Absorption kinetics of some carbohydrates in conscious pigs

1. Qualitative aspects

BY A. A. RERAT, P. VAISSADE AND P. VAUGELADE

Laboratoire de Physiologie de la Nutrition, Centre National de Recherches Zootechniques, INRA, 78350 Jouy-en-Josas, France

(*Received 1 November 1983 – Accepted 15 December 1983*)

1. Concentrations of reducing sugars, glucose, fructose and lactic acid in blood obtained from arterial and portal catheters were measured for periods of 8–24 h in twenty-three unanaesthetized pigs (mean body-weight 50 kg). From 6 to 8 d after implantation of catheters, the animals received experimental meals containing different levels (400, 800, 1200, 1600 g respectively) of different sugars (glucose ten meals, sucrose eighteen meals, lactose nine meals, maize starch sixteen meals) as well as a protein–mineral–vitamin premix.

2. After each meal the reducing sugars appeared in the portal blood in successive waves. The porto-arterial differences in the concentration of reducing sugars, representing the real appearance of sugar-hydrolysis products in the animal, varied greatly according to the sugar ingested and its level of intake. For each level of intake, these differences were larger, but of shorter duration, for glucose and sucrose than for maize starch. For these three carbohydrates, the higher the level of ingestion, the larger and the more persistent the porto-arterial differences. Lactose represented a special case, as the porto-arterial differences of reducing sugars were always much lower than those obtained with the other sugars and they did not vary with the level of intake.

3. Our findings show that the products formed by feeding glucose and sucrose appear more rapidly in the portal blood than those formed by feeding lactose. Accordingly, the length of time of digestion of glucose and sucrose is shorter than that of maize starch and lactose.

Sugars ingested by man and monogastric animals are mainly polysaccharides although, in certain circumstances, disaccharides and even monosaccharides are also ingested. In the pig, dietary energy generally comes from cereal starch, but sometimes from industrial by-products such as whey or molasses which contain other types of carbohydrate (lactose, sucrose). However, use of dietary sugars, such as lactose (Février, 1969) or sucrose (Brooks & Iwanaga, 1967; Aherne *et al.* 1969), does not lead to the same results especially for growth and nitrogen retention; in addition, their influence is variable according to age (Ekstrom *et al.* 1975). As the supply of energy-giving nutrients at the sites of protein synthesis should synchronize with the supply of amino acids to make the synthetic process optimal (Elman, 1953), variations in the nutritive value of sugars might be due to a different chronology in their digestion and in the absorption of their hydrolysis products, as well as to the different nature of the latter. Until now, estimation of the amounts of nutrients available for the animal during digestion was made by means of the digestibility method which allows the disappearance of nutrients during their oral–aboral transit to be measured but does not quantify the precise kinetics of their appearance in the organism and their transformation during transit and absorption. These kinetic aspects of the appearance of nutrients in the animal may be estimated from the enrichment of the blood which irrigates the gastrointestinal tract. To that end, the variations in the concentrations of nutrients in the intestinal efferent blood (portal blood) and those of the intestinal afferent blood have to be measured simultaneously and the net absorption can be quantified by determination of the intestinal blood flow-rate.

We therefore developed a technique to establish the kinetics of appearance of nutrients in the blood after a meal. It involves a continuous post-prandial recording of portal and

Table 1. *Composition (g/kg) of the diet offered before and between experiments and of the concentrate diet offered as a protein–vitamin–mineral supplement during experiments*

Diet offered before and between experiments		Concentrate diet*	
Maize starch	700	Soya-bean meal	500
Concentrate diet*	250	Fish meal	150
Pure cellulose†	50	Wheat gluten	120
		Peanut oil	100
		Pure cellulose	50
		Minerals	60
		Vitamins	20

* Dry matter 91·9 g/kg. Diet contained (g/kg dry matter): nitrogen × 6·25, 48·8, lipids 13·6, carbohydrates 17·7, minerals 10·6, crude fibre 7·5. The same concentrate diet was used as 250 g/kg diet before and between experiments, and as a unique load of 150 g for the test meals.

† Purified wood cellulose.

systemic blood levels of nutrients as well as the blood flow-rate in the portal vein and permits quantification of porto-systemic differences any time after the meal (Rérat *et al.* 1980). This provides an over-all representation of the digestion processes (gastric emptying, hydrolysis and absorption of nutrients) but not the analysis of each step separately. This method was used to study the over-all qualitative and quantitative aspects of digestion of a monosaccharide (glucose), two disaccharides (sucrose, lactose) and a polysaccharide (maize starch).

This first paper concerns the variations in the blood concentrations of hydrolysis products of the carbohydrates under investigation in relation to time elapsed after the meal and sampling site (carotid blood, portal blood) and the influence of previous fasting on these measurements. The second paper is devoted to the quantitative aspects of the kinetics of appearance of nutrients following digestion of these carbohydrates (Rérat *et al.* 1984).

