was found scattered in the lumen, which appeared to have come from the cells lining it (Fig. 3). Mitosis could be observed, though less than the control. Other tissues which were observed to be affected after 48 hr. were more or less normal.

It appears from the present series of experiments that with concentrations of 24×10^{-5} gm. per c.c. of extracts of *G. populifolia* some organs of the developing embryos are affected. In the neural tissue there is an antero-posterior gradient in the degree of susceptibility, the brain being more affected than the neural tube, thereby showing that regions having a high rate of mitosis are more susceptible. This might also account for the lower degree of susceptibility of the somite where the rate of mitosis is not so great as in the two former tissues.

For the expansion of blastoderm tension is necessary, as reported by New[2]. This tension might have been reduced by breaking the intercellular connexions, resulting in a reduction of its size. A similar explanation has been given by Overton[3].

Engorgement of the blood vessels and an increase in the mesenchymal cells in the embryo proper might be due to the reasons suggested by Overton[3] and Waddington *et al.*[4].

The presence of mitosis after 24 hr. treatment and more or less its complete absence after 48 hr. with cellular derangement suggest that the extract of *G. populifolia* might have acted as a cytotoxic agent.

We are grateful to Dr. Subodh Mitra, director of this Centre, for his interest in the work, and to Dr. P. De for valuable discussions.

S. K. Brahma
N. Kundu

Department of Cell Research and Chemotherapy,
Chittaranjan National Cancer Research Centre,
37 S.P. Mookerji Road,
Calcutta 26.

[1] Jurand, A., *J. Embryol. Exp. Morph.*, **6**, 357 (1958).
[2] New, D. A. T., *J. Embryol. Exp. Morph.*, **7**, 146 (1959).
[3] Overton, J., *J. Exp. Zool.*, **139**, 329 (1958).
[4] Waddington, C. H., Feldman, M., and Perry, M. M., *Exp. Cell Res.*, Supp., **3**, 366 (1955).

Pathological Parthenogenesis in a Viviparous Toothcarp

In viviparous toothcarps parthenogenesis has been described in *Lebistes reticulatus*[1,2] and *Xiphophorus helleri*[2], while in this species also some cases of development of ovarial teratomas, caused by pathological parthenogenesis, are known[3]. In addition to these cases we observed a new one, namely in *Heterandria formosa*. It concerns a female isolated immediately after birth and consequently free from any contact with the male sex. After reaching a length of 27 mm. the virginal animal dropped 4 young fishes, all female. After this the animal became pregnant twice again, resulting in the birth of 6 and 10 young fishes, respectively, also exclusively female. For microscopic confirmation of the virginity the ovarial sperm chambers were examined, no spermia being observed. This finding ruled out self-fertilization, a phenomenon incidentally found in viviparous Cyprinodonts and explained by the presence of a bisexual gonad producing oocytes as well as spermia. As in the cases already described[2,3], an infection process caused by the Phycomycete *Ichthyophonus hoferi* was present in the cranial part of the ovary and in the adjacent regions. If an extensive process had been localized in the caudal parts of the ovary and near the junction between ovary and oviduct, birth would undoubtedly have been prevented. The young females appeared without an exception to be haploid.

The coincidence of the *Ichthyophonus* infection on one hand and the parthenogenetic development on the other justifies the assumption that the pathological process in this case had caused the parthenogenetic development. It could be assumed that the *Ichthyophonus* toxin provided the artificial stimulus for the development of the unfertilized oocytes.

Summarizing, we may conclude that this case forms an argument for my theory of pathological parthenogenesis[2,3]. It would certainly be advisable in any new cases of parthenogenesis to carry out a systematic examination for the possible presence of a pathological process in the ovary or in the immediate surroundings of this organ.

A. Stolk

Department of Histology,
Free University,
Amsterdam.

[1] Spurway, H., *Nature*, **171**, 437 (1953).
[2] Stolk, A., *Nature*, **181**, 1660 (1958).
[3] Stolk, A., thesis, Utrecht (1950); *Proc. Kon. Ned. Akad. Wet. Amsterdam, C* **56**, 28 (1953); *Nature*, **183**, 763 (1959).

Diagnostic Significance of Heterogeneous Lactic Dehydrogenases in Malignant Effusions

There is no laboratory test available for the differentiation of benign effusions from malignant exudates. Wroblewski and Wroblewski[1] reported in 1958 that, in malignancy, concentration of lactic dehydrogenase is frequently higher in effusions than in the corresponding sera. Nevertheless, the diagnostic value of these determinations is restricted since in about one-third of all cases investigated the amount of the lactic dehydrogenase in serum and effusion was the same[1]. Recent work on lactic dehydrogenase of tissues[2] and serum[3–5] indicates that this enzyme occurs in a number of molecular species which can be distinguished on account of their kinetics, antigenicity, inhibition by diphosphopyridine nucleotide analogues and electrophoretic mobility. In serum, starch block[3,4] and agar gel[5] electrophoresis and chromatography on diethylaminoethyl (*DEAE*) cellulose[6] showed the presence of at least four dehydrogenases, which are best labelled according to their electrophoretic mobility, namely, α_1, α_2, β and γ-lactic dehydrogenase. A specific increase in particular fractions has been observed in the following diseases: α_1 in myocardial infarction[3–6], $\alpha_1 + \alpha_2$ in muscular dystrophy[4] and γ in liver disease[5,6].

No information about the heterogeneity of lactic dehydrogenase in effusions is available. Since differences between malignant and benign effusions might occur, some thirty effusions were investigated by starch block electrophoretic separation[7] and determination of lactic dehydrogenase of each fraction using the optical test[8]. Effusions were centrifuged and the sediment discarded. 0·2 ml. serum or effusion was separated on starch block electrophoresis into four main fractions: α_1, α_2, β and γ. Lactic