and BRYANT and BELLAIRS[2] showed that repeated autotomies lowered regeneration rates. Neither of these factors were involved in the present study, though failure to control them would presumably give rise to variation beyond that reported here.

Field studies have led to suggestions that the age and 'activity' of individuals may modify tail regeneration[14–16]. In view of the wide variations in regeneration obtained under relatively uniform experimental conditions, we suggest that conclusions based on field studies involving small sample sizes may be tenuous.

The peripheral innervation (see references in ZIKA and SINGER[17]) and the ependyma[18] have been shown to be important determinants of whether regeneration will occur, but their influence on the rates of tail regeneration has not been examined in detail. Such factors may underlie the large degree of variability in our study that was unaccounted for by factors generally thought to influence regeneration rates.

We emphasize the need for care in the selection of sample sizes in tail regeneration studies even when the conditions of the specimens and experimental regime are relatively uniform. The profound temperature effects on tail regeneration raise doubts regarding the validity of interspecific comparisons based on studies where 'comparable' temperatures were not used for all species. In this regard, we suggest that cognizance be taken of the species characteristic preferred body temperatures[4]. These considerations may also be useful in experimental design since regeneration rates most advantageous for study can be attained by selecting appropriate temperatures[19].

Résumé. L'étude du phénomène de régénération de la queue chez le mâle adulte du lézard *Anolis carolinensis*, à 32 °C, s'est montré extrèmement variable même dans des conditions uniformes. Ces variations n'étaient en relation ni avec les dimensions ni avec l'accroissement du corps, ni avec l'endroit de l'amputation dans la vertèbre, ni avec la condition de l'épiderme au moment de l'amputation. L'étude à 21 °C a montré que les effets de la température diffèrent selon les phases du processus régénératif. Le temps de formation du blastême a donné une moyenne de 36,2 jours, mais de 8 jours à 32 °C, $Q_{10} = 3,9$; pendant le mois suivant, la croissance fut à peu près de 0,15 mm par jour mais de 0,98 mm par jour à 32 °C, $Q_{10} = 5,5$. Au terme de sa régénération, la queue n'a atteint que le 17% de sa longueur, même 10 mois après l'opération, tandis qu'elle atteignait 28% au bout de 6 semaines à 32 °C.

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Avian Cardiovascular Parameters: Effect of Intravenous Osmotic Agents, Relation to Salt Gland Secretion

Interest in the avian (nasal) salt gland which serves as an extrarenal osmoregulatory system[1], led us to speculate what effects hypertonic i.v. osmotic agents, which cause the gland to secrete hypertonic sodium chloride, might have on representative cardiovascular and plasma electrolyte parameters. Also, we questioned what role these events might have in the initiation of the secretory response. Indirect evidence was obtained by measuring the following parameters in the pentobarbital anesthetized goose: blood pressure, heart rate, blood flow in both carotids and the right alar artery blood flow, blood volume, erythrocyte volume, and plasma sodium, potassium, and osmotic concentration values at various times before and after i.v. administration of 10 ml each of 10% sodium chloride and 20% sucrose. Analysis of the evidence suggests that the increases in blood volume may be the measurable initiating stimuli for salt gland secretion.

Two mongrel dogs and 7 Toulouse domestic geese were used in this study. The geese were maintained on 1.5% sodium chloride and 0.05% potassium chloride drinking water to hypertrophy the salt gland and make it functional. Purina pigeon chow was given ad libitum. Total carotid and alar blood flows were measured in the heparinized bird with precalibrated cannulating flow probes[2] (EMP-300 I.D. $^1/_{16}$ and $^1/_8$ inches) and a dual-channel flowmeter manufactured by Carolina Medical Electronics, Inc. Lateral blood pressure was recorded from a port in the blood flow probe on the alar artery using a Statham blood pressure transducer. Blood volume was determined by the RIHSA[3] isotope dilution method using a Volémetron[4] instrument set on the 0.5 l scale and using 2.0 ml specimen tubes. Preliminary experiments revealed that optimal mixing of RIHSA in the goose occurred by 4 min post injection time. Therefore, this dose-mixing time was considered as a minimum in all subsequent volume determinations. Erythrocyte volumes, at each blood volume sample time, were calculated from micro-hematocrit and blood volume determinations. Plasma sodium and potassium concentrations were determined at each blood volume sample time on freshly drawn plasma using an internal lithium standard flame photometer[5]. Plasma osmotic concentrations were determined using a microvolume Advanced Osmometer[6].

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[4] Volémetron BV-3 from Ames Atomium Inc., Billerica, Mass.
[5] Flame Photometer, Model 142, Instrumentation Laboratories; Boston, Mass.
[6] Advanced Osmometer, Type 9413. Advanced Instruments, Inc., Newton-Heights, Mass.

