due to hypoglycemia, are negligible in comparison to this high dose. Females in group C + GH ate normal pelleted food ad libitum, and were treated with growth hormone, and females in group C ate normal pelleted food and received no hormone. Group C served as control for groups R, R + GH, and C + GH.

Caesarian sections were done on the morning of the 22nd day of pregnancy, whenever possible, to recover the offspring and placenta. [Nutritional effects on placentas are reported by us elsewhere (*7*).] The offspring and placentas were weighed, and cerebral hemispheres were dissected out, weighed, and homogenized for determination of DNA (cell number) and protein content (*6, 7*).

Prenatal caloric restriction alone (Table 1, group R) resulted in highly significant decreases in neonatal body weight and placental weight (*7, 10*). Highly significant decreases were also found in cerebral weight, DNA (cell number), and protein content (*5, 11*). Treatment of the pregnant females with growth hormone (group R + GH) resulted in almost complete reversal of these effects; the increases were highly significant as compared to group R. The considerable (21 percent) increase in placental weight, concomitant with increases in cerebral weight, DNA, and protein, is consistent with our report of correlation between placental weight and prenatal brain development (*12*). Essentially similar results were obtained when rats totally deprived of protein between day 10 and day 20 of pregnancy (*7*) were treated during this time with growth hormone (*13*). Thus, growth hormone administered in pregnancy is a potent stimulator of placental and cerebral development in offspring of nutritionally restricted rats.

Growth hormone in physiologic quantities is unlikely to cross the placenta (*14*). It is not known whether this placental barrier also operates at the high doses of hormone we used; if it does, then the primary action of this hormone may be on the mother. The hormone might improve nutrient supply to the fetus by mobilization of maternal nutrient reserves (fat and glycogen), which compensate for deficiencies in maternal blood levels of nutrients (*15*). The hormone might directly increase uterine size and blood flow, with a secondary increase in placental size, or the hormone might enhance maternal hormonal mechanisms that influence the placental size. Decreases in body weight as a result of food restriction can be partially reversed by treatment with growth hormone after weaning (*16*). However, since this hormone treatment was after weaning, the phenomenon is probably different from the one we observed.

Treatment of normal animals with growth hormone (group C + GH) did not produce a significant increase in cerebral DNA. The discrepancy between this result and that previously reported (*2*) is probably due to differences in the animals used in the two experiments. The animals in the previous experiment were of nonuniform stock and were inferior to the present animals with respect to all measured parameters. Treatment with growth hormone did produce a significant increase in cerebral weight, in confirmation of our earlier experiments (*2*); this increase was not due to water but to increased content of cerebral protein.

STEPHEN ZAMENOF
EDITH VAN MARTHENS
LUDMILA GRAUEL

Department of Medical Microbiology and Immunology, Department of Biological Chemistry, Brain Research Institute, and *Mental Retardation Center, University of California School of Medicine, Los Angeles 90024*

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Strontium Accumulation by Sarcoplasmic Reticulum and Mitochondria in Vascular Smooth Muscle

Abstract. *Electron-opaque deposits of strontium were observed in the sarcoplasmic reticulum and in mitochondria of spontaneously contracting vascular smooth muscles that had been incubated in a strontium-containing solution prior to fixation. The deposits were present in those elements of the sarcoplasmic reticulum that are in close contact with the surface membrane and also in more centrally located portions. In vascular smooth muscle that does not contract spontaneously, similar deposits of strontium were only seen if the muscle was depolarized during or glycerinated before exposure to the strontium-containing solution. Strontium was also deposited in the sarcoplasmic reticulum of the endothelium. It is suggested that translocation of calcium from the sarcoplasmic reticulum that is in close contact with the surface membrane, and now shown to accumulate divalent cations, is responsible for the action potential–triggered contractions of rabbit and guinea pig mesenteric veins. Strontium may also be a suitable marker for identifying sites that accumulate calcium in other types of cells in which translocation of calcium plays a major regulatory function.*

The contraction-relaxation cycle of both striated (*1*) and of smooth (*2, 3*) muscles is mediated by, respectively, the rise and fall of the free calcium concentration in the cytoplasm. In fast striated muscles, the release of calcium from and its uptake by the sarcoplasmic reticulum regulates contractile activity. Because of its low electron opacity, ultrastructural localization of calcium to the sarcoplasmic reticulum in striated muscle fibers has been ac-

complished with the difficult techniques of ^{45}Ca autoradiography (*4*) or by precipitation with oxalate to enhance the accumulation of calcium and to prevent its loss during fixation (*5*). Until now, accumulation of divalent cations by the sarcoplasmic reticulum of vertebrate smooth muscles in situ has not been demonstrated.

