

From the Medical Department of the University Hospital, Utrecht, The Netherlands. (Director: Professor C. D. de Langen, M.D.)

THE CAPILLARY RESISTANCE IN ALLERGIC DISEASES

By

A. P. J. VAN DER BURG, M.D.

An impression of the resistance of the capillary vessels of the skin can be gained by examining their permeability to erythrocytes, a positive (propulsive) pressure or a negative (suction) pressure being applied.

The resistance of the endothelial wall depends on the tonus of the vessel, and on the strength and physical condition of the cells together with the intercellular cementing substance. These characteristics may change during diverse physiological and pathological circumstances.

According to the conceptions of *Nelemans* and *de Langen*, during rest—in basal conditions—the non-innervated true capillaries are closed by contraction of the entire endothelial wall. Blood supply is then provided by the hemoducts (called by the French “mesartérioles”) and by the anastomoses between arteriolae and venulae. When an organ becomes active, and is in need of an increased supply of blood, substances are formed in the tissue which cause a slight narrowing of the venulae and simultaneously a relaxation of the wall of the true capillaries, in which case the anastomoses are closed. These substances, CO₂, lactic acid, and especially histaminic substances, approach the capillaries from the tissue, dilate these vessels, and increase the permeability of the wall.

As a method of judging capillary resistance, propulsion

was introduced by *Auspitz* as long ago as 1874. Only in 1911 did this technic become generally known under the name of "Symptom of *Rumpel-Leede*". This method has been modified, and efforts made to find quantitative standards by many investigators, i.a. by *Goethlin* (1931) and *Elslo* (1948). A positive pressure method serves well to give a momentary

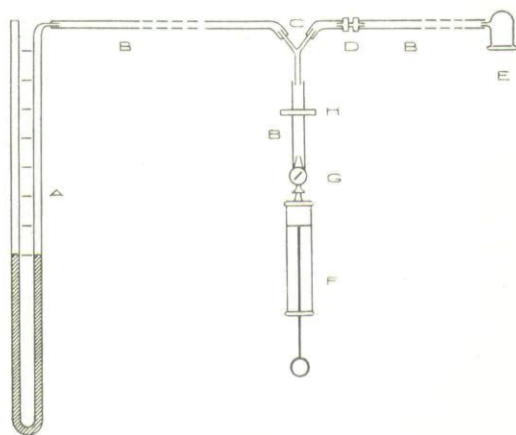


Fig. 1.

A - mercury-manometer; B - vacuum tube; C - metal Y-pipe; D - hose coupling; E - suction cup; F - syringe; G - cock; H - Hofman clamp.

impression of the general condition of the wall of the skin capillaries.

Changes in capillary resistance during disease or after administering drugs can be judged better with the suctiontest, derived from the ancient "cupping", and used by *Hecht* for the first time in 1907 as a method of examining capillary resistance. Since the publications of *von Borbély* (1930) the apparatus has consisted of a glass suction-cup (in our experiments with a diameter of 1 c.M.), a syringe or pump and a mercury-manometer or a springvacuumgauge, besides a convex lens. The capillary resistance is regarded as that negative pressure which causes the appearance of 1 or 2 petechiae in the centre of the base of the cup (Fig. 1).

The petechia found on the edge of the cup should not be taken into account, as they may arise by distortion. If to an arm which has been made empty of blood a negative pressure is applied which amounts to twice the value of the capillary resistance—and so normally would certainly cause petechia—then no petechia are seen to arise during the arterial closing. After reflux-stet of the blood petechia still do not appear in the centre of the site of suction although they do on the edge, where because of distortion a rent has appeared in the vessel. Therefore we are of the opinion that in the centre of the suction cup diapedesis takes place through the intercellular interstices of the capillary wall, or that those interstices become torn but are immediately resealed.

Almost all investigators, ourselves included, soon came to the conclusion, that the capillary resistance, measured in this way, is not the same in various parts of the body. The upper half of the body shows smaller values than the lower half. The highest capillary resistance is found on the palms and soles. In symmetrical places the values are fairly similar. Thus one should always measure in the same places. We generally used the skin infraclavicularly and cubittally.