Intravenous osmotics in the goose ± S.E. (n = 7)

Period	Time (min)	B.P. (mm Hg)	Heart rate (per min)	Total carotid flow (ml/min)	Alar flow (ml/min)	Blood vol. (ml)	Vol. RBC (ml)	Plasma Na(mEq/l)	Plasma K(mEq/l)	Plasma mOsmol.
Control	−2	89.5[a] ± 4.2	334[a] ± 8.2	17.3[a,b] ± 1.2	7.4[a,b] ± 1.0	288[a] ± 6.0	107[a,b] ± 8.5	152[a] ± 3.1	3.8[a] ± 0.1	314[a,b,c] ± 4.8
NaCl	0									
(10 ml, 10%)	4	95.5[a] ± 5.3	352[a] ± 9.1	31.0[a] ± 2.4	26.0[a] ± 2.2	415[a] ± 5.1	129[a] ± 5.7	159 ± 3.2	3.5 ± 0.2	327[b] ± 5.1
	8	76.5 ± 3.8	341 ± 7.3	21.5 ± 2.2	15.3 ± 2.1	391[a] ± 4.3	137[b] ± 5.2	170[a] ± 3.8	4.1 ± 0.3	342[c] ± 5.2
	16	73.0 ± 2.9	348 ± 6.4	17.5 ± 1.5	15.3 ± 1.9	282 ± 3.2	100 ± 4.1	158 ± 3.2	4.4 ± 0.2	353 ± 5.4
	36	76.7 ± 3.9	322 ± 8.3	21.5[b] ± 2.0	13.1[b] ± 1.8	287 ± 3.4	82.0 ± 3.2	155 ± 3.1	5.1[a] ± 0.4	357[a] ± 5.3
Control	40	75.0[b] ± 2.8	325[b,c] ± 7.7	21.3[c] ± 1.9	13.2[c] ± 1.5	290[b] ± 3.3	90 ± 3.7	147 ± 2.7	4.0 ± 0.3	320[b] ± 4.6
Sucrose	41									
(10 ml, 20%)	45	81.5[b] ± 4.1	350[b] ± 6.9	28.5[c] ± 2.6	19.8[c] ± 2.1	378[b] ± 4.1	95.0 ± 3.9	149 ± 2.8	4.0 ± 0.3	318 ± 4.6
	49	75.2 ± 3.8	358 ± 5.3	23.0 ± 2.4	15.6 ± 1.7	444 ± 5.1	134[b] ± 4.3	153 ± 3.2	3.2 ± 0.2	331[b] ± 4.9
	57	71.4 ± 3.7	350 ± 9.1	23.0 ± 2.2	15.6 ± 1.5	444[b] ± 4.1	134 ± 3.7	153 ± 2.7	3.2 ± 0.3	318 ± 4.5
	77	72.3 ± 3.8	369[c] ± 6.6	16.3 ± 1.7	7.5 ± 1.1	–	–	153 ± 2.9	3.5 ± 0.1	321 ± 5.0
Statistical evaluation		[a] $p < 0.95$ [b] $p < 0.95$	[a] $p < 0.2$ [b] $p < 0.2$ [c] $p < 0.005$	[a] $p < 0.001$ [b] $p < 0.01$ [c] $p < 0.1$	[a] $p < 0.001$ [b] $p < 0.02$ [c] $p < 0.05$	[a] $p < 0.001$ [b] $p < 0.001$	[a] $p < 0.1$ [b] $p < 0.02$	[a] $p < 0.005$	[a] $p < 0.01$	[a] $p < 0.005$ [b] $p < 0.2$ [b] $p < 0.01$

The data from the 7 geese are found in the Table. The control period data were collected 2 min before the i.v. sodium chloride (NaCl) osmotic load was given at 0-time. The blood pressure (BP), heart rate, total carotid flow, alar flow and blood volume measurements were made at selected 4 min intervals for 36 min. Blood samples were secured for determinations of erythrocyte (RBC) volume, plasma sodium (Na) and potassium (K). At 41 min the i.v. sucrose load was given and the same parameters as those listed for sodium chloride were measured at selected 4 min intervals for up to 77 min total time. Examination of the data in the Table and statistical evaluation shows no significant difference in the blood pressure or heart rate until one compares the heart rate value at the end of the sodium chloride period (36 min) with that at the end of the sucrose period (77 min). It is difficult to explain the high heart rate at 77 min in view of the other values obtained at this time. The total carotid blood flow increased significantly only briefly at 4 min following both the sodium chloride and sucrose osmotic loads. However, the alar (wing bed) flow shows a highly significant increase throughout the sodium chloride period and a further small but significant increase in the first 4 min period of the sucrose period.

