intensity was relatively high. Polarized UV light (with horizontal e-vector[5]) is thus distinguished from unpolarized UV light in an intensity-invariant manner. In the field the intensity of the UV component of the sky light is always several times higher than the intensity of the UV light reflected from water surfaces. It is therefore plausible that the dive into the water, the plunge reaction, is elicited exclusively by the polarized component of the reflected UV light.

The threshold intensity I_{thr} was found to be 1.5×10^{-7} W/cm^2. The UV light with which this result was obtained had $\lambda_{max} = 360$ nm, with a half-height width of 40 nm. The experiments employed animals that had been illuminated before the test with incandescent-bulb light containing little UV. Control animals, illuminated for half an hour before the flight by two additional 40-W UV fluorescent lamps, did not have higher thresholds. That the intensities naturally encountered by the animal are above this level is demonstrated by measuring the intensity of P in the laboratory at I_{thr} and that of an outdoor water surface on a cloudless summer afternoon through a Schott UG11 filter in combination with a Käsemann P-UV2 polarization filter with horizontal direction of transmission; the transmission properties of this filter combination correspond approximately to the absorption properties of the UV retinula cells of *Notonecta* with horizontal analyzer direction[7]. The results indicate that the light reflected from the water surface at an angle of for example 30° from the vertical is 100 times as intense for the UV receptors with horizontal analyzer as the polarized light in the laboratory at I_{thr}.

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Cerebrovascular reactivity to CO_2: modulation by arterial pressure[1]

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Summary. Cerebrovascular reactivity to CO_2 (CO_2R), measured in halothane-anesthetized rabbits, decreased as arterial pressure was increased either pharmacologically or mechanically. On the other hand, hypotension, induced by bleeding, led to an increase in CO_2R. These responses were unaffected by denervation of baroreceptors.
Key words. cerebral blood flow; arterial pressure; CO_2 reactivity; baroreceptors; halothane; rabbit.

We previously reported[3] that halothane-anesthetized rabbits whose mean arterial pressure (MAP) is at or above 70 mm Hg show less cerebral vasodilatation in response to hypercapnia than do those whose MAP is at a lower level. The present experiments were designed to test whether this difference in CO_2 reactivity (CO_2R) was causally, rather than coincidentally, related to MAP, and to investigate the possible role of arterial baroreceptors in the response. Accordingly, CO_2R was tested when MAP was altered by both pharmacological and mechanical means, and the role of the baroreceptors was assessed by testing before and after section of the sinus and vagus nerves.

Methods. Male New Zealand rabbits weighing 2.5–3.5 kg were initially anesthetized with 3.5% halothane in O_2 administered by mask, and then maintained on 1% halothane throughout the experiment. A femoral artery and an auricular vein were cannulated for recording of MAP and administration of drugs, respectively. The animals were tracheostomized, paralyzed by a continuous i.v. infusion of pancuronium bromide at 0.4 mg/h and mechanically ventilated to achieve an initial end-tidal CO_2 ($ETCO_2$) of approximately 3%, as measured by a Beckman LB-2 gas analyzer. From this low value, $ETCO_2$ was set at the desired level by adding CO_2 to the inspired gas mixture. Respiratory anesthetic concentration was monitored continuously with a mass spectrometer or with a Beckman infrared LB-2 analyzer with a suitable pick-up head.

Internal carotid blood flow (ICBF) was measured with a 1.0–1.5 mm noncannulating electromagnetic flowmeter probe and a Biotronex 410 flowmeter. The probe was placed on the left common carotid artery after ligation of the external carotid artery and any extracerebral branches from the internal carotid. A hydraulic occluder[4] was placed around the artery distal to the flow probe to provide mechanical zero calibration. The detailed procedure, as well as a validation of ICBF in the rabbit as an estimation of cerebral blood flow, is described elsewhere[5]. Mean ICBF, MAP and respiratory CO_2 concentration were recorded continuously on a Grass Model 5 Polygraph.

MAP was adjusted downwards by bleeding from the arterial line, or upwards either by i.v. infusion of phenylephrine (0.3–2.5 $mg \cdot kg^{-1} \cdot h^{-1}$) or by inflation of a no. 5 Swan-Ganz catheter advanced through the femoral artery into the thoracic aorta. In the latter case, MAP was recorded through the large lumen at the tip of the catheter just above the level of the occlusion.

