tained for Δ^{260}, while pancreatic ribonuclease (*10*) at *p*H 7.3 gave a Δ^{260} of 0.930 with ribonucleic acid and of 0.000 with the "core." Unlike the pancreatic enzyme, the *Euglena* ribonuclease therefore was capable of further degrading the "core"—an indication that it does not share the specificity of pancreatic ribonuclease for pyrimidine internucleotide bonds. This was confirmed by experiments in which the enzymes were allowed to act on polyadenylic and polyuridylic acid (*11*) and in which the reaction products were analyzed by paper chromatography. Both polymers were degraded by the enzyme from *Euglena*, while pancreatic ribonuclease attacked only the polyuridylic acid (*12*).

J. FELLIG*
CHRISTINE E. WILEY
Research Department,
Union Carbide Chemicals Company,
South Charleston, West Virginia

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10. The pancreatic ribonuclease was obtained from the Worthington Biochemical Corp., Freehold, N.J.
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* Present address: Tonawanda Laboratories, Linde Co., a division of Union Carbide Corp., Tonawanda, N.Y.

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Effect of Reserpine on Ventricular Escape

Abstract. Catecholamine depletion by reserpine diminishes the tendency of the ventricle to escape from vagal suppression. Neither spinal section nor adrenalectomy enhances the reserpine effect. Norepinephrine restores the characteristic occurrence of ventricle escape during vagal stimulation.

Krayer and his co-workers (*1*) have described the importance of catecholamines to the rhythmicity of the sinus pacemaker, and Roberts and Modell (*2*) have shown that catecholamine activity is even more important to the rhythmicity of the ventricular pacemaker. It has been recently indicated that an adrenergic mechanism may play a role in the phenomenon of vagal escape (*3*). If catecholamines are important to the intrinsic rhythmicity of the ventricle, then ventricular escape from vagal control should be influenced by catecholamine depletion. This report (*4*) describes experiments designed to explore this relationship. Reserpine was used to deplete the amines.

Table 1. The effect of reserpine on ventricular escape during vagal stimulation. S.E., standard error.

Procedure	No. of animals	Av. slowest sinus rate (induced by vagal stimulation) (beats/min. ± S.E.)	Av. ventricular escape rate* beat/min ± S.E.	Incidence of asystole†	Av. duration of asystole (sec ± S.E.)
		Anesthetized animals			
Control	15	49 ± 8	64 ± 8	2/15	7
Reserpine-treated	14	14 ± 5	28 ± 7‡	11/14	17 ± 2
		Animals with spinal-cord section			
Control	14	60 ± 6	66 ± 5	0/14	0
Reserpine-treated	4	7 ± 2	20 ± 11	4/4	14 ± 4

* See text for explanation. † Periods longer than 5 seconds. ‡ Based on 11 animals, since three developed only sinus escape.

In cats with the spinal cord sectioned at C1 or in cats anesthetized with Dial urethane (0.6 to 0.7 ml/kg), the right vagus nerve was stimulated. With appropriate vagal stimulation, the sinus rate may be slowed sufficiently to permit ventricular escape—that is, there is an unmasking of ventricular rhythmicity through the removal of sinus dominance. In each experiment the minimal intensity of vagal stimulation which would permit ventricular escape was used. Therefore, while the stimulus parameters varied, animals were compared at equivalent stimulus responses. Pulses of 6 to 14 volts, frequency of 10 to 40 cy/sec, and pulse duration of 0.1 msec applied for at least 20 seconds (*5*) were usually sufficient to produce ventricular escape. Reserpine (0.5 to 10.0 mg/kg) was injected intravenously 24 to 50 hours before the experiment. Since there was no significant difference in the effects of reserpine in the dose range employed, the results at all dose levels were pooled and averaged. The data are summarized in Table 1.

In 15 untreated, anesthetized animals, when the sinus rate decreased during vagal stimulation to an average of 49 beats per minute, the rate of ventricular escape averaged 64 beats per minute and stabilized at an average of 75 beats per minute. Untreated animals with spinal-cord section responded in a similar manner, indicating that ventricular escape is not primarily related to sympathetic tone. In all cases, a sinus rhythm reappeared promptly after the cessation of vagal stimulation.

