF (s)	47±32	18±8**	18±7**
pSBF (AU)	62±24	39±7**	45±16
PRH %	1 365±638	724±248**	841±553**

$^{*}p<0.05$; $^{**}p<0.01$; $^{***}p<0.001$.

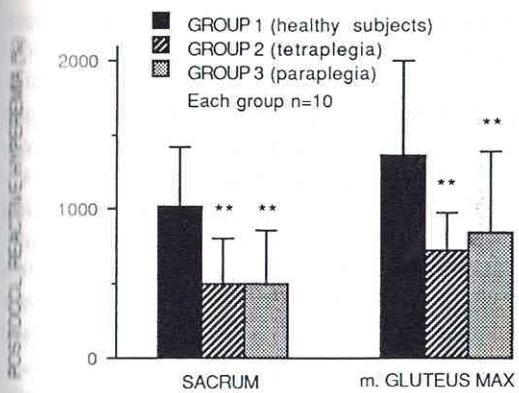

Fig. 3. The percentage increase of SBF (PRH%, mean ± SD) during postocclusive reactive hyperemia over the sacrum and the gluteus muscle. $^{**}p<0.01$.

significantly prolonged time to peak SBF in the subgroup without sensation (43±20 s) over the sacrum area, compared to the subgroup with sensation (16±5 s, $p<0.01$) and to the healthy subjects (27±14 s, $p<0.05$). The percentage increase of SBF during the postocclusive reactive hyperemia of G1 (Table II, Fig. 3) was significantly higher over both areas than in the SCI groups (sacrum: $p<0.01$; gluteus: $p<0.01$). In G2 the value over the sacrum was significantly lower compared to the value over the gluteus muscle ($p<0.05$). No significant difference was seen between the two subgroups.

Temperature measurements

The mean room temperature was 22.9±0.6°C (range 20.9–24.5°C). The skin temperature was measured at rest in all groups and over both areas (sacrum, G1: 32.1±1.3; G2: 31.4±0.8; G3: 32.0±1.0°C; gluteus, G1: 31.0±1.6; G2: 30.5±0.7; G3: 31.9±1.8°C). The only difference seen was a significantly ($p<0.05$) lower skin temperature over the gluteus muscle in G2 compared to the sacrum area. The skin temperature at rest was not found to be influenced by the room temperature.

During the arterial occlusion (Fig. 4) the temperature changes (ΔT) increased in all groups and all areas. The increase was significantly larger ($p<0.05-0.01$) over the gluteus muscle than over the sacrum in all groups.

During the postocclusive reactive hyperemia a more rapid increase of ΔT was seen during the first 1.5 to 4.0 min. There were large variations in the time to reach the maximum ΔT (range 0.5 to 8 min over the sacrum, and 0.5 to 4 min over the gluteus). The temperature pattern differed between the groups during the second part of the investigation. A strong response and a levelling off appearance was seen in G1, especially over the gluteus muscle compared to the sacrum ($p<0.01$). This pattern persisted in most of the subjects in G1 until the end of the investigation.

In contrast, a successive temperature decrease was seen in the SCI groups over both areas compared to G1 (gluteus: $p<0.05$; sacrum: NS).

When evaluating the ΔT in the two subgroups (Fig. 5) we found a strong temperature response ($p<0.05$) over the sacrum among those where the lesion obviously was not functionally complete compared to the other subgroup without sensation, which showed no such temperature response at all. The response in the subgroup with sensation over the sacrum area agreed with the response seen in the healthy subjects.

