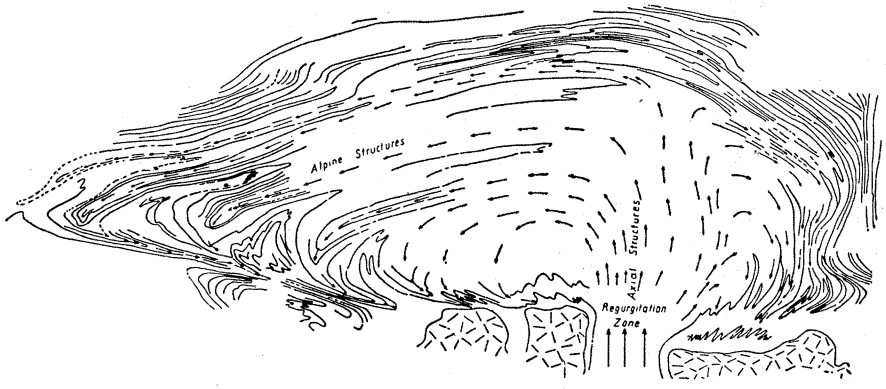

Fig. 2. Fold pattern of asymmetric orogen in cross section.

geodesists. These departures of the geoid from a rotational spheroid would still be in approximate fluid equilibrium and would not imply crustal loads of the order claimed by O'Keefe and his coauthors. They would cause a polar wandering with rheid adjustment until the rotation axis coincided with the maximum inertia axis. This also clears up the conflict reported by Munk (*6*) who found that, in the absence of such asymmetries, the pole should have migrated to Hawaii and, on accepted values for viscosity, should have arrived there within 10^5 years, whereas the palaeomagnetic data indicate that the pole has been moving slowly in the opposite direction and has not been far from its present position since the early Tertiary.

Crustal contraction has been axiomatic for so long that an expanding earth is still liable to rejection as irresponsible heresy. Originally the hypothesis of earth contraction stemmed from the idea of a cooling earth. Although this basis has long since been removed, and more recently reversed (*7*), geologists have still clung to contraction because of the false notion that the folding so universally observed in rocks necessarily indicates crustal shortening, even though I showed (*8*, p. 93) that complex folds of the type found in the hearts of orogens do not necessarily imply shortening. More recently (*4*, *5*) I have pointed out that at all but those shallow zones where the overburden load is less than the unconfined compressive strength, crustal extension yields a triaxial stress ellipsoid with all stresses positive, an environment which may produce boudins and tectonite fabrics and conjugate shears typical of orogenic zones, and further that even megastructures such as the orogens themselves do not necessarily imply crustal shortening. Figure 1 shows the fold pattern of the Malaspina Glacier, traced directly from Sharp (*9*), and Fig. 2 shows a typical cross-section of an asymmetric orogen such as the Alps, with a steep root zone and recumbent folds and nappes, with immense horizontal transport. However, Fig. 2 is drawn from Fig. 1 simply by up-ending it and reducing the ratio of the vertical and horizontal scales to simulate the flattening effect of gravity. Both the Malaspina Glacier in plan and the Alpine orogen in section exhibit similar features of fold and overthrust patterns, and in both the deformed material has developed complex tectonite fabrics, but whereas the lobe of the Malaspina Glacier has visibly increased in width tenfold during the generation of the folds, the Alpine orogen is imagined to have been reduced in width by a comparable order!

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The hypothesis of fluid equilibrium does not seem to be consistent with Carey's suggestion that the density in the mantle in the Southern Hemisphere is less than that in the Northern.

Since the density varies with depth, the two hemispheres can be compared only at corresponding depths—that is, on corresponding geopotential surfaces. But the condition of fluid equilibrium demands that the density on a given geopotential surface be constant. If it is not, then the pressure, which is the weight of the overlying mass, will be greater in those regions where the density is greater. This will cause the heavier fluid to move out laterally and underrun the lighter fluid everywhere, until perfect stratification is brought about.

Heiskanen and Vening Meinesz point this out, remarking on the subject of fluid equilibrium:

"The condition to be fulfilled is that all equipotential surfaces be at the same time surfaces of equal density" (*1*).

Jeffreys makes the same point (*2*). The result is very well established. It is impossible to have hydrostatic equilibrium at the same time that the density is less at a given depth in the Northern Hemisphere than in the Southern Hemisphere.

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25 August 1959

Instatement of Stuttering in Normally Fluent Individuals through Operant Procedures

Abstract. Normally fluent subjects were required to read from printed pages, and a recording was made of nonfluencies until a stable rate was established. A persistent shock was then introduced. Its cessation for a limited period was made contingent upon nonfluency. Chronic stuttering was instated and eliminated as a function of the change in stimulus.

In operant conditioning, the response is brought under stimulus control through the arrangement of consequences explicitly contingent upon the response (*1*). In a previous study, stuttering responses by chronic stutterers from a speech clinic were brought under stimulus control to a considerable extent by this procedure (*2*). When each stuttering response was followed by noxious stimuli, stuttering was attenuated, and when, with the same subjects, each stuttering response temporarily eliminated these stimuli, the stuttering rate rose.

Stuttering has often been considered an emotional blocking; it can, however, be regarded as a unit of verbal behavior (*3*); that is, breaks, pauses, repetitions, and other nonfluencies can be considered operant responses, having in common with other operants the characteristic of being controllable by

Table 1. Response slope for results reported by subject and experimenter, expressed in degrees.

