and in others more serious, of presenting a mirror reversal of the views. The views likewise appear somewhat small, though it would be easy to introduce lenses to magnify them. But the interest in the device is merely in its simplicity and in the fact that so direct an application of the Wheatstone plan should have lain so near at hand for so long a time and, to my knowledge, not have been intentionally sought for or accidentally hit upon by the many experimenters who have contributed to the literature of the stereoscope. And it is also fair to add that the practical preparation of a pair of mirrors at this angle is not an easy matter, if one is desirous of eliminating the seam or line at their point of junction the presence of which to some extent mars the perfection of the stereoscopic effect. It seems, however, worth while thus briefly to record the possibility of a reflecting stereoscope which is adaptable to the ordinary stereoscopic card. As a laboratory device for illustrating the variety of applications of the stereoscopic principle, it may possess interest if not value.

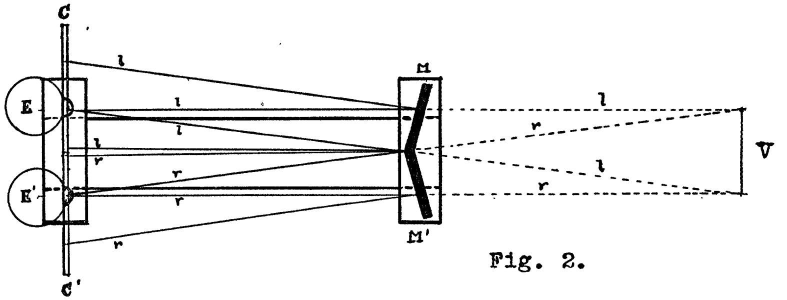

FIG. 2. The apparatus as seen from above. The letters as above: *V*, the combined view as projected by the eyes in stereoscopic vision; *r*, *r* and *l*, *l*, the paths of the rays in the right and left eyes from cards to mirrors and back.*

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* In this diagram no account is taken of a minor discrepancy due to the fact that stereoscopic photographers have agreed upon a separation (and size) of the stereoscopic views (3–3¼″) greater than the interocular distance (2¼–2½″). As a result practically so much of the views as corresponds to the interocular distance becomes completely stereoscopic, the marginal portions not participating in the stereoscopic effect. Yet for ordinary views this discrepancy is not serious; none the less the effect in views mounted within the limits of the interocular distance is distinctly more perfect.

THE NATURE OF THE ACTION OF DRUGS ON THE HEART (PRELIMINARY NOTE).*

THE analysis of the nature of the action of drugs and certain inorganic substances in solution on the isolated heart of vertebrates is rendered difficult by the intimate connection of the nervous with the muscular elements in the heart, making it practically impossible to study the effects of a solution on the nervous elements apart from that on the muscle, and *vice versa*. In the heart of *Limulus* the relation of the nervous to the muscular elements is such that this analysis can be made. The heart of *Limulus* can be prepared in a manner allowing the determination of the nature of the action of a solution: (1) On the ganglion cells, (2) on the motor nerves, (3) on the motor nerve endings and the muscle.

I have shown elsewhere that in *Limulus* the origin of the heart-beat is nervous, not muscular, and that the coordination or conduction in the heart is effected through the nervous, not through the muscular tissue. I have some evidence that a similar mechanism of the heart-beat obtains also in the molluscs and the crustaceans, and I have little doubt that we shall have to revise the generally accepted theory of the function of the ganglion

* The experiments were performed at the Marine Biological Laboratory, Woods Hole, Mass.

cells even in the vertebrate heart. This must necessarily lead to a revision of some of our views of the nature of the action of drugs and certain solutions on the heart, as these are based on the myogen theory of the heart-beat.

The preparation of the *Limulus* heart for the study of the action of solutions on the ganglion cells apart from their effects on the muscle is represented in Fig. 1. The ganglion or median nerve-cord is extirpated from the first and second segments, the lateral nerves being left intact. The removal of the nerve-cord removes the ganglion cells from that part of the heart. The extirpation of the nerve-cord in the first two segments diminishes the strength of the contractions in these segments, but it does not affect the rate of the beats or the strength of the contractions in the other parts of the heart. After removal of the ganglion from the anterior end of the heart the contraction of the muscle in these segments is caused by impulses from the ganglion cells situated in the nerve-cord of the fourth, fifth and sixth segments, these impulses passing forward to the muscle in the lateral nerves. The heart-muscle may now be removed for a distance of one centimeter or more in the region of the second pair of lateral arteries, leaving the two ends of the heart connected by the lateral nerves only. The anterior end of the heart is supported and connected with a recording-lever for graphic registration in the manner indicated in Fig. 1.