Strontium has a higher atomic number, and hence higher electron opacity than calcium, it is accumulated by isolated preparations of sarcoplasmic reticulum (*6*), it can activate myofibrillar adenosine triphosphatase activity (*7*) and therefore substitute for calcium in the triggering of contractions (*7, 8*), and it also combines with the calcium-sensitizing protein system (troponin-tropomyosin) of smooth and striated muscles (*9*). In isolated sarcoplasmic reticulum preparations, in the absence of oxalate, calcium and strontium are accumulated at approximately the same rate (*6*). We have successfully used strontium to demonstrate cation accumulation by the sarcoplasmic reticulum of smooth muscle (and of endothelial cells) in situ. The accumulation of electron-opaque deposits of strontium by the sarcoplasmic reticulum of intact cells was observed in the absence of oxalate and therefore may reflect the natural rate of divalent cation accumulation.

Portal-anterior mesenteric veins of rabbits and guinea pigs were examined as examples of vascular smooth muscles that generate spontaneous action potentials accompanied by rhythmic contractions, and rabbit main pulmonary arteries (MPA) were used as examples of electrically quiescent vascular smooth muscle (*10, 11*). Vascular strips were incubated for 1 hour in a modified (calcium-free) Krebs solution in which 10 m*M* $SrCl_2$ was isoosmotically substituted for NaCl. Some preparations were depolarized for 50 minutes with a high-potassium (136 m*M*) solution containing 10 m*M* Sr^{2+} and then returned to the Sr^{2+}-containing modified Krebs solution for an additional 10 minutes before fixation. Other preparations were placed in 30 percent glycerol for 15 minutes prior to 1 hour incubation in the Sr^{2+}-containing Krebs solution. Control vascular strips were incubated in Krebs solution without Sr^{2+} (*12, 13*).

Electron-opaque deposits were found in the lumen of the sarcoplasmic reticulum of portal-anterior mesenteric veins of rabbits and guinea pigs when strips of veins were incubated for 1 hour with 10 m*M* Sr (Figs. 1, A to C, 3, and 4). The spontaneous contractions of rabbit portal-anterior mesenteric veins persisted and even increased when they were transferred from the normal (calcium-containing) Krebs solution into the Sr^{2+}-containing solution. We assumed that Sr^{2+} entered these fibers during the action potential. Strontium was also found in the mitochondria, as was observed by Peachey in his studies of the toad bladder (*14*). Both the peripheral and the central elements of the sarcoplasmic reticulum contained the electron-opaque deposits, and Sr^{2+} accumulations were also found in endothelial cells (Fig. 5). Unstimulated MPA smooth muscle did not contract when trans-

