only from animals which were quiescent throughout the period of measurement. A layer of mineral oil on the floor of the chamber trapped any droppings and prevented distortion of the data by evaporation of water from this source. Chamber temperature (T_a) was monitored within $\pm 0.2°C$, and relative humidity in the chambers was estimated by the use of mixing ratios[3]. The rate of O_2 consumption was computed from appropriate equations[10], and the ratio of evaporative heat loss to heat production, hereafter called the e/p ratio, was calculated on the basis of 0.58 g-cal/mg of water evaporated and 4.8 g-cal/cc O_2 consumed. It was not feasible to measure body temperature (T_b) and respiratory exchange concurrently. In conditions duplicating the respiratory measurements but employing a quick-access chamber, core body temperature was determined to $\pm 0.05°C$ by a fine thermocouple inserted 1 cm into the cloaca. Measurements were made in a darkened room within 50 sec after opening the chamber.

Results and Discussion. The data in the Table show that the zebra finch has a capacity for evaporative cooling (maximum e/p = 1.37) greater than that known for any other passerine. These species attain maximum e/p ratios of ca. 0.5 or less when $T_a \leq 40°C$[4,11], and often exhibit decreasing e/p ratios at higher T_a, at which evaporative capacity is maximal but metabolic rate continues to increase with hyperthermia. The zebra finch shows a similar cooling capacity at $T_a \leqq 40°C$, but a continued exponential increase to at least $T_a = 43.3°C$. At this T_a, and at any greater than 36.5°C, the birds sustain a moderate hyperthermia which maintains a positive gradient for heat transfer, or at least minimizes heat loading when $T_a > T_b$. Thermoregulatory performance at higher T_a was not systematically investigated because of apprehension about heat death of irreplaceable birds of the imported stock, but at mean $T_a = 43.9°C$, mean T_b of 6 birds was $43.4 \pm 0.1°C$; a maximum difference of 1.2°C was observed in one bird at $T_a = 44.2°C$.

Pulmocutaneous water loss, evaporative cooling (e/p), and cloacal temperature (T_b) as functions of ambient temperature (T_a)

T_a °C	% R.H.	T_b °C	Pulmocutaneous evaporation mg H_2O/cm^3 O_2	e/p
20.4	21	39.8 ± 0.4[a]	1.93 ± 0.06[a]	0.23
30.3	10	40.8 ± 0.2	2.39 ± 0.06	0.29
36.5	7	41.5 ± 0.2	3.50 ± 0.31	0.42
40.0	8	42.4 ± 0.1	4.35 ± 0.33	0.53
43.4	24	43.3 ± 0.1	11.31 ± 1.06	1.37

[a] Mean ± standard error.

The standard metabolic rate of the zebra finches was 3.28 ± 0.12 cm^3 $O_2/g \times h$ (mean body weight = 11.7 g), compared with a predicted weight-relative value of 3.17 $cm^3/g \times h$[11]. The thermoneutral range extended from 30°C to ca. 40°C; metabolic rate increased to 4.86 ± 0.40 $cm^3/g \times h$ at $T_a = 43.5°C$. Heat production as a function of ambient temperature is thus similar to that of other passerines of comparable size and does not exhibit the adaptive diminution found in certain caprimulgids[7]. The unusually great capacity for evaporative cooling in the zebra finch results, rather, from a relatively large capacity for pulmocutaneous evaporation of water.

Zusammenfassung. O_2-Verbrauch und Gesamt-Wasserverdunstung wurden bei australischen Zebrafinken gemessen. Bei geringem Wärmeaustausch zwischen Körper und Umgebung durch Strahlung, Konvektion und Ableitung können diese Wüstenvögel den für die Wärmebilanz erforderlichen Wärmeabfluss einzig durch Verdunstung auf dem normalen Niveau halten. Eine solche Fähigkeit ist bei Passeres bisher nicht beobachtet worden.

