

9. The pentosans on the one hand and the albuminous compounds on the other are intimately intermixed or interwoven in the protoplast of the plant and in the nucleus of the animal cell. The conditions which accelerate the hydration of the first may not affect the volume of the other except to cause a slight shrinkage. The effect of the hydrogen ion is to increase the hydration capacity of albumin and its derivatives, while lessening the hydration of the carbohydrates.

10. In addition to these differential effects of the solutions upon the principal components of the plasmatic colloids, the changes in volume are not isotropic but may be determined by the manner in which the mesh, masses of colloidal material or organs of the protoplast are laid down or fall into place. These two classes of variables may well be considered as prolific sources of differentiation in the procedure of the cell.

20 (1480)

Profound effects of digitalis on the vagus producing severe detrimental subjective symptoms, as shown by simultaneous electro-cardiograms and pneumograms.

By **ROBERT H. HALSEY** (by invitation).

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In a case of syphilis, chronic interstitial nephritis, dilatation and hypertrophy of the heart, auricular fibrillation, arteriosclerosis, periodic breathing, Cheyne-Stokes type, in which because the auricle was fibrillating, digitalis had been given for the purpose of showing the ventricular rate. Counts of the pulse showed the ventricle to be contracting at a rate approximating 100 during apnea and slowing to about half this rate as soon as hyperpnea began. That is, the heart rate was highest while there was no lung ventilation (apnea) and the heart beat less frequently during the period the lung ventilation was greatest (hyperpnea).

As the digitalis was continued the man complained of increasing distress, most severe during his hyperpnea. The ventricle rate did not become slower and evidently the patient was much more uncomfortable. The thought occurred that releasing the heart

from vagus control might give relief, therefore, atropine gr. 1/30 was given by hypodermic injection. Record taken 27 minutes later shows the ventricle beating irregularly at the rate of 150 per minute but uninfluenced by breathing. The patient became comfortable and the cyanosis was less.

Discussion.—As the slowing of the ventricular rate followed the first inspiration after apnea and was maintained until the last part of the hyperpnea stage, it was considered to be due to the inhibition of the vagus which was stimulated by the reflexes from the lung generated by the respiratory efforts of hyperpnea. The average hyperpnea periods were of 37 sec. duration. The reflex from the lung became much less effective as a stimulant to the vagus during the descending phase of the hyperpnea; for, the ventricular rate became accelerated several inspirations before apnea began.

This loss of vagus effect upon the heart rate was due, probably, to the fact that reflex stimuli from the lung tissue when it is contracting are not so strong as when the pulmonary tissue is being distended, but not to true vagus escape of the heart. That the vagus controlled the ventricular rate was shown by absence of alterations of the ventricular rate while the atropine was effective. During atropine effect the hyperpnea persisted for 47 seconds, a ten second increase over the digitalis period.

The distress of the patient was due to the diminished interchange of O_2 and CO_2 between the lungs and blood. This interchange was least during apnea, when the pulmonary ventilation was lowest though the heart rate was highest, but during hyperpnea, when the ventilation of the lung was greatest, the heart rate was slowest. The sensitiveness of the vagus to the pulmonary reflex was increased by digitalis to such a degree that the number of ventricular contractions during hyperpnea were very much less than when digitalis was not given.

To state this in another way; it appears that without digitalis the amount of interchange of O_2 and CO_2 was sufficient, to keep the patient comfortable, notwithstanding the inverse periodic changes in rate of heart and breathing; but, when digitalis was given, the vagus became much more susceptible to the lung reflexes slowed the circulation so as to diminish the interchange of

O₂ and CO₂ between the alveolar air and the blood sufficiently to cause real distress. Relief followed the discontinuance of digitalis and the administration of belladonna.

Conclusion.—In this case of auricular fibrillation digitalis did not improve the circulation because it increased vagus affects which diminished the O₂ and CO₂ interchange between alveolar air and blood.

21 (1481)

The action of camphor on the central nervous system of the squid.

By A. R. MOORE.

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Newly hatched squid (*Loligo pealii*) if put into a solution of camphor gum in sea water, 1/10 saturation, show characteristic mantle spasms, involving play of the chromatophores, after a latent period which is about 40 seconds at 24° C. This effect, it has been shown, is due to the action of camphor on the stellar ganglia.¹ In appearance, camphor spasms are indistinguishable from those caused by nicotine.² The difference does not lie in the character of the response of the end organs, muscles and chromatophores, but in the locus and the nature of neuronc excitation.

The value of the temperature coefficient Q_{10} for the action of camphor, based on the lengths of the latent periods, is 2.4. The function connecting the velocity of the reaction with the concentration of the drug is, for camphor, expressed by $v = kC^{\frac{1}{2}}$ in

which $v = \frac{1}{\text{latent period}}$, C = concentration, and k is a constant whose value is approximately .75.

The camphor spasms soon pass off and the animals lie inert with chromatophores relaxed. This is not due to paralysis of the end organs for the reason that they may again be thrown into activity by treatment with strychnine or nicotine. The absence of any interference between the action of nicotine and that of camphor may be demonstrated by the following experiment. Let

¹ Moore, A. R., *Proc. Nat. Acad. Sc.*, 1917, III, 598.

² Moore, A. R., *J. Gen. Physiol.*, 1919, I, 505.