Table 1. Osmolality of protoplasm and vacuolar fluid in milliosmoles.

Vacuole	Medium	Protoplasm	Medium
31	5	109	3
33		116	
32	10	109	
32	2	110	9
24	1	116	
27	4	101	
38		94	10
26		87	10
38		106	
35	10	79	
36	8	92	
		88	
	Averages		
32	6	101	8

The osmolality of the contents of mature contractile vacuoles and medium surrounding the amoeba at the time of the formation of the vacuole are given in Table 1. The average concentration of the vacuolar fluid was 32 mosm and that of the medium was 6 mosm. The osmolality of protoplasm samples is also given in Table 1. The average concentration of protoplasm was 101 mosm. These samples were not obtained from amoebae which had already been punctured because of the possible changes that might have taken place in the protoplasm following the first puncture. In two cases only, two punctures were performed on the same amoeba. In one the vacuolar fluid was 36, the protoplasm 92, and the medium 8 mosm. In the other food vacuole and protoplasm samples were obtained, the concentrations being 91 and 88 mosm, respectively.

The finding that the vacuolar fluid is distinctly hypoosmotic to the protoplasm is in agreement with the hypothesis that the vacuole serves in osmoregulation. Until a few years ago it was believed that water was secreted actively into the vacuole (*5*). If this were the case the vacuolar fluid would be hypoosmotic to the protoplasm, as we have found it to be. Active water transport, however, is highly improbable on thermodynamic grounds (*6*) as well as from a comparative physiological point of view (*7*). Another possible mechanism for the formation of the vacuolar fluid was that a solute, possibly a nitrogenous waste product, was secreted into the vacuole with the water following passively. If this were the case, however, we would expect the fluid in the vacuole to be isoosmotic to the protoplasm, which is in disagreement with our findings. A third possible mechanism is that the formation of the fluid takes place in two steps: (i) solute is secreted with water following passively, and (ii) the solute is reabsorbed leaving a dilute solution behind. If this latter explanation is correct, it is possible that the secretion of the fluid takes place in the small vesicles surrounding the vacuole and that reabsorption of the solute takes place in the vacuole itself. According to Mercer (*8*) the mature vacuole is surrounded by numerous mitochondria which are not yet present in the small growing vacuole. A vast number of tiny vacuoles or vesicles are found between the layer of mitochondria and the membrane. These vacuoles presumably burst into the main vacuole.

It has been shown by Dunham and Child (*9*) that in *Tetrahymena* sodium is extruded against a chemical concentration gradient while potassium is retained inside the cell against a gradient. The intracellular chloride concentration is much lower than the cation concentration. In dilute solutions both cations, particularly K, are retained in higher concentrations than in the medium. These authors suggest that they are retained by a system of internal binding sites with a saturation level.

The membrane of the contractile vacuole is, according to Mercer (*8*), identical in structure to the plasma membrane. If they also function alike, we might expect the vacuolar membrane to reabsorb K and extrude Na. Consequently, when these possibilities are investigated, it is most likely that we will find a lower K concentration and a higher Na concentration in the vacuole than in the protoplasm (*10*).

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References and Notes

1. D. L. Hopkins, *Biol. Bull.* **90**, 158 (1946); W. B. Cosgrove and R. G. Kessel, *J. Protozool.* **5**, 296 (1958); J. Green, *Nature* **179**, 432 (1957).
2. S. Lövtrup and A. Pigon, *Compt. Rend. Trav. Lab. Carlsberg* **28**, 1 (1951).
3. The technique of placing a collodion film over the amoeba originated with Max Cerames-Vivas of this laboratory.
4. J. A. Ramsay and R. H. Brown, *J. Sci. Instr.* **32**, 372 (1955).
5. A. Krogh, *Osmotic Regulation in Aquatic Animals* (Cambridge Univ. Press, London, 1939); J. A. Kitching, *Symp. Soc. Exptl. Biol.* **8**(1954), 63 (1954).
6. W. A. Brodsky, W. S. Rehm, W. H. Dennis, D. G. Miller, *Science* **121**, 302 (1955).
7. B. Schmidt-Nielsen, *Am. Heart J.* **62**, 579 (1961).
8. E. H. Mercer, *Proc. Roy. Soc. (London)* **B150**, 216 (1959).
9. P. B. Dunham and F. M. Child, *Biol. Bull.* **121**, 129 (1961).
10. Supported by Public Health Service grant A-1956 and a grant-in-aid from the American Heart Association.