In animals treated with reserpine, ventricular escape still occurred, but there was a striking difference in the sinus rate at which this escape from vagal control appeared. Furthermore, ventricular rate after escape from vagal control also differed greatly from the comparable ventricular rates in the untreated animals. In 14 anesthetized animals, when the sinus rate was slowed by vagal stimulation to an average of 14 beats per minute, the independent ventricular rate averaged 28 beats per minute. These rates are significantly lower than those of the controls ($p < .01$). There was a similar response in four reserpine-treated animals with spinal-cord section. During vagal stimulation, asystolic periods of more than 5 seconds duration developed in only two of 29 untreated animals; such periods occurred in 15 of 18 reserpine-treated animals. In addition, the duration of asystole was considerably greater in the reserpine-treated animals (Table 1). The lower independent ventricular rate and the longer period of asystole after reserpine treatment indicate that the effect of reserpine on the ventricle is a consequence of an altered ventricular rhythmicity.

The appearance of ventricular escape during asystole suggests other factors not influenced by reserpine. In all reserpine-treated animals with spinal-cord section and in two reserpine-treated anesthetized animals, ventricular escape during asystole developed with the onset of convulsions. Thus, it seemed possible that such concomitants of anoxia as the liberation of potassium or the mobilization of cardiac catecholamine stores not depleted by reserpine (*6*) provoked ventricular escape. Other sources of catecholamines probably did not play a role, since bilateral adrenalectomy after reserpine treatment (in one anesthetized animal and in two animals with spinal-cord section) did not further diminish the ability of the ventricle to escape from vagal suppression.

In 12 animals treated with reserpine the administration of norepinephrine prevented, during vagal stimulation, the development of asystolic periods of more than 5 seconds' duration. The vagus was stimulated periodically during an infusion of 0.1 to 1 μg of norepinephrine per kilogram per minute. After 5 to 10 minutes, vagal stimulation permitted ventricular escape with much less slowing of the sinus rate. The reversal of the reserpine effect by norepinephrine implies that the effect of reserpine to reduce ventricular rhythmicity is the result of catecholamine depletion.

JAY ROBERTS
RICHARD P. STADTER
Department of Pharmacology, Cornell University Medical College, New York, New York

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Crystal Structure Refinement of Reedmergnerite, the Boron Analog of Albite

Abstract. Ordering of boron in a feldspar crystallographic site $T_1(0)$ has been found in reedmergnerite, which has silicon-oxygen and sodium-oxygen distances comparable to those in isostructural low albite. If a simple ionic model is assumed, calculated bond strengths yield a considerable charge imbalance in reedmergnerite, an indication of the inadequacy of the model with respect to these complex structures and of the speculative nature of conclusions based on such a model.

Recent conclusions important to the interpretation of the mineralogy and petrology of alkali feldspars have been presented by Ferguson (*1*). Evidence supporting the new theory is taken in part from the results of crystal-structure analyses of low and high albite (*2*) as to (Al, Si)-ordering in tetrahedral sites T_1 (0), T_1 (*m*), T_2 (0), and T_2 (*m*), and from the resulting charge balance calculated for these sites when a simple ionic model is assumed. Determination by x-ray diffraction methods of (Al, Si)-distribution in feldspar crystals must necessarily be indirect because of the near equivalence in x-ray scattering power of aluminum and silicon. A recently described mineral, reedmergnerite (*3*), $NaBSi_3O_8$, is isostructural with albite, $NaAlSi_3O_8$, and the elements boron and silicon can readily be distinguished by x-ray techniques. Furthermore, aluminum and boron have many chemical and structural similarities in crystals, both elements occurring coordinated by a tetrahedron of oxygens, with boron-oxygen distances of 1.46 to 1.48 A (*4*) and aluminum-oxygen distances of 1.75 to 1.79 A (*5*). It seemed probable that comparison of albite and reedmergnerite structures would help to clarify the (Al, Si)-distribution problem and provide additional information about charge balance assumptions. Accordingly, a detailed crystal-structure investigation of reedmergnerite has been completed, in which we started with the known low albite parameters (*2*).

Thirteen cycles of least-squares analysis of 2753 three-dimensional data for reedmergnerite have been carried out on a Burroughs 220 digital computer; the full matrix associated with the normal equations was used. A residual factor

$$R = \Sigma \mid \mid F_{\text{obs.}} \mid - \mid F_{\text{calc.}} \mid \mid / \Sigma \mid F_{\text{obs.}} \mid$$

of 11 percent has been obtained, excluding data recorded as zero. Reedmergnerite contains the typical feldspar framework found in low albite (*2*); the refinement shows that boron is wholly contained in site T_1 (0), and that the framework is distorted as compared to that in albite, so that the smaller boron-oxygen tetrahedra can be accommodated. The resultant shrinkage in framework accounts for an average 4-percent reduction in dimensions of the reedmergnerite cell as compared with those of the albite cell (*3*). The silicon-oxygen distances in reedmergnerite (Table 1) compare so closely with those reported for low albite (*2*) that considerable doubt is cast on the validity of (Al, Si) assignment from small variations in these bond distances, according to the relationship proposed by Smith (*5*) and used in the study of albite (*2*).

