Table 1. Time course of coiling of fully turgid tendrils of wild cucumber when gassed with O_2 or with CO_2.

Gas	Increase in curvature (deg) after (min):			
	2.5	5	7.5	10
O_2	0	0	20 ± 31	20 ± 31
CO_2	0	75 ± 78	405 ± 150	555 ± 196

bers into which the gas was injected. Ethylene at 10 to 20 parts per million produced slight but definite curling; the weakness of the response, compared with the coiling induced by IAA (Figs. 1 and 2) makes it unlikely that the latter was in fact caused by ethylene produced in vivo under the influence of high concentrations of IAA.

In the experiments with CO_2 the fully turgid tendrils were placed in chambers through which streams of gas (bubbled first through water) were passed. Table 1 shows that, whereas O_2 was almost totally ineffective, CO_2 elicited vigorous curling that started within 5 minutes—close to the average time for response to tactile stimuli. It is likely that this effect of CO_2 is intimately related to its action as a remarkably efficient and quick-acting promoter of extension growth (*6*).

The strong coiling in response to symmetrically applied IAA and CO_2 reported here argues against Boresch's theory that an asymmetric distribution of auxin, effected by contact, causes the thigmotropic response. [The possibility that CO_2 brings about an asymmetric release of auxin within the tissues may be discounted, since growth induced by CO_2 and by IAA differs widely in susceptibility to inhibitors (*6*).] I therefore propose that the thigmotropic response is due, not to an asymmetric distribution of auxin, but to an asymmetric response to auxin. The situation may closely parallel that proposed for the various tissues of the pea epicotyl (*7*). Under the influence of the very low concentrations of auxin present in an unstimulated tendril, the latter grows more or less straight. Higher concentrations, however, produce curvature, either because of differences in the shape of the growth-concentration curves for the tissues on the two sides of the tendril, or because the maximum capacity for growth on the two sides differs.

I suggest the following mechanism for thigmotropism: The contact stimulus gives rise to an action potential in the tendril [observed by Umrath (*8*)]. According to modern theory, this action potential is associated with changed permeability of cell membranes to ions, and a consequent redistribution of ions between, for instance, the vacuole and the cytoplasm. The changed ionic environment brings about an increase in free auxin (by either releasing bound auxin or promoting synthesis) to which the two sides of the tendril respond asymmetrically.

Two of my findings possibly weigh against this proposal: the relatively slow response of the tendrils to external auxin (though this is probably due to slow penetration, as I have noted), and the fact that in its early stages the response to auxin in the floating experiments often did not resemble in form contact coiling. An alternative suggestion is that the auxin-induced coiling represents not the primary swift response to touch—which is soon reversed by the straightening reaction (see *1*) and which may involve a contractile protein (*2*)—but the continued curvature under a sustained contact stimulus which leads to a permanent grasp of the support. On the basis of the experiment shown in Fig. 2, I suggest that this permanent grasp (as well as the coiling that subsequently occurs along the entire length of the tendril, bringing the stem closer to the support) involves a supply of auxin translocated basipetally from the point of contact.

It is noteworthy that the effect of CO_2 resembles the tactile response both in form and in speed. Since CO_2 action is comparable to that of IAA in that it produces a decrease in wall pressure (*9*), this interesting finding appears to favor the first of my two proposals.

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Hydraulic Compression of Mice to 166 Atmospheres

Abstract. *Hydraulic uniform compression elicited tremors, uncoordinated limb movements, and tonic convulsions in liquid-breathing mice at pressures ranging from 50 to 100 atmospheres. Such abnormal muscular activity was observed neither in control animals nor in mice caudally to a spinal transection. Uniform compression of isolated preparations of mouse muscle in saline failed to contract at pressures up to 200 atmospheres.*

The effects of pressure on marine organisms, isolated organs, and cells have been studied since 1884 (*1*), and a concise review of pressure physiology has recently been written by Fenn (*2*). The response to uniform compression of gas-breathing animals may be modified or obscured by the pharmacological properties of compressed gases, and pressure effects in intact mammals have only been postulated thus far on the basis of indirect evidence and extrapolation (*3*, *4*). Since mice with liquid-filled airspaces have been reported to survive submerged in hyperbarically oxygenated salt solutions (*5*) and in a synthetic liquid equilibrated with oxygen at atmospheric pressure (*6*), it has become possible to study the effects of increased ambient pressure per se in mammals directly. One can observe the behavior of intact mammals subjected to great hydrostatic pressures and compare it with the behavior of control animals at normal atmospheric pressure under otherwise identical conditions. We report here the preliminary results of a series of such experiments in which adult Swiss mice were used.