By the removal of the nerve-cord and the separation of the heart-muscle in the second segment the action of a solution applied to the anterior end of the heart can not be due to or complicated by the effects of this solution on the ganglion cells, care being taken, of course, that the solution does not touch the posterior end of the heart. But although this possibility is excluded, the solution applied to the anterior end of the heart may affect the rhythm of this part of the heart in either or all of these three ways: (1) It may act directly on the muscle, (2) it may act on the motor nerves and nerve-endings, or (3) it may act on sensory nerves and nerve-endings, the impulses being carried to the ganglion cells in the posterior part of the heart by means of afferent fibers in the lateral nerves. This would be a true reflex action. I have positive proofs of the presence of such a local reflex mechanism in the *Limulus* heart.

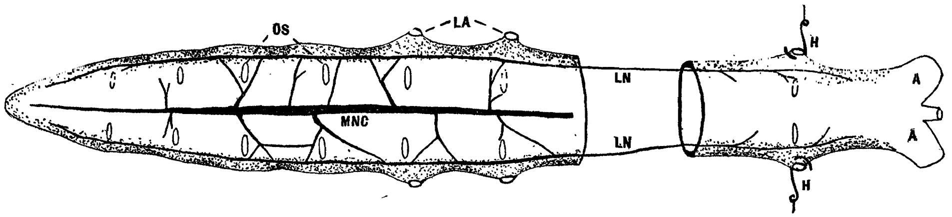

FIG. I. Heart of Limulus as prepared for studying the effects of solutions on the ganglion cells apart from that on the muscle. Dorsal view. *A*, anterior arteries; *LA*, lateral arteries; *LN*, lateral nerves; *MNC*, dorso-median nerve-cord or ganglion; *OS*, ostia; *H*, attachments for graphic registration of the contractions of the ganglion-free segments.

Conversely, if we apply the solution to the posterior end of the heart, that is, to the nerve-cord behind the lesion in the second segment, taking care that none of the solution reaches the isolated anterior end, the changes in the rhythm of the two anterior or reacting segments must be ascribed to changes in the activity of the ganglion cells due to the direct influence of the solution. It is possible to remove the greater part of the heart-muscle of the portion of the heart including the fourth to the seventh segments, leaving the nerve-cord, the lateral nerves and their main connections intact, without greatly disturbing the activity of the ganglion. The effects of the solutions on this isolated ganglion do not differ essentially from the effects of applying the solution to the nerve-cord left in connection with the muscle. It is, therefore, probable that the solutions worked with act on the ganglion cells directly rather than indirectly by stimulation of sensory nerves or nerve-endings.

The alkaloids were dissolved in filtered plasma from the animal, or in sea water. Sea water is isotonic with the blood (Dr. Garrey) and is so slight a stimulant to the ganglion as to be almost neutral. On the heart-muscle I could not find that sea water had any effect.

The Effects of the Alkaloids on the Muscle.—Atropin, curare, pilocarpin and physostigmin of the strength of 1–100 in sea water or plasma have apparently no effect on the heart-muscle, the strength or the rate of the beats not being altered. Digitalin and nicotin of the same concentration produce extreme tonus contraction when applied to the ganglion-free segments. At a dilution of 1–1,000 digitalin applied to the muscle causes increased strength of the contractions, followed by a slight tonus. Nicotin produces tonus contraction even at the dilution of 1–2,000. Veratrin at the dilution of 1–1,000 causes an initial diminution of the amplitude of the beats, followed by strong tonus. At a dilution of 1–100,000 or 1–500,000 the amplitude of the beats is diminished, but no tonus contraction is produced. Aconite was worked with at a dilution of 1–100,000 and weaker; at that dilution it has apparently no effect on the muscle.

