

A STUDY OF THE AETIOLOGY OF CONGENITAL STENOSIS OF THE GUT

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Summary

EXPERIMENTS ARE REPORTED which show that congenital stenosis of the small bowel of the chicken embryo may be produced by interrupting the blood vessels to the gut as it lies in the umbilical hernia which normally exists between the 9th and 17th days of development. Stenosis, associated with a mesenteric defect, was found in 3 out of 5 embryos which survived 1-7 days after the operative procedure. This provides further evidence that ischaemia during development can cause stenosis of the bowel at birth.

Introduction

ORIGINALLY CONGENITAL ATRESIA and stenosis of the intestine were thought to be due to failure of the gut to recanalize after being occluded by epithelium¹. In the first few weeks of intrauterine life the gut is a hollow tube lined by epithelium and attached to the posterior abdominal wall by a mesentery. The gut then elongates rapidly and protrudes into an umbilical hernia between the 5th and 10th weeks of intrauterine life because the abdominal cavity is too small to contain it. After this period it returns to the enlarging abdomen and undergoes a counterclockwise 270° rotation to become distributed in the adult position. At the stage when the gut is elongating rapidly there is also epithelial proliferation, which may occlude the lumen, and it was postulated that failure of this solid core to recanalize was the cause of congenital stenosis or atresia of the small bowel. The oesophagus and duodenum definitely pass through this stage of occlusion, but it is now doubtful whether this ever occurs below the duodenum². It seems unlikely therefore that stenosis of the small bowel can be attributed to this mechanism. Further evidence against this theory was the discovery of lanugo, meconium, and squamous cells in the gut below a completely obstructed segment of small bowel in a newborn infant, suggesting that the gut had been patent at a stage when hair was present, when the fetus could swallow amniotic fluid, and while bile was being secreted from the liver³. These occur at a later time than that at which the gut was thought to be occluded by epithelial proliferation.

An alternative suggestion has been that atresia and stenosis can be caused by ischaemia in utero⁴. In the adult, for instance, stenosis of

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the small bowel has been described in more than 80 patients following obstruction in a hernia, usually inguinal⁵. In the human fetus an umbilical hernia is present in the early developmental stages and, since it contains loops of small bowel, a similar obstructive mechanism could cause congenital stenosis. There have been two previous experimental studies which support the theory that ischaemia could cause stenosis in utero. In one puppies were operated upon through a hysterotomy and webs, stenoses, and atresias were successfully produced by twisting the gut to form a volvulus⁶. In the other rectal stenosis and atresia were produced in rabbits by dividing the blood vessels and freeing the rectum in utero⁷. Both these procedures interfered with normal development at a late stage, when the natural umbilical hernia had closed.

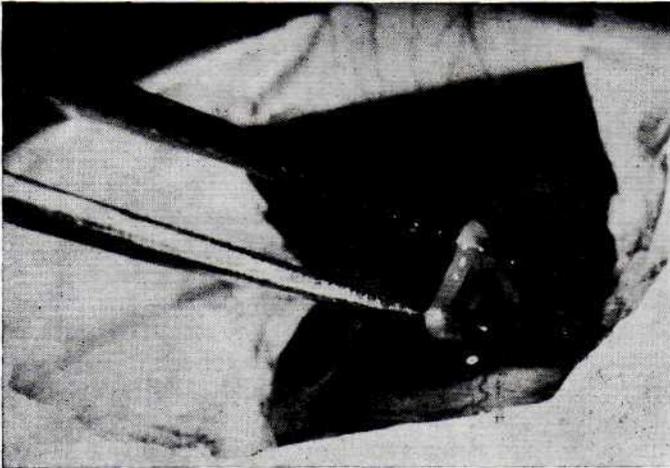


Fig. 1. A loop of small bowel is lifted up through the opening in the egg, demonstrating the mesenteric defects.

In the present study an attempt was made to cause ischaemia of the chick embryo gut early in its development, when a large umbilical hernia containing gut is still present.

