which are scattered in the connective tissue and between muscle cells. According to our present observations, these macrophages contain fewer inclusion-bodies than those in regressing tails.

The evidence obtained from enzyme histochemistry leads to the conclusion that the increase in activity of acid phosphatase and cathepsins is due to the local activation of macrophages in the regressing tail. This view is also supported by our observation[9] that during tail atrophy there is a change in the pattern of cathepsins. It is therefore conceivable that the tail macrophages are the sites of this activity. Future work will be directed to the elucidation of those events which initiate the activation of macrophages in the tail tissue[10].

Zusammenfassung. Histochemische Reaktionen an regredierenden Schwänzen von Krallenfroschlarven haben ergeben, dass die saure Phosphatase und die kathepsinartigen Esterasen vorwiegend in Makrophagen des subepidermalen Bindegewebes angereichert sind. Somit dürfte die erhöhte Aktivität dieser Fermente im regredierenden Schwanzgewebe auf der lokalen Aktivierung von Makrophagen beruhen.

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[9] R. WEBER, in preparation.

[10] We are greatly indebted to Dr. R. HESS (CIBA Basel) for generous advice on histochemical techniques. Our thanks are also due to Prof. E. LÄUPPI (Gerichtlich-medizinisches Institut, Bern) for permission to use his cryostat and to Mr. B. NICK for assistance in microphotography.

Reactivity of Cat Skeletal Muscle Vessels to Noradrenaline During Induced Respiratory Alkalosis

Several workers[1-3] have shown that alterations in the acid-base balance of the blood will change the pressor responses to i.v. adrenaline or noradrenaline injections. Usually, uncompensated acidosis diminishes and uncompensated alkalosis increases the pressor responses. However, the site and mechanisms of these effects are to a large extent still obscure[4]. The present study shows that the overall response of a peripheral vascular bed, consisting mainly of skeletal muscle vessels (the skinned hind leg of the cat) to noradrenaline increases during uncompensated respiratory alkalosis. The results suggest further that this increase is primarily due to the increase in pH rather than the decrease in carbon dioxide tension.

Methods. Ten cats (weighing 2.0–4.3 kg), anaesthetized with pentobarbital, were used. One leg was tied off at the upper part of the thigh with only the large vessels and nerves remaining untouched. This leg was subsequently skinned and the paw tied off just above the ankle joint, leaving the femoral artery to perfuse the skeletal muscles. The blood in the femoral artery was exteriorized in a polyethylene loop and the flow maintained by means of a constant output pump (Sigmamotor Inc., U.S.A.). Changes in perfusion pressure during the course of an experiment thus reflected changes in the vascular resistance of the perfused region. The preparation also included resection of the ipsilateral sympathetic chain at the midlumbar level and the splanchnic nerves on both sides.

The following physiological functions were continuously recorded on a Grass polygraph: (1) systemic blood pressure (femoral artery), (2) perfusion pressure (in the loop, distal to pump), (3) arterial pH (in the loop, proximal to pump) and (4) carbon dioxide tension (in end tidal air). The blood pressures were recorded via transducers (Statham Inc., U.S.A.). The arterial pH was recorded via a pH-meter (PHM 22, Radiometer Inc., Denmark). End tidal carbon dioxide tension was determined by drawing the expired air through a gas-analyzer (Beckman Spinco model LB-1). Spontaneous respiratory movements were abolished by a slow i.v. drip of succinylcholine iodide (Celocurin, Vitrum Inc., Sweden) in a low-molecular weight dextran (Rheomacrodex Mw 40000, Pharmacia Inc., Sweden) and the animals were ventilated artificially.