Reporter	Base line period			Escape period			Extinction period			
	1	2	3	1	2	3	1	2	3	4
Session 1										
Subject	4	12	9	28	32	30	18	16	12	
Experimenter	4	10	8	26	31	27	20	13	14	
Session 2										
Subject	12	14	16	55	52	52	55	49	30	19
Experimenter	12	10	13	51	50	50	52	41	27	15

ensuing consequences. For the chronic stutterer, such nonfluencies may have been immediately followed by consequences which did not occur in connection with regular speech, thereby becoming isolated as response units. Some consequences, such as attention on the part of the listener, noninterruption, and the like, may increase the likelihood of occurrence—that is, may be reinforcing. If stuttering can develop in the manner indicated, then it should be possible to turn nonfluencies of normal subjects into chronic stuttering.

In the experiment previously reported, speech therapists defined moments of stuttering. In the experiment discussed here, the subject was trained prior to the experiment to press a microswitch with each nonfluency. Training continued until the experimenter considered the subject's responses valid. The instructions for this training, plus the instruction to read, the warning that there might be occasional shocks, and the advice that the subject could stop the experiment, constituted the experimenter's only communication with the subject. All recordings and scheduling involved automatic electrical equipment. Tape recordings were kept of the readings. Stuttering was operationally defined as any hesitation, stoppage, repetition, or prolongation in the rhythmic flow of vocal behavior.

The subject sat in a small room and read from printed pages. When the cumulative recorder depicted a smooth base line—that is, indicated regularity of response rate—conditions were changed. An electric shock (24 v, direct current) was pulsed at the rate of ten shocks per second into a 5:1 transformer with 2000-ohm resistance, connected to the secondary circuit; the room lights simultaneously flickered at the same rate. Each pressing of the microswitch by the subject turned off the shock and eliminated the flicker for 10 seconds. The subject could thus completely eliminate both, by responding at 10-second or shorter intervals.

Responses by one subject are recorded in Fig. 1. In the first session, during ordinary reading, nonfluent responses occurred at a fairly regular rate, as depicted in the low slope of the base-line period. At point A_1, the shock-flicker period began, each marker indicating onset of shock and flicker. The curve shows an increase in nonfluencies, leading to less frequent shock, as shown by less frequent markers. At point B_1, extinction began. The shock-flicker was disconnected, and the response rate dropped toward its base-line value.

In the second session, 2 days later, the slope of the base-line period was similar to that of the preceding session. At point A_2, the shock-flicker condition was reintroduced. Upon presentation of the first shock, the response rate rose so markedly that only one further shock, indicated by S_2, occurred during the entire period, when the response rate started to fall. At point B_2, the shock apparatus was disconnected. Since nonoccurrence of shock while the apparatus is connected results in a high rate of response, this rate of response is maintained when the apparatus is disconnected. Eventually, some slackening of response rate occurs, and since the consequences of a high rate of nonfluencies no longer differ from the consequences of a low rate, the response rate returns to base level.

A validity check of the subject's reports of stuttering was made independently from a tape recording, by a speech therapist who pressed a microswitch connected to the cumulative recorder at each response he defined as stuttering. Each test period was divided into three equal periods (except for the extinction period on the second day, which was divided into four parts), and slopes for the responses reported by the experimenter and by the subject were plotted and compared. The results are presented in Table 1, which indicates similar ordinal relationships.

The subject's speech outside the booth was normal. In a third trial, a week later, stuttering was controlled by the flickering light, without shock, and the sessions were discontinued for obvious reasons.

When questioned after the sessions, the subject of Fig. 1 reported that he had stuttered a great deal but ascribed this to his anxiety over his inability to read simple passages. He reported disliking the shock but said it was irrelevant to his behavior in the booth.

A second subject responded in the same way but did not return. A third subject did not initially show the same effect, but a change in procedure brought his stuttering responses under stimulus control: When the light was steady, there was no shock, but when the light flickered, a shock was occasionally presented. Response shut off the flicker for 10 seconds, after which it reappeared. The response rate rose rapidly, and periods of flickering dropped from 131 during the first 20 minutes to 3 for the next 12 minutes, when the session was terminated. A fourth subject exhibited nonfluencies so

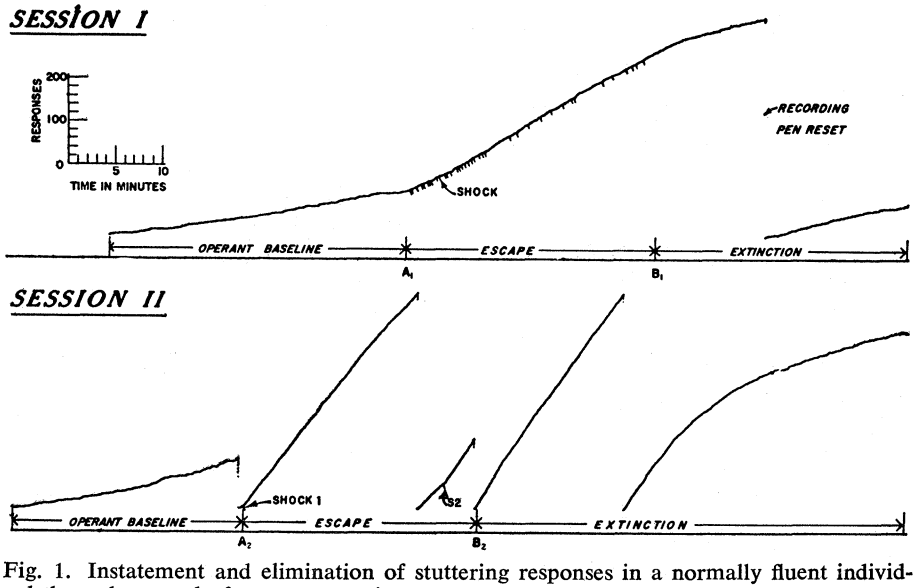

Fig. 1. Instatement and elimination of stuttering responses in a normally fluent individual through control of response-contingent consequences.

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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7 August 1959

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.