support to these observations. It has been found that pullets fail to hydrolyse a considerable proportion of the phytic acid present in their rations. Calcium carbonate supplements appear to lower the proportion hydrolysed, just as in the case of the rat[2]. The experiments also suggest that when a ration is supplemented with calcium (*a*) in the form of carbonate and (*b*) in the form of tribasic calcium phosphate, a greater proportion of the phytic acid is hydrolysed in the case of the ration supplemented with calcium phosphate than in the case where the supplement is calcium carbonate.

It is also clear from the experimental results secured to date that the conclusions of Knowles, Watkin and Hendry[3] with regard to the nature of the calcium and phosphorus compounds present in the excreta of the domestic fowl require modification, in so far as a considerable proportion of the phosphorus excreted by pullets on rations similar to those used by these workers is in the form of phytic acid phosphorus. Furthermore, while the rule found by Knowles, Watkin and Hendry[3] with regard to the ratio of 'non-carbonate calcium' to total phosphorus in the excreta has been verified in the case of non-laying pullets, it does not appear to hold for the excreta of laying pullets, even when the excreta contain appreciable amounts of carbonate.

It is hoped to publish a fuller account of these experiments at a later date.

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A Metabolic Explanation for Irritation and Coma Produced by Rise in Intracranial Pressure

INCREASE in pressure inside the cranial cavity, whether local or general, first produces symptoms of cerebral irritation such as convulsions, contraction of the pupil, vomiting and rise of blood pressure, and is followed by coma, dilatation of the pupil, and other symptoms of paralysis of the nerve centres. The current explanation for this train of symptoms, which is seen in many pathological processes (for example, intracranial hæmorrhage, œdema, tumour, etc.), supposes that irritation is caused by congestion of the cerebral vessels and cyanosis, while further increase in pressure produces profound cerebral anæmia and coma.

The first result of a rise in intracranial pressure is to cause partial collapse of the venous sinuses of the dura mater and of the cerebral veins[1], accompanied by a diminution in the rate of venous return. Accordingly the rate of oxygen supply to the brain is much diminished and approximately anærobic conditions are realized in some of the cells. Now, it has been shown that under anærobic conditions the rate of katabolism of glucose by cerebral cortex is enormously increased[2]. Thus the rate of catabolism of glucose by rabbit's cortex has a low value in oxygen ($Q_{c6} = -3$), whereas in nitrogen this quotient is increased manyfold ($Q_{c6} = -8$). Recent observations which I have made on the metabolism of human cortex (kindly given to me by Mr. McConnell of the Richmond Hospital, Dublin) indicate that here anoxæmia has essentially the same effect in increasing metabolism. Thus the Q_{c6} of human cortex, calculated from measurements of respiration and glycolysis, is -3 in oxygen and -10 in nitrogen. This stimulation of metabolism may well be accompanied by stimulation of the nerve centres, and thus convulsions and other symptoms of irritation are really due to the intracerebral anoxæmia, which is itself the result of rise in intracranial pressure. Still further increase in intracranial pressure causes capillary collapse and so seriously impedes the cerebral circulation as to deprive the brain cells of glucose, their main metabolic substrate. The rate of cerebral metabolism is now substantially diminished even below its normal level, and irritation thus gives place to paralysis. In this manner, a progressive rise in pressure within the cranial cavity may first produce a stimulation of metabolism associated with irritation, and is later followed by diminution or even cessation of metabolism, accompanied by coma and other terminal symptoms of compression.

The convulsions produced by heart block in man and by ligation of the carotids and vertebrals[3] in animals have probably the same cause as those produced by rise in intracranial pressure. In these cases also, anoxæmia causes increase in katabolism of sugar by the brain cells with increase in nervous irritability. Further, Loewenhart, Lorenz and Waters[4] have found that intravenous injection of sodium cyanide causes stimulation of the respiratory centre and also a remarkable cortical stimulation in stuporous cases of dementia præcox. They suggest that the cyanide, by inhibiting oxygen absorption, stimulates anærobic metabolism. Stone[5] also has shown the lactic acid content of brain increased by cyanide convulsions and suggests that convulsions may be associated with increased tissue activity. I have found the Q_{c6} of rabbit's cortex, as above calculated, to be increased from -2 to -7 by the addition of 2×10^{-3} *M.* sodium cyanide. The respiration is simultaneously diminished by 50 per cent, but the ærobic lactic acid production is markedly stimulated. Here again increased nervous activity is connected with increase in katabolism of glucose.

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Proteins of Rattlesnake Venom

RECENTLY, Slotta and Fraenkel-Conrat [1,2] reported that they have succeeded in separating the whole of the neurotoxin and hæmolysin of *Crotalus-t-terrificus* venom in a crystalline form, and that repeated recrystallization of this substance did not change its physiological properties or its chemical composition. This crystalline substance containing both neurotoxic and hæmolytic activities was considered by them to be a pure substance. In the course of our investigation on the separation of the various active principles of *Crotalus-t-terrificus* venom we have found that the hæmolysin can be partially separated from the neurotoxin by adopting the following procedure :

100 mgm. of venom was dissolved in 10 c.c. wate