The Relationship Between Pressure Ulcers and Skin Blood Flow Response After a Local Cold Provocation

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Objective: To study the relationship between an impaired blood flow response after a local cold stimulus, testing nerve regulation of the local blood flow response, and an increased risk of developing pressure ulcers.

Design: An observational, longitudinal, prospective study.

Setting: Dutch nursing home.

Patients: Eighty-two newly admitted somatic nursing home patients, age 60 years and older.

Intervention: A local cold stimulus (17°C) applied to the trochanter major.

Main Outcome Measures: On admission, blood flow response to a local cold stimulus. As the stimulus was withdrawn, the temperature measured at the skin increased asymptotically toward the final temperature, Tf. The velocity of this rise was characterized by the time constant, τ, of the process. On admission, and weekly during a 4-week follow-up period, the presence or absence of pressure ulcers was verified.

Results: The blood flow response time correlated significantly with the risk of developing pressure ulcers. The patients who developed pressure ulcers during the follow-up period had a significantly longer response time than the patients who did not.

Conclusions: Malfunction of the nervous regulatory mechanisms of the local blood flow is partially responsible for an increased susceptibility to pressure ulcer formation.

Key Words: Autonomic nervous system; Elderly; Pressure ulcers; Rehabilitation; Skin temperature.

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EARLY RECOGNITION OF PATIENTS at risk for developing pressure ulcers has always been an important clinical problem. The risk of developing pressure ulcers results from a combination of 2 different sets of factors, the so-called external factors and internal factors.1,2 External factors are those related to the pressure (intensity, duration, direction); the internal factors (eg, fever, anemia, endothelial dysfunction, malnutrition) together determine a patient’s susceptibility to developing pressure ulcers. This susceptibility is a patient-bound characteristic that changes with time, depending on health status.4 Measuring this susceptibility has always been difficult because the underlying pathophysiologic mechanisms are unclear. Therefore, in most pressure ulcer risk assessment scores, a patient’s susceptibility to developing pressure ulcers is influenced by general physical condition, diabetes mellitus, nutritional status, and temperature.3-8

However, Meijer et al1 found that the recovery time of the local blood flow after relief of a local test pressure assessed the susceptibility to pressure ulcer formation. Susceptible patients were found to have substantially prolonged recovery times. Meijer1 calculated a patient’s risk of developing pressure ulcers by combining the outcome of the measurement of susceptibility with a measurement of the external forces acting on tissues (pressure, shear forces). The method used by Meijer,1 the so-called pressure-time-temperature (PTT) method, was based on an intense local pressure stimulus, which could be painful or induce local damage to the tissues. The objective of our study was to investigate the relationship between impaired blood flow response and increased risk for developing pressure ulcers, without using an external pressure stimulus to provoke this blood flow response.

Blood flow response after pressure relief is determined by various individual mechanisms. In local autoregulation, vasoactive chemical agents formed during ischemia play an important role.1 The elastic mechanical properties of the tissue must be of importance because these properties at least determine the penetration depth of an exercised pressure, and thus the severity of damage resulting from ischemia or distortion of the local endothelium and other tissues. The method used by Meijer,1 based on pressure as an external stimulus for the cessation of local blood flow, mainly tests local autoregulatory mechanisms. However, blood flow is also regulated by the autonomic nervous system. In a study9 of diabetic patients with autonomic neuropathy, it was suggested that impairment of the local blood flow response was the result of both endothelial damage and autonomic neuropathy.

Because the main function of the autonomic vasoregulation of the skin is thermoregulation, applying a temperature stimulus can test the autonomic nervous system. The vascular response after a low-temperature stimulus is not dependent on muscle tone and mechanical deformation of the tissue, but solely on sympathetic regulation. The use of a low-temperature stimulus to provoke a local blood flow response is neither painful nor harmful to the patient.

Our hypothesis was that the risk for developing pressure ulcers can be assessed by measuring the velocity of the blood flow response after a local cold provocation.

METHODS

Design

An observational, longitudinal, prospective design was used to study the relationship between the blood flow response after a local cold stimulus and the susceptibility to the development of pressure ulcers in a cohort of newly admitted, elderly nurs-
The skin with adhesive tape. The measurements were performed in a temperature-controlled room.

While lying on their side, patients were covered with a blanket, leaving 1 trochanter major exposed. They were then allowed to stabilize in the measurement position and to adapt to the environment during a period of 15 minutes before measurement. A physicist, who was unaware of the medical status of the patient, performed the measurements. Corresponding with the PTT measurements by Meijer et al., in which pressure was used as an external stimulus for provocation of the blood flow response, the method using temperature as a stimulus can be named the temperature-temperature-time (TTT) method.

Figure 1 shows a typical example of registration. The initial skin temperature is $T_0$. As the stimulus is applied, the skin temperature drops toward a new equilibrium (descending part of the curve). This process is dominated by the stimulus, and was not taken into account in the analysis. As the stimulus was withdrawn, the temperature measured at the skin increased asymptotically toward the final temperature, $T_f$. The velocity of this rise can be characterized by the time constant, $\tau$, of the process ($\tau \approx 1.44$ times the half-life, $t_{1/2}$, of the process). Both $\tau$ and $T_f$ are calculated by means of exponential regression analysis, in which an exponential function is fitted to the ascending part of the curve. Although the amplitude of the response is considered to be dominated by the amplitude of the stimulus, and to contain no clinically relevant information, the difference in final temperature and initial temperature ($T_f - T_0$) may be clinically relevant. In this study, both $\tau$ and $T_f - T_0$ were included as independent variables.