It appeared to us that with this method of negative pressure one can also measure the capillary resistance of the mucous membrane on the inside of the lips, provided that before and after the suction the excess blood is well pushed away with a little glass or convex lens. We always found the capillary resistance of this mucosa to be about equal to that found infraclavicularly.

Many have tried to establish "normal" values for the capillary resistance, but this seems to be impossible. With *Frischknecht* (1945) and *Matti* (1950) we found that among healthy people there is a great spread between high and low values which are distributed according to a biological curve, most cubital values being found between 100 and 200 m.M. Hg., and most infraclavicular values between 75 and 150 m.M. Hg. (Fig. 2).

When measuring one should take into account small 24-

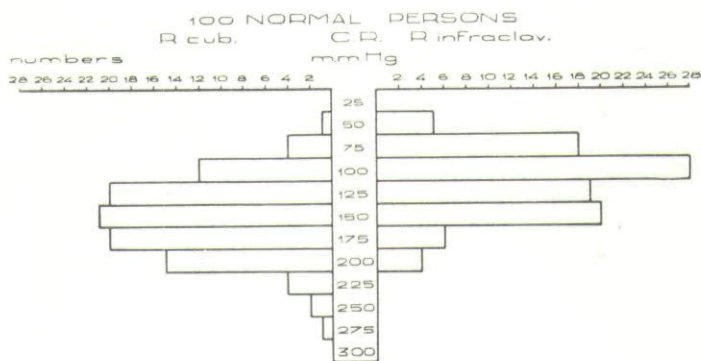


Fig. 2.

hour fluctuations in the capillary resistance. *Matti* and we, too, found a slight fall averaging about 34 m.M. Hg. (about 20 %) in the morning and early afternoon. Towards 15 O'clock (3 p.m.) the capillary resistance rises again to attain the highest level during the night.

We have found no indications that the capillary resistance falls with increasing age. Nevertheless children have a very high capillary resistance in the first months of life, viz. cubitally and infraclavicularly about 500 m.M. Hg. and higher.

Omitting measurements just before, during, and just after

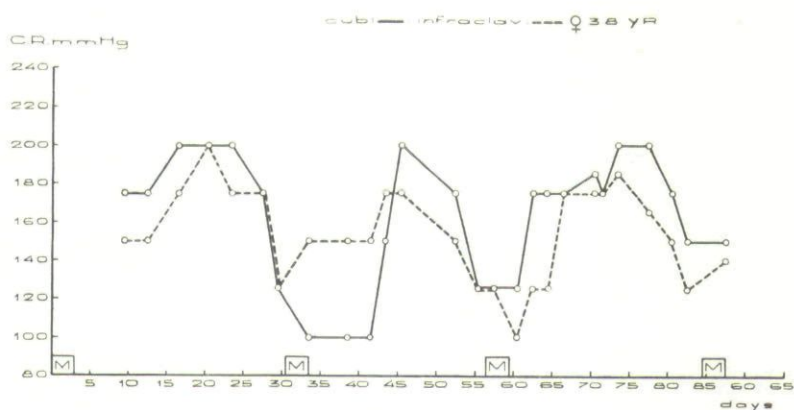


Fig. 3.

the menstruations, we found no differences in the capillary resistance of healthy men and women. But it appeared to us, and also to *von Borbély* a.o. that capillary resistance falls considerably close to or during menstruation. (Fig. 3). A menopausal woman examined during the same period did not show these falls.

Da Silva Mello (1929), *Bexelius* (1933) and *Sack* (1938) report that the psychic condition may influence capillary resistance. *Da Silva* often found in nervous people entirely negative reactions with the negative pressure method, even when applying a negative pressure of 400–500 m.M. Hg. He is of the opinion that psychic factors may have a vasoconstrictive influence. *Da Silva Mello's* observations are supported by a personal case:

At the first investigation of a nervous woman the capillary resistance was 200 m.M. Hg. infraclavicularly on the right side. After 10 min. infraclavicularly on the left 50 m.M. Hg. After half an hour again below the right clavicle 75 mm Hg. Similar observations occurred more than once, even in patients who had not been examined for a long time. Not too much value should therefore be attached to capillary resistance values found during the patient's first visit to the physician. Values found when the patient has got used to the investigator and the surroundings are more reliable. The same observation applies in the estimation of blood pressure.