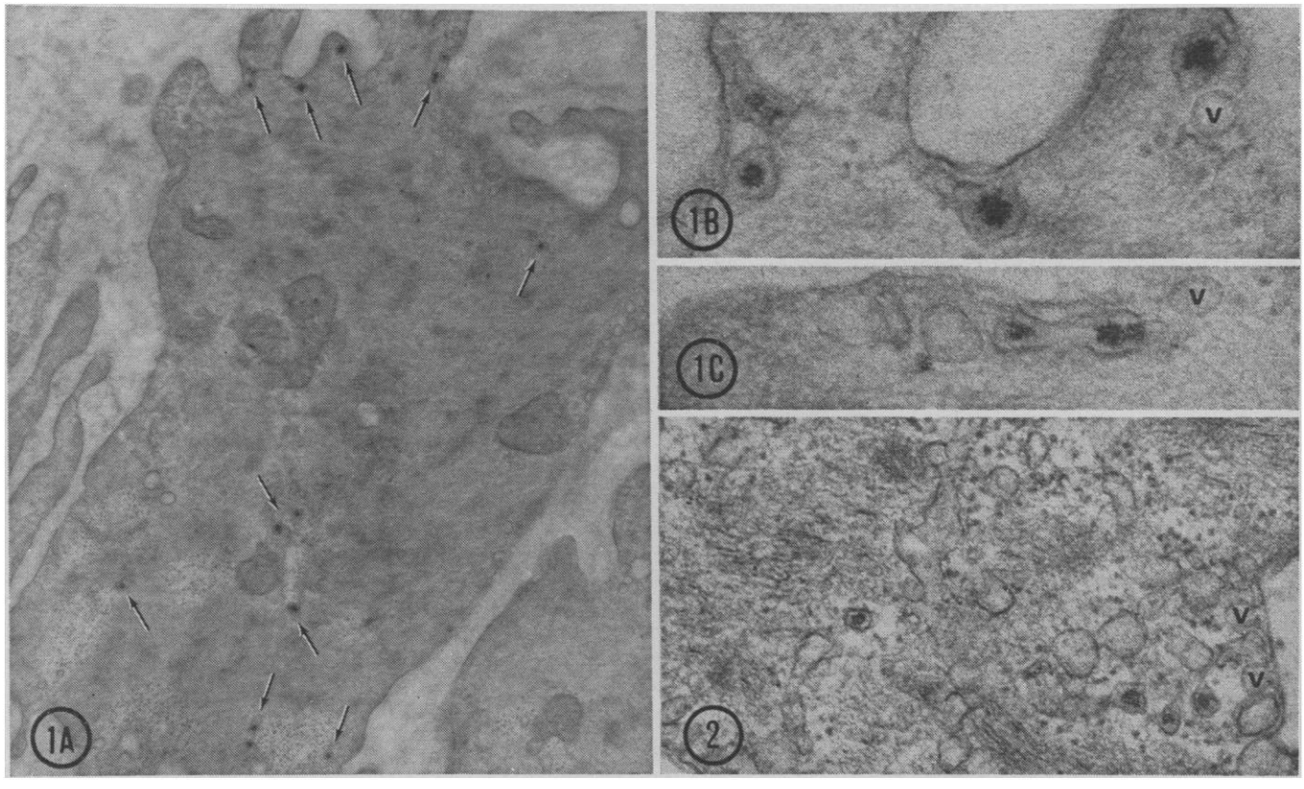

Fig. 1. Oblique section of smooth muscle cell in guinea pig portal-anterior mesenteric vein incubated in Krebs solution containing 10 m*M* Sr^{2+} for 1 hour before fixation. Electron-opaque deposits (arrows) of strontium are present in both the peripheral and central portions of the sarcoplasmic reticulum. (A) Magnification is 19,500 ×. (B and C) Higher magnification (100,000 ×) of the surface couplings of strontium-containing sarcoplasmic reticulum in the fiber shown in (A). *V*, surface vesicles. Fig. 2. Transverse section of a portion of a smooth muscle cell in a rabbit main pulmonary artery that had been placed in 30 percent glycerol for 15 minutes and then in Krebs solution containing 10 m*M* Sr^{2+} for 1 hour before fixation. Electron-opaque deposits are in the lumen of the sarcoplasmic reticulum. Block-stained with uranyl acetate (60,000 ×). *V*, surface vesicle.

