

tracings. Further observations indicate that this type of pulse is common in fever patients and that it is rarely marked in normal individuals. It is the type of pulse that has frequently been described as bounding, poorly sustained, pointed, etc.—terms which refer to the sudden fall of pressure immediately after the primary pulse wave.

This type of pulse as it occurs in febrile patients may be converted into a normal form by therapeutic doses of a pituitary preparation.¹ Following such an injection the pulse form usually showed a definite change in from ten to fifteen minutes, the maximum effect was reached in about an hour, and the effect did not pass off for two or three hours. The degree of change varied in different patients. Frequently it was so marked that not a trace of the original backflow remained and the pointed character of the volume pulse from the arm was entirely lost. Thus far we have not been able to determine any fixed relation between the change in pulse form and changes in the systolic blood pressure or changes in the rate of blood flow throughout the arm. The change in form however was regularly accompanied by a diminution in the size of the volume pulse in the arm. These changes may be explained by assuming a constriction of the larger arteries in the arm or a constriction of vascular areas elsewhere in the body, particularly in the head and splanchnic region. The pulse changes produced by therapeutic doses of pituitary substance are precisely opposite to those which usually follow a therapeutic dose of nitroglycerin.

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The effect of carbon dioxide on the eggs of *Ascaris*.

By THEOPHILUS S. PAINTER (by invitation).

[From the Osborn Zoological Laboratory, Yale University.]

The undivided eggs of *Ascaris megalcephala* (var. *bivalens*) were kept in an atmosphere of carbon dioxide for three months. On the removal of the eggs from the gas, a few smears were allowed to undergo full development. Only about one third of the embryo,

¹ 1½ c.c. of Parke, Davis, and Co.'s pituitrin were injected intramuscularly.

were normal, the remainder being either masses of disorganized cells, or embryos in which the posterior end was differentiated, together with the primordial germ cells. The problem was to determine the causes of the abnormalities. Eggs were preserved in all stages, stained and mounted *in toto*.

Two distinct and independent causes were found for the abnormal development.

The first of these was the fusion of the chromosomes in the equatorial plate phase of the dividing S_1 -blastomere. (This is the "Ur-somatic" cell in which the diminution process takes place.) The fusion resulted in one of two things: (a) When the fusion involved the greater part of all the chromosomes, the blastomere did not divide. At the next division cycle, a tetraster appeared with eight chromosomes (or their equivalents in small "diminished" chromosomes) lying in the spindles. The tetraster divided very irregularly and the result was the total disorganization of the cells of the ectoderm. Such eggs gave rise to embryos which failed to invaginate. (b) When the fusion involved the ends of the chromosomes only, then division took place, but the chromatin was unequally distributed to the two daughter blastomeres, *A* and *B*. This led to an upsetting of the cleavage rhythm of these two cells, the blastomere with less chromatin dividing more rapidly than its mate. The P_1 blastomere (the cell which gives rise to the cells of the entoderm, mesoderm, the stomadeum cells, and the primordial germ cells) and its derivatives divide normally throughout development. It should be added that the P_1 cell plays a dominant rôle in the formation of the posterior end of the embryo. When the division of the chromatin was very unequal, the ectoderm cells became so scattered that they did not take up their proper places and a mass of disorganized cells resulted. When the division was more nearly equal, it seems probable that partially normal embryos resulted.

The second type of abnormality consisted in a shifting of the positions of the *A* and *B* blastomeres and their derivatives. Due to this, the cells from the P_1 blastomere which come into relation to certain of the ectoderm cells to form the stomadeum do not find their proper places. The posterior end of the embryo, coming, for the most part, from the derivatives of the P_1 cell was normally differentiated.

It is interesting to note that while the chromatin of the somatic cell (the one in which the diminution process occurs) is affected by the treatment with the carbon dioxide, the chromosomes of the "Urgeschlechtszelle" are perfectly normal.

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Apnea as an after-effect of pulmonary distension and its dependence upon the vagus nerves. Demonstration.

By **T. S. GITHENS** and **S. J. MELTZER.**

[From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.]

In recent years the conception became dominant, due especially to the investigations and teachings of Haldane and his pupils, that apnea as an after-effect of distension of the lung is essentially of chemical origin, due to a reduction of CO_2 in the blood circulating through the respiratory center; this has been designated as apnea vera. It was recently stated that there is no experimental evidence for a possible claim that "true apnea" could depend exclusively upon the intactness of the vagus nerves. At this meeting G. and M. demonstrated the following three facts. (1) A fairly prolonged and complete apnea followed a short distention of the lungs in dogs without any previous artificial respiration; the duration of the apnea depended upon the degree of pressure used for the distension (Meltzer's pleural canula was used for the graphic presentation of respiration). (2) The same apneic after-effect was obtained when the air used for the distension of the lungs contained 5 per cent. CO_2 . (3) No such apneic after-effect could be obtained after both vagus nerves were cut.

These experiments demonstrate that the mere distension of the nerve endings of the pulmonary vagus without the aid of a chemical factor (acapnia) is capable of producing a prolonged apnea as an after-effect of the mechanical stimulus. The restriction of the use of the term "true apnea" for a condition produced exclusively by chemical changes does not seem to be well founded.