The Effects of the Alkaloids on the Ganglion Cells.—Atropin, curare, pilocarpin and physostigmin of the concentration of 1–100 (a strength which has practically no effect on the muscle) stimulate the ganglion cells intensely, the beats becoming very rapid and in some cases reduced to a minimum. The stimulating action of these drugs is best shown at a dilution of 1–500 or 1–1,000; at this concentration their effect on the nerve-cord is in the direction of augmenting the rate and the intensity of the nervous discharge from the ganglion cells, that is, the rate and amplitude of the beats in the ganglion-free reacting portion of the heart are augmented. The latent-time of the action of these drugs is very short, or from one half to five seconds, the shorter, the stronger the solution and the more excitable the nerve-cord.

Nicotin, digitalin, veratrin and aconite at the concentration of 1–100 stop the activity of the ganglion cells instantaneously. That this is a case of over stimulation or paralysis and not a true depressor action is shown by the fact that the stimulating action of these alkaloids is plainly evident on greater dilutions. Veratrin is the most intense stimulant of any of the alkaloids worked with. It must be diluted 1–1,000,000 or 1–2,000,000 in order not to kill or paralyze the ganglion almost instantaneously by over stimulation. At this great dilution it still causes great augmentation of the rhythm of the ganglion cells. Aconite stimulates the ganglion similarly at the dilution of 1–100,000 or 1–500,000; nicotin at the strength of 1–5,000 or 1–10,000; digitalin at the dilution of 1–1,000 or 1–2,000.

The latent period of the stimulation is short, or from one half to five seconds. At the dilution of one to two millions veratrin has often an initial depressor effect, which is invariably followed by acceleration or stimulation. Such an initial depressor action was not observed in the case of any of the other alkaloids.

None of these alkaloids proves fatal to the ganglion unless the application is long continued or their concentration great enough to stop the activity of the ganglion cells at once, and even in these cases the ganglion cells can usually be brought back to almost normal activity by continued bathing in plasma or sea water. After a bath of ten seconds in 0.5 per cent. nicotin, 1 per cent. digitalin, or 1/500 per cent. veratrin I never succeeded in restoring the ganglion.

The action of these alkaloids is, therefore, primarily on the ganglion cells in the heart rather than on the nerve-fibers or the muscle-cells. This is especially true of atropin, curare, pilocarpin and physostigmin, as these drugs have slight, if any, effect on the muscle and motor nerve-endings at the concentration of 1–100. And it is also true of the other alkaloids, or nicotin, digitalin, aconite and veratrin, for although these drugs act strongly on the muscle even at a greater dilution than 1–100, their action on the nerve-cord is much more rapid and intense, and they, furthermore, act on the ganglion at a dilution which fails to affect the muscle. Part of the difference in the reaction of the muscle and the ganglion to these drugs is probably to be sought in difference in permeability of the muscle cell and the nerve cell.

The failure of these alkaloids (with the exception of veratrin) to produce initial inhibitory or depressor effects on the ganglion or the muscle is not due to the absence of an inhibitory nervous mechanism in the *Limulus* heart. Two pairs of inhibitory nerves pass from the posterior and dorsal surface of the peri-œsophageal ganglion to the median nerve cord on the dorsal side of the heart.

Before the action of these drugs on the ganglion and the heart muscle of *Limulus* was taken up an extensive study of the action of curare, atropin and nicotin on the molluscan and the crustacean heart had been carried out with the view of finding a drug that would paralyze the cardio-inhibitory nervous mechanism in these animals. None of the crustaceans or the molluscs allow a determination of the point of action of the drugs in the heart similar to the heart preparation of *Limulus*, but the reaction of the crustacean and the molluscan heart to these three alkaloids is identical with the reaction of the ganglion-free segments of the heart of *Limulus* on application of these drugs to the ganglion, which fact suggests that the relation of the ganglion cells to the heart-beat in these animals in no wise differs from that in the heart of *Limulus*.

We know of no drug which will paralyze the motor nerves or nerve-endings in the heart without destroying the excitability and contractility of the muscle. The heart-muscle responds to the stimulation of the nerves that pass from the nerve-cord to the muscle fifteen to twenty hours after the activity of the ganglion has ceased from exhaustion. After bathing the heart in one per cent. curare, one per cent. atropin, or one tenth per cent. nicotin for two or three hours the stimulation of these nerves is still effective. Nor does curare paralyze the motor nerve-endings in the body muscles of crustaceans and molluscs as maintained by some observers. In these animals curare acts like strychnia in the vertebrates, that is, causing primary excitation of the central nervous system, leading to tetanus and spasm and subsequent paralysis if the dose is sufficiently strong, but after such paralysis the body muscles still respond to stimulation of the motor nerves.

