gone a state of precocity indicative of exhaustion, where some tubules even contained the desquamated epithelial cells and debris only. This gives evidence of hypotrophy and reduction in size. In all cases, a marked increase in interstitial tissue is evidently accompanied by vascular tissue growth, so that the testes are dense and compact in transection, as contrasted to those in controls. One may postulate that this interstitial hypertrophy of the stroma is a direct result of an attempt on the part of the testes to counteract the excess of the gonadotropins, and that this may terminate in sterility if the treatment is prolonged. It is well-known that many of the sex hormones have the power to suppress the hypophyseal functions.[22]

Summary. (1) This paper reports for the first time the possibility of complete sex-reversal in the adult female *Xiphophorus helleri* by administration of gonadotropic (human chorionic) hormone from pregnancy urine. (2) Gonadotropin when administered to the adult males of this species increases not only the amount of interstitial stroma (Berkowitz), but hastens spermatogenesis and brings about an exhaustion effect simulating a condition of sterility. (3) There were no significant effects on the secondary sex characters of the adult males and unaffected females under this treatment.

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Exploration of a Method for Standardizing Hemorrhagic Shock.

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The urgency in developing standardized laboratory procedures for production of shock which will enable us to evaluate the efficiency of remedial measures with greater certainty is generally recognized. While no claim to such achievement can be made, we desire to report results which point to certain trails that ought to be explored more thoroughly.

Since the bulk of experimental work[1] strongly suggests that the progressive decline in arterial pressures is associated with reduced

22 Moore, C. R., and Price, D., *Am. J. Anat.*, 1932, **50**, 13.

1 Wiggers, C. J., *Physiol. Rev.*, 1942, **22**, 74.

circulating volume, it appeared probable that the greatest hope of developing a standard laboratory procedure for producing shock lay in reducing the blood volume by bleeding. However, our studies showed that the dangerous factor in shock, *viz.*, its irreversibility, is not as definitely related to the percentile reduction in blood volume as it is to the degree and duration of hypotension which eventuates. As is well known, stray dogs differ greatly in regard to the percentile reduction in blood volume withstood before they develop a protracted low arterial pressure. Consequently, attempts to standardize procedures for creation of experimental hemorrhagic shock which are based on withdrawal of definite volume percent of the estimated blood are doomed to disappointment.

We have sought to determine the degree and duration of posthemorrhagic hypotension which leads to a circulatory state that cannot be benefited more than temporarily by substantial infusions of the withdrawn blood (heparinized), and is generally accompanied by severe congestion, edema and hemorrhage of the upper intestinal mucosa.

Results. Fig. 1 shows graphically representative results selected from 75 experiments.

The data plotted in section A are from experiments in which mean arterial pressures were maintained by regulated bleeding between 46-60 mm Hg (average 50) for 1¾-4 hours. Irreversible shock never resulted. This emphasizes (1) the importance of distinguishing between posthemorrhagic hypotension and hemorrhagic shock, (2) the remarkable ability of tissues to withstand reduced blood

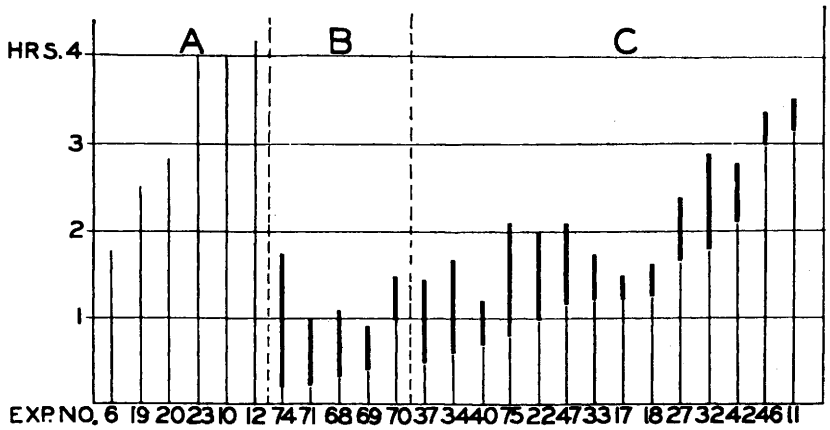

FIG. 1.

Diagrams showing intervals of moderate posthemorrhagic hypotension (narrow lines) and of extreme hypotension (broad lines) which proved effective and ineffective in causing irreversible shock. Discussion in text.

flow and (3) the impracticability of such a procedure for production of experimental shock.

The data plotted in Section B are from relatively fewer experiments in which an attempt was made to reduce arterial pressures to levels barely consistent with life (30-35 mm Hg). Regardless of the detailed methods of bleeding used, the compensatory reactions which come into play always result in brief preliminary periods of moderate hypotension before a drastic hypotension can be created. The tabular results show that a drastic hypotension (30-40 mm Hg) of an hour or more may not produce shock. In experiment 74, 105 minutes did not suffice.

The data of selected experiments plotted in Section C are from a group of animals in which varying intervals of moderate post-hemorrhagic hypotension ($60 > 40$), followed by severe hypotension ($30 < 40$) after additional bleeding led to irreversible shock according to the criteria outlined. It is apparent that this group includes conditions which, in comparison with those of Section B, would not be expected to induce shock (*e. g.*, Exp. 40, 17, 18). Such variation in resistance or susceptibility of stray animals to hypotensive states precludes any scientific attempt at statistical analysis. However, a survey of our experiments shows that when the initial period of moderate, and the subsequent period of drastic hypotension were 90 and 45 minutes respectively, all animals tested developed irreversible shock. When these intervals were reduced to 60 and 30 minutes respectively, some animals did and others did not develop shock. It is possible that tests on a larger number of animals may establish minimum effective periods of moderate and severe hypotension between those ranges which will produce irreversible shock with reasonable safety. We suggest for ourselves and others who have access to cheaper animals for such studies, trials of 60-60 minutes and 60-45 minutes as minimum effective periods.