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Sudden death of experimental animals following intrapericardial injections of tincture of iodine.

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During the course of some experiments on rabbits, it was noted that injection of tincture of iodine (U. S. P.) into the pericardial sac was followed by some unidentified disturbance of cardiac mechanism which resulted in the death of the animals within a few minutes. The same series of events followed in each of the five animals employed. Six dogs were then studied. Under ether intratracheal anesthesia, the chest was opened and from 1.5 to 2 cc. of tincture of iodine injected, with much the same results as with the rabbits. As soon as the first few drops of the solution touched the epicardium there was an obvious visible effect on the heart muscle which continued until the death of the animal within five to ten minutes. Examining the heart grossly after death, it was found that the iodine had diffused over the entire epicardium but there was no visible evidence of staining of the heart muscle beneath the serous covering. In order to determine, if possible, the sequence of events following the injection of the irritant, the next two experiments were carried out in the Department of Physiology with the animals attached to the electrocardiograph. The first animal alone succumbed to the first dose of iodine. The second animal failed to show the usual prompt response and death did not occur until three injections of iodine had been given. We have no positive explanation to give of the phenomena observed. The rapidity of the effect of the injection would make it improbable that there was any systemic disturbance; rather it would point to a purely local action of the alcohol on the heart muscle or the electrical mechanism of the organ. The electrocardiograms showed paroxysms of ventricular tachycardia, flutter and fibrillation. After the paroxysms a peculiar rhythmical rising of the T wave was noted coming off the down stroke of the R wave at higher and higher levels and then dropping back, but finally coming off per-

sistently at a higher level and rising above the peak of the R wave, the latter appearing merely as a notch. The resulting complexes were bizarre. A.-V. block finally appeared. A shifting of the position of the auricular pacemaker was noted in P waves which became negative and returned to upright positive position. In one instance the auricular electrical phenomena continued, while in another the ventricular electrical changes continued after the auricle stopped.

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A structural characteristic of the cardiac poisons.

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Former investigations[1] have shown that strophanthidin is unsaturated and that the double bond is situated within the lactone ring, between the β and γ carbon atoms, so that strophanthidin may be designated as a $\Delta\beta$-γ crotonic lactone. Characteristic of strophanthidin and all of its derivatives which still possess this unsaturated lactone ring is their reducing action on Tollens' solution. On the other hand, dihydrostrophanthidin and its derivatives, or isostrophanthidin, in which the double has been either hydrogenated or shifted to another position, no longer react with Tollens' reagent, or at least far more gradually than in the case of strophanthidin and its derivatives. The behavior towards Tollens' solution is thus a very characteristic test for the unsaturated lactone group of these compounds.

Results of a similar and most striking character have been recently obtained by the use of the sodium nitroprusside test. Strophanthidin and all of its derivatives which still possess the unsaturated lactone ring give positive reactions with this reagent. But as soon as this group is hydrogenated or lost by saponifica-

[1] Jacobs, W. A., and Collins, A. M., *J. Biol. Chem.*, 1925, lxiv, 383; 1925, lxv, 493.