We have briefly discussed the differences in capillary resistance occurring in physiological conditions which should be recognized in order to judge rightly the results of our measurements during disease and under the influence of medical treatment. It is widely known that all kinds of affections, e.g. haemopathies, avitaminoses, intoxications and infectious diseases may influence the permeability of the capillaries. The classical example of this is scarlet fever.

We will now discuss the relationship between capillary resistance and allergy. To this end we must first direct our attention to that interesting substance, histamine. Reports are unanimous that histamine (0.5–1 mg. subcutaneously) causes

an immediate and marked decrease of the capillary resistance, which rises to its normal value after $\frac{1}{2}$ –1 hour. (Sack, 1938; Franke, 1943; Koller and Diem, 1945; Wyss and Gianoli, 1946; Knoll, Wilbrandt and Wyss, 1949; Matti, 1950).

Our own investigations, in 5 patients, which were carried out with 0.5 mg. histamine subcutaneously, because of the

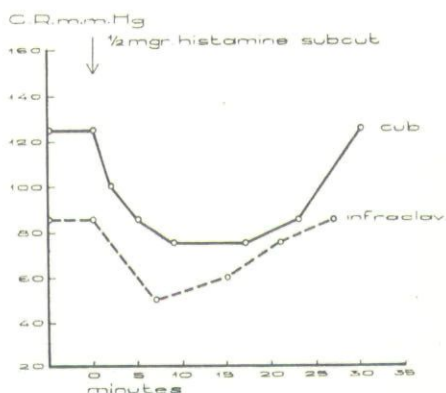


Fig. 4.

untoward effect following the administration of 1 mg., confirmed these results with a single exception, where no "histaminic flare" occurred, neither could a decrease in capillary resistance be observed.

In the other patients the decrease in capillary resistance set in almost immediately after the histamine injection and reached its lowest point after 7–20 minutes, with a decrease of the resistance of 25–50 m.M. Hg. The action lasted for $\frac{1}{2}$ –1 hour (Fig. 4).

The injections were given in the thigh, and the measurements of the capillary resistance were made infraclavicularly or antecubitally.

After subcutaneous injections of histamine in the forearm we often easily succeeded in provoking a positive Rumpel-Leede phenomenon.

Franke (1943) administered 2-3 mg. histamine orally without any effect. If he rubbed imadyl ointment (containing 2 % histamine) into the skin, he observed locally and at some distance from the site of rubbing a decrease in capillary resistance.

Koller and *Diem* (1945) gave interesting data. Following intracutaneous injection of 0.1 mg. histamine into an arm or a leg they observed a reaction absolutely contrary to that following 1 mg. subcutaneously, viz., 15 minutes after making the wheal they found a marked increase in capillary resistance, exceeding the range of their mercury manometer, reaching its maximum after an hour and sometimes lasting for days at a stretch.

Because the action of intracutaneously administered histamine was contrary to that of histamine administered subcutaneously, they presumed that it was the stimulation of the skin which caused the elevation in capillary resistance. Intracutaneous injection of bee venom, normal saline solution, and even procaine, also gave rise to an increase in capillary resistance.

Together with *Krogh* (1929) these investigators assume that some cutaneous stimuli liberate a histamine-like substance, if not histamine itself, from the injured tissues, which reaches the blood stream. Histamine possesses a vasodilator action, but after intracutaneous injection they observed an increase in capillary resistance. The rise might be induced by an exceedingly strong counter-action of the body, following the liberation of histamine.

Koller and *Diem* observed this reaction not in all, but in most of the test persons whom they called "normal".

Up till now we have injected 2 test persons with 0.1 mg. histamine, measuring the capillary resistance in the forearm and subclavicularly. The results reported by *Koller* and *Diem* were not confirmed. One test person showed in the forearm an increase in capillary resistance of 50 m.M. Hg., whereas subclavicularly the values remained the same, the other showed subclavicularly an increase of 50 m.M. Hg. and ante-

cubitally a decrease of 25 m.M. Hg. It is possible that these persons were accidentally non-reactors.