Alcohol in concentrations of 1–100 or 1–200 stimulates the ganglion cells both as regards the rapidity and the intensity of the nervous discharges.

The Action of Certain Inorganic Salts on the Ganglion Cells.—Barium chloride, rubidinum chloride, potassium chloride and sodium chloride stimulate the ganglion cells, the chlorides of calcium and cæsium depress the activity of the ganglion without primary stimulation.

When the nerve-cord is removed from the heart and the heart immersed in isotonic ($\frac{5}{8}$m.) sodium chloride the heart begins to contract more or less rhythmically after a latent period of from thirty to forty-five minutes. The addition of a small amount of calcium chloride prevents the development of this rhythm or stops it after it is once developed. The action of these salts on the ganglion-free heart-muscle of *Limulus* is thus identical with their action on the apex of the frog's or the tortoise's ventricle. The $\frac{5}{8}$m. NaCl solution applied to the nerve-cord stimulates at once or after a latent time of less than a second. The stimulation of the ganglion cells appears in increased rate as well as amplitude of contraction, the amplitude of the beats becoming gradually diminished, until after ten to fifteen minutes the activity of the ganglion ceases completely. A small amount of calcium chloride counteracts the stimulating and subsequent depressor action of the pure sodium chloride.

Calcium chloride in isotonic concentration down to a dilution represented by one part $CaCl_2$($\frac{5}{8}$m.) to twenty parts of sea water or plasma depresses the activity of the ganglion cells. This effect appears immediately on application of the solution to the ganglion.

When the strength of $\frac{5}{8}$m. is used the activity of the ganglion stops short at once, or a few diminutive beats separated by greatly prolonged diastoles precede the complete abolition of the activity. In weaker concentrations the depressor actions on the ganglion cells appear only in diminished rate and amplitude of the contractions in the reacting portion of the heart. After complete abolition of the activity of the ganglion by $CaCl_2$ the normal activity can be restored by bathing with plasma, sea water or isotonic sodium chloride solution. The resumption of functional activity is very gradual.

The action of calcium chloride on the ganglion is almost duplicated by that of cæsium chloride, only that the latter salt does not appear to depress quite as strongly as the former. The recovery of function is also somewhat quicker after the cæsium chloride bath. At $\frac{5}{8}$m. concentration cæsium chloride stops the activity of the ganglion almost at once, but in concentrations represented by one part of $\frac{5}{8}$m. of the salt to ten or twenty parts of sea water or plasma the depressor action appears in diminished rate and amplitude of the beats. The depressor action of this salt on the ganglion cells is further shown by its antagonism to the stimulating action of the alkaloids veratrin, aconite, niconite and digitalin. The addition of cæsium chloride to the solutions of these drugs greatly decreases their stimulating power, and bathing the nerve-cord in isotonic (or even weaker) solutions of the chloride after previous application of the alkaloid solutions quickly counteracts their stimulating effects.

The most powerful stimulants to the ganglion cells are the chlorides of barium, potassium and rubidinum. It is very difficult to work with the chloride of barium because of the fact that the barium is precipitated by the sulphates in the plasma or sea water. The isotonic solution of the salt can not, therefore, be diluted with either sea water or plasma, but a dilution is necessary to study its effect on the ganglion cells because the $\frac{5}{8}$m. concentration stops their activity instantaneously. This is due to over stimulation or paralysis. The barium chloride solution was diluted with a mixture of fifteen parts of $\frac{5}{8}$m. NaCl to one part $\frac{5}{8}$ m. $CaCl_2$. This mixture proved nearly neutral for the nerve-cord. The addition of even one part of barium chloride to twenty parts of this mixture produced a marked augmentation of the rate and strength of the beats, which under the conditions of the experiments meant an augmentation of the rate and intensity of the nervous impulses reaching the muscle. Rubidinum chloride stimulates the nerve-cord at a dilution represented by one part of the $\frac{5}{8}$m. solution to twenty parts of sea water or plasma. The isotonic solution of the salt stops the activity of the ganglion instantaneously.

