It is well to provide means for opening the circuit automatically in case the cooling water is accidentally shut off to prevent the resistor from burning out. This can be done readily by connecting in series with the resistor a short piece of the same wire of which it is made and placing this piece so that it is within the stream of water issuing from the end of the resistor when the flow is normal but is out of the stream when the flow is reduced. By varying the angle which the emerging stream of water makes with the vertical and the distance of the piece of wire from the orifice any desired sensitivity can be had. With proper adjustment, this piece of wire will function as a fuse, burning out before the water has quite stopped flowing and thereby protecting the resistor.

No careful study of the durability of the resistors has been made. The following tests will, however, serve to show that their useful life is reasonably long. A fixed resistor made of No. 22 wire carried 20 amperes for 20 hours and then 25 amperes for 20 hours more. At the end of the test, it was in perfect condition and its resistance had changed by only about ½ per cent. A variable resistor carried 25 amperes for 20 hours and was operating satisfactorily at the end of the test. Other resistors have given satisfactory service in the laboratory for longer times, but no record of their performance has been kept.

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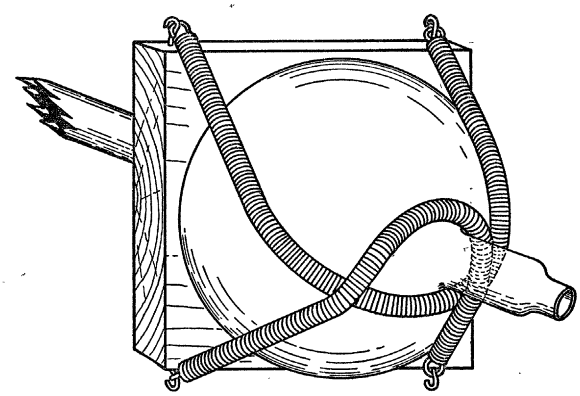
GLASS BLOWERS' HOLDER FOR LARGE FLASKS

THE holding of flasks, larger than a liter, in glass-blowing operations is usually a troublesome matter. The ordinary snap type of holder is not generally supplied in these sizes. An extremely simple device which the writer has found to be quite satisfactory is depicted in the accompanying sketch and requires only a few explanatory comments.

The flask is held against the base by two long U-shaped springs, which are looped over the neck of



the flask. The springs are those used to close screen doors and, where necessary, can be kept from contact with the glass by strips of asbestos paper. Round-bottom flasks are held more firmly if the base is provided with a slight hollow or if a thin cork ring is placed between the wooden base and the flask. A cork ring around the neck of an erlenmeyer flask will keep the springs from slipping down and permit such a flask to be held securely. For convenience in manipulating this holder it is desirable to have a detachable handle. A suitable handle and base is at hand in most laboratories in the form of old wooden funnel stands.

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SPECIAL ARTICLES

THE ORIGIN OF PIGMENT CELLS IN AMPHIBIA

IN the present study the methods of transplantation and explantation have been employed to decide between the divergent views that have been held regarding the origin of pigment cells in the amphibian embryo—whether from connective tissue, leucocytes, epidermis or ganglion crest. Embryos of *Amblystoma punctatum, A. tigrinum* and *A. mexicanum* (axolotl, both black and white) have been used in the experiments.

Explantation experiments: Pieces of the neural folds (anlagen of the ganglion crest) from the trunk region of *A. punctatum* neurulae were explanted into Holtfreter's solution. Melanophores appeared in every explant within six days (37 cases). Prospective flank ectoderm was explanted in the same way. In only one explant out of 20 did melanophores develop. It is probable that in this one case some ganglion crest cells were included at the time of operation. Flank ectoderm and mesoderm explanted at older stages up to stage 32 (non-motile embryo with elongated tail bud) gave negative results. When transplanted to the abdominal wall or explanted at stage 32 or older, melanophores invariably developed in the graft or explant. It is apparent that the flank tissues are not capable of forming melanophores until a much later stage than the ganglion crest cells. It is probable that the cells of the ganglion crest migrate into the flank at stage 32. Hence they are included in flank explants after this stage.

Defect experiments: The neural folds in the trunk

region of *A. tigrinum* and *A. punctatum* neurulae were removed bilaterally. In these cases melanophores, xanthophores and the dorsal fin failed to develop in the entire trunk of the embryo, while the head and tail were normally pigmented. This experiment is similar in results to a slightly different experiment by Holtfreter on Triton,[1] and affords striking evidence in favor of the ganglion crest origin of pigment cells.

