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An Experimental Study of Hensen's Node in the Chick Embryo.*

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In making chorio-allantoic transplantations of the entire area pellucida of chick blastoderms in the head-process stage, Willier found that only head structures and heart developed in the grafts and the more posterior trunk structures failed to appear.[1] Since the only differentiation was of parts lying anterior to Hensen's node, the suggestion was made that the node may normally have a rôle in the formation of trunk structures at this stage but for some unknown reason did not realize its capacities in the grafts.

It was then suggested by Dr. B. H. Willier that I make an experimental analysis of the rôle which Hensen's node has in the development of the embryo. The problem was attacked by isolating and transplanting to the chorio-allantoic membrane transverse levels of the area pellucida at different stages of development as follows: (1) the node and other levels of the area pellucida of blastoderms in a stage prior to the appearance of the head-process, (2) the node and other levels of blastoderms in the advanced head-process stage and (3) the node level of embryos ranging from one to 12 somite stages.

The grafts obtained from the transverse levels of the area pellucida of blastoderms, in which a head-process was not yet present, showed that the node level has a great capacity for differentiation and is essentially totipotent. The structures which develop include all levels of the brain, neural cord with ganglia and nerves, epiphysis, eye, notochord, heart, gut and liver, cartilages with attached muscles, integument with feather germs, and mesonephros. Other levels of the area pellucida had very little capacity for differentiation. The transverse level anterior to the node and the streak posterior to the node showed only a development of gut, cartilage, muscle and integument.

Grafts of the node level from blastoderms with a head-process and beginning head fold showed a development of hind brain, neural cord, ganglia and nerves, notochord, heart, gut and liver, cartilages with attached muscles, mesonephros, integument with feather germs,

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* This work was done at the University of Chicago under the direction of Professor B. H. Willier.

[1] Willier, B. H., *Anat. Rec.*, 1926, xxxiv, 158.

and adrenal gland. There is thus a diminishing capacity of the node for differentiation when compared with the node of the earlier stage since the more anterior structures such as fore brain, eye, epiphysis and hypophysis do not appear. However, these structures and others develop from the level anterior to the node which indicates that there has been a determination of some of the anterior parts. The primitive streak posterior to the node has no greater capacity for differentiation than previously. However, if the posterior part of the node is included with it, additional structures such as neural cord, mesonephros and adrenal gland develop.

Grafts from the node level of blastoderms having from one to 12 somites show that there is a further reduction in the capacity of the node. However, at all stages considered there develops neural cord with ganglia and nerves, notochord, cartilage and muscle, integument with feather germs; mesonephros, adrenal gland and gut.

The totipotency which the node exhibits, *i. e.,* the capacity to differentiate structures which represent different levels of the embryo, indicates that the node is a center of differentiation in the normally developing embryo. That it continues to be such a center in the later formation of the trunk may be seen by its continued although diminishing capacity for differentiation.

Since at all stages considered the node has the capacity to differentiate such structures as the neural tube and notochord, and since the primitive streak posterior to the node lacks this capacity, it is suggested that the primary rôle of the node is to form these axial structures. The factors which control the development of these structures have not yet been completely worked out but the preceding experiments would seem to indicate a definite correlation between the development of the central nervous system and the head-process. These experiments show that in stages just prior to the appearance of the head-process, the nervous system does not develop in grafts of either the level anterior or posterior to the node in spite of the fact that brain and neural cord form in these respective levels at a later period of development (Kopsch).[2] However, if the head-process is present or a part of the node is included in the grafted part, then the nervous system does develop from either level. A further indication of correlation is seen in the successfully grown grafts of the node from blastoderms of the various stages used. In 75% of such cases the notochord which develops from the head-process is observed to be in close proximity to the neural tube. It is consequently suggested that the head-process may be the first structure

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[2] Kopsch, F., *Z. f. Mikr. Anat. Forsch.,* 1927, viii, 512.

to differentiate and subsequently acts as an inducing agent for the differentiation of the nervous system both in the grafts and in the normally developing embryo.

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An Inquiry into the Cause of Congenital Absence of the Gall Bladder.

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Out of some 2700 cats examined during the last few years, the writer has noted 3 kittens (2 females and one male) in which no trace of the gall bladder could be found. In one of these there was a large empty *fossa vesicae felleae* suggesting that perhaps the gall bladder had once occupied it and then been absorbed. In the other 2 there was neither fossa nor any fibrous remainder to indicate that the gall bladder had ever been formed; so that we may assume that it either failed to develop in the embryo or had disappeared before birth. According to Golob,[1] known instances of a congenitally absent gall bladder in man do not exceed 40. In some species of animals, as in the pigeon, a biliary vesicle is formed embryonically but its duct subsequently atrophies, leaving a detached bladder that soon degenerates; in others, as the rat, it is doubtful whether any anlage ever forms (Scammon[2]). But in rats, according to McMaster,[3] the loss of the biliary reservoir is compensated for by an increased concentration of bile, the bilirubin concentration being 8 times that in the hepatic ducts of the mouse—an animal having a gall bladder. In this connection it is interesting to note that one of the 3 cases of congenital absence of the gall bladder in cats was found in a kitten which was being operated upon. When a sample of the hepatic bile from this animal was examined with a colorimeter it was seen to be 2.2 more concentrated than a sample of hepatic bile from a control kitten, suggesting that if the gall bladder is lost early enough in fetal life, the deficiency is partly overcome by a compensatory mechanism.

In the hope of finding a possible explanation for this anomaly the

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[1] Golob, Meyer, *J. Am. Med. Assn.*, 1927, lxxxix, 691.

[2] Scammon, R. E., *Anat. Rec.*, 1916, x, 543.

[3] McMaster, P. D., *J. Exp. Med.*, 1922, xxxv, 127.