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Spinal Cord Transection and CCl_4-Toxicity

In rats, cervical transection of the spinal cord markedly reduces the hepatotoxic effects of a single dose of CCl_4[1–4]. Spinal cord transection has manifold effects on the animal. The manner in which it protects in CCl_4 intoxication has not been fully elucidated[4,5], although elimination of sympathetic centers in the brain has been suggested[1,2].

We have observed that as the level of cord transection is lowered, the degree of protection conferred diminishes[3,4]. Furthermore after cervical transection the rats become poikilothermic; this response also becomes less marked as the level of transection is lowered. These observations prompted an investigation into the possible role of hypothermia as a factor in the protective effect of spinal cord transection.

Male Holtzman rats weighing from 180 to 260 g were used throughout the study. The animals were subjected to light ether anesthesia for spinal cord transection at the level of C-7. Twelve cord-sectioned rats were placed in an incubator (34°C) immediately after surgery. These were to be compared with a second group of twelve similarly sectioned rats maintained at ambient laboratory temperatures (about 24°C). 1 h after surgery both groups received 0.625 ml/kg CCl_4, p.o. 24 h later liver sections were taken and fixed in cold, neutral-buffered formalin for histological examination[6]. The level as well as the complete-

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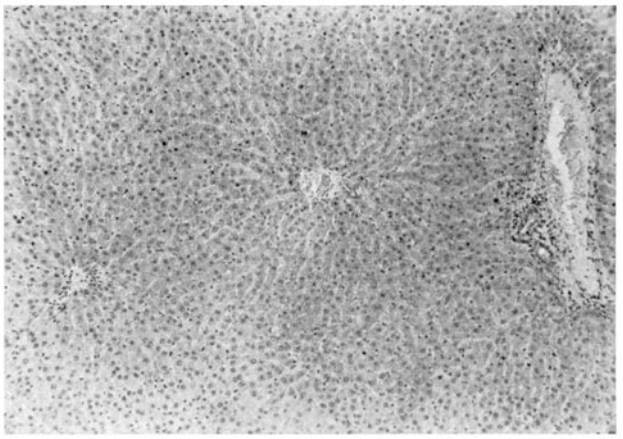

Fig. 1. Liver section from cord-transected rat (maintained at room temperature) 24 h after CCl_4. H. & E. stain, 55 ×.

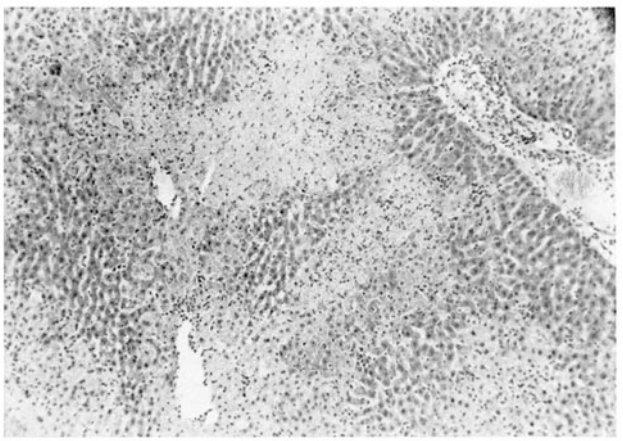

Fig. 2. Liver section from cord-transected rat (maintained at 34°C) 24 h after CCl_4. H. & E. stain, 55 ×.

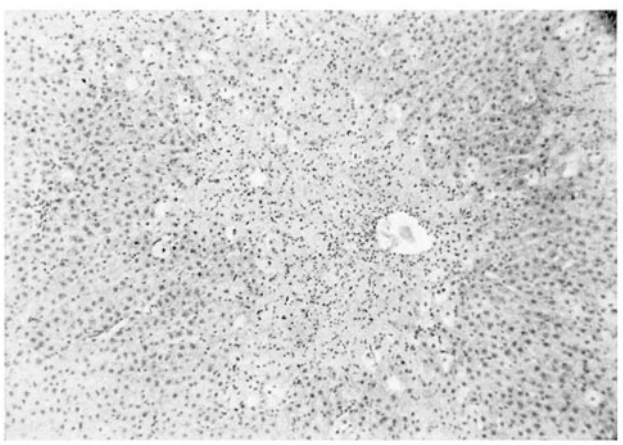

Fig. 3. Liver section from cord-transected rat (maintained at room temperature) which had received 3 doses of CCl_4 12 h apart. H. & E. stain, 55 ×.