Before measurements of ICBF were taken, a 1-h stabilization period was allowed after completion of all surgical procedures. The dependence of CO_2R on MAP was calculated for each animal as follows. First, CO_2R was determined at the resting, undisturbed level of MAP by varying $ETCO_2$ in steps between 3

Dependence of cerebrovascular reactivity to CO_2 (CO_2R) on mean arterial blood pressure (MAP)

Groups	Intact A		B		Denervated C	
	Slope	Intercept	Slope	Intercept	Slope	Intercept
Mean	−0.15	24.5	−0.19	27.6	−0.15	31.7
SE	0.03	0.97	0.02	2.4	0.05	2.9
n	6	6	7	7	6	6

Mean values represent the average of slopes or intercepts of the regressions of CO_2R (Δ% ICBF/Δ% $ETCO_2$) on MAP (mm Hg) in the three experimental groups. MAP was lowered by bleeding in all groups; it was elevated by phenylephrine infusion in groups A and C and by an intra-aortic balloon in group B. Baroreceptors were denervated by severing cranial nerves IX and X at the neck in group C. All three slopes were significantly different from zero; neither the slopes nor the intercepts differed significantly from each other.

and 8% and recording the change in ICBF after a 2–3 min stabilization period. These paired values of $ETCO_2$ and ICBF were then used to calculate the regression equation of ICBF on $ETCO_2$, the coefficient of which represented the quantitative expression of CO_2R (Δ %ICBF/Δ %$ETCO_2$). Second, MAP was varied above and below the resting level and CO_2R determinations were then repeated for each MAP level and used to calculate the slopes and intercepts of the regression of CO_2R on MAP for the particular animal. These values were then averaged within each experimental group to yield the mean slopes and intercepts shown in the table. Four to six steps of MAP, over the range of 45–105 mm Hg, were studied in each animal. Results were assessed by analysis of variance and LSD test[6].

Results. Average CO_2R prior to interventions designed to modify MAP was 14 ± 1.5 (SE) (Δ %ICBF/Δ % $ETCO_2$). Average MAP was 78 ± 2.3 mm Hg.

Reduction in MAP by bleeding led to a progressive increase in CO_2R while increase in MAP by infusion of phenylephrine produced a corresponding reduction of CO_2R (table, A). Equivalent increase in MAP produced by aortic occlusion was accompanied by the same reduction in CO_2R as observed with infusion of phenylephrine (table, B).

Normocapnic ICBF was essentially constant over the MAP range of 60–110 mm Hg, with 10% decrease at 50 mm Hg. Thus the increase in CO_2R with low MAP was a result of a higher absolute level of ICBF with hypercapnia.

The relation of CO_2R to MAP was unaffected by baroreceptor denervation (table, C).

To give a more direct indication of the magnitude of the change in CO_2R with MAP, data from groups A, B and C were pooled and mean CO_2R was calculated at four MAP intervals: 45–60, 61–75, 76–90 and 91–105 mm Hg. Mean CO_2R's and standard errors (SE) for these intervals were 17(1.7), 15(1.6), 13(1.8) and 11(1.7), respectively.

Discussion. Conflicting observations on relation of CO_2R to MAP have been reported. In dogs, for instance, both a direct correlation of CO_2R with MAP[7] and absence of such dependence[8] have been observed. In rabbits, on the other hand, a pressure dependence of CO_2R has been described, but only for low MAP levels[9]. Since anesthetics are known to affect CO_2R[3] this divergence among reported findings may well be related to the effects of the anesthetics used (barbiturates, urethane), and the difficulty in controlling their effective levels when they are given i.v. For this reason, we used inhalation anesthesia where the anesthetic level can be finely adjusted and maintained constant.

The present results confirm our preliminary observations[3] that CO_2R is inversely dependent on MAP. Moreover, this effect is independent of whether the MAP is increased pharmacologically or mechanically, and it apparently does not involve the baroreceptor reflex, since denervation of the arterial baroreceptors did not affect the correlation of CO_2R and MAP. It is most likely that the alterations in CO_2R resulted from changes in myogenic tone of the cerebral vessels. Thus, high MAP, by increasing the stretch on the vessel walls, might enhance the vascular tone and decrease the amount of vasodilatation produced by CO_2, while low MAP would have the opposite effect. An additional consideration is that hemodynamic changes accompanying the induced alterations in MAP may also have affected the apparent CO_2R through modification of the peak arterial PCO_2 during the CO_2 test, or through a change in the effective alveolar (and plasma) concentration of the inhaled anesthetic.

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A somatotopic organization of leg afferents in the spider *Cupiennius salei* Keys. (Araneae, Ctenidae)

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Summary. Axonal anterograde degeneration after ablation of different leg segments of the spider *Cupiennius salei* was traced using LM- and EM-methods. The pattern of degeneration seen in cross sections of the leg nerves close to their entry into the subesophageal ganglion shows a somatotopic organization of afferents within the leg nerves coming from different leg segments. All afferents run through the ventral part of the nerve.
Key words. Spider *Cupiennius salei;* leg segments; axonal anterograde degeneration; somatotopic organization; leg afferents.

In a nervous system, the arrangement of axons within peripheral nerves and the patterns of axon distribution which may exist, i.e. a somatotopic organization, are of interest for the interpretation of developmental and regeneration processes. Moreover, electrophysiological studies of the activity of specific receptors and investigations of the central projections require an exact knowledge of the courses of afferents within the ascending nerves.

In arthropods only a few reports are available concerning such somatotopic organization; all of them deal with insects[3]. We have studied this problem in a spider (Arachnoidea, Araneae). Using Wallerian anterograde degeneration we examined the arrangement of afferents within the leg nerves coming from different leg segments. Information about projections of leg afferents is essential for current studies of leg reflexes in *Cupiennius*[4].