reduces the acetylcholine-induced net K^+ release, as a result of enhanced K^+ uptake. Stimulation of active K^+ uptake by cGMP seems to be the consequence, furthermore, of a stimulatory effect of the nucleotide on the Na^+, K^+ ATPase responsible for active K^+ uptake, since addition of ouabain prevented the observed stimulation of K^+ uptake by the nucleotide. Shi et al. have shown that cGMP stimulates the hydrolysis of pNPP, a substrate for the Na^+, K^+ ATPase[13]. The mechanism by which cGMP stimulates the Na^+, K^+ ATPase was not directly investigated in our study, but could involve effects on a protein kinase or on a regulatory protein that inactivates the pump mechanism.

Active K^+ uptake by the Na^+, K^+ ATPase is also observed in rat submandibular[4] and parotid[14] slices after stimulation of α-adrenergic receptors. However, a recent study failed to demonstrate an increase in cGMP formation in rat submandibular gland slices following α-adrenergic receptor stimulation[6]. It is possible that active K^+ uptake during the response to α-adrenergic stimuli is mediated by a mechanism which does not involve cGMP. However, differences in the time course of cGMP generation by cholinergic and α-adrenergic stimuli should be considered, since α-adrenergic stimulation is effective in stimulating cGMP formation in other tissues, including the parotid gland[8,9], and it is not clear why the submandibular gland should be an exception. Our results indicate that the function of cGMP in the K^+ release mechanism of the rat submandibular gland is to enhance active K^+ uptake. This function is physiologically important, since recovery of the K^+ lost as a result of the passive efflux induced by receptor stimulation would restore the ionic composition of the salivary cells to the prestimulation levels, and, thus, render them responsive to subsequent secretory stimuli. The activation of the $Na+$, K^+ pump during the response to secretagogues will also result in the active extrusion of Na^+ from the salivary cells and contribute, therefore, to the formation of saliva. The energy required for the ionic movements responsible for fluid secretion may derive from that stored in the Na^+, K^+ gradient across the cell membrane.

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Extracellular volume, electrocardiogram and anion distribution in hibernating hamster ventricle[1]

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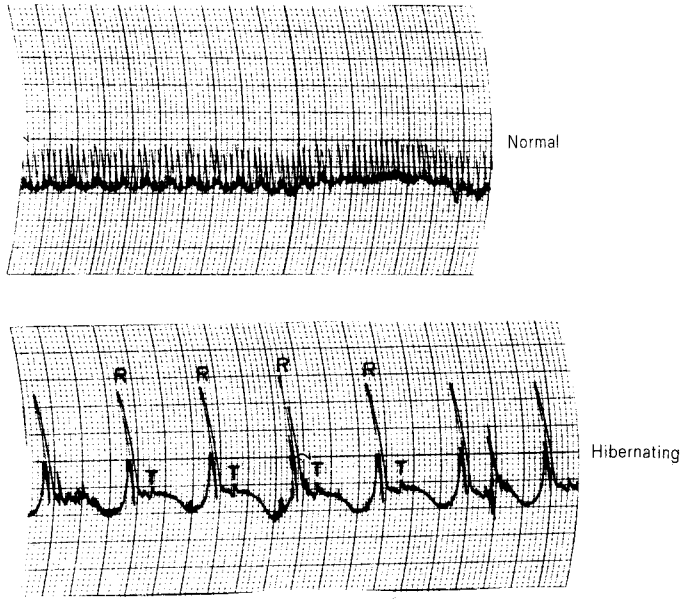
Summary. The intracellular Cl concentration, $[Cl]_i$, was found to be significantly larger and the extracellular volume (ECV), much smaller in ventricles of hibernating hamsters as compared to non-hibernators. The decreased ECV in ventricles of hibernators was consistent with an increased R wave component of the ECG, as well as a lower mean fraction tissue water, f_{H_2O}, of these tissues.

It is still uncertain whether Cl ions are distributed passively or actively across the membrane of cardiac muscle cells. It is largely accepted that the Cl concentration in heart muscle exceeds values compatible with a passive distribution[2–4], however, evidence is available which supports a passive Cl distribution in some cardiac tissues[5–7]. This apparent discrepancy in the literature is in part due to the fact that it has been difficult to measure Cl content of muscle cells with sufficient precision[8]. This difficulty rests in the fact that the cellular Cl content and concentration in striated muscles are small relative to the high extracellular content and concentration; and the relatively large statistical dispersion of extracellular space measurements observed when muscles are examined in vitro has led to unacceptable large dispersions when intracellular Cl, $[Cl]_i$, and cellular Cl content are derived by conventional compartmental analysis.