The animals were initially ventilated with oxygen and the degree of the ventilation was adjusted to maintain pH approximately 7.4. Blood flow to the perfused leg was such that the perfusion pressure equalled the systemic blood pressure. The animals were, at this stage, in a state of compensated metabolic acidosis with a low carbon dioxide tension in the end tidal air. This was later compensated by a slow i.v. infusion of 0.6 N sodium bicarbonate in an amount sufficient to restore the normal carbon dioxide tension of 40 mm Hg. To maintain a constant ventilation and blood pH, at the same time, the percentage of carbon dioxide in the inspired gas was increased. Later respiratory alkalosis could easily be produced by again administering oxygen. With this experimental procedure it was possible to follow the peripheral effect of i.a. noradrenaline injections during three different types of alterations in the acid–base balance: (1) Compensated metabolic acidosis with a low carbon dioxide tension and a normal blood pH; (2) 'Normal' stage with a normal carbon dioxide tension and normal blood pH; (3) Uncompensated respiratory alkalosis with a low carbon dioxide tension and a high blood pH.

To obtain larger changes in the carbon dioxide tension, the compensated metabolic acidosis (1) was aggravated in some experiments by a slow infusion of 5–10 cm³ 0.2 N hydrochloric acid i.v.

The effect of noradrenaline in different doses (administered i.a. in the loop) on the perfusion pressure was tested repeatedly during the experiments, and the recorded changes were expressed in two different ways: (1) The percentual change in the response of a noradrenaline dose sufficient to produce a 50 mm Hg increase of the perfusion pressure at stage 2; (2) The relative increase or decrease of the dose required to maintain a constant 50 mm Hg pressure response.

Results. In 8 experiments out of 10, uncompensated respiratory alkalosis increased the overall response of muscular blood vessels to i.a. injections of noradrenaline. An increase of the pH to approximately 7.56 (5 cats) increased the response of an i.a. noradrenaline injection to an average of 114%. A more severe alkalosis (pH 7.80; 5 cats) increased the response to 132%. The dose necessary

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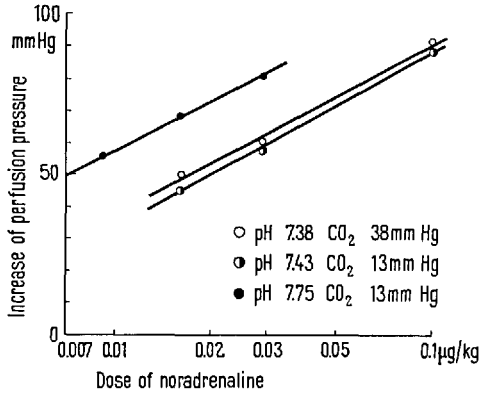
to elicit a 50 mm Hg response could at the same time be decreased to 0.71 and 0.45 of the control dose. The mean carbon dioxide tension in the end tidal air decreased from 40 to 19 and 13 mm Hg respectively. A corresponding fall in carbon dioxide tension at a constant pH, on the other hand, caused a decrease of the noradrenaline effect in 4 cats and an increase in three cats. Only in 1 of these 3 cats was the increase similar to that obtained during a subsequent alkalosis.

Dose-response curves in a typical experiment are given in the Figure. Only uncompensated respiratory alkalosis was accompanied by a significant change. The control response of 50 mm Hg increased during the alkalotic period to 68 mm Hg and the dose producing a 50 mm Hg increase of the perfusion pressure could be decreased from 0.018 μg/kg to 0.0076 μg/kg.



Cat 4.3 kg. Increase of perfusion pressure following i.a. injections of noradrenaline to the skinned hind leg perfused at a constant rate of flow (7.2 ml/min). o—o normal acid-base balance; ◗ – ◗ compensated metabolic acidosis; ● – ● uncompensated respiratory alkalosis.

The average increase of the pressure responses during alkalosis appeared to be of the same magnitude per pH unit as the decrease earlier described during respiratory acidosis[5].

Conclusions. Respiratory alkalosis increases the overall response of skeletal muscle vessels to noradrenaline. An average pH change of 0.2 units is required to induce a noticeable change. The present results also suggest that the increase is primarily due to the pH change and not to the concomitant decrease in carbon dioxide tension.

A more detailed report of these studies will appear elsewhere[6,7].

