CHART II.

Comparison of stable antigen emulsion and Kahn antigen dilution in the microscopic slide precipitation test for syphilis.

Number of sera tested. Courtesy of Dr. H. J. Knapp, Cleveland Health Department	1 Three day old emulsion of concentrated chilled Kahn extract No. 4 by new formula	2 Freshly prepared emulsion of concentrated chilled Kahn extract No. 4 by new formula	3 Freshly prepared Kahn antigen dilution (same extract as 1 and 2)	4 Three day old emulsion of concentrated chilled Kahn extract No. 1 by new formula	5 Freshly prepared emulsion of concentrated chilled Kahn extract No. 1 by new formula
49	—	—	—	—	—
4	—	—	—	—	—
1	±	—	—	—?	±
1	±	±	—	—	—
2	±, +	±	—	—	—
1	+	±	±	—	—
1	++	++	++	++	++, +++
1	++	+	+	++	+, ++
1	+++	++	—?		++
1	++++	++++	++++	+++	++, +++
3	++++	++++	++++		
7	++++	++++	++++	++++	++++

Total sera tested, 73.

3. Because more cholesterin may be dispersed in a given total by the new formula, it is possible to make the emulsions more sensitive, yet no less specific, than Kahn antigen dilutions from the same extract.

4018

Bronchial Perfusion of Isolated Lung as a Method for Studying Pharmacologic Reactions of Bronchiolar Muscle.

TORALD SOLLMANN AND W. F. VON OETTINGEN.

From the Department of Pharmacology of the School of Medicine of Western Reserve University.

The methods for studying the changes of bronchiolar tone in intact animals are often complicated by changes in the circulation which make their interpretation uncertain. It would therefore be desirable to check them under conditions in which the circulation would be excluded, namely outside of the body. The use of ring preparations of tracheal or bronchial muscle for this purpose has not been altogether satisfactory, partly because these are confined to the trachea and larger bronchi which might react differently from

the smaller bronchioles that play the major part in the bronchial reactions; and partly because it is difficult to reproduce the natural conditions of tension. These objections are avoided in the following method, which is based on measuring the rate of flow of a Locke solution through the bronchial tree, allowing the fluid to drain off by filtration.

The arrangement is shown diagramatically in Fig. 1. A Mariotte bottle (M) filled with Locke solution is connected with a Woulff bottle (W), also filled with Locke solution, which sits in a bath (B) regulated to keep the temperature of the solution at 39° to 40° C., as indicated by the thermometer (Th). The outflow passes to a ⊣ tube (T). The upper limb of this is furnished with a short piece of rubber tubing (R) which may be closed by a screwclamp (S). The lower limb is connected with a cannula tied into the short stump of the trachea of the excised lung (L). This should be used either promptly after the death of the animal, or else after lying on ice.

In starting the experiment, the T tube is raised so that no fluid flows from the Mariotte bottle; the screwclamp (S) on the upper limb is opened, and the lung is attached to the lower limb. The T is then gradually lowered so that just enough liquid flows into the bronchi to distend the lung approximately to its normal size. The lung is then gently squeezed to expell the air through the vertical limb. When foam ceases to rise, the screwclamp is closed and the height of the lung is adjusted so that the air enters the Mariotte bottle at the rate of about 20 bubbles per minute. When the rate of bubbling has become nearly constant, indicating that the volume of the lung has become adjusted to the pressure of the system, the preparation is ready for the experiments.

The drugs are injected in 1 cc. of solution from a syringe into the rubber tube, between the T and the lung; followed immediately by the withdrawal of the same volume of fluid, through the tubing near the Woulff bottle, to balance the changes of pressure in the system. The rate of flow should not be estimated by the outflow of liquid from the lungs, since this is complicated by the alterations of capacity through edema and stretching. It should be measured by the entrance of air into the Mariotte bottle; either simply counting the number of bubbles per minute by a watch; or connecting the Mariotte bottle through Ta to a tambour registering on a drum, directly or through an electric relay. However, such registration is generally a needless complication.

Figure 2 illustrates the results obtained with this method. In Fig. 2A 1 cc. of pilocarpine, 1 :1000, was injected into the trachea,

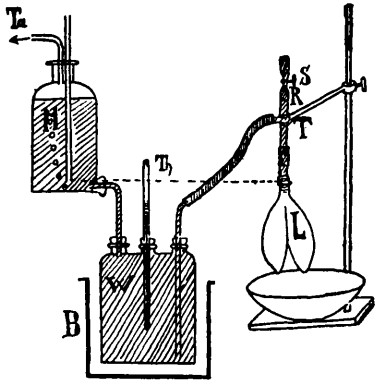

FIG. 1.

