		RI (in 0.5 sec)	PT (sec)	RD (in 1 sec)	RT (sec)
so	acetic acid (1/160*M*)	14.5	0.9 ± 0.2	3.6	1.5
sa	NaCl (1/2*M*)	1.8	0.6 ± 0.3	1.9	1.35
sw	sucrose (1/2*M*)	5.7	1.3 ± 0.2	3.4	1.45
b	QHCl (1/160*M*)	7.7	1.6 ± 0.2	5.0	1.85

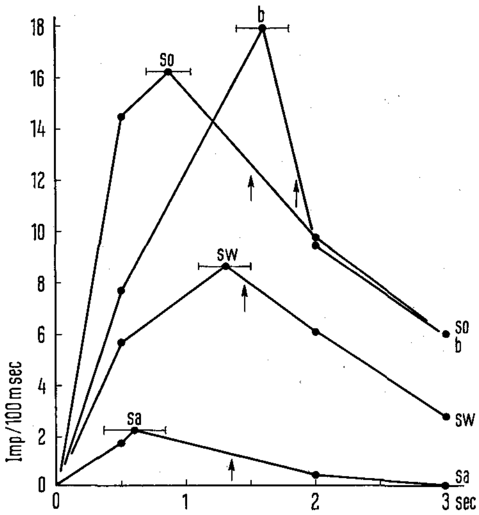

Fig. 3. Temporal sequence of summated responses of the most popular representatives of 4 primary taste qualities. Horizontal lines represent fiducial limit ($p < 0.05$). The difference between the peak time of bitter and that of sweet is statistically significant ($p < 0.05$). RT means the reaction time in sec obtained by us in man[8]. Other explanations in Figure 2.

Figure 2 shows summated response of four primary taste qualities as a function of time after the stimulation. Two or six kinds of chemicals were used for each quality. HCl, NaCl, KCl and LiCl were prepared as $1/2\,M$ solutions, while the others as $1/160\,M$ ones. The Table in the Figure shows mean values of the rate of increase (RI), the peak time (PT) and the rate of decrease (RD) of each quality calculated from the figure. As is clearly seen from the Figure and the Table, the numerals underlined characterize each quality, while the rate of decrease has nothing to do with the taste quality. The same thing can be more definitely said on the summated response curves obtained from the most popular representatives of four primary taste qualities (Figure 3).

From these results it could be concluded that the impulse train of the greatest rate of increase mediates as ensemble the sourness, and that of the smaller one with the shortest peak time mediates as a whole the salty sensation. The bitter sensation is evoked by impulse pattern with the longest peak time, while the sweet sensation by that with an intermediate one, both almost equal to each other in respect to the rate of increase.

Zusammenfassung. Neurophysiologische Untersuchung über die Basis der Geschmacksdifferenzierung in die vier Hauptqualitäten bei Kröten und Nachweis eines offenbar spezifischen Impulsmusters im N. glossopharyngicus.

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Effect of Aluminium Phosphate Gel on Whole-Body Retention of Simultaneously Administered ^{226}Ra, ^{85}Sr and ^{47}Ca in Mice

Aluminium phosphate inhibits intestinal absorption of Sr in man[1] and rat[2] and lowers the whole-body retention to about 10% of control values for ^{85}Sr and to 50% for ^{47}Ca[3], thus being somewhat less specific for Sr than sodium alginate[4]. Increasing the phosphate level of the diet also reduces Sr uptake[5]. We compared the capacity of aluminium phosphate to inhibit intestinal uptake of the heaviest alkaline earth metal ^{226}Ra with that obtained for ^{47}Ca and ^{85}Sr when a mixture of the 3 isotopes is administered by gastric tube to 3-month-old male black C 57 mice, previously fasted for 24 h with free access to water. The total body-burden was measured in vivo by a Ge(Li) detector coupled to a translating and rotating sample holder, as described previously[3]. Each animal received 15 μCi ^{226}Ra, 7 μCi ^{85}Sr and 3 μCi ^{47}Ca at pH 5 in 0.25 ml volume, intubated in the stomach. The tested aluminium phosphate gel was the commercially available 'Phosphalugel' (Laboratories Biotherax, 93 Saint-Denis, France) containing a mean quantity of 3% Al and 10.5% phosphate, together with some other substances such as agar, pectin, sorbic acid and calcium sulfate. Some of these substances might also be able to reduce Sr uptake[6]. The gel was administered by gastric tube (0.4 ml/mouse corresponding to 480 mg Al/kg body weight, and 1680 mg PO_4/kg). The control animals received the same volume of a boiled 0.2% agar gel in water having about the same consistency as the phosphate gel.