Zusammenfassung. Die respiratorische Alkalose steigert den Effekt des Noradrenalins auf Skelettmuskelgefässe. Eine durchschnittliche Steigerung des pH von 0.2 Einheiten wird benötigt, um eine merkliche Änderung herbeizuführen. Die vorliegenden Resultate lassen vermuten, dass die Steigerung in erster Linie auf die pH-Veränderung zurückzuführen ist und nicht auf die gleichzeitige Senkung der CO_2-Spannung.

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Department of Physiology, Karolinska Institutet, Stockholm (Sweden), March 25, 1963.

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[7] Research grants from The Swedish National Association for Heart and Chest Diseases and from Knut and Alice Wallenbergs Foundation are gratefully acknowledged.

Einfluss der Temperatur auf den Vitamin-C-Gehalt während des Wachstums von englischem Spinat (*Rumex patientia* L.)

Zur Frage der Abhängigkeit des Vitamin-C-Gehaltes verschiedener Spinatsorten von Jahreszeit und Vegetationsperiode konnten in Freilandversuchen für die Praxis der menschlichen Ernährung zwar aufschlussreiche Ergebnisse gewonnen werden; infolge der sich dauernd ändernden Umweltbedingungen ist es jedoch hierbei praktisch unmöglich, alle die Faktoren zu ermitteln, die für eine Veränderung des Vitamin-C-Gehaltes während des Wachstums der Pflanze verantwortlich sind. Das Arbeiten in Klimakammern (Phytotron) gestattet, alle Aussenbedingungen bis auf eine konstant zu halten und das Pflanzenmaterial so unter einem ganz bestimmten Gesichtspunkt während einer Vegetationsperiode zu untersuchen. Die Ergebnisse von Versuchen dieser Art gelten natürlich nur für die jeweils eingehaltenen Versuchsbedingungen und lassen sich nicht ohne weiteres auf praktische Verhältnisse übertragen. Nachfolgend wird über Ergebnisse von Versuchen berichtet, in denen der Einfluss der Temperatur auf den Gehalt an Vitamin C in Englischem Spinat überprüft wurde.

Die Versuche wurden im Phytotron der landwirtschaftlichen Fakultät der Universität Giessen in Rauisch-Holzhausen[1] durchgeführt.

Durchführung der Versuche: Als Pflanzenmaterial wurde *Rumex patientia* L. gewählt, eine Gemüsepflanze aus der Familie der Polygonaceen, die sich einmal durch eine kurze Keimdauer und zum anderen durch einen hohen Vitamin-C-Gehalt auszeichnet[2]. Die Aussaat erfolgte am 5. Juli 1962 in zwei verschiedenen Klimakammern unter den folgenden Bedingungen:

1. Belichtungszeit: 17 h am Tage. Belichtungsstärke: 14000 Lux maximal. Tagestemperatur: 20°C; Nachttemperatur: 5°C.

2. Belichtungszeit und Belichtungsstärke wie bei 1. Tages- und Nachttemperatur konstant 20°C.

Zum Vergleich wurde im Freiland eine dritte Versuchsreihe mit dem gleichen Pflanzenmaterial zur gleichen Zeit angesetzt.

Die Vitamin-C-Bestimmungen wurden bei den 3 Versuchsreihen zweimal pro Woche parallel durchgeführt. Etwa von der vierten Untersuchungswoche an wurden, als es die Menge des Untersuchungsmaterials zuliess, Blattstiel und Blattspreite getrennt untersucht. Um den Vitamin-C-Gehalt mit den einzelnen Stufen des Pflanzenwachstums vergleichen zu können, wurden während der

[1] Herrn Prof. Dr. v. Boguslawski, dem Direktor des Instituts für Pflanzenbau und Pflanzenzüchtung der Univers tät Giessen, danke ich verbindlichst für die Möglichkeit, im Phytotron seines Instituts arbeiten zu dürfen, Herrn Kustos Dr. Brettschneider-Herrmann für die freundliche Beratung und Überwachung der Klimaversuche.

[2] A. Seybold und H. Mehner, *Über den Gehalt von Vitamin C in Pflanzen*. 10. Abhandlung, Tab. 27 (Springer Verlag, Heidelberg 1948).