rarely that conditioning could not be established during the session.

None of the subjects in these tests reported a history of any previous speech difficulties, and their nonfluencies outside the laboratory are of the type that occur in normal speech.

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References and Notes

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Effects on Palate Development of Mechanical Interference with the Fetal Environment

Abstract. Operations employing various combinations of amniotic fluid withdrawal and release of the embryo from enveloping membranes were performed just prior to the time of normal palate closure. Observations on subsequent palate development led to the hypothesis that fetal membranes can compress the fetus and cause cleft palate when pressure in the amniotic sac is reduced.

Puncturing the amniotic sac with a hypodermic needle just prior to closure of the secondary palate can cause cleft palate in fetal mice (*1*). To explain this effect, it has been suggested that amniotic fluid leaks out and hydrostatic pressure in the amniotic cavity is reduced, the uterus thus being allowed to compress the fetus. The fetus' head would then be pressed against its chest, the lower jaw and tongue being forced up toward the nasal septum. Since the palatine shelves have to force the tongue away from the nasal septum before they can come together and fuse (*2*), pressure on the tongue from below could prevent normal palate closure (*1*).

To test this hypothesis of uterine compression, several modifications of the amniotic sac puncture experiment have been tried (*3*). Thirty-four pregnant mice of heterogeneous origin were treated 13⅔ days postconception, and 25 were treated 14⅓ days postconception. The treatment consisted of exposing the uterus by an abdominal incision under ether anesthesia and subjecting all the embryos of one uterine horn to one of the following manipulations: (i) withdrawal of amniotic fluid; (ii) withdrawal of amniotic fluid and release of the embryo (with its amnion-yolk sac) by slitting the uterus lengthwise; (iii) release of the embryo from the uterus without removing any amniotic fluid; (iv) release of the embryo from both the uterus and amnion-yolk sac membranes. The other horn was left untouched or else was subjected to a manipulation other than the one used on the first horn. Amniotic fluid was removed with a No. 25 or No. 27 needle (these gave the same results, qualitatively) on a microsyringe, and the quantity withdrawn was approximately 1/50 ml (although the total loss varied because of leakage through the puncture hole). The females were reopened at 18⅓ days, the condition of the fetuses was recorded, and the living fetuses within their membranes were transferred to Bouin's fixative.

Table 1. Pooled results for fetuses treated at 13⅔ and 14⅓ days postconception and collected at 18⅓ days.

Treatment	No. of fetuses with		No. of fetuses resorbed
	Normal palate	Cleft palate	
Amniotic fluid withdrawn	24	23	104
Fluid withdrawn + release from uterus	34	18	89
Release from uterus	37	0	51
Release from all membranes	3	0	37
Control (to release from uterus)	13	2	29

The results are shown in Table 1. Release from the uterus obviously does not protect the fetus from developing a cleft palate. However, the cleft palates that develop in released fetuses are still dependent on loss of amniotic fluid, since the control fetuses released from the uterus did not develop clefts. Perhaps the amnion-yolk sac membranes can exert sufficient pressure to account for the cleft palates that developed in the released, amniotic fluid-deficient fetuses. Release of fetuses from both the uterus and fetal membranes would have provided a good test of this possibility, but the resorption rate was so high following this radical procedure that not enough fetuses could be collected to provide a reliable answer (Table 1).

To investigate further the cause of clefts in the released, fluid-deficient fetuses, 20 females were treated at 13⅔ days, and 64 living fetuses were collected at 14⅔ or 15⅓ days, postconception. It had been observed for fetuses collected at 18⅓ days that those subjected to a decrease in amniotic fluid were severely compressed if they had been left within the uterus, but were not noticeably compressed if they had been released from the uterus at the time of treatment. However, when collected at 14⅔ or 15⅓ days, both groups showed mild signs of compression, such as grooving of the body by the umbilical cord or limbs twisted out of position. Since the degree of fetal compression at the critical time for palate development is comparable in both groups, compression may be due to the amnion-yolk sac membranes in both cases. The radical compression caused by the uterus arises at a later time and is not necessarily involved in the development of the cleft palate. In Table 1, two cleft palates are listed for fetuses that were untreated except that the adjoining uterine horn had been incised. Under these circumstances there is some tendency for the fetuses to shift out of position toward the open horn (that is, the lateral pressure had decreased). Perhaps this is another expression of the sensitivity of palate development to changes in pressure relationships (*3*).

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Long-Term Effects of Tenotomy on Spinal Monosynaptic Response in the Cat

Abstract. Section of the Achilles tendon in the cat resulted in a shortening of the latent period and an increase in the amplitude of the spinal monosynaptic response from gastrocnemius nerves investigated 28 to 42 days after tenotomy. No change was observed in the course of post-tetanic potentiation.

Following tenotomy, the muscle is prevented from exerting effective tension and is also deprived of passive stretch. It is thus quite feasible to assume that as long as regrowth of the tendon does not take place, the muscle proprioceptor end-organs (both muscle spindles and tendon organs) are considerably restricted in function, since the natural stimuli for their activation —that is, active and passive tension— are practically eliminated by tenotomy.