Nevertheless *Koller* and *Diem's* view is attractive, and one could imagine the liberation of adrenalin or ACTH, both of which increase the capillary resistance. Therefore one of the first objections that can be made is, why does the supposed

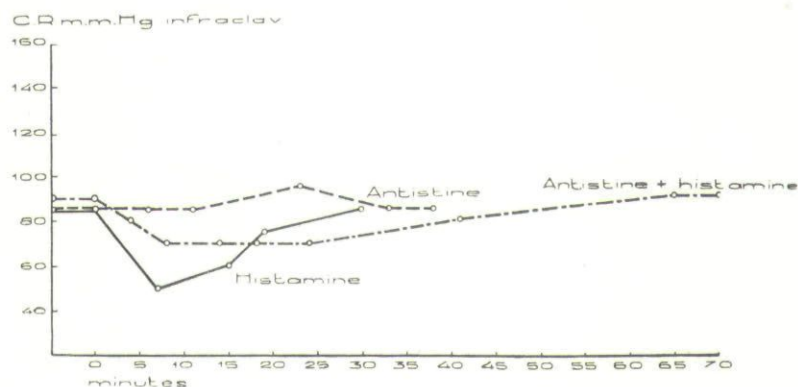


Fig. 5.

counter-action not occur following the subcutaneous injection of histamine?

What do the antihistaminics do?

Knoll, Wilbrandt and *Wyss* (1949) observed the influence of antistine on the capillary resistance in 4 persons. In 3 they found a decrease and in 1 an increase. They did not state, however, what type of patients these persons were.

Matti (1950) writes that antistine influences capillary resistance slightly or not at all. Our experiments agree with this observation, at least in healthy people.

Matti reports that antistine is capable of neutralizing histaminic action considerably. In order to verify this we followed the effect on a test person of 0.5 mg histamine subcutaneously on the first day, of 100 mg. antistine intravenously on the second, and on the third day, of 100 mg. antistine intravenously, immediately followed by 0.5 mg. histamine

subcutaneously. From the graphs made it can be concluded that in this test person antistine partly neutralizes and also delays the action of histamine (Fig. 5).

Experimentally, however, adrenalin exerts a stronger antagonistic action towards histamine than antistine. A 26-year-old female subject, who reacted to 0.5 mg. histamine with a

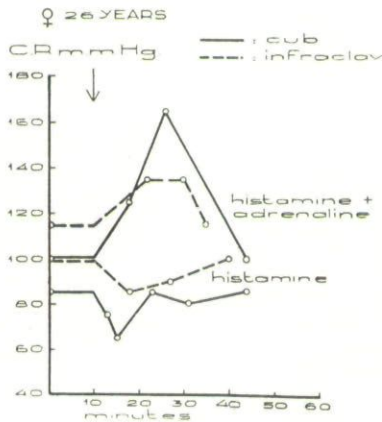


Fig. 6.

decrease of the capillary resistance and the appearance of a pronounced "flare", was given simultaneously 0.5 mg. histamine and 1 mg. adrenaline subcutaneously at various sites on the following day. This resulted in a rise in capillary resistance and a much less marked "flare" (Fig. 6).

Observations made by *Bayer, von Borbély, Franke* and ourselves show that calcium, which is much used in allergic diseases, nearly always yields a marked but transient increase in capillary resistance after intravenous administration (Fig. 7).

The suggestion that adrenalin, while raising the capillary resistance, stimulates ACTH production, occasioned *Robson and Duthie* (1950) to suppose that an increase in capillary resistance could be a consequence of an increased adrenocorticotrophic action. To this end they examined the capillary

resistance in 10 patients treated with ACTH. They found that a single dose of 25 mg. ACTH led to considerable rise of capillary resistance within a period of four hours, with one exception, who reacted 8 hours after the injection with increase in capillary resistance, when in other patients it was already decreasing.