Six treatments were tested on 5 to 6 mice each:

group 1 received $AlPO_4$ gel just before isotope intubation;
group 2 received agar gel just before isotope intubation;
group 3 received $AlPO_4$ gel just after isotope intubation;
group 4 received agar gel just after isotope intubation;
group 5 received $AlPO_4$ gel 1 h after isotope intubation;
group 6 received agar gel 1 h after isotope intubation.

In group 1 to 4, there was maximum 1 min time lapse between the 2 intubations. The total body retention 4 days after the intubation is given in the Table. Most of the mice which received phosphate treatment close to the moment of intubation of the isotopes had a ^{226}Ra content lower than the detection limit of the Ge(Li) detector, corresponding to less than 0.3% of the administered dose.

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Total body retention in % of the administered dose of ^{47}Ca, ^{85}Sr and ^{226}Ra, 4 days after gastric intubation in mice: influence of $AlPO_4$ gel

Time of treatment relative to isotope intubation	Retention (% of dose)			Reduction factor = agar controls/PO_4 treated		
	^{47}Ca	^{85}Sr	^{226}Ra	^{47}Ca	^{85}Sr	^{226}Ra
1. $AlPO_4$ just before	8.0 ± 1.8	0.8 ± 0.2	< 0.3	3.3	14	>26
2. Agar just before	27 ± 8	11 ± 4	7.7 ± 5.9			
3. $AlPO_4$ just after	7.1 ± 1.2	0.8 ± 0.2	< 0.3	3.5	12	> 27
4. Agar just after	25 ± 8	11 ± 4	7.9 ± 5.5			
5. $AlPO_4$ later (1 h)	24 ± 5	9.6 ± 2.4	8.8 ± 3.7	1.3	1.5	1.7
6. Agar later (1 h)	32 ± 11	15 ± 6	15 ± 9			

Limits are the 95% fiducial limits ($x \pm ts_{\bar{x}}$), 5 to 6 mice/group.

In a second experiment, 20 μCi ^{226}Ra without other isotopes was intubated to each of 10 mice. The first group of 5 animals received 0.4 ml of phosphate gel just before the Ra intubation, the second group 0.4 ml of agar gel.

After 4 days, the Ra retention was measured in the $AlPO_4$ treated animals with a more sensitive method. The mice were killed with an overdose of ether anaesthesia, and slowly incinerated up to 520°C. Directly hereafter, the ashes were measured in a NaI(Tl) well crystal with a 400 channel analyzer on the 186 KeV emission of ^{226}Ra; thus avoiding the interferences of all the daughter isotopes build up by the Rn-gas (= the first daughter of the whole chain) and giving a detection limit of about 10^{-5} μCi. The whole-body retention for the $AlPO_4$-treated group was 0.01% (± 0.005 at the 95% fiducial level) of the dose; in the agar-controls (measured with the GeLi detector) this was 8.0% (± 4.0).

The limiting effect of a massive quantity of aluminium phosphate gel on the intestinal uptake in mice is thus similar to that obtained in man [1] and rat [2] for ^{85}Sr and ^{47}Ca, but is very much higher for Ra than for Sr. The heavier alkaline earth thus seems to be more strongly fixed, as was observed with sodium alginate also [4]. The simultaneous administration of both ^{226}Ra and $AlPO_4$ reduced the Ra-burden 800 times in mice, while the maximal effect observed with sodium alginate [4] was a 135-fold reduction.

Résumé. L'administration presque simultanée d'un gel de phosphate d'alumine et de $^{226}RaCl_2$ réduit de 800 fois l'absorption intestinale du ^{226}Ra chez la souris. La charge corporelle en ^{85}Sr et ^{47}Ca est réduite d'environ 10 resp. 3 fois.

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Development of Human Foetal Inotropic Responses to Catecholamines

The blood pressure response of foetuses to catecholamines have been studied for many years. Mature foetal rabbits [1] and new-born dogs [2] respond the same as adult animals. Blood pressure responses in young foetal lambs, however, are less than in mature foetal lambs [3], suggesting that the pressure responses to catecholamines are not fully developed until the foetus nears term. This decreased response to catecholamines in immature foetuses has also been observed in foetuses of rabbits and guinea pigs [4], cats [5] and rats [6]. There is little information on the development of inotropic responses to catecholamines in foetuses; a previous study has shown great variations in positive inotropic responses to adrenaline and noradrenaline of a human foetal Langendorff preparation [7]. We have previously reported [8] that inotropic and electrophysiological responses of human foetal myocardium to carbamylcholine are not fully developed at 12–22 weeks gestation and that inotropic responses develop before certain electrophysiological responses. The present study was undertaken to investigate the development of inotropic responses in human foetuses to catecholamines.

Atrial and ventricular tissues were dissected from 17 human foetuses of 12–22 weeks gestation, as judged from nomograms relating crownrump length and dry weight of the excised heart to the period of gestation. A total of 30 left and right atria, 6 ventricular strips and 3 papillary