Treatment of Shock with Vasodilators Measuring Skin Temperature on the Big Toe
Ten Years' Experience in 150 Cases

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Since August, 1953, an intensive care unit run by the Department of Anesthesiology has been in operation at Kommunehospitallet in Copenhagen. After a few years, the unit had to be extended, and it was found advisable, both for therapeutic and scientific reasons, to install air conditioning equipment in two rooms. They were built according to a new design evolved by Svend Clemesen, in cooperation with Mr. Georg Werner, an engineer, and myself.

The rooms were provided with "cold ceilings." The heat loss by radiation can then be varied and individually adjusted in each separate case by changing the temperature of the ceiling. These rooms have been of great importance in the development of the therapeutic procedure, the description of which is the main purpose of this paper. It is now possible to maintain constant atmospheric environment. A change in the skin temperature on the feet or the thumb can then only be the result of a change in the patient's condition caused by the disease or the treatment. Correlation of the skin temperature on the feet with the rectal temperature can then serve as a guide in the proper fluid and blood volume replacement.

The following account is based on experience gained the last ten years on more than 150 patients in all forms of shock, except cardiogenic, treated with a vasodilator agent.

The treatment is based on the influence of chlorpromazine on the peripheral blood flow and on heat regulation as well. This effect can be demonstrated by the following two simple experiments.

Experiment 1 (Fig. 1)

This figure shows a donor in an air-conditioned room. The donor has an open peripheral circulation in order to get rid of his body heat. He is comfortable, not sweating, and in heat balance with the room.

When the donor is bled 500 ml of blood, the blood pressure remains unchanged, but a drop in skin temperature of 8° C on the thumb shows that this blood pressure is now maintained by means of peripheral vasodilation. If an intravenous injection of

![Figure 1: A donor in heat equilibrium with the room. After removal of 500 ml of blood, the skin temperature on the thumb drops 8° C. When 25 mg of chlorpromazine is given intravenously, skin temperature rise is followed by a drop in blood pressure.](attachment://image-url)

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15 mg chlorpromazine is given, this vasoconstriction is abolished. The blood pressure therefore drops, and peripheral vasodilatation is accomplished, reflected by a rise in skin temperature. This is comparable to the effect of spinal analgesia in which a drop in blood pressure is due to too low volume in relation to the size of the vascular bed.

This observation shows that when a person can accomplish vasoconstriction, low blood pressure is a late sign of reduction in blood volume, but when vasoconstriction is abolished, blood pressure fall is an early sign of discrepancy between the blood volume and the size of the vascular bed.

If, after the administration of chlorpromazine, the donor receives 500 ml of blood, his blood pressure will rise.

A patient's thumb becomes warm before the feet. That is why the skin temperature on the big toe is used to show that general vasodilatation has occurred. When his feet become warm, he is out of shock.

**Experiment 2 (Fig. 2)**

If a person is placed on a cool room, he will protect himself against heat loss by vasoconstriction. When vasoconstriction is abolished, the body temperature will depend on the ambient temperature.

What are the possibilities for a patient in shock to regulate his body temperature?

The patient in shock who has cold feet and a subnormal rectal temperature has such bad circulation that he cannot produce heat enough to maintain the body temperature. This is what is seen in some cases of coronary heart failure and hemorrhagic shock. The patient in shock with cold feet and a high rectal temperature has such bad circulation that it cannot carry his heat to the body surface. There is sometimes a temperature difference of 20° C within the same body. Such a patient is

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**Figure 2**: A normal person in a cool room. When the skin temperature on the thumb has fallen, the patient complains about freezing. The rectal temperature has been maintained during this. After the administration of 25 mg of chlorpromazine intravenously, the skin temperature rises and the rectal temperature starts to fall. When vasoconstriction is abolished, the body cannot protect itself against the heat loss.
like one treated with a noradrenaline drip—the uneven distribution of heat is related to an uneven blood flow as a result of a poor peripheral circulation.

Elevated body temperature in seriously ill patients is usually considered to be the result of increased heat production—fever from infection, etc.—and not as a result of vasoconstriction and poor peripheral blood flow, making it impossible for the patient to impart the heat to the body surface.

If such a patient is treated with chlorpromazine to open up the peripheral circulation, followed by further infusions to improve blood volume necessary for the larger vascular bed, he is, as far as the heat regulation is concerned, put in just the opposite situation. Now he cannot protect himself against heat loss and his rectal temperature depends on ambient temperature, and can be set at will, which we have seen again and again in a larger number of cases.

The cold ceiling here comes in very useful, since the rectal temperature of the patient can be maintained at a normal or subnormal level in a very convenient way which does not interfere with the nursing care. By varying the temperature of the ceiling, the heat loss by radiation can be adjusted to that rectal temperature which is considered beneficial to the patient. We usually maintain 36.5-37° C.

Once these principles have been understood, they can be applied in the treatment of seriously ill patients, even without an air-conditioned room,—but the air-conditioned room has been a necessity for the evaluation of the treatment.