8 November 1962

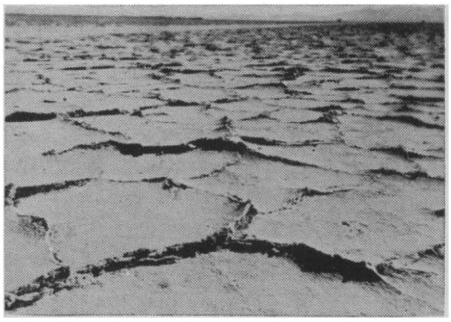
Polygonal Fracture and Fold Systems in the Salt Crust, Great Salt Lake Desert, Utah

Abstract. *Small folds and fractures (thrusts) up to 50 feet apart that have produced polygonal patterns in the Bonneville salt crust, western Utah, are believed to be caused by the annual expansion of the salt crust due to the growth of salt crystals within the salt layer plus the effect of increased summer temperature. It is suggested that these strain systems are caused by positive (compressional) isotropic planar stresses developed within the salt layers of the salt crust.*

Periodic evaporation of a veneer of surficial saline water and seasonal capillary upflow and evaporation of water have caused precipitation of a crust of mineral salts, chiefly sodium chloride, over the well-known Bonneville Salt Flat racing course near Wendover, Utah. The surficial salt crust developed annually (¼ to 1 inch) and the upper layer of the perennial salt unit (2 inches) cover an area of more than 200 square miles. They overlie the main stratified salt body which ranges up to several feet in thickness (10 to 25 inches on the racing course) and which overlies unconsolidated, saline-water-saturated, thin-bedded clays and granular gypsum deposits.

Precipitation and growth of salt crystals in the upper perennial salt layer and in the surficial annual salt crust plus the effect of increased summer temperature cause essentially planar isotropic positive (compressional) stresses to develop within the tabular shaped salt units. This stress has caused the upper 2-inch layer of perennial salt to fracture (thrust) polygonally, and, over a period of time, to develop the reticulate or polygonal strain systems illustrated in Fig. 1. Polygonal fold systems develop most frequently in the annual thin (¼ to 1 inch) salt crust (Fig. 2).

The development of the polygonal strain systems does not take place simultaneously. The fractures are usually initiated at specific points on the salt layer and grow laterally at about 120 degrees from the points of inception. The points of initial strain development are believed to be determined by points of weakness in the salt layer, and they tend to be equally spaced over the salt layers. This spacing, it is suggested, is largely controlled or determined by points or areas of relative weakness within the salt layer, which fall in or near the zones where



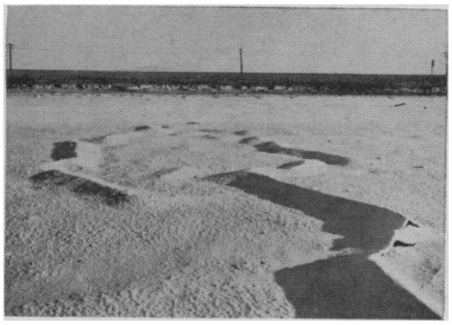


Fig. 1 (left). Polygonal or reticulate fracture systems in the salt crust. Fig. 2 (right). Polygonal fold systems in the annual salt crust.

the theoretical hexagonal strain systems would have developed had all theoretical conditions prevailed. The distance between the points of fracture inception, which eventually determines the size of the intervening polygonal cell, is largely determined by the strength and thickness of the tabular body; the greater the strength and thickness the larger the cell.

The strain system consisting of two fractures joining at about 120 degrees (biradial) shown in Fig. 3*G* tends to develop during the earliest stages of deformation. The two fractures grow radially from points or zones of inception. This two-fracture system may be modified at a later time by a third fracture which may grow out at about 120 degrees from the point or zone of intersection of the two fractures, thus developing the three-fracture system or triradial system illustrated in Fig. 3*H*. The triradial system may also develop by simultaneous radial growth of three fractures.

When an out-growing fracture approaches an existing one of an adjacent system at angles less than 90 degrees it will veer into and intersect it at about 90 degrees, forming the right angle or orthogonal system illustrated in Fig. 3*F*. If marked inhomogeneities exist in the paths of growing fractures the fractures may bifurcate at the points of weakness, forming triradial or closely related systems. Furthermore, along some of the longer straight fractures, subsequent smaller fractures may grow out at right angles, also forming the specific orthogonal type.

Sinuous to arcuate systems tend to develop along linear belts of weakness or other inhomogeneities in the salt crust, such as thinner or thicker belts or linear zones of greater porosity. Although in this system maximum thrust displacement occurs at about right angles to individual segments of the sinuous fracture, there is a tendency for minor fractures to develop at the points of maximum curvature of the thrust zone as illustrated by the dashed lines in Fig. 3, *K* and *L*. The pattern of this system may be angular (Fig. 3*L*) or curved (Fig. 3*K*). Weathering of the initially angular parts of an overthrusted cell margin may cause it to become rounded or curved. The angular pattern in a sinuous fold system is illustrated in the right half of Fig. 2.