Methods

Fertile eggs of the white Leghorn chicken (*Gallus gallus*) were incubated and then opened between the 9th and 17th days of development. Transillumination enabled the air bubble to be located, to see if the egg was fertile and whether the chick was alive. A 1-cm square hole was then cut in the shell with a drill and Carborundum bit. This opening was enlarged by chipping away the shell, and the underlying layers were separated so that the air bubble could be decompressed. With a sterile technique the shell membrane was reflected as a flap 1 cm wide

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and 1-2 cm long. The amnion was carefully divided without losing too much amniotic fluid. If the opening was in a favourable position the vitelline vessel would be in the operation field and could be followed to the umbilical hernia, where loops of small bowel were visible. Otherwise it was necessary, especially in the younger embryo, to lift a leg out and rotate the chick so that the gut appeared. In the 14-day-old chick the diameter of the small bowel is 0.8 mm^s. A small loop was lifted out gently and, with fine-pointed forceps, the mesentery was divided and then torn downwards, disrupting the blood vessels (Fig. 1). Before the gut was returned it was carefully examined to see that no



Fig. 2. In this postmortem specimen the marker passes through a small mesenteric defect and under a stenotic area of gut (arrowed). In this fresh specimen the proximally dilated segment is not as clearly seen as in the histological section.

damage to the wall had been sustained. The flap was replaced and a sheet of thin wax was placed over the opening in the shell to prevent loss of fluid. The eggs were replaced in the incubator and examined daily. None was allowed to survive to the stage when the chick started pecking through the shell on the 21st day.

Results

A total of 106 technically successful attempts were made to open the eggs. In approximately half, the loop of bowel was lifted out, but only in 38 were the mesenteric vessels disrupted, the remainder serving

as controls. Twenty embryos survived longer than 24 hours (maximum survival 7 days), of which 15 were controls and 5 had the vessels divided. The major cause of death was presumed to be blood loss occurring either when the membranes were opened or when the mesenteric vessels were torn. The embryos did not usually survive if there had been a large leak of amniotic fluid or if the flap had not sealed itself off. An area of stenosis was found in 3 of the 5 embryos in association with the defect in the mesentery where the vessels had originally been disrupted (Fig. 2). The degree of stenosis was sufficient to cause proximal dilatation (Fig. 3). No such lesions were found in the 15 control embryos.

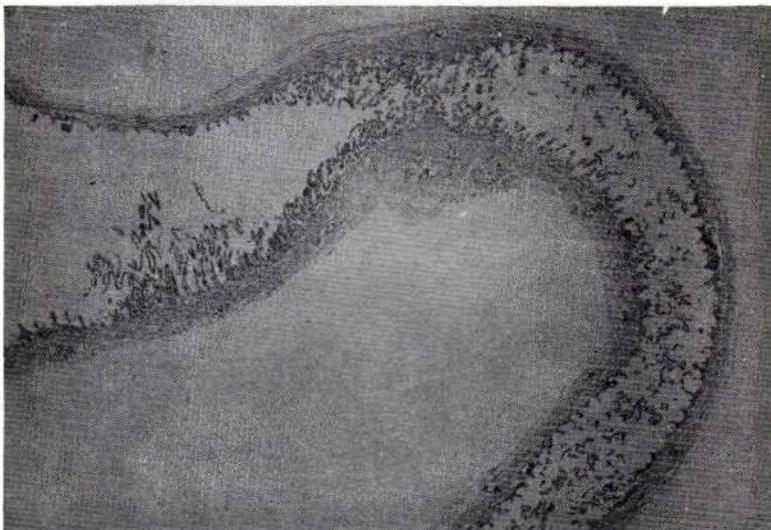


Fig. 3. A section through a stenotic area, showing dilated bowel on the left, the stenosis, and then normal bowel on the right.

Discussion

The present work confirms that congenital stenosis of the small bowel can be produced by interrupting the blood supply to the gut. Only small loops of bowel and small arteries were divided so that it was unlikely that atresia would follow. This technique differs from previous ones because it was carried out while the growing animal still had an umbilical hernia containing gut. In previous experiments the operation was performed at a later stage of development, when the umbilical hernia had closed. If ischaemia of the small bowel were to occur naturally it would presumably be caused by obstruction in the umbilical hernia. In the human this contains gut between the 5th and 10th weeks

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of intrauterine life. Small-bowel lesions could be explained in this way, but it is unlikely that rectal lesions could, since, as seen in the adult position, there is less possibility of the large bowel being in the hernial sac. But at the 5th week of development almost all of the bowel is in the hernia and has a long mesentery containing blood vessels. At this stage the arteries would be occluded if the rotational effects that the bowel normally undergoes were excessive. After this accident the large bowel would return to the pelvis but the ischaemia it had suffered might have destroyed the muscle wall sufficiently to cause atresia or stenosis.

This work, together with that of Stone *et al.*⁷ and of Barnard⁶, confirms that a basic mechanism which could cause congenital intestinal atresia and stenosis may be ischaemia due to interruption of the arterial blood supply to gut which might be caught in an umbilical hernia. Allowing for the different growth rates of dog, rabbit, and chicken compared with man, it seems likely that this usually occurs at an even earlier stage in intrauterine life than that at which the present experiments were performed.

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