ness of the transection were verified by *post mortem* examination. At the time of sacrifice the rectal temperatures of the cord-sectioned, incubated rats were from 35 to 36°C while those of the rats maintained at room temperature were from 24 to 25°C. Other simultaneously run controls consisted of: (1) non-sectioned, CCl_4-treated rats kept at either incubator or laboratory temperature, and (2) sectioned and non-sectioned rats kept at either temperature but given corn oil alone. The non-sectioned rats under all conditions maintained rectal temperatures of from 36 to 38°C.

The CCl_4-treated, cord-sectioned rats, maintained at room temperature, were protected in that their livers showed only minimal changes (i.e. scattered, fine vacuolization and slightly decreased basophilia in the centrilobular area), as seen in Figure 1. On the other hand, the livers of the CCl_4-treated, cord-sectioned, incubated rats showed severe centrilobular necrosis (Figure 2). Thus the development of the lesion after CCl_4 ingestion was apparently related to the animals' body temperature. Incubation of the non-sectioned rats did not potentiate the effects of CCl_4; this is consistent with the hypothesis since their body temperatures were the same under both conditions.

A possible explanation for the protection seen could be that the metabolic activity of the liver was reduced by hypothermia. If this were true we thought that it might be possible to produce hepatic necrosis in cervically transected animals kept at room temperature by prolonging the exposure of the liver to CCl_4. Accordingly, twelve C-7 transected rats received CCl_4 1 h after surgery as before. In addition they received second and third doses 12 and 24 h later. 24 h after the third dose liver sections were taken as described above. These animals had been maintained at room temperature (24°C) for the entire 48 h period. At about 10 h after surgery their rectal temperatures were from 24 to 25°C and remained so until sacrifice. The livers of these rats showed the severe centrilobular necrosis and bands of swollen vacuolated cells typical of the picture seen in livers of non-sectioned rats 24 h after CCl_4 (Figure 3). No necrosis was seen when a single dose of CCl_4 was administered 24 h after cervical transection and the animals (maintained at room temperature) sacrificed 24 h after dosing.

Reduced metabolic activity resulting from hypothermia following cervical cord-transection is strongly implicated by these results as an important factor in the protection afforded by this surgical procedure against CCl_4 hepatic necrosis. It is difficult to reconcile these results with the hypothesis of a primary action of CCl_4 on sympathetic centers in the brain, since in the present studies sympathetic outflow had been mechanically eliminated and necrotic changes were still produced[7].

Zusammenfassung. Typisch CCl_4-verursachte Leberschäden wurden bei Ratten mit Rückenmarkdurchtrennung und Körpertemperaturen von 35 bis 36°C beobachtet. Minimale histopathologische Veränderungen wurden bei gleich behandelten Ratten bei Zimmertemperatur beobachtet. Verlängerung der Beobachtungszeit an spinalisierten ausgekühlten Ratten lässt aber auch bei Zimmertemperatur typische Leberverletzungen entstehen. Diese Ergebnisse deuten auf Veränderung des Leberstoff-

[7] This study was supported in part by U.S.P.H.S. Research Grant A-5802 and U.S.P.H.S. Training Grant 2G-141.

wechsels durch Hypothermie hin, und es ist daher anzunehmen, dass dies die Ursache ist, weshalb die Leber von Ratten mit Rückenmarkschnitt gegen CCl_4-Schäden relativ geschützt ist.

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DISPUTANDUM

Ferrous Complexes in the Catalase Reaction

In a recent article[1] I have pointed out that polarographic evidence exists for the formation of a complex between ferrohaem and hydrogen peroxide.