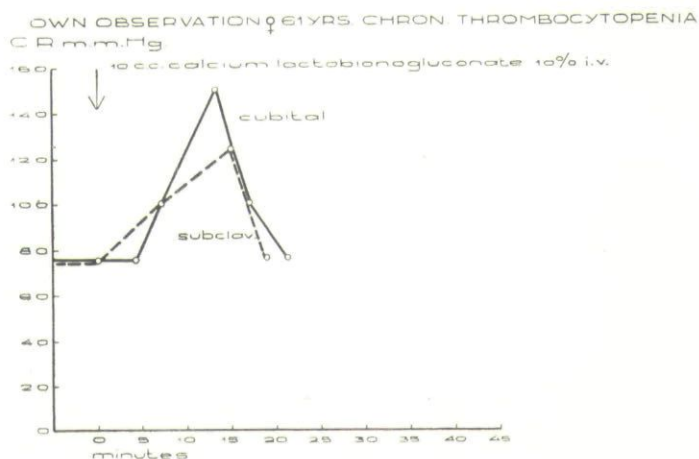


Fig. 7.

In all the cases the continued administration of 25 mg. ACTH every 8 hours resulted in a considerable increase of the capillary resistance, with its maximum after 72 hours. After cessation of the therapy the initial values were reached in 10-14 days.

In 2 rheumatic patients treated with ACTH we found after administration of ACTH a considerable increase of the capillary resistance of 50-75 %.

The findings in patients with allergic affections are now discussed.

Observing the Rumpel-Leede phenomenon in a positive Pirquet reaction, numerous petechiae are seen to arise around the positive skin reaction during the stasis, which are absent around the control-scratch. Marked differences are also found

in capillary resistance with the negative pressure method. We found the following values:

	A Fresh positive Pirquet	B Fresh positive Pirquet	C Fresh positive Pirquet	D Fresh positive Pirquet	E 8-day-old positive Mantoux	F 8-month- old positive Pirquet	
Capillary resistance							
middle forearm inside ...	300	250	200	250	400	350	Hg. mM
id. at the site of a posi- tive tuberculin reaction ...	75	50	25	50	300	250	

The capillary resistance also decreases in a localized Schwartzman phenomenon. (This is obtained by preparing the skin with the intracutaneous or subcutaneous injection of a bacterietoxin followed by administration of the same toxin intravenously after some hours. A haemorrhagic necrosis occurs in the pre-treated area of the skin (*Soffer and Schwartzman 1950, Schwartzman 1952*)).

Hoigné, Koller and Storck (1952) studied the localized Schwartzman phenomenon in animal experiments in order to clarify the pathogenesis of the haemorrhagic diathesis. Using the negative pressure method after pre-treatment of the skin, and especially after having provoked the phenomenon by a second injection, they observed a marked fall of the capillary resistance to 0 (not measurable on account of severe spontaneous haemorrhage) at the site of the pre-treated skin, and a decrease at other sites of the skin to half the initial value. They also found a decrease in the number of thrombocytes.

Soffer, Schwartzman, Sneyerson and Gabrilove (1950) report that ACTH, if given 2 hours before the provocative intravenous injection of the toxin, prevents the appearance of the phenomenon (the only other methods of suppressing the phenomenon are with nitrogen mustard, benzole or X-rays).

Hoigné, Koller and Storck (1952), also reported this,

and further observed that if ACTH was administered before the provocative injection, the capillary resistance in the parts of the skin not pre-treated did not decrease following intravenous injection of the toxin and did so to a lesser degree at the pre-treated site than without ACTH.

The use of cortisone requires doses 6 times larger to suppress the phenomenon.

Under allergic conditions fragility of the capillaries or decrease of the capillary resistance is often described. *Rapaport* (1941) saw this in 49 % of the allergic diseases in children. *Wyss* and *Gianoli* (1946) indicated that with the development of an asthmatic condition the density of the capillaries of the skin decreased, and increased with the recovery of the patient.

In a woman with a primula-urticaria on the upperarm, we found, repeatedly, on the site of the exanthema a capillary resistance of 60 m.M.Hg., on the healthy skin next to the exanthema of 110 m.M.Hg.

Berger (1940) tried to give an acceptable explanation of this phenomenon. The allergic antibody reaction should provoke an irritation with a functional tissue reaction. Part of the tissue reaction is the local shock of the bloodvessels: first vasoconstriction, after that dilatation by paralysis of the contractive elements of the blood vessels, hemostasis, stretching and damaging of the endothelial cells and the cementing substance, with exudation as a consequence. *Tsuji Kwanji* is of the opinion, that this process, that should cause exudation of the lung capillaries, is also the decisive factor for the outbreak of the attack of asthma. The improvement of the bronchial asthma and capillary resistance, which *Wyss* saw in high mountains, fits in fairly well with these theories.