The $\frac{5}{8}$m. solution of the chloride of potassium stops the activity of the nerve-cord at once, just like the similar solutions of the chlorides of barium and rubidinum. Even at the dilution of one part of the potassium chloride solution to five parts of sea water or plasma the activity of the ganglion is usually arrested immediately, that is, without previous stimulation. That this is not a true depressor action is shown by the fact that at greater dilutions a strong stimulating action appears. At a concentration of one part $\frac{5}{8}$m. KCl to twenty parts of plasma the rate and strength of the beats are greatly augmented and this stimulating action is maintained for a relatively long time. Potassium chloride is a much greater stimulant to the nerve-cord than is sodium chloride. The concentration of the latter salt, represented by one part $\frac{5}{8}$m. NaCl to twenty parts of sea water or plasma, has no appreciable effect on the nerve-cord. Yet under some conditions potassium chloride appears to have a true depressor action. When the ganglion has been bathed for some time in a solution of one part $\frac{5}{8}$ m. KCl to twenty parts of sea water and this solution replaced by sea water for some minutes, a reapplication of the former solution to the ganglion may result in a partial depression of the activity preceding the stimulation. The action of the potassium ion is thus a complicated one and in part determined by the condition of the ganglion cells.

Application of distilled water to the nerve-cord causes great acceleration of the rate of the beats. This may be accompanied by an increased amplitude of the contractions for a few seconds, but the contractions become diminutive very quickly, and the rapid diminutive beats are followed by a prolonged diastole. The function of the nerve-cord is restored by plasma or sea water.

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QUOTATIONS.

THE COLLEGE YEAR.

THE beginning of the college year, a month ago, brought several interesting facts under discussion. For instance, in almost every college there was an increase in the number of students—in some colleges a very large increase. The demand for higher training keeps pace with the growth of wealth and population—perhaps outruns it, by mere physical measurement. Endowments and gifts to colleges continue to be made in ever-increasing sums. Yet the demands, especially of the larger universities, become greater every year. Columbia University, in New York City, for instance, has immediate need of more than two millions of dollars; and President Wilson, of Princeton, it will be recalled, formulated a plan of enlargement and improvement, last year, that calls for about twelve millions.

Dr. Alfred G. Mayer, a little while ago, put into concise form in SCIENCE the statistics of higher education in the United States, which show that the number of our universities and colleges in 1902 was 638, and the number of students, including graduate students, was 112,433. The number of colleges has increased by 50 per cent., and the number of students by about a hundred per cent., during the decade. But how small a part the college-bred are of the whole population is yet somewhat startling, for they comprise but one in every 700. There were twice as many teachers in 1902 as there were in 1889. The value of college property was multiplied by almost three; the endowment funds were two and a half times as great; gifts for other purposes were nearly three times as great; and the total income, exclusive of benefactions, was more than trebled. The number of books and libraries was doubled.

In spite of this increased prosperity, the average salary of teachers has probably declined. In one of our largest universities, the average, ten years ago, was $1,500. It is now only $1,257. In another one, the average was $1,454, and now it is $1,355. This low average has been caused by the engagement of an increasing number of instructors and other subordinate members of the teaching force. The salaries of the professors themselves have not declined, but the increasing proportion of college instruction is now done by subordinate members of the faculties. Sir William Ramsay, during his recent visit to the United States, made more than one plea for increasing the salaries of teachers of high grade.

College training, except in those universities that are maintained by the states, is yet paid for by rich men and dead men. The students, even at those institutions where fees are highest, pay not more than one third of the cost of the training that they receive. It is an industry that must yet be endowed—a fact that hints of its ecclesiastical history. In the perfect economic state, the state will pay for the training of all its children. But we need not yet bother ourselves about the ideal economic state. There is enough work for us to do in training well as large a number of capable youth as possible, at the expense of rich men, living or dead, at the expense of the state, or in any other way, if only enough youth be trained, and be trained well enough.—*The World's Work* for November.

BOTANICAL NOTES.

BOTANY AS A FACTOR IN EDUCATION.

IN a suggestive and helpful article in the October number of the *School Review* Professor J. M. Coulter discusses botany as a factor in education, noticing first its special function in secondary education, and then its general function as a representative scientific study. He says truly that since plants enter very largely into human experience their study 'must relate the pupil to his most common