EXPERIMENTAL

Animals

Twenty-three castrated male pigs of mean body-weight 50 kg (SEM 0·7) were used. For a period of 3–4 weeks before the trial, they received a pre-experimental diet (800–1000 g/meal) twice daily at 09.00 and 17.00 hours, the composition of which is given in Table 1. The mean growth of the animals during this period was 600 g/d. Each animal was fitted with an electromagnetic flow probe for measuring the blood flow-rate in the portal vein and with two catheters, one placed in the portal vein and the other in the left brachiocephalic artery through the carotid route by a procedure described elsewhere (Rérat, 1971; Rérat *et al.* 1980). The animals began to eat 1–2 d after the operation and rapidly recovered their normal growth rate. They were given penicillin ($1{\cdot}2 \times 10^6$ I.U./d) and streptomycin (1 g/d) for 3–5 d after surgery.

Diets and feeding conditions

Before the operation and until the end of the experiments, the animals were placed in restraining cages. The experiments began 6–10 d after the cannulas had been placed in position and when the animals had recovered a normal appetite and growth rate. After a fasting period of 20 h to avoid the effect of the previous meal (Rérat *et al.* 1980), the animals received in one daily meal (09.00 hours), 400, 800, 1200 or 1600 g of the carbohydrate studied (maize starch sixteen meals, lactose nine meals, sucrose eighteen meals, glucose ten

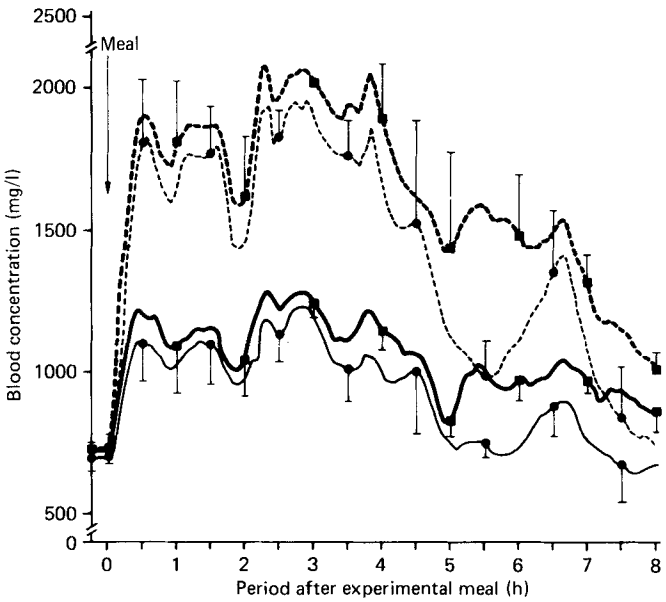

Fig. 1. Variations in blood concentrations (mg/l; mean with SEM) or reducing sugars (■) and glucose (●) in the portal vein (- - - -) and carotid artery (——) after intake of glucose (800 g). The last non-experimental meal was offered 20 h before the experimental meal. Each experimental meal was supplied with 150 g concentrate diet (Table 1). Vertical bars represent the standard error of the mean. Experimental meals (N3) in three different animals.

meals) in addition to 150 g protein–vitamin–mineral concentrate diet (Table 1). For each level of intake, meals which were consumed very slowly (in more than 20 min) were eliminated in order to have the same conditions of intake for all animals. The same animal was given one to six different experimental meals at a rate of one or three per week, the experimental period being interrupted by periods of recovery during which they received the well-balanced concentrate diet (Table 1) under the same conditions as before the experiment (two meals per day, 800–1000 g/meal). It has to be emphasized that the animals did not accept more than 800 g lactose and glucose and 1200 g sucrose. In some cases, the same animal was offered the same amount of the same experimental meal twice in order to test variability between meals: these replications were of 400 g glucose and of 400, 800 and 1200 g sucrose. The replications in the same animal are shown by the difference between the number of meals (N) and the number of animals (A) given in Table 2 (see p. 508).

Measurements

Blood was sampled over the whole periprandial period, i.e. from 30 min before until 8–12 h (exceptionally 24 h) after the meal. The cannulas were connected directly and continuously to an automatic analyser according to a procedure described elsewhere (Aumaitre *et al.* 1975). Total reducing sugars (Hoffman, 1937), true glucose (glucose oxidase technique; Hill & Kessler, 1961), fructose (Roe, 1934) and lactic acid (Ling, 1951) were determined either simultaneously or separately according to the type of studies made. In order to avoid excessive removal of blood, the number of simultaneous analyses in one and the same animal was restricted, explaining the different number of observations reported.

