temperature lies between 1 and 2. Investigations are planned to determine other properties of the nuclear colloid, particularly with regard to its radiosensitivity. The present knowledge of temperature effects will serve as a guide to the proper experimental conditions.

Summary. By means of the falling nucleolus method it was determined that a 10°C increase in temperature lowers nuclear viscosity in the Asterias egg by approximately 2.7 centipoises. Thixotropic properties disappear at temperatures above 25°C and coagulation occurs at about 35°C.

17041

Effect of Stretch and Pressure on Stimulus Formation in the Dog's Auricle.

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The application of a few crystals of aconitine or of 0.05 cc of a 0.05% solution of aconitine to the auricular appendix of the exposed heart of the dog results in a regular tachycardia with a rate of 300-500 beats per minute. Invariably this tachycardia responds to faradic stimulation of the vagus nerves with an increase of rate.[1] The administration of aconitine to the site of the sinus node also leads to a tachycardia which spontaneously or following stimulation of the vagus or sympathetic nerves is transformed into auricular fibrillation.[2,3] Because of its high rate and the ease of its transformation into fibrillation the auricular tachycardia was considerd to be auricular flutter. Cooling the small area to which the aconitine has been applied stops the fibrillation (or flutter); it reappears immediately when cooling is interrupted.[2] Earlier it was pointed out that these observations are not compatible with the assumption that auricular fibrillation is due to a circus movement mechanism. An extremely rapid stimulus formation with secondary formation of reentry waves was considered the responsible mechanism.[2]

This report deals with observations made during application of stretch and pressure on the right auricle of the dog's heart in the presence of an aconitine-induced tachycardia.

Method. The technic employed was the same as in previous investigations. The heart was exposed with the dog under nembutal anesthesia and artificial respiration. Aconitine was injected into the region of the head of the sinus node. All electrocardiograms were recorded in lead II. Stretching of the auricle was accomplished by one of several methods: 1) by attaching a weighted hook to the right auricular appendix or the right auricle at the junction between the vena cava superior and the right appendix; 2) by stretching the tip of the appendix of the right auricle by means of a blunt forceps; 3) by rapid intravenous infusion of 50 cc of 0.9% saline at body temperature into the jugular vein or the vena cava superior. Pressure on the area into which aconitine had been injected was exerted by a probe. In all experiments we satisfied ourselves that the site of application of aconitine actually was the focus of stimulus formation because cooling of this site invariably resulted in a cessation of flutter or fibrillation as long as cooling continued.

Results. In 11 experiments stretching led to an increase of auricular rate and to auricular fibrillation. The fibrillation sometimes developed so quickly that the transitional period of tachycardia could not be analyzed. More often a short period of increased rate preceded the fibrillation. The duration of the