Heteroplastic transplantations: Dorsal ectoderm of the trunk region with the adhering neural crest cells was transplanted between *A. punctatum* and *A. tigrinum* and from black axolotls to *A. punctatum*. All operations were performed on tail bud stages. Melanophores and xanthophores of the donor type appeared in all grafts from the axolotl to *A. punctatum* and from *A. tigrinum* to *A. punctatum*. The graft areas were easily distinguished from the host skin by the pigment differences which were maintained throughout larval life and after metamorphosis. In the grafts from *A. punctatum* to *A. tigrinum* melanophores of *A. punctatum* (donor) type developed in 9 out of 17 cases. In all cases they were replaced by melanophores of tigrinum (donor) type before metamorphosis. Flank or limb ectoderm and mesoderm transplanted between any of these species at tail bud stages acquires host pigmentation[2] in contrast to the results with dorsal ectoderm and neural crest cells. However, limbs transplanted to *A. punctatum* from *A. tigrinum* at older stages (stage 38, limb bud beginning to elongate) and from the axolotl at stage 34–37 develop the pigmentation as well as the size and form of the donor limb.

Experiments involving the white axolotl: The white axolotl develops melanophores only on the dorsal parts of the head and trunk, and none on the flank. Epidermal melanophores are not present, and the epidermal cells do not produce melanin as they do in pigmented species of Amblystoma. Flank ectoderm of *A. punctatum,* carefully cleaned of mesoderm cells, was transplanted to the flank of the white axolotl. In all cases subepidermal melanophores appeared under the grafted ectoderm. Later epidermal melanophores and xanthophores appeared in the graft region. Ectoderm from the abdomen was transplanted to the flank of the axolotl with exactly similar results. The melanophores induced by the grafted ectoderm are undoubtedly of axolotl (host) origin since they are similar in size and shape to those of the host and not to those of the donor. Furthermore flank or abdominal ectoderm of *A. punctatum* never gives rise to melanophores when explanted or transplanted to the abdomen.

[1] Holtfreter, *Roux' Archiv,* Bd. 127, 1933.

[2] Harrison, SCIENCE, Vol. 74, found similar results in limb transplantations.

It is clear that there are subepidermal cells in the white axolotl which acquire melanin and become typical melanophores when the non-pigmented axolotl epidermis is replaced by the pigmented epidermis of *A. punctatum.* Harrison (unpublished data) has found that if Amblystoma ectoderm is grafted to the limb area of *Triturus torosus* melanophores appear under the graft long before they develop in the other limb. The relationship here is the same as that between *Amblystoma punctatum* and the white axolotl.

It is suggested that in both of these cases the transplanted epidermis supplies some substance or substances to the underlying cells which they utilize in melanin formation. In the light of Bloch's experiments[3,4] the substance supplied might be dopa or dopa oxidase, which would then react with the one or the other present in the underlying cells to form melanin.

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HYPERIMMUNE ANTIPOLIOMYELITIC HORSE SERUM[1]

WITHIN recent years various workers[2–7] have demonstrated the successful hyperimmunization of horses to the poliomyelitic virus. That a potent antiviral serum can thus be produced in a refractory animal is again indicated in the following report.

Three horses were inoculated over a period of 2½ years with 10 per cent. poliomyelitic virus filtered through a Berkefeld N candle. Four different strains of virus were used, 2 monkey passage and 2 isolated from human cases during the New York epidemic of 1931.[8] Injections were given both intracutaneously and subcutaneously at bi-weekly intervals for the first 3 months, reduced later to weekly periods with a dosage of 200 cc, given by the combined intramuscular and subcutaneous routes. Trial bleedings were taken several times and the serums eventually neutralized the virus in dilutions of at least 1–200 or 1–300. The

[3] Bloch, *Zeit. exp. Med.,* Bd. 5, 1917.

[4] Bloch, *Centralbl. f. Haut-u. Geschlects-Krankheiten.* Bd. 8, 1923.

[1] Supported by a fellowship grant from the Eli Lilly Company.

[2] M. Neustaedter and E. J. Banzhaf, *Jour. Am. Med. Assn.,* 68: 1531–1533, 1917.

[3] R. W. Fairbrother, *Brit. Jour. Exper. Path.,* 11: 43–53, 1930.

[4] A. Pettit and B. Erber, *Bull. de l'Acad. de méd.,* 107: 455–459, 1932.

[5] E. R. Weyer, W. H. Park and E. J. Banzhaf, *Jour. Exper. Med.,* 53: 553–566, 1931.

[6] E. W. Schultz and L. P. Gebhardt, *Proc. Soc. Exper. Biol. and Med.,* 31: 260–261, 1933.

[7] M. Schaeffer, *Proc. Soc. Exper. Biol. and Med.,* 31: 1232–1234, 1934.

[8] One strain of virus was kindly supplied by the laboratory of the New York City Department of Health and the other (F1) by Dr. John Paul, of Yale University.