port is the basis of this increase in sodium outflow. The effect might be related to the marked tendency of epinephrine to produce depolarization of the membrane potential as observed in the sinus venosus[3,4]. It seems improbable that changes of the action potential produced by epinephrine are responsible for the increase in sodium outflow, since the beating rate of the preparation does not affect the time constant of the slow component of the loss curve.

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Piezoelectric Effect in Bone

There is convincing evidence that bone has an orderly morphological and microscopic structure. This evidence is derived mainly from electron microscopy, ordinary histological preparations, and microradiography[1]. Such a structure, consisting essentially of apatite crystals embedded in an organic matrix, might be expected to exhibit piezoelectric properties, as in the case of many other multicrystalline structures.

This has been conclusively demonstrated by Fukada and Yasuda[2], who measured the piezoelectric constants of small specimens of compact bone cut from human and ox femurs. These authors conclude from the observed relationship between polarization and stress that the effect is truly piezoelectric, and they suggest that it results from the slipping of collagen fibres past one another.

We have observed the same stress-induced electrical effect in a number of whole bones from different anatomical sites and species, both in bending and compression modes. The bones were cleaned of soft tissue and periosteum, then placed in an ultrasonic cleaner with detergent and finally dried. Electrodes were painted on the bones with conductive paint (DuPont silver paint No. 4817), leads were attached to these electrodes and connected to a vibrating reed electrometer (Applied Physics Corporation model 31), the output of which was displayed on a chart recorder. To minimize electrical pickup the bone being tested was kept in a shielded box and stressed from the outside. Bones which were stressed by bending, such as human ribs or rabbit femurs, were clamped at one end and bent by applying a force normal to the other end. Shorter bones, such as a human toe phalanx, were stressed by applying known compressive forces lengthwise along the bone axis.

Our results may be illustrated by the following example: the ends of a human toe phalanx were squared off with a saw to permit uniform application of the compressive force. Two silver bands, spaced 12·7 mm apart, were painted completely around the mid-portion of the bone. The d.c. resistance between these electrodes was approximately 10^9 ohms. Sudden application of a static force resulted in a potential difference between the electrodes proportional to the stress and with a decay time of about 0·5 sec; the latter appeared to be characteristic not only of the electrical circuit constants but also of some mechanical property (relaxation phenomenon) of the bone. On releasing the stress the same voltage pulse appeared with opposite polarity. The magnitude of the effect was of the order of 0·3 mV/kg of applied force.

It has long been known that the architectural structure of bones depends to a large extent on the mechanical forces acting on them. The fact that surface charges appear on stressed bone may be the controlling factor in bone formation. The local electric fields resulting from such surface charges might be expected to influence the orientation and deposition of ions or polarizable molecules. As evidence for the effect of electric fields on bone formation, Yasuda *et al.*[3] showed that the development of callus in living bone could be induced by electrical energy. Similarly, Bassett[4] has influenced the direction of bone growth in tissue culture by means of electric fields, as well as by inhomogeneous magnetic fields.

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A Possible Mechanism of Neurosecretion: Release of Vasopressin by Depolarization and its Dependence on Calcium

It is recognized that the various influences, nervous and osmotic, which bring about release of the hormones vasopressin and oxytocin from the neurosecretory terminals in the posterior lobe of the pituitary gland act through the parent cells in the supra-optic and paraventricular nuclei. Such a scheme demands that these cell bodies in the hypothalamus regulate release of hormone from their terminals a considerable distance away and it is simplest to suppose that this regulation is effected by action potentials propagated down the supra-opticohypophysial tract. In this view the cells of the supra-opticohypophysial system possess the characteristic property of neurones—the propagation of action potentials—as well as the characteristic property of endocrine organs—the liberation of hormones into the blood stream[1,2].

The purpose of this communication is to give a preliminary account of experiments indicating that depolarization of the endings of the supra-opticohypophysial tract, such as might be caused by action potentials, is an adequate stimulus for the release of vasopressin and suggesting an explanation of the way in which depolarization may act.

The experiments were performed on rats weighing about 150 g. The infundibular stem was cut and the posterior pituitary glands rapidly removed, halved and suspended *in vitro* in Locke's solution at 37·5° C. Five glands were used in each experiment. At intervals of 20 min the incubation medium was drawn off and replaced with fresh medium of the same or different composition. The pressor activity of the incubates was then assayed against pitressin using the method described by Dekanski[3]. After 20–40 min incubation in Locke's solution the resting output of vasopressin was generally about 20 μU per gland per min. A much higher rate of secretion occurred when the potassium concentration was raised ten-fold (to 56 mM). A typical effect is shown in Fig. 1*a*. This powerful stimulant effect of excess potassium was found to be dependent on the presence of calcium; there was no increase in vasopressin output when excess potassium was given in calcium-free Locke's solution after a period of 40 min incubation with the calcium-free medium (Fig. 1*b*). The inhibitory effect of calcium omission was readily reversible and potassium regularly regained its effectiveness as a stimulus for vasopressin release after calcium had been restored to the incubation medium. Finally, it was found that calcium itself (2 mM) caused a powerful release of vasopressin when added to a preparation exposed to