In allergic persons antisthine influences the capillary resistance, i.e. by increasing it.

In two cases of ulcerative colitis *Koller* and *Diem* observed a marked rise in capillary resistance immediately following intramuscular injection of torantil. The same was observed in an asthmatic patient following administration of

antergan, which was confirmed by *Wyss* and *Gianoli* (1946) in 2 cases of asthma.

A patient with a prolonged asthmatic attack was injected by us with 100 mg. antistine intravenously. First the subclavicular capillary resistance decreased from 250 to 125 m.M.Hg., but then it increased to 275 m.M.Hg. after 21 minutes. The antecubital capillary resistance rose in 23 minutes from 125 to 275 m.M.Hg.

That cortisone increases the capillary resistance, though with some delay, was observed by us in a 48-year-old woman, no longer menstruating, with a frequently recurring status asthmaticus which did not react to any other treatment. In this patient the capillary resistance rose considerably in the 14 days after the administration of cortisone, viz., in the forearm from 125 to 225 m.M.Hg. and infraclavicularly from 100 to 175 m.M.Hg. Simultaneously her general condition improved greatly: the quantity of sputum diminished and the eosinophil count decreased from 16 % to 1 %.

In relation to the increase of the capillary resistance by ACTH and cortisone, we should like to mention the observations of *Scarborough*, who 24 hours after major operations (without blood transfusion) found an increase in capillary resistance of 80-100 % in 23 cases.

The results in some personal cases reported below, also show an increase in capillary resistance after operations.

	Capillary resistance:	
	Ante- cubitaly	Infracla- vicularly
	m.M. Hg.	
<i>1st patient:</i>		
C.B.-S., 44 years old	200	100
19 hours after arthroplasty of the left hip (with blood transfusion,.....	250	150
<i>2nd patient:</i>		
A.P.L.-A., 51 years old	350	200
16 hours after hysterectomy	500	300
<i>3rd patient:</i>		
T.M.D.-B., 39 years old	150	150
24 hours after hysterectomy and appendectomy	200	225

This is reported here, though *Scarborough* does not give any explanation for it, but it must be considered possible that a major operation is a "stress" in Selye's sense and that the consequently increased circulation of ACTH is responsible for the increase in capillary resistance.

Of great importance for the study of the correlation between allergy and capillary resistance is the haemorrhagic diathesis.

The fragility of the capillaries in various forms of purpura has been closely studied by many investigators. Pathomonomically three classes of purpura can be distinguished:

1. Purpura with thrombocytopenia (thrombocytopenic forms).
2. Purpura with a normal number of thrombocytes, which are, however, inferior (thrombasthenic, thrombopathic and thrombolytic) forms.
3. Purpura without changes in the thrombocytes (athrombocytopenic forms).

The forms mentioned under item 2 seem only to occur hereditarily. The forms of purpura with thrombocytopenia and those with a normal amount of normal blood platelets are moreover mainly essential (due to unknown causes) or they are caused by drugs to which the patient is hypersensitive, or by (other) allergens.

All the chronic forms of purpura show exacerbations during which the haemorrhagic tendency becomes manifest or increases. In the athrombocytopenic forms this is actually the only symptom of the disease. In addition in thrombocytopenic purpura a lower platelet count, a delayed retraction of the blood clot and sometimes a prolonged bleeding time are found, quite apart from the exacerbations. In the forms of purpura with abnormal thrombocytes, experienced investigators have observed morphological changes in the blood platelets or in the megacaryocytes. In Glanzman's thrombasthenia the contraction of the blood clot is said to be delayed; in Willebrandt's thrombopathy this is described as normal, but

the elasticity of the coagulum is said to be diminished (*Juergens* and *Forsius*, 1951).

The exacerbations of the above-mentioned three groups of purpura have in common that an increased tendency to haemorrhage can occur together with a measurable decrease in capillary resistance.