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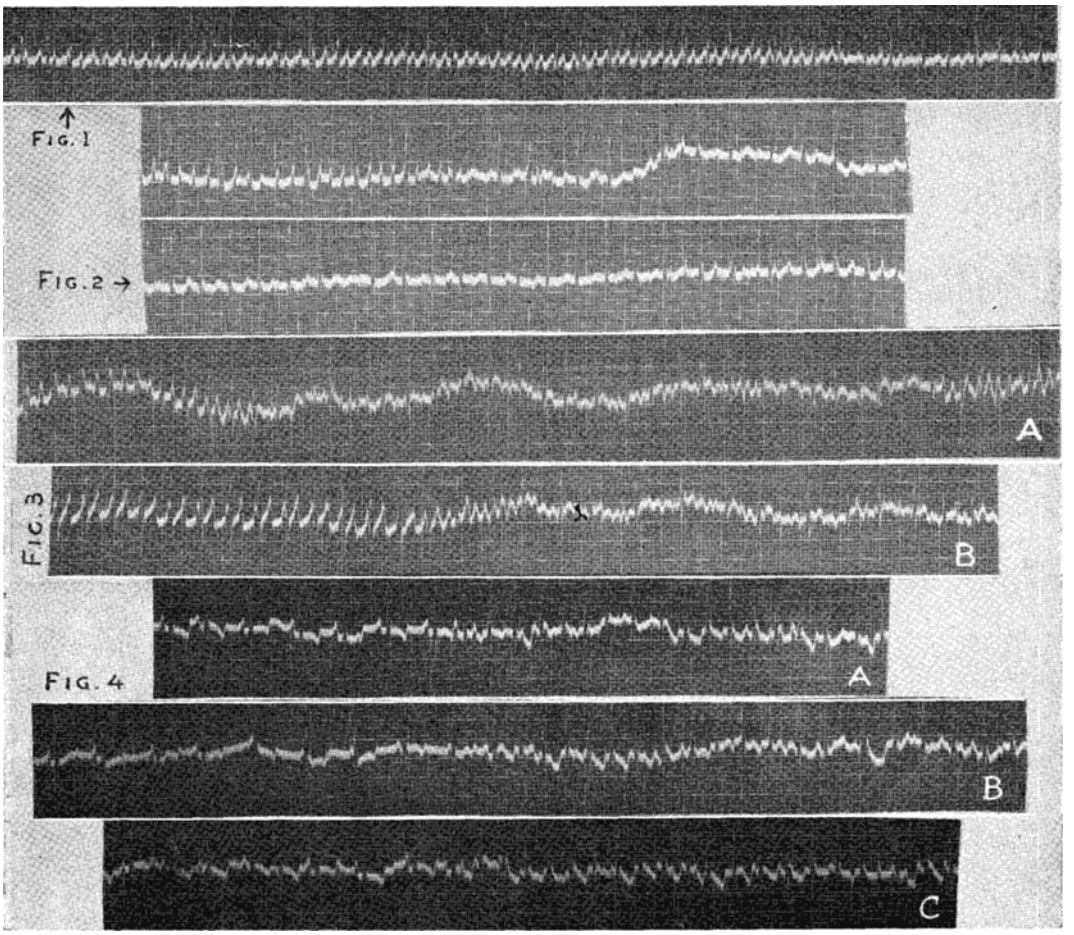

FIG. 1. Stretching of the right auricle changes auricular flutter into fibrillation.

FIG. 2a and b. The tracings are continuous. Stretching of the right auricle causes temporary auricular fibrillation and return to flutter.

FIG. 3a and b. Stretching of the right auricle causes a marked increase of the flutter rate and fibrillation.

FIG. 4a, b and c. In Fig. 4a stretching changes sinus rhythm into transient auricular fibrillation; rapid dilatation of the right auricle caused by intravenous injection has the same result (Fig. 4b). Pressure exerted on the site of injection causes auricular fibrillation (Fig. 4c).

fibrillation thus elicited varied from a few seconds to minutes.

Fig. 1 was obtained in an experiment on Sept. 23, 1947. Application of the aconitine solution to the head of the sinus node caused an auricular flutter with a rate of 460 per minute and a 2:1 A-V block. Stretching of the auricle with a weight of 20 g increased the auricular rate to 540 and finally to 600; fibrillation then suddenly appeared.

In the experiment of May 6, 1948 auricular flutter with a rate of 400 and an irregular A-V block was elicited in a similar manner. With stretching the rate increased to 500 and then to about 600; at this moment fibrillation immediately followed (Fig. 2a). Fig. 2b represents the direct continuation of Fig. 2a and shows the return of auricular flutter after approximately 10 seconds.

In the experiment of Jan. 25, 1949 the fibrillation induced by stretching lasted only about 7 seconds (Fig. 3a). The auricular rate before the stretching at the beginning of Fig. 3a measures 420 per minute. This rate was increased with stretching to about 600 and again fibrillation appeared. With the

return of flutter, at the end of the tracing, the auricular rate is again 600 and gradually fell to values of around 460. The stretching was repeated about 12 minutes later with the same result. The auricular rate increased from 420 to about 660 beats per minute and auricular fibrillation appeared (Fig. 3b).

Of particular interest are the tracings of Fig. 4. In this experiment auricular stretching had converted auricular flutter into fibrillation which persisted for 58 minutes. The stretching was performed shortly after the injection of aconitine and it is possible that the stretching hastened the appearance of fibrillation which would have developed anyway. After the disappearance of fibrillation repeated stretching of the auricle by traction on the appendix regularly elicited transient fibrillation.

Fig. 4a shows a regular sinus rhythm with a rate of 126. Stretching caused auricular fibrillation after the auricular rate had increased from 200 to 300. Two P waves which are situated within the QRS complex just before fibrillation started, follow each other with a rate of about 600. Shortly after this fibrillation subsided, 50 cc of saline were rapidly injected into the jugular vein in order to cause acute dilatation of the right auricle. Fig. 4b obtained during this maneuver shows that after a short period of sinus node inhibition and A-V escape beats fibrillation developed. Repetition of this experiment twice yielded the same results. Finally pressure was exerted on the site of the injection of aconitine with a blunt probe and fibrillation reappeared immediately (Fig. 4c).