This finding forms an analogy (or precedent) for one of the five reactions postulated by WESTHEIMER[2] to explain the catalatic decomposition of hydrogen peroxide and thus makes this mechanism *ceteris paribus* more likely. It certainly does not prove the WESTHEIMER mechanism. NICHOLLS[3] has recently criticized WESTHEIMER's mechanism, and to this criticism I feel it is not my task to reply.

However, to further weaken the mechanism proposed by WESTHEIMER, NICHOLLS has also attacked the polarographic evidence for the formation of a complex between ferrohaem and H_2O_2 and in fact, polarographic kinetics in general. This criticism is completely without foundation and has been made obviously without thorough knowledge of this field of study. The points taken are on the level of 1943 and to refute them it would be necessary to republish all the work, many times reviewed by BRDIČKA[4], which has been done since.

Nicholls claims that the oxidation of ferrohaem to hemine by H_2O_2 may be the explanation of the polarographic reaction. This has been considered and disproved. He tries to support his claim by a quotation from KOLTHOFF and PARRY[5] to the effect that there are great discrepancies between the true and polarographic rates of reaction.

This statement was made in 1951 and is based on a completely incorrect calculation of a system the treatment of which has now been clarified for years[4]. Wherever it was possible to make a direct comparison between the values of rate constants determined by conventional methods and those derived from kinetic currents the agreement was excellent[6–8].

This is also true of the specific case cited by KOLTHOFF and PARRY. The agreement here is also excellent[9] when the calculation is done properly.

Furthermore, in the case of the polarographic reaction of the hemine/H_2O_2 system no real comparison of rates is involved. There is no doubt that the polarographic reaction is an 'extremely rapid reaction', i.e. under conventional conditions instantaneous. On the other hand, many ferrohaem complexes which give the polarographic reaction are oxidized by H_2O_2 very slowly or not at all.

Zusammenfassung. Vor kurzem wurde von NICHOLLS der WESTHEIMER-Mechanismus des H_2O_2-Zerfalls durch Katalase mit Argumenten einer unseres Erachtens abwegigen Interpretation der polarographischen Kinetik kritisiert. Die Argumente werden durch Hinweise auf unberücksichtigte Arbeiten widerlegt.

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PRO EXPERIMENTIS

Zur Registrierung von Rückatmung in Nicht-Rückatmungsventilen (Non-Rebreathing Valves)

Das Nicht-Rückatmungsventil hat bei der Beatmung die Aufgaben, das mit dem Inspirationsdruck zugeführte Gasgemisch in die Luftröhre zu leiten; während der Exspiration soll das aus den Alveolen zurückströmende Gasgemisch nicht in das Beatmungssystem gelangen, sondern in den umgebenden Raum geleitet werden. Als Regelgrössen für die Steuerung des Ventils wirken die von der Atemphase abhängigen Druckgradienten. Je genauer das Beatmungsventil Ausatmung und Einatmung voneinander trennen kann, desto besser ist das Ventil. Die Möglichkeit, dass bei einigen Ventilen diese Anforderung nicht genügend erfüllt ist, schien uns grundsätzlich gegeben, so dass wir uns veranlasst sahen, die Arbeitsweise von Nicht-Rückatmungsventilen zu registrieren.

Beobachten wir die Arbeitsweise eines Ventils bei Beatmung im einzelnen und verfolgen wir gleichzeitig den Druck in seinen drei Anschlußstutzen (A inspiratorische Seite, B Patientenseite, C exspiratorische Seite), so ergeben sich folgende Bilder (Figur 1a).

Zu Beginn der Inspiration steigt auf der Inspirationsseite der Druck, bis die Feder- oder Magnetkraft überwunden ist, die den Ventilstempel in Exspirationsstellung fixiert; nach Öffnung der Inspirationsseite steht der Stempel zunächst in einer Mittelstellung und die Luft strömt sowohl zum Patienten als auch zum Ausatmungsstutzen. Ein gewisser Teil des Atemgases geht damit verloren; das spielt hinsichtlich des Beatmungsausmasses des Patienten keine Rolle. Hat der Stempel seine Inspirationsstellung erreicht (Figur 1b), strömt während der übrigen