We have compared for 1½ years the bleeding tendency, bleeding time, thrombocyte number and capillary resistance in a woman with chronic essential thrombocytopenia. The conclusion is that there is no clear correlation between bleeding tendency, bleeding time and thrombocytic count, nor between the blood platelets count and the capillary resistance. An increased bleeding tendency, however, is accompanied by a measurable decrease in capillary resistance. These findings are confirmed in the literature (*Hecht* 1907, *Roskam* 1929, *Bayer* 1930, *Bruehl* 1933, *Brock* and *Malcus* 1934, *Guggisberg* 1936, *Wright* and *Lilienfeld* 1936, *Peck*, *Rosenthal* and *Erf* 1937, *Sack* 1938, *Elliot* 1938).

On these grounds *de Vries*' remarks in his "Leerboek der Bloedziekten" should be emphasized: "it is not the thrombocyte count which is the essential factor, but the changes in the vessels".

It is therefore not quite correct to refer to vascular purpura as only that form without changes in the number or quality of the blood platelets. Any form of purpura is vascular.

Drug purpura is an affection well known to every physician, which may be caused by simple, frequently used drugs.

Schrader (1922) described the case of a woman who developed an extensive haemorrhagic exanthem with a positive "endothelium symptom" following 10 drops of tinctura of opium. *Deggeler* (1936) observed a patient with innumerable petechiae and a strongly positive Rumpel-Leede symptom following adaline.

In 1946 in Surabaya we had a patient who showed the same condition after a small dose of extract of opium, in

whom the Rumpel-Leede phenomenon became positive and transient thrombocytopenia occurred.

Last year we saw a woman with very peculiar eruptions. Following the use of powders containing caffeine, asperin and phenacetin a rash resembling erysipelas, with a dark-red sharply defined border arose on the right lower leg. Percocid was administered, but the eruption extended and the left leg also became affected. At other sites of the body large, itching, red, urticarial spots developed. When we examined the patient the rash on the lower legs appeared to contain numerous petechiae, so that the capillary resistance could not be determined there. The capillary resistance on the unaffected skin of the left forearm amounted to 100 m.M.Hg., that on the urticarial spots without spontaneous petechiae 25 m.M.Hg., while the Rumpel-Leede phenomenon was positive on these spots but not on the unaffected skin around them. The leucocyte and thrombocyte counts were normal when the eruption was maximal, but an eosinophilia of 9 % was found. The symptoms disappeared after withdrawal of all the drugs.

Erythema and purpura with or without thrombocytopenia and with lowered capillary resistance (or the latter alone) were also described following administration of salvarsan, neo-salvarsan and neo-arsphenamine, either after the first injection, or after sensitization (*Stephan* 1921, *Schrader* 1922, *von Borbély* 1930, *Falconer*, *Epstein* and *Weaver* 1936, *Horne* and *Scarborough* 1940).

On personal observation of patients who were not hypersensitive, no influence of neo-salvarsan on the capillary resistance was found. *Stephan* and *von Borbély* advise continual checking of the capillary resistance during a course of arsenic in order to discover hypersensitivity early. *Falconer et al.* found that patients hypersensitive to neo-arsphenamine tolerated mapharsen, so that in such cases the preparation can be changed.

Athrombocytopenic purpura in active periods also shows a greatly increased permeability of the capillaries. The best

known forms of this affection are Schönlein's rheumatic purpura and Henoch's abdominal purpura, also mentioned in combination, and both occurring mainly in children. Two personal cases with athrombocytopenic purpura were followed up for some years.

The first was a woman of about 48, who had complained for years of frequently recurring petechial skin haemorrhages on the whole body with abdominal pains and sometimes joint pains. She underwent a thorough examination. This revealed a strongly positive Rumpel-Leede phenomenon and a very low capillary resistance. When spontaneous petechiae occurred the resistance was 20–30 m.M.Hg. (infraclavicularly and antecubitally), in the intervals it was 40–60 m.M.Hg. She was therefore just under or just above the limit of spontaneous haemorrhages. She never had accchymoses or perceptible internal haemorrhages. When the petechiae were disappearing they often itched severely. Exhaustive and repeated internal and dermatological examination (Dr. Laurie C. Bolle) yielded no other abnormalities than some calcified glands situated anterior to the duodenum (Prof. Dr. R. H. de Waard). The blood sedimentation rate and the morphological blood picture, number and quality of thrombocytes, bleeding time and coagulation time, bone marrow picture and vitamin C content of the blood were all normal. Infectious foci and familial disposition were ruled out. The patient herself has composed a menu that she tolerates best, consisting mainly of bread, raw vegetables and some potatoes; yet this does not prevent the periods with spontaneous petechiae nor can it be clearly determined whether there are certain foods that provoke these attacks. The diagnosis of purpura abdominalis was made.