Similar observations were made in other experiments for a period of 15-20 minutes after the disappearance of flutter or fibrillation.

Discussion. In all 11 experiments identical results were obtained. If the auricular muscle containing the sinus node into which aconitine had been injected was stretched by traction or by rapid intravenous introduction of fluid invariably an increase of auricular rate and then auricular fibrillation were observed. Even after the aconitine induced fibrillation (or flutter) had disappeared they could be reinduced by these measures.

The appearance of rhythmic stimulus formation or the increase of rate of an already existing stimulus formation in a response to the mechanical stimulus of stretch or pressure is known to exist in sensory nerves, in motor nerves, the skeletal muscle, the heart muscle of the frog[4] and the bundles of specific fibers of the heart of the dog.[5] In lower animals, the mollusks for example, the stretch caused by cardiac filling is the physiologic stimulus for cardiac activity. Stretching and pressure facilitate depolarization of the cell membrane;[6] stronger mechanical stimuli of this kind cause local injury of the type seen during continuous chemical or electrical stimuli which also lead to rhythmic stimulus formation. On the other hand conductivity does not seem to be altered by mechanical stretching. This fact has been demonstrated for the skeletal muscle,[7] for the junctional A-V fibers of the heart of the frog[8] and finally for muscle strips from the frog's ventricle.[9] In the experiments mentioned last stretching which increased by 20% the distance between two points on which the electrodes were attached did not increase conduction time.

In view of the fact that the mechanical devices used in our experiments influence only the formation of stimuli and not the conduction of the impulses we see in these observations further justification for the assumption that in auricular flutter and fibrillation we are dealing primarily with a disturbance of stimulus formation. The rate of stimulus formation is increased by stretching and when a certain limit is reached—in our experiments a rate of about 600 per minute—fibrillation appears. There are no data available which permit us to assume

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that stretching causes an increased rate of conduction of a circus wave. As soon as the rate of stimulus formation reaches values of approximately 600 per minute the appearance of refractory islands of muscle makes a regular response impossible and flutter changes into fibrillation.

The appearance of fibrillation following stretching, particularly on stretching by intravenous infusion (Fig. 4b) is of interest because it may explain the attacks of paroxysmal fibrillation on sudden physical exertion. The increased filling of the right auricle particularly at the beginning of sudden severe physical exercise[10] may in a predisposed heart elicit paroxysmal fibrillation.[11-13]

We fully appreciate the fact that the assumption of a rapid stimulus formation as the cause of auricular flutter or fibrillation[2] will necessitate an answer to one pertinent objection: Why does stimulation of the vagus nerves increase the auricular rate of one type of auricular tachycardia (auricular flutter) and stop the stimulus formation during sinus rhythm or other auricular tachycardias? Auricular extrasystoles caused by the intravenous injection of minute doses of aconitine disappear during the faradic stimulation of the vagus nerves. They increase in number after the stimulation of the vagus is discontinued.[14] At the present time we are not prepared to offer an explanation for this fundamental difference. It is possible that local application of aconitine causes the establishment of a continuous stimulus; this would elicit more frequent responses with a shortening of the refractory phase during stimulation of the vagus nerve. If extrasystoles occur due to discontinuous stimulus formation vagal stimulation inhibits them.

Summary. Stretching of the auricular muscle fibers during a tachycardia induced by aconitine leads to an increase of rate and to transient auricular fibrillation. Transient auricular fibrillation (or flutter) can be elicited by stretching during a period of 15-20 minutes after the aconitine arrhythmia has subsided and sinus rhythm prevails.

Because there is much evidence that stretching changes impulse formation and not impulse conduction these results are believed to bring further evidence against the circus movement hypothesis.

The experiments offer an explanation for the appearance of paroxysmal fibrillation induced by physical exertion.

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17042

Cortical Projection of Proprioception in the Cat and Monkey.*

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In spite of the abundant evidence of the importance of proprioception for the regulation of movements in general and those elicited by stimulation of the motor cortex in particular[1-3] little is known about the action of proprioceptive impulses on the cortex.†

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