situations this duration of occlusion seldom occurs, some types of clothing and therapeutically applied dressings, bandages, tapes and several topically used vehicles including soft paraffin may result in occlusive or semi-occlusive conditions lasting for various periods of time, mostly as short-term occlusion. Therefore, the results of this study may not be fully comparable with the conditions of everyday life for these patients. Whereas occlusion promoting hydration of the usually dry epidermis in atopic dermatitis could be of some benefit for these patients, the quantitative increase in the skin microflora—particularly of *S. aureus*—after 24 hours' occlusion may become harmful to the patient and his/her environment. Similar considerations should be borne in mind regarding psoriasis: the high rate of desquamation and thus the dispersal of *S. aureus* as a public health hazard has been noted (6). According to these current findings, one should be aware of the possible need for antibacterial measures in the management of both skin diseases, especially for atopic dermatitis, when prolonged occlusion is required. The experimental data here concern 24 hours' occlusion; unfortunately we note (unpublished data) more than the uncommon staphylococcal infection with even our more limited occlusion (8 hours) utilized in clinical practice.

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Blood Hyperviscosity in Psoriasis

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Abstract. A study of platelet function and whole-blood viscosity in 11 patients with the confirmed diagnosis of psoriasis revealed a significant elevation in whole-blood viscosity (3.33±0.37 cP) as compared with that found in a control group (2.80±0.1 cP). The platelet count and platelet aggregation with ADP, epinephrine and collagen as well as platelet malonyldialdehyde were all within the normal range in all the patients. It is suggested that the increased blood viscosity may contribute to the higher incidence of occlusive vascular disease occurring in patients with psoriasis.

Key words: Psoriasis; Hyperviscosity of blood; Occlusive vascular disease

The high incidence of occlusive vascular episodes occurring in psoriatic patients treated with Azaribine prompted a number of investigators to carry out retrospective studies. In a recent study of dermatological patients, McDonald & Calabresi (4) noted that psoriatic patients have a significantly higher incidence of occlusive vascular disease than non-psoriatic patients.

The reports in the literature confine themselves largely to clinical observations and statistical analyses of such cases, and little has been done to elucidate the mechanism responsible for this phenomenon. We report here the findings of a study of platelet functions and whole-blood viscosity made in psoriatic patients.

Table I. *Blood viscosity and results of platelet function tests in controls and psoriatic patients*

	Viscosity (cP)	Platelet aggregation			Malonyl-dialdehyde (nmol/10^9 platelets)	Platelet count (per mm^3)
		ADP%	Epineph-rine %	Collagen%		
10 Controls	2.80±0.1	82±15.2	80±10.1	75±8.2	1.01±0.20	212 000±35 000
11 Patients	3.33±0.37	83.5±14.6	78.6±9.2	77.0±7.6	1.07±0.17	214 000±34 700

MATERIAL AND METHODS

The material comprised 11 patients of both sexes, aged from 10 to 71 years, who were hospitalized for the treatment of psoriasis. All had extensive, plaque-type psoriasis involving at least 30% of the body surface. In none was there any history of hematological or cardiovascular disease or of any complications such as clinical joint damage. None had any current or recent infection. None was taking any kind of medication at the time of the study and in none was there excessive alcohol consumption. For control purposes, 10 samples of blood were obtained from the blood bank and subjected to analysis. Analysis comprised the platelet count, assessment of platelet function on the basis of aggregation with ADP, epinephrine and collagen as well as malonydialdehyde content, and whole-blood viscosity.

A 20 ml sample of venous blood was obtained from each of the subjects. Two ml of the sample was anticoagulated on EDTA–heparin and used for the platelet count (2), using a Zeiss microscope and an improved Neubauer chamber (normal range 150 000–400 000/mm^3). The remaining 18 ml was anticoagulated with 2 ml sodium citrate 3.8%. Of this, 5 ml of whole blood was used to determine viscosity, using a kinematic viscometer Ostwald-Cannon-Fenske (3) (normal range 2–3 centipoise (cP)). The remainder was centrifugated at 85 *g* for 15 min and the platelet-rich plasma (PRP) obtained was used for the tests on platelet function. Platelet aggregation was determined by the method of Born & Cross (1) using an Evans Electroselenium (EEL) aggregation meter, model 169, with recorder. The platelet concentration was adjusted to 200 000±20000/ml. To 0.6 ml PRP were added the following aggregating substances: ADP to a final concentration of 2 μmol, epinephrine to a concentration of 1 μmol and native collagen 0.05 cc. Platelet-poor plasma was considered 100% and PRP 0%. The maximum percentage of aggregation at 4 min was calculated. The time required for each platelet aggregation determination was 6 min. (Normal laboratory range for all three aggregations was 70–100%.) Platelet malonyldialdehyde was determined using arachidonic acid as an activator (5) (normal 0.60–1.30 μmol/10^9 platelets).

RESULTS

The platelet counts were within the normal laboratory range—214 000±34 700/mm^3. Hemoglobin, hematocrit, red cell count, white cell count and the differential white cell count were all within normal range in all the samples. Whole-blood viscosity was significantly elevated in the psoriatic patients (3.33±0.37 cP) as compared with the control group (2.80±0.1 cP). Platelet aggregation with ADP, epinephrine and collagen and platelet malonyldialdehyde were all within normal range in all the subjects (Table I).

DISCUSSION

This study failed to reveal any abnormalities in platelet function, indicating that these are not involved in the higher incidence of occlusive vascular disease in psoriatic patients. The high values of blood viscosity, however, suggest that this abnormality may contribute to the vascular complications accompanying this disease. Whether this hyperviscosity of the blood in psoriasis is the result of a primary haematological abnormality or is secondary to the disease process cannot be determined from the available evidence. Whole-blood viscosity is influenced by many factors such as fibrinogen, other plasma proteins and altered properties of the red cell surface, none of which was analysed in the present study. The mechanism of the increased viscosity in this disease remains to be further clarified.

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