probably common to the filtrable agents of tumors and of leucosis although they have not been satisfactorily investigated in this connection. These are as follows:

Resistance of certain individual animals to these transmissible agents is not based on immunological principles but appears to be governed chiefly by hereditary factors. Ellermann observed that fowls that resisted one inoculation might occasionally succumb to reinoculation. We have found that the blood of a fowl that had recovered from leucosis did not exert any protective action when inoculated simultaneously with leucemic blood into 5 fowls, for all of them developed leucosis about the same time as the corresponding controls.

Some fowls resist inoculation although they are given several hundred times the amount necessary to cause leucosis in susceptible individuals. In one passage, for example, 4 fowls were inoculated with 0.2 cc., 4 with 0.001 cc. and 4 with 0.00005 cc. of plasma (not cell-free). One of the fowls inoculated with 0.2 cc. resisted the disease although 2 of those receiving 0.001 cc. and one of those receiving 0.00005 cc. developed leucosis.

Filtrable tumors of the fowl unlike other filtrable virus diseases of the fowl could not be transmitted to other species of birds. The transmissible agent of filtrable tumors appears to be in large part attached to cells or to cell fragments.

These properties as well as the character of the disease produced (neoplastic growth) seem to justify a separation of the filtrable agents of tumors within the apparently heterogenous group of filtrable viruses and suggest the possibility that the agent of filtrable tumors is not a virus in the ordinary sense.

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Blood Lactic Acid and the Coronary Circulation.*

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Increasing information on the distribution of lactic acid in the body tissues has suggested that it plays a more complex rôle than has generally been suspected. Evidence was produced[1] to show

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[1] McGinty, D. A., *Am. J. Physiol.*, 1929, **88**, 312.

that brain tissue removes lactic acid from the blood under normal conditions and that lactic acid is added to the blood circulating through the brain during impaired oxidations induced experimentally. It is evident that this equilibrium between lactic acid absorption and outward diffusion must play a part in the acid-base balance of the blood as well as in the brain. Under conditions in which lactic acid is produced in excessive amounts, its removal from the blood is a matter of importance. Himwich and his collaborators[2] showed that excess blood lactic acid produced chiefly in muscles is carried to the liver and stored as glycogen to be converted subsequently into glucose. These findings have been confirmed and extended by Cori and Cori.[3] In an effort to find further sources of blood lactic acid or further means of its disposal, a study was made of the lactic acid content of arterial and coronary venous blood of the heart *in situ*.

Dogs were anesthetized either with morphine and urethane or with Gréhant's mixture of chloroform and alcohol. The chest was opened during constant artificial respiration and a cannula placed in the coronary sinus. Volume flow of coronary blood was recorded in most of the experiments. Simultaneous samples of arterial and coronary venous blood were analyzed for lactic acid, a difference of 3 mg. % or more being considered as of definite physiological significance.

In 118 out of 120 pairs of samples in 11 dogs under a variety of experimental conditions, coronary venous blood contained less lactic acid than arterial blood by amounts ranging from within the analytical error to 23 mg. %. No parallelism could be observed between the extent of absorption as shown by the arterial-venous differences in the coronary system and the general arterial lactic acid levels which ranged from well within normal limits to values exceeding 100 mg. %.

In 5 out of 7 observations with stimulation of the stellate ganglion followed by increased heart rate, blood pressure and coronary volume flow, arterial-venous differences fell from an average of 8.0 mg. % to 3.8 mg. %. Usually this was followed by recovery. In 2 experiments in which only a slight change in volume flow took place, no significant change in lactic acid absorption was observed.

Stimulation of the vagus, in 9 of 13 observations caused an increase in the arterial-venous difference from an average of 5.4 to

[2] Himwich, H. E., Koskoff, Y. D., and Nahum, L. H., PROC. SOC. EXP. BIOL. AND MED., 1928, **25**, 347.

[3] Cori, C. F., and Cori, G. T., The Harvey Lectures, 1927-28.

8.6 mg. % during the fall in volume flow of blood. In 2 experiments no change occurred. A decrease in arterial-venous difference appeared in 2 observations in which blood samples were taken at least 2 minutes after the end of stimulation and much later than ordinarily.

In 2 experiments with rapid intravenous injection of large doses of pitressin, following which a considerable reduction in coronary volume flow occurred, an initial pre-injection absorption of 7.5 and 10.1 mg. % lactic acid was replaced by an outward diffusion of 4.1 and 4.2 mg. % respectively. On recovery absorption again took place.

In 4 experiments a reduction of arterial-venous difference occurred during the height of the pitressin effect on the heart, and in 3 observations with considerably smaller amounts of pitressin an increased lactic acid absorption was observed during the moderate fall in volume flow of blood.

The gradient of inward or outward movement of lactic acid between blood and heart muscle is dependent on the relative concentrations in the blood and muscle. The concentration in muscle may be regarded as being dependent on the state of equilibrium between disposal and production of lactic acid. With moderate changes in coronary volume flow during vagal and stellate stimulation and with small doses of pitressin, the rate of absorption of lactic acid is believed to be primarily dependent on the velocity of the blood through the heart capillaries. With more pronounced diminution in flow of blood with a subsequent reduction in oxidations in heart muscle, increased lactic acid production may reduce the gradient between blood and muscle or with larger injections of pitressin establish a reverse gradient from muscle to blood.

These experiments demonstrate that the heart under normal conditions must be regarded as one of the organs concerned with the removal of lactic acid from the blood. The fate of the lactic acid thus absorbed and its relation to oxidations in heart muscle is under investigation.