By diminishing peripheral resistance with a vasodilator agent, blood flow has been improved. After the Swedish investigators (Gelin et al) have demonstrated the improvement of blood flow by decreasing the viscosity of blood with low molecular dextran, the latter agent has been used to combine the two principles. It must be emphasized that low molecular dextran shall not be used as a substitute for fluids, but only when fluids have been replaced without re-establishment of good peripheral circulation. This precaution is necessary if production of thick, sticky urine is to be avoided. This complication has never been seen by us, but has been observed by others when the above mentioned precaution has not been respected.

This regimen has been used even as the guide in infusions and transfusions to patients with bleeding peptic ulcer. The greatest danger involved is that somebody starts the injection of chlorpromazine before filling of the jugular vein has occurred as the result of the previous treatment.

The main idea in the treatment is illustrated by Fig. 3.

A 55-year-old man was admitted to the hospital with perforated peptic ulcer of five days duration. Preoperatively he received 5500 ml of different fluids intravenously.

Postoperatively, chlorpromazine was given since he had nearly 20° C of temperature difference between the rectum and
the big toe in spite of attempts of treatment before and during the operation.

After eight hours, he was in the hemodynamic condition which is considered most beneficial. Further intravenously given regimen shall be restricted to the maintenance of this state. When the feet get warm, the patient is out of shock.

I want to emphasize that the above-mentioned relationship between heat regulation and peripheral blood flow, and the re-establishment of the latter as the principal guide in the treatment when vasodistraction is abolished, are such an essential key to the understanding of the treatment of shock that it can be applied in any type of shock when the heart still has a functional reserve capacity — in the same way as underventilation of lungs with high oxygen gives CO₂ accumulation, irrespective of the reason for the respiratory insufficiency. But I want to stress that this method is not a substitute for other necessary procedures, such as electrolyte balance, fluid balance, antibiotics, etc. Its main purpose is to ensure that the patient is brought into a hemodynamic condition in which he has the blood volume most beneficial in his condition.

The motto could be: Open up and fill up — and stop when the feet get warm.

SUMMARY

During the last ten years, more than 150 patients in shock who did not respond to conventional treatment have been treated with small doses of chlorpromazine and pethidine to create universal vasodilation. Temperature measurement in the rectum and the skin temperature on the big toe was then a guide to the proper intravenous administration of fluids.

The regimen has been conducted in air-conditioned rooms where changes in temperature of the patient can only be related to changes in his condition due to disease or treatment.

During vasodistraction due to shock or administration of vasoconstrictors, one can see 20° C difference between rectal temperature and skin temperature of the big toe.

The patient cannot get rid of his heat because blood flow in the skin and subcutaneous tissue is so decreased that he cannot carry his heat to the body surface. When vasodistraction is achieved after the administration of chlorpromazine, the hemodynamic condition is characterized by a fall in rectal temperature and a difference between this and the skin temperature of only 3-4° C. In this condition, the patient cannot protect himself against heat loss and the rectal temperature will be dependent upon the temperature of the surroundings. The value of the regimen is its simplicity since only the following measurements are necessary: blood pressure, pulse, respiration, hourly urinary output, skin temperature of the big toe and rectal temperature. The filling of the external jugular veins can be observed and does not have to be measured.

Resumen

Durante los últimos diez años más de 150 pacientes en shock, que no respondieron al tratamiento usual, han sido tratados con pequeñas dosis de clorpromazina y petidina con el fin de inducir una vasodilatación general. La temperatura rectal y la cutánea en el dedo grande del pie fueron tomadas como norma para la administración intravenosa de fluidos.

El tratamiento ha sido llevado a cabo en un local termicamente estabilizado, en el que los cambios en la temperatura del paciente tienen que estar en relación con cambios en su estado debidos al proceso patológico o al tratamiento. Durante la vasodistención debida al shock o a la administración de drogas vasocostructoras pueden observarse diferencias de 20° C entre la temperatura rectal y la cutánea al nivel del dedo grueso del pie, el paciente no puede irradiar el calor corporal porque el aflujo de sangre a la piel y tejido subcutáneo es tan limitado que el calor no es conducido a la superficie. Cuando se produce la vasodilatación después de la administración de clorpromazina el estado hemodinámico se caracteriza por una caída en la temperatura rectal y una diferencia entre esta y la cutánea de solo 3-4° C. En este estado el sujeto no puede protegerse contra la pérdida de calor y la temperatura rectal dependerá de la temperatura ambiente. El valor de este método se sitúa en su sencillez, ya que solo son necesarias las siguientes medidas: presión sanguínea, pulso, respiración, cantidad de orina por hora, temperatura cutánea en el dedo grueso del pie y tem-
TREATMENT OF SHOCK WITH VASODILATORS


A-V BLOCK AND ATRIAL SEPTAL DEFECT

Two cases of atrioventricular block associated with atrial septal defect of the fossa ovalis (secundum) type are presented. In the first case, the block was truly congenital, while in the second, it was acquired after birth. In the first case there was no connection between the defective musculature of the atrial septum and a remnant of the A-V node. In the second case, there was nitis of the A-V bundle and bifurcation. It is hypothesized that injury of the musculature of the approaches to the A-V node and of the bundle are related to the malformation of the atrial septum and consequent hemodynamic weakening of the septum and central fibrous body.