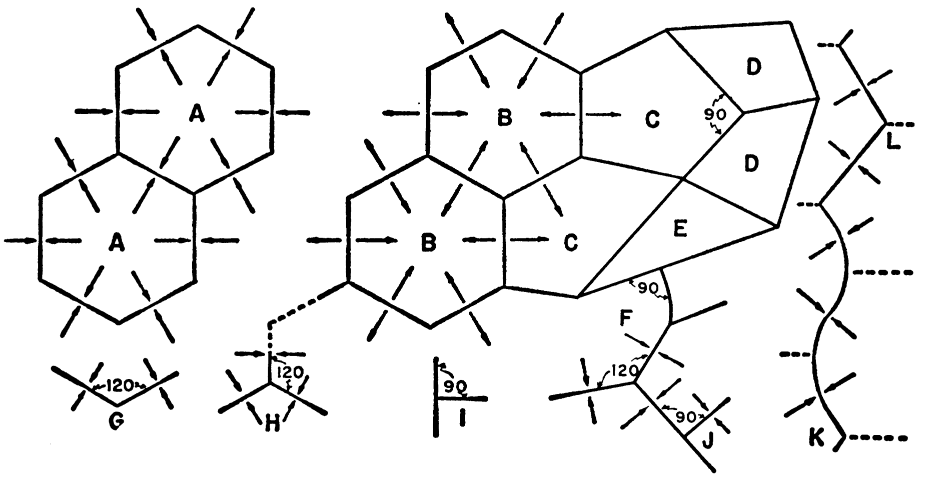


Fig. 3. Various strain systems related to polygonal yielding.

The relative intensity or degree of deformation within the polygonal systems is greatest at the points of maximum curvature of the sinuous and biradial structures and at the centers of the triradial structures (Fig. 3).

As previously noted, the size of the cell is largely determined by the thickness and strength of the tabular-shaped strained solid body. Variations from the theoretical hexagonal pattern, as noted above, are probably related to inhomogeneities in composition and the extent of strain development within the salt crust. Pentagonal, rectangular, and triangular cells may eventually develop along with the hexagonal cells, but generally the cells with fewer than six sides increase in abundance toward the zone of termination of the salt crust or in other areas of marked inhomogeneities (Fig. 3, *C–E*).

Polygonal fold systems develop most frequently in the thinner annually developed salt crust (Fig. 2). They are initiated by doming at points of weakness which tend to be equally spaced. From these domes two or three anticlinal folds may grow radially at about 120 degrees. The order of formation or paragenesis of the biradial, triradial, orthogonal, sinuous, and polygonal fold systems is essentially identical with that of the fracture systems.

The theory of polygonal deformation involves considerations of the manner of failure of infinite, homogeneous, tabular shaped solid bodies which are subjected to isotropic compressional stresses of sufficient intensity to cause failure by either fracturing or folding. Under these theoretical conditions, it is believed that a hexagonal strain system will develop simultaneously (Fig. 3*A*). This strain system provides a maximum release of stress, per unit volume, with a minimum amount of strain. Although the strained salt crusts are not infinite in extent or homogeneous in composition, nor have they been subjected to strictly isotropic compressional stresses, it is believed that the conditions related to their failure approach those of the theoretical conditions noted above.

The individual tabular shaped units of a physically or dynamically layered sphere may, in a sense, be considered infinite in extent since they do not terminate. Specific layers of such a sphere, if caused to differentially expand or contract sufficiently to cause deformation, will fail polygonally (*1*).

Desiccation of wet, tabular shaped clay bodies at room temperature and field observations of similar phenomena indicate that the polygonal fracture systems apparently caused by negative (tensile) stress (Fig. 3*B*) develop in the clay layers in essentially the same order as the thrust and fold systems of the salt crust. The ratio of layer thickness to cell width varies considerably. Polygonal cells developed in 3-mm-thick clay layers, drying at room temperature, had a thickness-to-width ratio of 1:12 (*2*).

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References and Notes

1. F. H. Norton, *Refractories* (McGraw-Hill, New York, 1949), p. 426.
2. Investigation supported by a grant from the University of Utah Research fund.