DISCUSSION

In a previous study, we have shown that a locally applied pressure of 110 mmHg (14.7 kPa) decreased

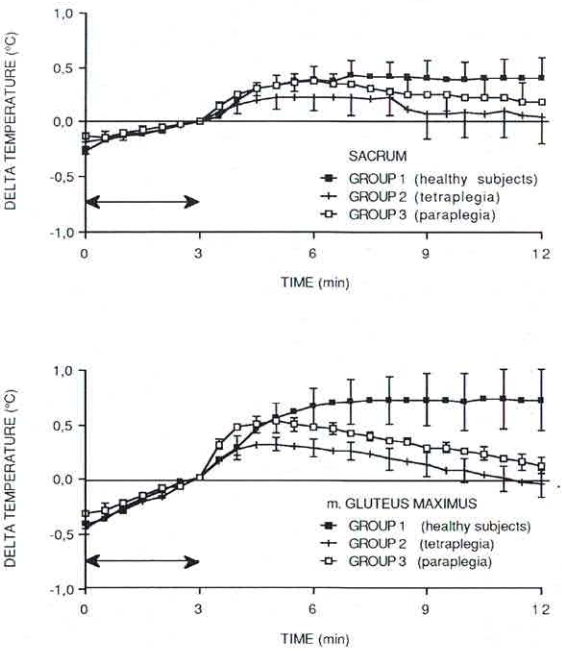

Fig. 4. Temperature changes (ΔT, mean ± SE) over the sacrum (upper part) and the gluteus muscle (lower part) areas respectively during (marked with an arrow) and after 3 min of arterial occlusion.

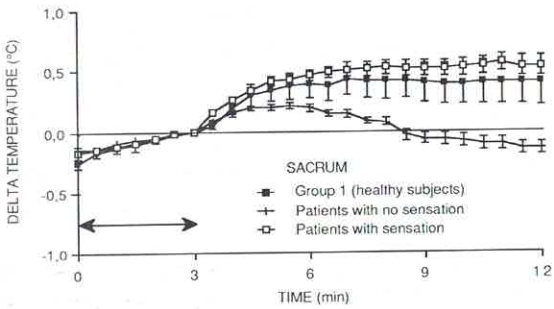

Fig. 5. Temperature changes (ΔT, mean ± SE) over the sacrum during (marked with an arrow) and after 3 min of arterial occlusion in the subgroups with ($n=8$) and without ($n=12$) sensation over the sacrum area. (For clinical data see Table I.)

the SBF over the sacrum in healthy subjects by approximately 43%, while the same pressure over the gluteus muscle did not give any decrease at all (14). In the present study, when using the same pressure device, we found that a locally applied mean pressure of about 260 mmHg (34.7 kPa) was needed to stop the blood flow in the skin microcirculation over both the sacrum and the gluteus muscle in healthy volunteers. In paraplegic and tetraplegic patients almost the same pressure had to be applied over the gluteus area to stop the skin microcirculation, while a significantly lower pressure was needed to stop the skin blood flow over the sacrum area. This increased sensitivity to pressure over sacrum may be one of the major factors for the higher incidence of pressure sores found over this area in patients with SCI. In most of the SCI patients in our study, the skin overlying the sacrum area also seemed to be thinner than in the healthy subjects, which may reflect a locally disturbed microcirculation in the region. A general weight loss and malnutrition appears to play an important role for the chronically ill patients with pressure sores (10) and in dementia patients. A low weight to height index indicates a certain degree of malnutrition (1). However, in our study, all subjects had a normal weight to height index, and consequently malnutrition could not be of importance for the observed differences. The SCI patients in our study had shown a high incidence of pressure sores soon after the injury while the patients still were in hospital. That may in part be explained by a disturbed microvascular reactivity during the early spinal shock but also reflects difficulties in the acute care of these patients. A better organization will most probably decrease the frequency of pressure sores.

As the control center of the blood pressure is located in the brain stem the transection of the spinal cord causes major disturbances of blood pressure regulation (8). When evaluating the arterial blood pressure in our patients, we found that the tetraplegic subjects, and also the subgroup without sensation over the sacrum area, had a significantly lower systolic blood pressure compared to healthy subjects. Similar findings have been reported previously in SCI patients (9). A low systolic blood pressure may consequently in some patients with SCI contribute to an increased risk for developing pressure sores over the sacrum.