Using improved techniques for measuring Cl content and concentration[8], Golnick et al.[9] recently measured values of $[Cl]_i$ in in situ hamster ventricle which was significantly less than reported earlier for heart muscle, but consistent with the recent observations of Polimeni and Page[6] for rat ventricle. These data would suggest that Cl is passively distributed in the intact rat and hamster ventricle. To further support the idea of a passive distribution in these tissues, it would be desirable to compare our calculated Cl equilibrium potential (derived from the in situ distribution of Cl) to the in situ cardiac membrane potential. These data, however, are difficult to interpret since the distribution of Cl in contracting heart muscle is fixed to a mean value of membrane potentials, E_m, which are continuously changing (because of the continuous firing of action potentials). In considering this problem, we thought it conceivable that an accurate comparison between E_m and $[Cl]_i$ could be made in in situ hearts of hibernating animals (where the heart rate is extremely slow). However, in searching the literature, we found little if any information regarding Cl distribution in the ventricles of hibernating animals. We decided, therefore, to first examine the Cl distribution in the ventricles of our experimental animal,



This figure represents an ECG recording of a normal anesthetized hamster (top) and a hibernating anesthetized hamster (bottom). The strip chart speed (abscissa) was 5 mm/sec and the recorder was calibrated to record 5 mV/cm (ordinate).

the hibernating Syrian hamster. Here we report the in situ determination of ECV, ECG and $[Cl]_i$ in the ventricle of the hibernating hamster. These measurements show interesting differences in the Cl distribution, ECV and ECG of hibernating vs normal hamster ventricles.

Female Syrian hamsters (~200 g) were induced to hibernate by placing them in a cold room kept at constant temperature (3-5 °C) and controlled photoperiod. In most cases, it took approximately 6-10 weeks before the hamsters were induced into hibernation, during which time there was a significant gain in body weight (~30%). Shortly before the hamsters entered hibernation, their body weight decreased by approximately 10-20%. Following at least 2 months of intermittent hibernation, an ECG was recorded by placing the hamster in a refrigerated box permitting enterance of the recording wires. The figure gives a typical ECG recording of both a non-hibernating and a hibernating hamster. Both groups of animals were anesthetized with lidocaine s.c. before making the electrophysiological measurements. In non-hibernating animals, the heart rate was 300-350 beats per min and the electrical systole (corresponding to Q-T in the human ECG) accounted for 36% of the cycle length (n=90 ECGs). In hibernators, the heart rate was 15-20 beats per min and electrical systole 24% of the cycle length; the amplitude of the R wave in hibernators was approximately 3.75 times that of non-hibernators. This difference cannot be ascribed to a poor frequency response of the ECG recorder (Beckman Type R411 Recorder) since a check by means of an oscilloscope (Tektronix Model 5111) revealed that the peak of the conventionally recorded R wave was cut by the order of < 5%. Following the ECG recording, a blood sample was removed from the ventricle with a heparinized syringe and the heart immediately excised. The tissues and blood plasma were prepared and analyzed for its Cl contents according to Macchia et al.[8]. The ECV was determined using morphometric techniques as described by Page[10]. From the ECV, plasma and tissue Cl contents, the $[Cl]_i$ was determined. From the table, the $[Cl]_i$ (in μmoles/g cell water) was found to be significantly larger in ventricles from hibernating hamsters (25.2 ± 1.7, n=20) as compared to non-hibernators (7.2 ± 1.3, n=31).

The increased ventricular $[Cl]_i$ in hibernating hamsters were observed to correspond to a significant decrease in the ECV. The calculated ECV (in g EC water/g muscle water) of 0.081 ± 0.004 (n=8) for hibernating hamsters was found to be significantly lower than reported for non-hibernators (0.240 ± 0.007, n=12). The decreased ECV of ventricles from hibernators was found to be consistent with the lower mean fraction tissue water in these tissues (0.773 ± 0.003, n=20) as compared to the mean water content of 0.791 ± 0.004 (n=49) as measured in non-hibernators.