Administration of large doses of calcium, vitamin C and P, ACTH and adrenoxyl per os and by injection gave no distinct improvement.

The second patient with athrombocytopenic purpura is an otherwise healthy young man of 20, who in the spring of the last three years has developed spontaneously a large number

of petechiae on arms, upper legs and ankles. The Rumpel-Leede phenomenon is at that time strongly positive and the capillary resistance extremely low: 10 m.M.Hg. infraclavicularly, 20 m.M.Hg. antecubitally. There are no other symptoms, he feels well, and has a normal temperature. Full examination, as in the first case, yielded normal results. Infectious foci, hypersensitivity to food or a familial disposition could not be determined in this case either.

Calcium and vitamins C and P had no results. In October of the first year of this treatment the capillary resistance was only 40 m.M.Hg. (antecubitally and infraclavicularly). From the beginning of November, with a capillary resistance of 65 m.M.Hg. (at the same sites) no new spontaneous petechiae occurred. It was not before February that the values were 150 m.M.Hg. antecubitally and 100 m.M.Hg. infraclavicularly.

These vascular lesions are usually ascribed to the action of a "toxin" of histamine-like composition (*de Vries*) which would damage the semi-liquid colloid substance between the endothelial cells in particular (*Hijmans van den Bergh* and *de Langen*). These considerations led to investigation of the influence of antihistaminics in these cases. As mentioned before, antistine has no influence on the capillary resistance of normal persons. The two patients mentioned above were given 100 mg. antistine intravenously at about 9 a.m. The female patient in two hours reacted with an increase of the capillary resistance from 30 to 70 m.M.Hg. antecubitally and from 30 to 60 m.M.Hg. infraclavicularly. With daily oral administration of 320 mg. antallergan for a week these values were maintained.

The result in the young man was most surprising and definite. After injection of antistine his capillary resistance rose within 3 hours from 15 to 90 m.M.Hg. antecubitally and from 20 to 60 m.M.Hg. infraclavicularly. After seven daily oral doses of 320 mg. antallergan the values were 125 and 70 m.M.Hg. respectively, no spontaneous petechiae appearing from then on (Fig. 8).

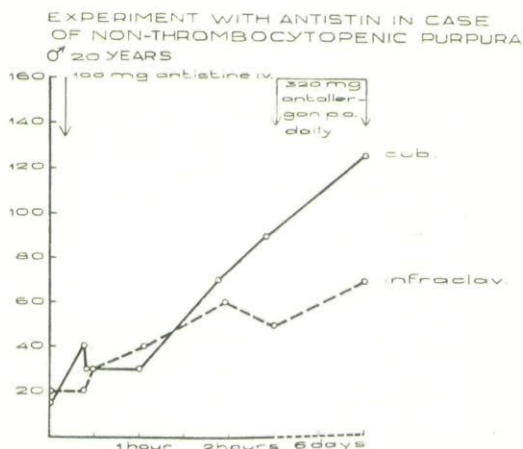


Fig. 8.

This year he visited us very early with the first petechiae. From the experience of the previous year we succeeded in preventing further development of the purpura by means of one injection of antistine.

It might appear from further experiments in other patients with athrombocytopenic purpura that determination of the capillary resistance after intravenous injection of an antihistaminic constitutes a new method for the differential diagnosis between allergic and other forms of purpura.

We hope that these examples make clear the importance of determining the capillary resistance in cases of allergy.

SUMMARY

After a description of the method of estimating the capillary resistance by means of negative pressure, the physiological differences in the permeability of the skin capillaries are discussed. The changes in capillary resistance following administration of histamine, antihistaminics, calcium, adrenalin, ACTH and cortisone in normal and allergic persons are mentioned.

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