Table 1. Some interatomic distances for reedmergnerite and low albite (*2*).

	Interatomic distances (A)	
	Reedmergnerite (all ± 0.01 A)	Low albite
	$T_1(0)$ tetrahedron	
(B or Al)-$O_A(1)$	1.47_3	1.76_2
(B or Al)-$O_B(0)$	1.48_4	1.70_2
(B or Al)-$O_C(0)$	1.44_0	1.76_2
(B or Al)-$O_D(0)$	1.47_3	1.74_4
Av.	1.46_8	1.74_2
	$T_1(m)$ tetrahedron	
Si-$O_A(1)$	1.58_9	1.59_9
Si-$O_B(m)$	1.60_0	1.62_6
Si-$O_C(m)$	1.60_2	1.55_3
Si-$O_D(m)$	1.62_1	1.58_3
Av.	1.60_3	1.59_0
	$T_2(0)$ tetrahedron	
Si-$O_A(2)$	1.62_7	1.61_8
Si-$O_B(0)$	1.57_6	1.62_7
Si-$O_C(m)$	1.63_0	1.64_9
Si-$O_D(m)$	1.62_0	1.64_9
Av.	1.61_3	1.63_6
	$T_2(m)$ tetrahedron	
Si-$O_A(2)$	1.65_0	1.64_0
Si-$O_B(m)$	1.61_4	1.59_7
Si-$O_C(0)$	1.61_8	1.61_8
Si-$O_D(0)$	1.60_9	1.61_1
Av.	1.62_3	1.61_6
	Oxygen-Na	
$O_A(1)$	2.48_1, 2.50_3	2.61_5, 2.68_1
$O_A(2)$	2.39_2	2.36_3
$O_B(0)$	2.40_6	2.46_0
$O_C(0)$	(3.44_1)	2.88_9
$O_C(m)$	2.84_4	(3.24_7)
$O_D(0)$	2.37_8	2.46_5
$O_D(m)$	2.85_2	(2.99_7)

The sodium atoms in reedmergnerite are located in the cavities within the feldspar framework. Anisotropic temperature motion of the sodium atoms has not yet been fully investigated, but the isotropic temperature factor *B* is 1.23 A^2, in marked contrast to the maximum *B* of 0.78 A^2 found for the other atoms in the structure. The coordination of oxygen atoms about sodium in reedmergnerite is comparable to that found in albite for the five closest oxygens (Table 1), but some

Table 2. Bond strengths contributed by (B or Al)$^{3+}$, Si^{4+} and Na^+ to O^{2-} atoms in tetrahedral groups of reedmergnerite and low albite (*2*). (*a*) $T_1(0)$=B (reedmergnerite), Al (low albite), remainder = Si; Na coordinated by five closest oxygens (reedmergnerite, Table 1), by six closest oxygens (low albite, Table 1). (*b*) $T_1(0)$=B (reedmergnerite), Al (low albite), remainder = Si; Na coordinated by seven oxygens (Table 1). (*c*) Al-Si distribution taken as follows: $T_1(0)$ = 0.72 Al, $T_1(m)$ = Si, $T_2(0)$ = 0.20 Al, $T_2(m)$ = 0.09 Al, remainders = Si; Na coordinated by six oxygens (Table 1). (*d*) Al-Si distribution as in (*c*); Na coordinated by seven oxygens (Table 1).

Tetrahedral site	Bond strength					
	Reedmergnerite		Low albite			
	(*a*)	(*b*)	(*a*)	(*b*)	(*c*)	(*d*)
$T_1(0)$	7.80	7.56	7.83	7.71	8.02	7.90
$T_1(m)$	8.15	8.31	8.08	8.18	8.03	8.13
$T_2(0)$	8.15	8.31	8.08	8.18	7.93	8.03
$T_2(m)$	7.90	7.78	8.00	7.93	8.00	7.93
$\Sigma\lvert\Delta\rvert$	0.60	1.28	0.33	0.72	0.12	0.33