Measurement of Health Condition

Health condition was operationalized according to the Norton score. A change in Norton score of more than 3 points during the follow-up period was considered to indicate a substantial change in health condition. The Norton score of each patient was routinely measured by the research physician on admission and after 4 weeks, or if necessary more frequently during the study period.

Measurement of Pressure Ulcers

The examinations to determine the existence of pressure ulcers during the study period were performed by a research
physician who was unaware of the results of the measurement of the blood flow response. The first examination was performed within 48 hours after admission to the nursing home.

Each patient was examined from head to toe for the presence or absence of pressure ulcers. A pressure ulcer was defined as any degenerative change, caused under influence of pressure and shear forces acting on biologic tissues. In this study, nonblanching hyperemia was considered to be the initial manifestation of a pressure ulcer.

### Analysis

The mean response time (τ) and the difference between final and initial temperature measurement (T_f − T_0) of the group of patients with newly developed pressure ulcers (ulcers developed during follow-up period regardless of location) were compared with those of the group of patients with no newly developed pressure ulcers by means of Student’s t test.

The relationship between the time difference in skin temperature, the response-time, and the risk of developing pressure ulcers was calculated by means of logistic regression analysis. These relationships were adjusted for age, gender, and prevention given.

### RESULTS

Of the 145 patients who met the inclusion criteria, 29 patients were not measured for the following reasons: refusal (n = 6), assessed as unsuitable by the nursing home physician for medical or ethical reasons (n = 13), or measurement not performed within 48 hours after admission (n = 10). Of the remaining 116 patients who were measured, 34 patients (29%) were excluded for the following reasons: the presence of a pressure ulcer on admission that remained stable or was cured during the follow-up period (n = 20), discharge or death within 2 weeks after admission without developing a pressure ulcer (n = 9), and substantial change in health condition (n = 5).

Data on 82 patients were included in the analyses.

In total, 21 patients (26%) developed a pressure ulcer during the study period. Blood flow responses of patients in the group who developed pressure ulcers (D+ group) were compared with the responses of those who did not, develop pressure ulcers (D− group) (Table 1). D+ group patients showed a significantly slower response time compared with the D− group (Table 1).

### DISCUSSION

The objective of this study was to investigate whether a relationship exists between the blood flow response after a local cold stimulus, using the TTT method, and the risk for developing pressure ulcers. In this prospective evaluation, a significant relationship was found between τ (the response time of the skin temperature after relief of the low-temperature stimulus) and the risk for developing pressure ulcers. The D+ group showed a significantly slower response time compared with the D− group (Table 1).

From these observations, it can be concluded that malfunction of the nervous regulatory mechanisms of the local blood flow is at least partially related to increased susceptibility to pressure ulcer formation. These findings support the observations reported by Mein et al, who investigated the influence of spinal anesthesia on the local blood flow response as a model for autonomic neuropathy. They reported a doubling of the blood flow response time after relief of a pressure stimulus during spinal anesthesia. However, in their study, no distinction could be made between the influence of blockage of the autonomic nerves and the motor nerves, resulting in paralysis of muscle tissue. Moreover, the stimulus used in their study (local pressure) induced tissue strain.

Because in our study no tissue strain was induced, it can be concluded that the delayed blood flow response, which was also reported by Mein, has to be attributed to malfunction of the autonomic nervous system.

The studies performed by Meijer and Mein, based on a pressure stimulus, show a much greater variation in the blood flow response, compared with the relatively small variation observed in the response provoked by a temperature stimulus, whereas both responses correlated with the risk of developing pressure ulcers. This may imply that both responses are related, at least partially, to different sets of subfactors of susceptibility.

However, the reliability of the 2 methods may differ, so this assumption should be made with caution. It cannot be excluded that the experimental setting influenced the variations. There is an important difference between the pressure stimulus test and the temperature stimulus test. With the pressure stimulus, at the end of the stimulation period there is thermal equilibrium in the tissue, whereas with the temperature stimulus, there is an absence of thermal equilibrium. This implies that the velocity of the temperature response after relief of the temperature stimulus is modulated by the thermal diffusion flux, which complicates comparison of the responses of the 2 methods. The thermal diffusion flux may well be influenced by the amount of subcutaneous fat tissue.

A substantial variation in the difference between final and initial skin temperature (T_f − T_0) was observed. The skin temperature did not return to its original value after relief of the temperature stimulus in either group of patients. These observations suggest a persisting vasoconstriction. However, no relationship with the risk for developing pressure ulcers could be established. A similar, substantial variation was found in a previous study based on a comparable cold provocation test, in which fingers were cooled. However, the reported possible influence of ambient temperature on T_f − T_0 does not apply to the results of our study because room temperature was kept at a minimum of 24°C during the tests. Further research to determine the significance of this parameter seems to be indicated.

An important limitation of our study was the indirect measurement of blood flow by skin temperature. The use of other,
more direct, techniques for assessing skin blood flow (eg, the laser Doppler technique) can give more detailed information on vasoactivity after an external stimulus and therefore should be used in future research.

CONCLUSION

The information provided by this study can help in the assessment of patients at risk for developing pressure ulcers. In clinical practice, pressure ulcer risk assessment is mainly performed by using risk scores. An important shortcoming of these scores is the fact that their validity is based on specific patient populations. Patient outcome on a risk score is always a reflection of the mean risk for all patients with the same score in that specific population. Direct measurement of the patient’s individual susceptibility therefore should be preferred. Further research should focus on developing techniques that can do this and that can be used in clinical practice.

References


Supplier

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