The use of dessication of typing serums makes it unnecessary to use precaution as to sterility in handling and storage and simultaneously eliminates the possibility of precipitate formation, which causes confusion in interpreting results in stored liquid phenolized serums. This method makes it possible for the practicing physician to carry the typing serums in his case for emergencies, and hospital technicians can also rely on more accurate results from these preserved serums.

Slides of phenolized serums have been kept at room temperature and 8°C. without deterioration for 11 months, whereas the liquid phenolized serums give a very confusing precipitate in that time. Desiccated and liquid stained serums have kept perfectly to date or 7 months. This study is being continued to determine the maximum length of time the serums can be desiccated and still give accurate results.

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Independence of Ventricular Arhythmia from Insufflations to Coronary Flow in Rabbits.

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In a previous paper[1] it was demonstrated that a left ventricular arhythmia produced from insufflations of benzol in tracheotomized animals was contingent on impulses which descend the median part of the lateral columns of the spinal cord and reach the heart by way of the stellate ganglia. The purpose of this report is to record some observations which suggest that this arhythmia is not dependent on the nutrient supply of the heart. This arhythmia is normally preceded by a moderate rise in carotid pressure, a change which Markwalder and Starling,[2] Anrep and Segall,[3] Hochrein, Keller and Mauke[4] would place first, as a means for producing an increased coronary supply. The arhythmia never appeared when blood pressure was low (40 mm. and below). Double vagotomy in no way delayed the onset or shortened the interval of this arhythmia, except possibly for a short interval immediately after sectioning.

[1] Allen, W. F., *Am. J. Physiol.*, 1931, **96**, 243.

[2] Markwalder and Starling, *J. Physiol.*, 1914, **47**, 275.

[3] Anrep and Segall, *Heart*, 1926, **13**, 239.

[4] Hochrein, Keller und Mancke, *Arch. Exp. Path. u. Pharm.*, 1930, **151**, 146.

Porter,[5] Mass,[6] Wiggers,[7] Anrep and Segall,[3] Greene[8] and others demonstrated that the constrictor fibers for the coronaries run in the vagi and Anrep and Segall[3] have demonstrated that double vagotomy causes a considerable increase in the coronary flow.

If a change in the nutrient supply to the nerve-intact heart is the cause of the arhythmia from insufflations, it should be due to an increase in the coronary flow rather than to a decrease. To demonstrate the effect on the pulse of a brief interval of anemia of the left ventricle, the left interventricular artery and the left ventricular vein* were ligated close to the atrium in a number of animals for several minutes, and in a few animals the right interventricular artery was also ligated. In no instance did these ligations evoke a premature systolic arhythmia. At the time of occlusion of each vessel there was a little drop in blood pressure and usually one or two ectopic beats. Since these beats occurred from similar ligations of any part of the ventricle, they doubtless correspond to the premature beats that Hering[9] and many others have obtained from mechanical and electrical stimulation of various parts of the heart.

To determine whether an increased coronary supply to the ventricles is essential for the production of the premature systoles in the arhythmia obtained from insufflations, the following experiment was done: A rabbit under 75 to 85% surgical anesthesia dosage of sodium barbital was prepared for taking carotid and trachial respiratory tracings. The heart was exposed from the left side and an arhythmia was obtained from benzol insufflation. The right interventricular artery, the left coronary vein and the left interventricular artery were then slit longitudinally for some distance, beginning close to the atria (a procedure which never elicited an arhythmia). This required but a few seconds and within 30 seconds benzol was insufflated into the nostrils in an attempt to produce an arhythmia while all the main arteries to the left ventricle were bleeding pro-

[5] Porter, W. T., *Boston Med. and Sur. J.*, 1896, **134**, 39.

[6] Mass, P., *Pflüger's Arch.*, 1898, **71**, 399.

[7] Wiggers, C. J., *Am. J. Physiol.*, 1909, **33**, 391.

[8] Greene, C. W., *Internat. Physiol. Cong. Abst.*, 1929, **13**, 103.

* A chrome-yellow gelatin injection of the rabbit's heart from the thoracic aorta discloses a different arrangement of the coronary veins than is found in the dog and other laboratory animals. A large left ventricular or great cardiac vein (See Vogt und Yungs' Anatomie) takes origin from the apex of the left ventricle and follows superficially in the ventricle about midway between the 2 interventricular arteries to enter the atrio-ventricular groove and end in the coronary sinus. Several right ventricular veins drain the right ventricle.

[9] Hering, H. E., *Pflüger's Arch.*, 1900, **82**, 1.

fusely. Under these conditions arterial pressure remained above the necessary height for obtaining an arhythmia much longer than was expected from the hemorrhage. The pulsating heart prevented clotting.

Five animals yielded 1 to 3 premature systolic arhythmias following a like number of benzol insufflations at the time the above mentioned coronary arteries were bleeding profusely. A representative record discloses a perfectly regular arhythmia from insufflations in that it is preceded by a moderate rise in blood pressure (16 mm.) and a slowed and strengthened pulse. Every animal which gave the 'insufflation' arhythmia with the heart exposed, produced one from insufflations when the above mentioned coronary vessels were slit. In one animal the arhythmia was obtained after the left interventricular artery and the left ventricular vein had been ligated close to the atrium.

Since the ventricular arhythmia following insufflations of benzol takes place readily under conditions where the blood supply to the ventricles has been greatly reduced and cannot be increased appreciably, when it normally occurs under conditions which should cause a pronounced increase in the coronary intake, suggests that this arhythmia is not contingent on the coronary flow to the ventricles.

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On the Property of Certain Normal Animal Sera to Neutralize the Virus of Poliomyelitis.*

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It has been known for a long time that the sera of children and monkeys, who have suffered from poliomyelitis, are capable of neutralizing the specific virus, although the frequency of this occurrence appears by no means to be as regular as was formerly believed[1] More recently a high percentage of normal adults, giving no history of a previous attack or of contact with the disease, have also been found to possess virucidal substances in their blood.[2] A

* Under a grant from the International Committee for the Study of Infantile Paralysis, whose work is being financed by Jeremiah Milbank.

[1] Jungeblut, C. W., and Smith, L. W., *J. Immunol.*, 1932, in press.