The above results show that the distribution of chloride ions is markedly different in hibernating hamster ventricles than in normal hamsters. The decreased Cl content in hibernating hamster ventricles, concomitant with a 3.5-fold increase in $[Cl]_i$ (see table), suggests that at least part of this increase is due to the large decrease in the ECV. The increase in $[Cl]_i$ in hibernators is not at this time completely understood. It is not possible from these data to rule out the unmasking of an inwardly directed Cl pump or perhaps inhibition of an outwardly directed Cl pump. However, it is conceivable that the increase in $[Cl]_i$ could reflect changes in the equilibrium distribution of Na, or K-concomitant with a markedly reduced resting membrane potential (V_m). The answer must await further measurements of V_m and cation distribution in ventricles of the hibernating hamster. More interestingly, in hibernators, the large decrease in

Cl content and concentration, morphometric space and fraction tissue water of ventricles of hibernating and non-hibernating hamsters

	$[Cl]_i$ (μmoles/g cell water)	$(Cl)_m/W_d$ (μmoles/g dry wt)	Morphometric space (g EC water/ g muscle water)	f_{H_2O}
Hibernating hamster	25.2 ± 1.7 (n=20)	100.9 ± 6.6	0.081 ± 0.004 (n=8)	0.7729 ± 0.003 (n=20)
Normal hamster	7.2 ± 1.3 (n=31)	124.3 ± 4.3	0.240 ± 0.007 (n=12)	0.7911 ± 0.004 (n=49)

Values are mean ± SEM.

ECV relative to changes in the intracellular volume (ICV) is consistent with the large increase observed in the R wave component of the ECG. This idea rests in the observation that the R wave component of the ECG reflects changes in the total longitudinal current in cardiac tissue. Since the extracellular component of the total current depends not only on the ratio of the external and internal specific resistances (R_o and R_i), but also on the ratio of the cross-sections of these two compartments[11,12], a change in these values will cause a change in the R wave amplitude. Here the 3-4-fold increase observed in the R wave component of the ECG of hibernating hamster is in good agreement with the 3-4-fold decrease in ECV observed in the ventricles of these animals.

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Thermal concomitants and biochemistry of the explosive discharge mechanism of some little known bombardier beetles[1,2]

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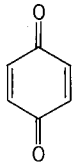
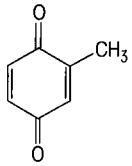
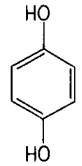
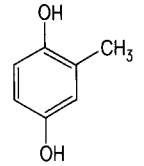
Summary. The quinonoid defensive spray of 2 carabid beetles of the subfamilies Metriinae and Paussinae is ejected hot (55 °C and 65 °C), with a heat content of 0.19 and 0.17 cal/mg. Hydroquinone(s) and hydrogen peroxide are identified as precursors of the quinones, indicating that in these lesser known 'bombardier beetles' the explosive discharge mechanism is similar to that of the familiar bombardiers of the genus *Brachinus* (subfamily Carabinae, tribe Brachinini).

Bombardier beetles include species of 3 different subdivisions of the family Carabidae: a) the tribe Brachinini of the subfamily Carabinae, b) the subfamily Paussinae (including its 2 tribes, the Ozaenini and Paussini), c) the subfamily Metriinae. All discharge 1,4-benzoquinones from their paired abdominal defensive glands, and do so audibly, hence their name[6-9]. Details of the discharge mechanism have been worked out for the Brachinini only, which includes the best known and most commonly collected bombardiers (e.g. the genus *Brachinus*). Hydrogen peroxide and hydroquinones, stored in the sac-like inner chamber of the glands, are passed through a 2nd chamber (the reaction chamber) containing crystalline catalases and peroxidases, causing the hydroquinones to be explosively oxidized to benzoquinones[6,10] and the reacting mixture to be expelled as a hot spray (100 °C)[11]. The question remained whether in the Paussinae and Metriinae the bombarding mechanism might be similar. We here present evidence that it is. Only a single species of the tribe Paussini had previously been studied and shown to generate its quinones in brachinine fashion, from hydrogen peroxide and hydroquinone[12]. No comparable precursor determinations had been made with Ozaenini or Metriinae, nor had thermal measurements been made of the spray of any bombardiers other than Brachinini.

We had available for study several live specimens of

Chemistry of spray, and of the glandular fluid that gives rise to the spray, in the two bombardier beetles studied

	Previously identified principal components of spray[9]			Compounds detected in glandular sac			
			$CH_3(CH_2)_{13}CH_3$			H_2O_2	$CH_3(CH_2)_{13}CH_3$
Subfamily Paussinae (tribe Ozaenini) *Goniotropis nicaraguensis*	+	+	+	+	+	+	+
Subfamily Metriinae *Metrius contractus*	+	−	+	+	−	+	+