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Hypothalamic Temperature in the Cat during Feeding and Sleep

Abstract. *Anterior hypothalamic temperature is reported for the unanesthetized cat resting at an air temperature of 22° to 25°C during the ingestion of cold or warm liquids, and during sleep. Drinking cold (5°C) milk resulted in an immediate depression of hypothalamic temperature and a period of peripheral vasodilation in the ear and forepaw foot and toe pads, followed by a drop in rectal temperature. Drinking warm (body temperature) milk did not bring about these changes. Hypothalamic temperature during sleep is lower by approximately 0.5°C and is characterized by widely varying, slow-frequency oscillations, compared to the higher, more precisely controlled temperature seen when the animal is awake.*

The anterior hypothalamus in several species of homoiotherms has been identified as a central nervous system site intimately involved in body temperature regulation. Not only has this area been shown to play a neural integrating role for the maintenance of biothermal control (*1*), but the temperature level of this portion of the diencephalon has also been shown to be one of the inputs for internal thermal control mechanisms (*2*).

Anterior hypothalamic temperature was measured in the cat by a small thermistor bead (VECO 32A7) housed in the tip of a 23-gauge stainless steel needle chronically implanted in the region of the supraoptic nuclear groups, and recorded on a single-point, calibrated, constantly recording Honeywell potentiometer. Rectal temperature was measured by a thermistor probe inserted 10 cm into the lower colon. Skin and extremity temperatures were measured with 36-gauge copper-constantan thermocouples attached to the skin by a single layer of thin plastic tape and recorded on a multiple-point, continuously recording potentiometer. All measurements were made on unanesthetized cats resting in a plastic wire mesh hammock. None of the animals tested in this portion of the study demonstrated any thermoregulatory consequences of this type of restraint. Ambient temperature during these measurements was between 23° and 25°C.

Figure 1*A* shows the pattern of anterior hypothalamic temperature in an awake, attentive animal through the 12 minutes of measurement. The 0.1°C fluctuations seen in this record have been reported earlier for this species under similar testing conditions (*3*). In the present study, after the animal became accustomed to the restraining procedures it would spontaneously sleep if left unattended for longer than an hour. Hypothalamic temperatures during these sleeping periods are reported in Fig. 1, *B* and *D* and the first half of tracing *C*. Rectal or room temperature during these periods did not change.

During sleep, hypothalamic temperature became more labile (tracings *B* and *D* in Fig. 1) and fell to a lower level than when the animal was awake, as shown in Fig. 1*C*. As the cat was awakened (arrow in Fig. 1*C*), anterior hypothalamic temperature increased and assumed a greater stability than the pattern recorded during sleep (compare the first and last half of tracing *C* in Fig. 1). The changes in this temperature pattern were not attributable simply to the dependent head position, since these altered patterns of hypothalamic temperature did not develop until after the animal had assumed a sleeping position for a number of minutes. Furthermore, head dependency per se associated with feeding (see below) did not result in modified hypothalamic temperature.

Considering the vascularization in this brain area and the rapidity with which hypothalamic temperature changed under these conditions, it would seem more likely that convective (local blood flow) rather than local metabolic thermal influences are responsible for these patterns. More direct measurements of blood flow in these areas would be interesting in relationship to the recently demonstrated changes in neural activity with sleep (*4*) and in view of the possibility that blood flow within the brain may show some degree of local control.

Local temperature changes in the anterior hypothalamus have been reported to bring about peripheral vascular adjustments appropriate to whole body thermal exposure (*5, 6*). Since drinking volumes of cold liquid has been used to readjust internal body temperatures in humans in which more direct local control is unfeasible, it was of interest to examine the thermoregulatory consequences of ingested cold liquids in cats with thermistors chronically implanted in the anterior hypothalamus.

Figure 2 (top) indicates that coincident with the beginning of drinking milk at 5°C, anterior hypothalamic

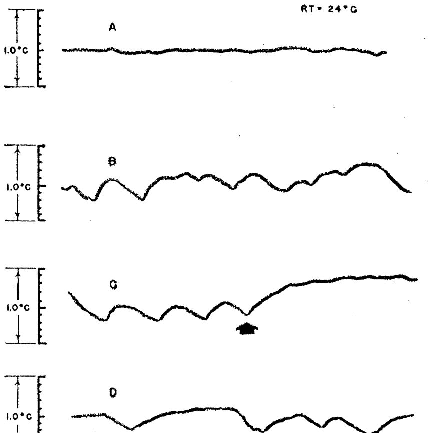

Fig. 1. The hypothalamic temperature shown in tracing *A* was recorded while the animal was awake and attentive to its environment; records *B* and *D* and the first half of tracing *C* were obtained while the same animal was asleep. The arrow in record *C* marks the point at which the sleeping animal was awakened. *TR* shows rectal and *RT* indicates room temperature during these measurements.