Diagram of bronchial perfusion. The letters are explained in the text.

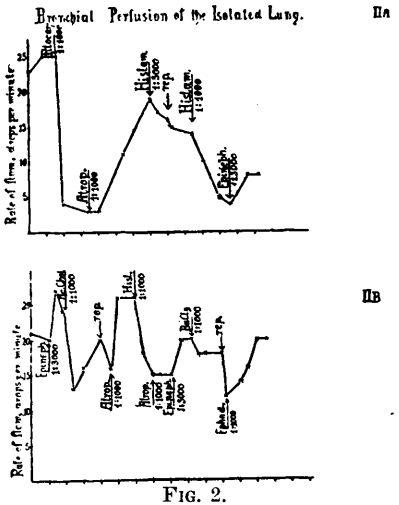

FIG. 2.

Charts of two experiments, illustrating the effects of drugs on the bronchial flow. The solutions were always injected in the volume of 1 cc. The time is marked in 5 minutes. "Rep."—repetition of injection.

which was immediately followed by a reduction of the inflow from 25 to 4 bubbles. About 10 minutes later 1 cc. of atropine sulphate, 1:1000, was injected, which produced a dilation indicated by an increase from 3 to 19 bubbles within 15 minutes. 1 cc. histamine, 1:5000, produced a slight constriction which became more pronounced by repeating the injection with a solution of 1:1000, as indicated by a decrease of the inflow from 19 bubbles to 4 within 25 minutes. Epinephrine, 1:3000, after histamine, produced only a slight dilation characterized by an increase from 4 to 8 bubbles per minute.

Fig. 2B illustrates a similar experiment. 1 cc. of epinephrine, 1:3000, produces a short increase of the inflow of 35%, acetylcholine, 1:1000, reduces it by 35%. Atropine, 1:1000, after acetylcholine produces dilation above the normal, and histamine, 1:1000, produces a rather marked constriction of 55%. Atropine, 1:1000, after histamine shows no effect, whereas epinephrine, 1:3000, produces a slight dilation. Barium chloride, 1:1000, produces another constriction of 40%, which is abolished by the subsequent injection of epinephrine, 1:1000.

As may be seen from these experiments, the method permits the study of the responses of the bronchial muscle to autonomic and muscular poisons, and gives a clear-cut picture of their action,

without the interference of changes of the circulation or of the volume of the heart.

4019

A Device for Determining Refractory Period of the Mammalian Heart During Normal Sinus Rhythm.

HAROLD A. WHEELER AND E. COWLES ANDRUS.
(Introduced by Edward P. Carter.)

From the Department of Physics, Johns Hopkins University, and the Cardiographic Laboratory, Johns Hopkins Hospital and University.

In previous determinations of the refractory period of the mammalian heart an artificial rhythm has been established by means of a series of break induction shocks at a rate necessarily more rapid than that of the normal sinus rhythm. This rhythm has then been interrupted by break shocks timed by some mechanical device to fall at various points in diastole. Such measurements have, therefor, yielded the refractory period of an artificially induced excitation.

The authors have devised an apparatus utilizing the action current after amplification with which it is possible to interrupt the *sinus* rhythm at any interval following a normal excitation. The amplifier is designed to be connected to the terminals of the galvanometer string without affecting the performance of the latter. The electric impulse to be amplified is on the order of 1 millivolt for 1/50 second, and the current supplied to the amplifier must be negligible as compared with about 1 microampere, the galvanometer current. A 5-stage, resistance-coupled amplifier is used, with 4 UX-240 tubes and 1 UX-112A. The string terminals are connected directly in the first grid circuit. The 4 coupling condensers are large, so as to give the entire amplifier a discharging time constant of 1/6 second. The first 2 plate circuits have condensers in parallel, so as to obtain a charging time constant of 1/50 second. In this way, the amplifier is rendered free from impulses of much longer or shorter duration. A 10,000-ohm magnet carries the plate current of the last tube, normally less than ½ milliampere because of a large negative grid voltage. An impulse of ½ millivolt for 1/50 second, applied to the amplifier, is amplified to over 20 volts for 1/10 second at the magnet terminals, about 200,000 times the applied impulse.