profound anaphylactic symptoms. In 6 of the families, we were unable to produce respiratory anaphylaxis in the mother and in 2 of the families, respiratory anaphylaxis was induced in the mother, but was not transmitted to the offspring.

The establishment of respiratory anaphylaxis has been adequately controlled by innumerable experiments, for in no instance has an animal been utilized who showed the slightest evidence of dyspnea during its sensitization period, and it is only after the incubation period has elapsed that respiratory anaphylaxis is manifested. The new-born animals were adequately controlled by the six negative groups, indicating that even animals as young as one day old show no dyspnea when placed in a cage unless they are sensitive.

That a dry substance entering the respiratory tract may go into solution in the maternal circulation, traverse the placental barrier, and enter the fetal circulation is demonstrated by the above experiments, and may offer another explanation[3] for the transmission of allergy or anaphylaxis from a mother to her baby. This transmission of hypersensitiveness from a mother to her offspring we must regard as strictly congenital.

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Changes in the Peripheral Circulation Accompanying "Tobacco Angina."

ELAINE P. RALLI AND B. S. OPPENHEIMER.

From the Cardiographic Laboratory of Mount Sinai Hospital, New York.

It is now fairly well established that coronary artery disease, occlusion, and consequent myocardial infarction constitute one of the pathological conditions associated with the syndrome, *angina pectoris*. There is, however, great diversity of opinion as to the mechanism of this symptom-complex; the probability is that typical *angina pectoris* is due to a single mechanism. There are several well recognized exciting causes of angina; such as physical effort, emotional excitement, exposure to cold or to fresh air, overeating, and in some patients tobacco smoking. We had observed a very few patients, beyond middle age, predisposed to *angina pectoris,* in whom the attacks could be provoked very promptly by smoking a cigarette. The attacks of pain induced by smoking were relieved by

[3] Ratner, B., *Am. J. Dis. Child.*, 1928, xxxvi, 277.

administering a nitroglycerin tablet dry under the tongue, but were not relieved by a tablet similar in appearance but not containing any nitroglycerin. The relief of pain was, therefore, not due to a psychic factor; no attempt was made to substitute any form of smoke other than that of tobacco. These few patients, therefore, offered an opportunity to study the peripheral circulation before, during, and after an attack of *angina pectoris*. This was done by making observations of any change in limb volume by means of a plethysmograph on the left arm, and simultaneously of the systolic and diastolic blood pressures in the other arm by the ausculatory method.

A simple air plethysmograph was used. This was made of glass and was approximately 13 inches long; the diameter at the open end was 5¼ inches, and the jar tapered down so that the diameter at the closed end was 1½ inches. The open end was covered with thick rubber membrane into which a small opening was cut, so that the patient could insert his hand and forearm without any undue pressure. A glass tube led from a rubber stopper at the top of the plethysmograph, and this was connected by rubber tubing to a glass tube containing water. In this way after an even baseline was established, changes in volume in the plethysmograph could be readily observed, but for the sake of simplicity no attempt was made to record these volumetric changes by means of a bellows recorder and smoked drum, or other apparatus.

Observations were made on 6 patients, all giving a history of precordial pain; also on 2 normal individuals, one male and one female, as controls. One typical example will be given here.

Male patient, baker, 45 years of age, formerly a heavy smoker, whose resting blood pressure was elevated, averaging 165/100-110 mm., would experience a typical attack of *angina pectoris* following 4 or 5 deep inhalations of a cigarette. (Deep breathing alone did not excite an attack.) Each attack consisted of precordial pain, radiating into the left arm, with *angor animi*, ashy color, cold perspiration. This pain was associated with a marked rise in both the diastolic and the systolic blood pressure, and by evidences of peripheral *vasoconstriction* as registered by the plethysmograph. The peripheral constriction *preceded* the onset of pain by 15 to 45 seconds. There was a marked rise of systolic and diastolic blood pressures (of about 40 mm. of mercury) which followed the peripheral vaso-constriction but just preceded the onset of pain. Following the administration of 1/100—1/50 grain of nitroglycerin tablet dry under the tongue, the plethysmograph would give evidence of peripheral dilatation which preceded the cessation of pain.

The interval of time elapsing between the peripheral dilatation and the cessation of pain depended somewhat on the severity of the pain. Three separate sets of observations were made on this patient, and in each case the results were the same.

In three of the 6 patients there was definite evidence that first peripheral vaso-constriction, and later a rise in arterial tension preceded the onset of pain. In 2 other cases, there was evidence only of an elevation of systemic blood pressure, but not definitely of peripheral vaso-constriction. The sixth patient refused to smoke, but exercise with a foot ergograph resulted in a rise in arterial tension with each attack of pain, but there was only slight evidence of peripheral vaso-constriction.

There are, therefore, instances of patients predisposed to *angina pectoris,* in whom cigarette smoking induces a peripheral vaso-constriction, which precedes the onset of precordial pain. At the same time an elevation of blood pressure was observed, so that it is reasonable to suppose that there was a general vaso-constriction, in which the coronary arteries may have taken part.

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Studies on Inhibition of Insulin Activity.

SAMUEL KARELITZ, PHILIP COHEN AND SIDNEY D. LEADER.
(Introduced by Bela Schick.)

From the Pediatric Department and Laboratories, Mount Sinai Hospital.

In the fall of 1925 at the suggestion of Dr. Bela Schick we investigated a diabetic child with refractory periods to insulin. The study of this case led us to investigate the inhibitory effect of blood on insulin activity.

Our experiments have all been done with rabbits. Unless otherwise stated, all work was done as follows. One physiological or 3 clinical units of insulin were mixed with the substance investigated and incubated at 37° C. from 1 to 2 hours, then injected subcutaneously into rabbits, weighing 2 kilos, starved during the preceding 24 hours. Hourly blood sugar determination and clinical observations were made.

The results summarized were as follows:

2-4 cc. of human plasma caused hardly any inhibition of insulin action.

5 cc. of human plasma caused moderate inhibition of insulin action.