A tendency to a higher rest SBF over the sacrum was seen in the SCI groups, although it was significantly increased only in the paraplegics, and in the subgroup without sensation over the sacrum. Wallin et al. (18) reported that cutaneous vessels might be more dilated at rest in patients with spinal cord injury due to sparse ongoing sympathetic activity and this is in accordance with our findings, but only in some patients with SCI. An increased total skin microcirculation, as evaluated by laser Doppler, but a normal capillary blood flow, has been found in low pressure areas of patients with moderate peripheral arterial insufficiency, and seems to indicate a vasodilatation caused by the decreased local blood pressure (2). An increase in the rest SBF may consequently not necessarily mean that there is a disturbed nutritional blood flow in the region, but only reflects a locally disturbed reactivity in the microvascular bed. An increase in SBF may not in itself be a risk factor for the development of pressure sores in SCI patients.

The postocclusive reactive hyperemia response, as evaluated by the percentage increase of SBF above the rest value, was significantly impaired in the SCI groups, both over the sacrum and the gluteus muscle, indicating a disturbed microvascular reactivity. Similar disturbances have been reported in diabetic patients with sympathetic neuropathy (17). The reason for this impaired reactivity may be that the responsiveness of the vascular smooth muscles to e.g. constrictor agents, such as epinephrine and norepinephrine, could be increased after denervation (19), but other studies provide no evidence for an increased sensitivity to norepinephrine in SCI patients (15). However, the regulation of the cutaneous blood flow is complicated and includes both sympathetic vasoconstrictor and vasodilatory activity. A disturbed balance between these two activities in the absence of supraspinal control could lead to a decreased ability of small arteries and arterioles to dilate during postoc-

clusive reactive hyperemia, and may explain the significantly reduced response seen in the SCI groups.

The main purpose of the skin microcirculation is to regulate the body temperature (6) and only a small portion is used for nutrition (5). The thermoregulation becomes poor when the spinal cord is damaged in the neck section above the sympathetic outflow, and the hypothalamus can then no longer control neither skin blood flow nor the degree of sweating (6). In our study, the temperature reaction after pressure release was markedly impaired in the patients with SCI, especially in the subgroup without sensation over the sacrum area. A high temperature over the sacrum area has been reported to be a predictor of sacral pressure sores (11). However, no patient had any sign of tissue damage at the time for the investigation.

During the arterial occlusion the temperature increased, and this is most probably due to lack of evaporation under the plexiglass (14). After pressure release there was a successive temperature increase during the early phase of the postocclusive reactive hyperemia, which is caused by the increase of blood flow in the region. In the healthy subjects and in the subgroup with sensation over the sacrum, this temperature increase persisted at a high level for at least 12 min, while it started to decrease after two to three minutes in the SCI groups in both areas, and especially in the subgroup without sensation over the sacrum.

Most of the tetraplegic patients complained of difficulties in the temperature regulation such as shivering and perspiration and some paraplegic patients also complained of cold feet. It is known that in subjects with cervical cord lesion, the occurrence of visible shivering is related to a low central temperature and can be seen in muscles innervated from the spinal cord above the lesion (7). However, many spinal lesions regarded as complete in fact are not (4), and there is evidence that a normal efferent sympathetic function in patients with lower injuries may be present below the lesion, so that the patient can still feel both sensation and cold (12).

When the subgroups of SCI patients were evaluated we found that though the lesion seemed to be clinically complete, there were patients with sensation over the sacrum area, and these patients showed a temperature response similar to the healthy subjects. The impaired thermal response to pressure in the subgroup without sensation over the sacrum is most probably due to the impaired autonomic nerve function in these patients.

In conclusion, the present study indicates that the reactivity of the skin microcirculation to locally applied pressure is significantly impaired in subjects with SCI as compared to healthy volunteers. Especially the subgroup without sensation over the sacrum seems to have a disturbed local microvascular reactivity indicated by a pathologic postocclusive reactive hyperemia response. These microcirculatory and thermal disturbances are most probably due to an impaired autonomic nerve function, and may be a contributing factor to the high number of pressure sores seen in SCI patients over the sacrum area. The simple technique used for studying the skin microvascular reactivity may be an effective tool for evaluating the risk of pressure sores to develop in patients at danger of this complication.

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