The blood volume increased significantly during the first 2 sample periods of the sodium chloride period and the first 3 samples measured during the sucrose period. Sucrose thus caused a more prolonged elevation in blood volume than sodium chloride. Erythrocyte volumes reach their highest values in the period just following the maximal increases in blood volume. The hypertonic solution may have caused red blood cell damage, though no visual evidence of hemolysis was seen in the plasma. This increase in erythrocyte volume probably demonstrates the ability of the goose to release stored erythrocytes. The values for plasma sodium (Na) demonstrates the rapid equilibration of sodium chloride with the interstitial space. At 4 min following the administration of 1.0 g of the sodium chloride solution, the plasma sodium is not significantly increased. Whether the significantly higher value of 170 mEq/l at 8 min represents a rebound event, we cannot state with certainty. It is interesting to note that the sodium re-enters the vascular space to maintain a constant plasma sodium in the face of expanding blood volume during both periods. Finally the plasma potassium (K) values show a progressive increase which becomes highly significant at the end of the sodium chloride period. This increase may be related to erythrocyte damage and release of erythrocyte potassium into the plasma. Plasma potassium values during the sucrose period suggests that sucrose may be causing a clearance of potassium from the plasma. This might indicate that sodium ion or ionic strength is more important than osmotic pressure in depleting potassium from erythrocytes.

Finally, measurement of plasma osmotic concentrations show significant increases above control values from 8 min throughout the remainder of the sodium chloride period. Also, the osmotic concentration increased significantly in the sucrose period for a short time at the 8 min post-injection time (49 min). It is interesting to note that when osmotic concentration attains a significant increase, erythrocyte volume and plasma sodium also reach significantly higher levels than their respective controls in the sodium chloride period. Further, these events were subsequent to

significant increases in the parameters measured with the exception that plasma sodium did *not* increase significantly along with erythrocyte volume and osmotic concentration. Therefore, the first event likely to initiate secretion from the salt gland is an expanding blood volume. However, osmotic concentration may be involved also.

In order to obtain some comparable data in another species, the response of the same variables in 2 dogs to i.v. sodium chloride and sucrose osmotics was studied. The increases in blood volume to equivalent doses of the above osmotic solutions in the dogs were only about one-half as great as those observed in the geese. Also, the period of increased blood volume measurements in the dogs rarely lasted past 10 min whereas in the geese, especially with sucrose loading, the blood volume measurement lasted at least twice as long.

Other experiments in this laboratory indicate that the unanesthetized goose starts salt gland secretion 3–5 min following administration of 10 ml of 10% sodium chloride, i.v. and that the secretion always lasts more than 60 min[7].

Therefore, the evidence suggests that the changes in blood volume caused by the hyperosmotic solutions used constitute the most probable stimulus for initiation of the secretory response from the avian salt gland. The long duration of the secretory response, however, cannot be explained by our data.

Zusammenfassung. Die Wirkung auf Blutdruck, Puls, Durchblutung, Blutmenge, Plasma-Kationen und osmotischen Druck zu i.v. hypertonischem NaCl und Saccharose im Laufe der Zeit wurde in bestimmten Zeitabständen bei 7 anästhetisierten Gänsen, die der Salzdrüsensekretion fähig sind, gemessen. Die Analyse der Reaktion führt zu der Annahme, dass die Zunahme der Blutmenge und/oder die osmotische Konzentration die primären Ereignisse sein könnten, die die Sekretion der Salzdrüsen auslösen.

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TERMINOLOGIA

'Chiral-Optical Effects', a Common Term for Both ORD and CD

More and more often, the 2 closely related phenomena, optical rotatory dispersion (ORD) and circular dichroism (CD), have to be discussed together in the scientific literature. Such discussions could be much simplified if a brief common term were available which would cover the essential aspects of both phenomena. It seems that 'chiral-optical effects' would be a suitably short and descriptive expression, which would include both ORD and CD (also, if desired, the magnetically induced ORD and CD, i.e. the Faraday effect) and would thus be useful. We therefore wish to propose its adoption. The desirability of having available a common term for both ORD and CD was discussed with Prof. A. DREIDING during a stay in his laboratory at the University of Zürich in 1966; Prof. DREIDING has now informed me that he has used a very similar term in his lectures.

Zusammenfassung. Es wird vorgeschlagen, die Terminologie auf dem Gebiet des Zirkulardichroismus und der Rotationsdispersion durch Einführung der gemeinsamen Bezeichnung «Chiral-optische Effekte» zu vereinfachen. Auch die analogen magneto-optischen Phänomene, d.h. der Faraday-Effekt, können in den vorgeschlagenen Ausdruck einbezogen werden.

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CONGRESSUS

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Like the first one, the Second International Symposium of Radioprotective Drugs will be under the auspices of the European Society for Biochemical Pharmacology. The Symposium is planned to provide an opportunity for the exchange of information on recent advances in the field of radiation protection and sensitization. Further information may be obtained from either one of the following scientific secretaries of the Symposium: Dr. H. Moroson, Sloan Kettering Institute for Cancer Research, Donald S. Walker Laboratory, 145 Boston Post Road, Rye, New York, USA. Dr. M. Quintiliani, Istituto Superiore di Sànità, 299, Viale Regina Elena, Roma 00161, Italia.