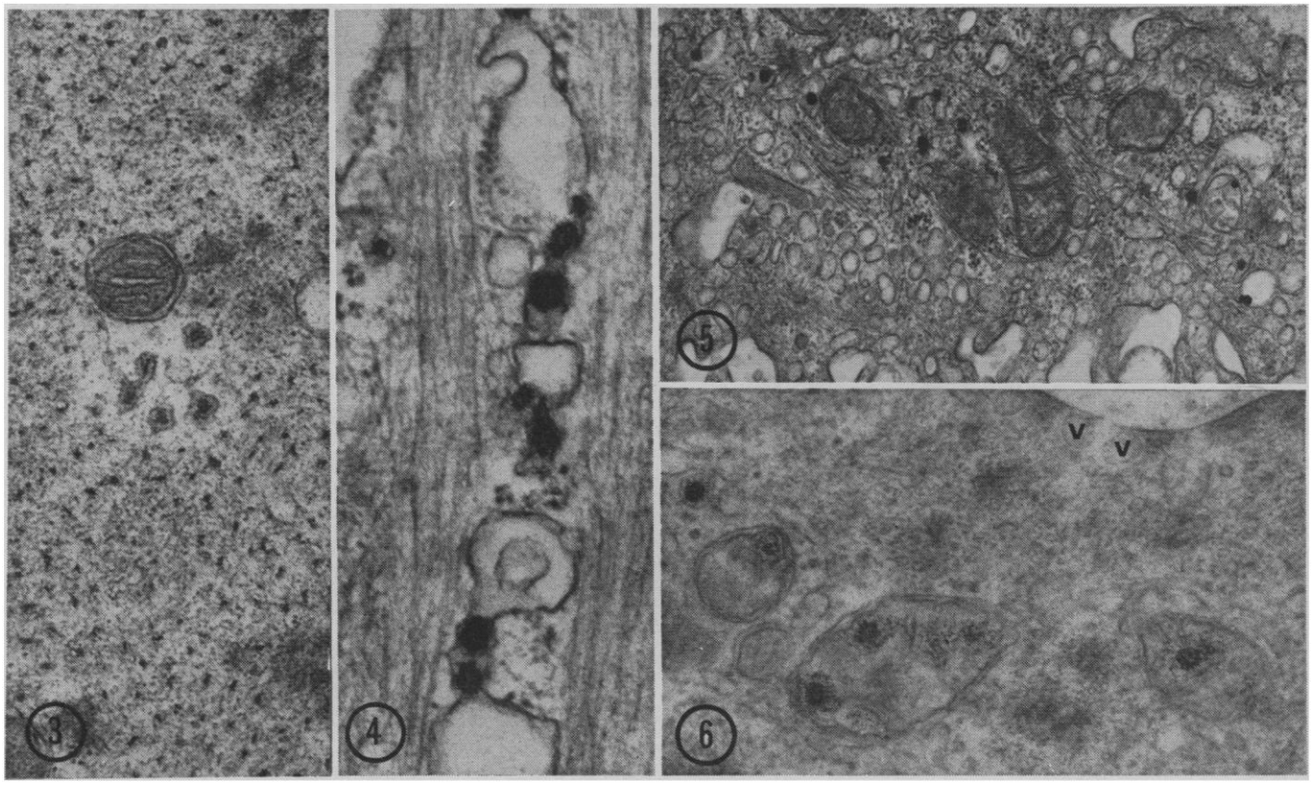

Fig. 3. Transverse section of rabbit portal-anterior mesenteric vein incubated in Krebs solution containing 10 m*M* Sr^{2+}. Electron-opaque deposits are in the lumen of the centrally located sarcoplasmic reticulum. A prominent array of transversely sectioned thick myofilaments is also present. Block-stained with uranyl acetate (60,000 ×). Fig. 4. Longitudinal section of a central portion of a rabbit portal-anterior mesenteric vein smooth muscle cell. Electron-opaque deposits occlude the lumen of the longitudinal tubules of the sarcoplasmic reticulum that are continuous with somewhat dilated portions that have ribosomes adhering to them. Longitudinally sectioned thick filaments are also seen. Block-stained with uranyl-acetate (60,000 ×). Fig. 5. Portion of an endothelial cell of guinea pig portal-anterior mesenteric vein incubated for 1 hour in Krebs solution containing 10 m*M* Sr^{2+} before fixation. The sarcoplasmic reticulum contains electron-opaque deposits. Block-stained with uranyl acetate, section-stained with alkaline lead citrate (30,000 ×). Fig. 6. Portion of a smooth muscle cell in a rabbit main pulmonary artery that had been depolarized with a high-potassium (136 m*M*) solution containing 10 m*M* Sr^{2+} for 50 minutes and then placed in Krebs solution containing 10 m*M* Sr^{2+} for an additional 10 minutes before fixation. Deposits of Sr^{2+} are present in the mitochondria (60,000 ×). *V*, surface vesicle.

ferred into Sr^{2+}-containing Krebs solution, and no intracellular electron-opaque deposits were observed in these preparations. Both findings show that, as expected, the polarized resting membrane is relatively impermeable to strontium. However, after glycerination or depolarization—procedures that increase the permeability of cell membranes—Sr^{2+} deposits were seen in the sarcoplasmic reticulum (Fig. 2) and mitochondria (Fig. 6) of MPA smooth muscle.

These results indicate that the structures identified as a sarcoplasmic reticulum in smooth muscle (*15*) can accumulate divalent cations that activate contraction, and are consistent with reports of calcium uptake by isolated microsomal fractions of smooth muscle (*16*), and by subsarcolemmal vesicles of invertebrate (paramyosin) smooth muscle (*17*). The sarcoplasmic reticulum may be one of the sites at which Sr^{2+} can exchange for Ca in aortic smooth muscle (*18*). The close apposition of the cation-accumulating sarcoplasmic reticulum to the surface membrane (Fig. 1, B and C) provides morphological support for the suggestion (*2, 10, 15*) that the twitch of vascular smooth muscle fibers is due to the translocation of calcium from the sarcoplasmic reticulum by the action potential. The relative contributions of the sarcoplasmic reticulum and of mitochondria to divalent cation movement and contractile activation and inactivation remain to be established. Barium contractures of rabbit portal-anterior mesenteric veins in calcium-free solution are followed by spontaneous relaxation accompanied by massive accumulation of barium into the mitochondria, without any significant uptake by the sarcoplasmic reticulum (*19*). It is possible that both the sarcoplasmic reticulum and the mitochondria contribute to the regulation of contraction and relaxation in smooth muscle.