The mice were placed in a small (150-ml) pressure chamber consisting of a thick-walled perspex cylinder sealed between two circular aluminum plates which are held together by steel bolts. The mouse enters into the chamber through a hole in the top plate which is then closed by means of a plug incorporating a valve. Gas or

liquid is introduced into the chamber from the bottom through a three-way valve, one end of which is connected with a hydraulic pump (*7*). The chamber containing the mouse is flushed thoroughly with oxygen and then flooded with oxygen-equilibrated FX-80 fluorocarbon liquid (*8*), replacing gas which escapes through the vent. Once the system is completely filled with liquid, the vent-valve is closed and the pressure in the chamber is raised by activating the hydraulic pump. The measured partial pressures of oxygen in the fluorocarbon liquid were 700 mm-Hg or less; the temperature of the liquid ranged from 17° to 25°C. Since the solubility of oxygen in FX-80 fluorocarbon liquid (at standard temperature, pressure and density) is approximately $9 \times 10^{-4} cm^3/cm^3$ per millimeter of Hg at 25°C, the liquid contained, roughly, 60 percent (by volume) of oxygen at the beginning of each experiment.

Eight control mice were kept at normal ambient pressure in the liquid-filled pressure chamber up to 1 hour; pressure experiments were completed within ½ hour.

Forty liquid-breathing mice were subjected to hydrostatic pressures up to 166 atm. The first pressure effect observed in most animals was trembling of the limbs, and voluntary movements became jerky and uncoordinated. These phenomena occurred at pressures ranging from 50 to 80 atm. When the pressure was increased further, the mouse was seen to clench its paws tightly, and almost immediately afterward the neck bent sharply and the upper portion of the body arched so that the lower jaw almost touched the chest. The hindlimbs and forelimbs stretched caudally in full extension. After a few seconds, the limbs bent slightly again and the forepaws seemed to be drawn together. Respiration ceased and the mouse appeared to be dead. These tonic convulsions occurred at pressures ranging from 50 to 100 atm. If the pressure is kept constant when the mice develop these muscle spasms, some of the animals recover: the muscles seem to relax slowly and gradually, and after approximately 1 minute the mouse appears to be normal again. Raising the pressure by another 5 atm or so provokes the next, often irreversible, tonic convulsion. These tremors, uncoordinated limb movements, and tonic convulsions were not observed in control animals.

Three mice were compressed with helium to 100 atm. Tremors and uncoordinated movements, but no tonic convulsions, were observed. Five mice were precooled in a water bath until the rectal temperature had dropped to approximately 20°C. When the animals were then compressed with helium, tonic convulsions, similar to the ones observed in hydraulically compressed mice, occurred in three animals at pressures ranging from 69 to 86 atm. Three fresh preparations of mouse hindlimbs failed to contract when hydraulically compressed in Ringer solution at 20°C up to 200 atm, while the muscles readily contracted after electrical stimulation, before as well as after compression. In three mice with transected spinal cords, typical pressure-contractions occurred cranially to the lesions, whereas the muscles caudally to the lesions remained flaccid.

One fluorocarbon-breathing mouse survived uniform compression to 100 atm for 30 seconds; it was decompressed in 3 seconds, resumed air-breathing, and was alive and in apparent good health 1 month after the experiment. Such a rate of decompression is equivalent to surfacing from 3000 feet (1000 m) underwater at a vertical speed of 700 miles/hr (1200 km/hr) without signs of decompression sickness. This confirms tolerance to rapid decompression of fluorocarbon-breathing mice reported earlier (*9*).

Kylstra *et al.* (*5*) have previously described reversible tonic convulsions in hyperbarically oxygenated saline-breathing mice at pressures between 20 and 70 atm and survival of a liquid-breathing mouse at 160 atm, but the inspired gas tensions were not adequately controlled in these experiments. Brauer and co-workers (*10*) recorded abnormal electroencephalographic tracings in monkeys that were compressed with mixtures of helium and oxygen at 45 to 47 atm. MacInnis *et al.* (*11*) saw tremors in mice breathing mixtures of helium and oxygen at pressures between 75 and 80 atm, but no convulsions at pressures up to 122 atm. It is possible that pharmacological properties of compressed helium inhibited convulsions in their animals. Moreover, the temperature of the chamber was carefully controlled and maintained at approximately 30°C, and compression was carried out very slowly so that maximum pressures were reached only after 3 hours. In our experiments maximum pressures were reached within 10 minutes; the body temperature of the mice dropped rapidly and approached the temperature of the liquid environment. Pressure-induced biological phenomena are temperature-dependent (*2*), and it seems likely that convulsions would occur at higher pressures in mammals with a normal body temperature than in hypothermic animals, as suggested by our observations in mice compressed in helium. Unfortunately, attempts to keep mice alive while submerged in hyperbarically oxygenated fluorocarbon, at temperatures greater than 30°C, have failed. Retention of carbon dioxide, resulting from inadequate alveolar ventilation and diffusion limitations (*12*), would seem to be the limiting factor.

Our observations in hydraulically compressed fluorocarbon - breathing mice clearly reveal profound biological changes brought about by uniform compression of intact mammals. The pressure-induced musclar activity seems to reflect changes in function of the central nervous system. Our results tend to support the conclusion reached by Miller and co-workers (*4*) that mammalian tolerance to high environmental pressures is probably limited to pressure per se rather than by the pharmacological effects of inert gases, such as helium and neon.

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