tive differences in specific activity noted on oral administration of testosterone propionate and methyl testosterone may be related to a difference in the route of absorption from the intestinal tract, rather than to a lesser destruction of methyl testosterone by intestinal ferments as has been suggested.[2] If absorption is mainly by way of the lacteals and lymphatics, thus avoiding the liver, a greater specific effect should be present than if absorption occurs into the portal circulation. It is likely that the specific chemical structure influences the route of absorption. There is one other possibility, although a slight one. Methyl testosterone may be absorbed through the esophagus during ingestion and thus enter the systemic circulation by way of the esophageal veins; or if it is less rapidly destroyed in the gut, or less rapidly absorbed, enough might reach the lower gut to enter the systemic circulation by way of the hemorrhoidal veins.

Summary and Conclusion. Methyl testosterone, like testosterone propionate, does not exert its specific effect on the genital organs of castrate male rats when implanted in pellet form in the spleen. When the pellet is placed in the transplanted spleen, with the splenic vessels ligated, the specific effect returns. As both androgens appear to be destroyed in the liver, it is suggested that the differences in the specific effects of these two substances when administered orally may be due to different routes of absorption from the intestinal tract (*e. g., via* the lymphatics), rather than different sites of inactivation.

11164

Proof of Excitability of Mammalian Ventricles During Systole.

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In a recent paper[1] we reported that a single, short, strong, localized shock applied directly to different regions of the left ventricle of dogs anesthetized by sodium barbital can produce permanent ventricular fibrillation. The same results have since been obtained on dogs under chloralosane and in cats under dial anesthesia. A shock, if strong enough and given during the vulnerable period near the end of ventricular systole, produces one extra-systole followed by about 4 more or less coördinated beats and then by true ventricular fibrillation. If

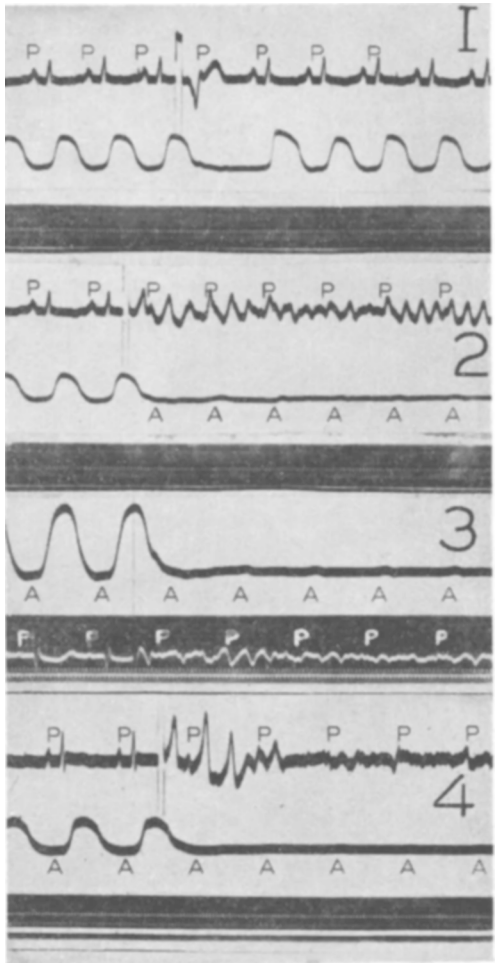
1 Wiggers and Wégria, *Am. J. Physiol.*, in press.

the shock is not quite strong enough or comes a little early in systole, instead of producing ventricular fibrillation, it gives a single premature contraction and only rarely several.

It is important to establish with certainty that these effects are due to ventricular excitation by the shock applied during systole rather than to a spread of current to the atria and subsequent ventricular excitation by supraventricular impulses arriving during diastole.

By recording simultaneous electrocardiograms and left ventricular pressure curves the latter possibility can be definitely excluded for the following reasons:

1. The premature electrical variation starts too shortly after the stimulus to allow for the A-V conduction time (Figs. 1-4).



2. The electrical variation has the form of a ventricular premature beat and not that of a ventricular beat due to an auricular impulse (Figs. 1-4).

3. The P-wave occurs after the premature ventricular complex and the spacing of the P-wave remains normal, showing that the auricular rhythm has not been disturbed (Figs. 1, 2-4).

4. The premature contraction when not producing fibrillation is followed by a full compensatory pause which, although possible in case of auricular premature systoles, is a rare occurrence (Fig. 1).

5. A stimulus applied a little too early in ventricular systole to produce an extra contraction never induces premature auricular contraction as it should if it were spreading to the atria.

When ventricular fibrillation follows as in Figs. 2, 3, 4, the undisturbed and regular sequence of the P waves and of small pressure elevations of atrial origin shows that the stimulus acts directly on the ventricle.

The conclusion is reached that a single short strong localized shock applied during the latter portion of systole is directly responsible for the premature contraction or ventricular fibrillation.

11165 P

A New Reagent for Quantitative Estimation of Estrone.

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Most present colorimetric methods of assay[1-4] for the estrogenic hormones have two serious defects: a lack of specificity and the production of interfering colors with inactive impurities. Bachman,[5] however, has recently reported a modification of the Kober reaction which appears to be specific for estriol.

This report deals with an investigation of guaiacolsulfonic acid:

1 *a.* Kober, S., *Biochem. Z.*, 1931, **239**, 209—modified by: *b.* Cohen, S. L., and Marrian, G. F., *Biochem. J.*, 1934, **28**, 1603; *c.* Cartland, G. F., Meyer, R. K., Miller, L. E., and Rutz, M. H., *J. B. C.*, 1935, **109**, 213; *d.* Venning, E. H., Evelyn, K. A., Harkness, E. V., and Browne, J. S. L., *J. B. C.*, 1937, **120**, 225; *e.* Bachman, C., *J. B. C.*, 1939, **131**, 455.

2 Zimmerman, W., *Z. physiol. Chem.*, 1935, **233**, 257; 1936, **245**, 47.

3 Smulovitz, M. J., and Wylie, H. B., *J. Lab. and Clin. Med.*, 1935, **21**, 210.

4 Kober, S., *Biochem. J.*, 1938, **32**, 357.

5 Bachman, C., *J. B. C.*, 1939, **131**, 463.