The use of Sr^{2+} to follow divalent cation accumulation by the sarcoplasmic reticulum in situ appears to be a promising method for following spontaneous and drug stimulated translocations of divalent cations in intact cells, and could perhaps be applied profitably to other systems in which divalent cation (that is, calcium) movement into cells is considered to play a major function, such as stimulus-secretion coupling and neurotransmitter release.

AVRIL V. SOMLYO
ANDREW P. SOMLYO
Department of Pathology, Presbyterian-University of Pennsylvania Medical Center, and *School of Medicine, University of Pennsylvania, Philadelphia 19104*

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Stereoscopic Depth Movement: Two Eyes Less Sensitive than One

Abstract. *Visual sensitivity to stimuli with sinusoidal movement was examined under a number of conditions of binocular stimulation. Sensitivity to stereoscopic movement in depth was reduced in comparison to that for monocular movement. The reduced sensitivity appeared to be due to the presence of stereoscopic depth movement, as opposed to stereoscopic stimulation, binocular movement, or fusion of the images.*

The dynamic properties of human stereoscopic depth perception may be investigated by the use of moving stimuli viewed stereoscopically. Lit and other workers (*1*) have carried out a number of studies on the stereoacuity for physical stimuli that either move linearly or oscillate sinusoidally in a frontal plane. However, because the stimuli contained both types of movement, these studies do not provide information on the relative effects of cues of monocular and stereoscopic movement in stereoacuity. Using stereoscopic random-dot stimuli of correlated stereograms, Julesz and Payne (*2*) studied stereoscopic apparent movement. Two types of stimulus presentation were studied: stimuli that could be seen as moving both monocularly and stereoscopically, and stimuli that could be perceived as moving only in the stereoscopic mode of presentation with all monocular movement cues eliminated. (In none of these studies was movement in depth produced; frontal plane movement of stimuli perceived in depth was the mode of movement investigated.) Julesz and Payne measured the alternation frequency of the two apparent positions for each type of stimulus at which the apparent movement percept disappeared and the stimuli appeared simultaneous. The critical frequency for simultaneity was significantly higher for the monocular apparent movement than for the stereoscopic apparent movement, although Julesz and Payne do not comment on this fact. This observation corresponds to a reduction in sensitivity to apparent movement with a stimulus providing only stereoscopic cues for movement as compared with one providing stereoscopic and monocular cues. I could find no other experiments in the literature that compared monocular and stereoscopic movement thresholds.

To investigate the problem of movement perception in stereoscopic vision and to extend the findings of Julesz and Payne, I used real movement that oscillated sinusoidally in depth at a range of oscillation frequencies. The display stimuli consisted of thin, bright vertical lines, subtending 1 degree in height and 2 minutes in width, seen against a dark background. The stimuli were produced on the face of a fast-phosphor Dumont oscilloscope by a sawtooth signal at 30 khz with a luminance of 3.4 cd/m^2 and were viewed with a natural pupil, because there was no overall change in stimulus luminance. The stimuli were displaced sinusoidally (that is, in simple harmonic motion) in a horizontal plane with an amplitude that could be varied by the subject. The stimulus configuration observed by the subject, produced by conventional orthogonal polarizers, consisted of a stationary line and an oscillating line seen with each eye at an average separation of 20 minutes. The moving lines could be oscillated in phase, which corresponded to a real movement version of the movement used by Julesz and Payne, or in antiphase. When the subject stereoscopically fused the stimuli to his two eyes to obtain the Cyclopean view, he perceived a stationary line and a moving line suspended in space in the dark. If the moving lines to each eye oscillated in phase he would perceive the line moving from side to side (binocular frontal movement). When the oscillations were in antiphase, the movement appeared toward and away from him (stereoscopic depth movement).

The visual sensitivity to monocular and stereoscopic depth movement was compared. The subject set the amplitude of the movement until it appeared just not visibly moving. The peak-to-peak amplitude of the movement at this threshold setting was measured. The monocular movement amplitude was taken as the measure in the case of the stereoscopic movement, in order to make a comparison of monocular movement information required for monocular as compared to stereoscopic movement threshold.