Table 2. *Magnitude and time of appearance of concentration peaks of reducing sugars in the blood and value of porto-arterial differences 8 h after intake of some carbohydrates**

(Mean values with their standard errors calculated from the no. of animals)

				First peak										Highest peak				Porto-arterial differences 8 h after the meal			
				Percentage increase above initial concentration‡				Magnitude of porto-arterial differences‡ (% of initial portal concentration)		Period after meal (min)				Percentage increase above initial concentration‡		Period after meal (min)					
				Portal blood		Arterial blood				Portal blood		Arterial blood		Portal blood		Portal blood		mg/l		% of initial portal concentration	
Carbohydrate	Intake level (g)†	*N*	*A*	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Glucose	400	6	5	167	33	93	20	73	14	43	9	43	7	179	25	71	16	15	18	2	2
	800	4	4	194	17	88	16	106	4	42	8	41	10	218	10	241	62	240	97	34	14
Sucrose	400	9	8	125	22	70	14	55	10	34	3	28	2	166	20	135	19	45	19	6	3
	800	6	5	115	19	49	9	67	20	46	10	35	2	144	13	181	18	291	78	34	8
	1200	3	2	199		96		103		35		27		262		180		527		73	
Maize starch	400	7	7	113	16	56	14	57	8	44	6	45	6	118	15	51	8	101	19	14	3
	800	5	5	178	22	96	18	83	12	104	12	97	22	188	19	122	13	165	27	25	5
	1200	2	2	231		53		80		90		35		263		127		415		65	
	1600	2	2	168		46		122		68		68		201		250		495		68	
Lactose	400	5	5	77	19	41	16	39	7	44	10	44	11	89	20	146	46	46	16	7	3
	800	4	4	41	10	23	6	32	5	42	4	40	4	48	13	94	16	57	14	6	2

N, no. of test meals; *A*, no. of experimental animals; *N*−*A*, no. of replicates in the same animal.

* Sites of sampling: carotid artery, portal vein; preliminary fasting period: 20 h.

† Fresh weight of carbohydrates. A constant load of 150 g concentrate diet was given.

‡ Initial concentration recorded at the beginning of the meal.

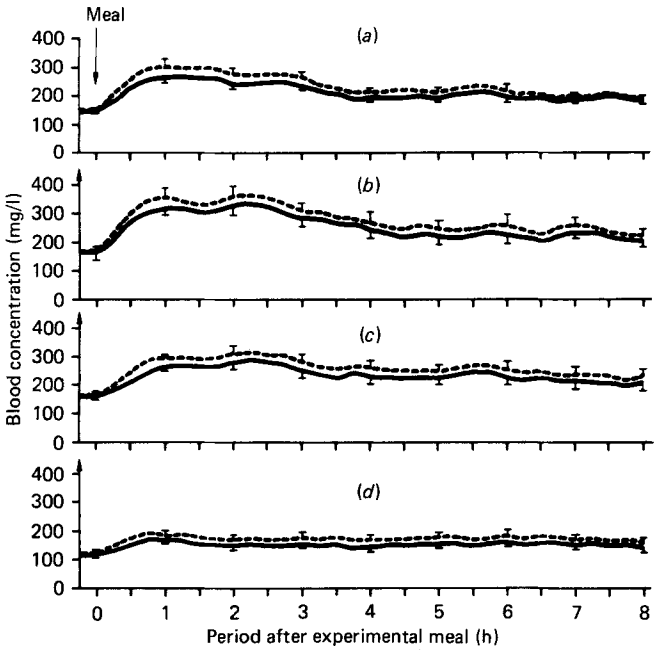

Fig. 2. Variations in blood concentrations (mg/l; mean with SEM) of lactic acid in the portal vein (- - - -) and carotid artery (——) after intake of carbohydrates. Mean intake (g): (*a*), glucose 619; (*b*), sucrose 542; (*c*), maize starch 825; (*d*), lactose 691. Each experimental meal was supplied with 150 g concentrate diet (Table 1). The last non-experimental meal was offered 20 h before the experimental meal. No. of experimental meals (*N*) and animals: glucose ten animals *N*13, sucrose five animals *N*6, maize starch eleven animals *N*15, lactose six animals *N*8.

Statistical methods

Statistical analyses (Snedecor & Cochran, 1967) involved standard error of the mean and comparison of two groups of equal size and groups of unequal size by *t* test. When the same meal was offered to an animal on two occasions, the mean result was used rather than the individual values in order to prevent values for this animal from prevailing over those of other animals receiving the same test meal only once.

RESULTS

Post-prandial variation in blood concentrations after intake of various carbohydrates

Table 2 summarizes percentages pertaining to the concentration rise of reducing sugars during the first absorption wave, the delay of appearance of the peak after the beginning of the meal, the upper limit reached and the level of porto-arterial differences 8 h after intake of the meal for all the levels of intake. Detailed values are shown in Figs. 1–5 for experiments in which three or more replications of the same level of intake (800 g) with simultaneous determinations of reducing sugars, glucose and, in some cases, fructose were made.

Glucose

Intake of a glucose meal (800 g, Fig. 1) caused an immediate and marked rise in the portal and arterial blood levels of reducing sugars, i.e. changes of 194 and 88% respectively, from the initial level within 40 min (Table 2). The concentration was maintained at a high level with an alternation of maxima and minima, the range of which decreased with the time

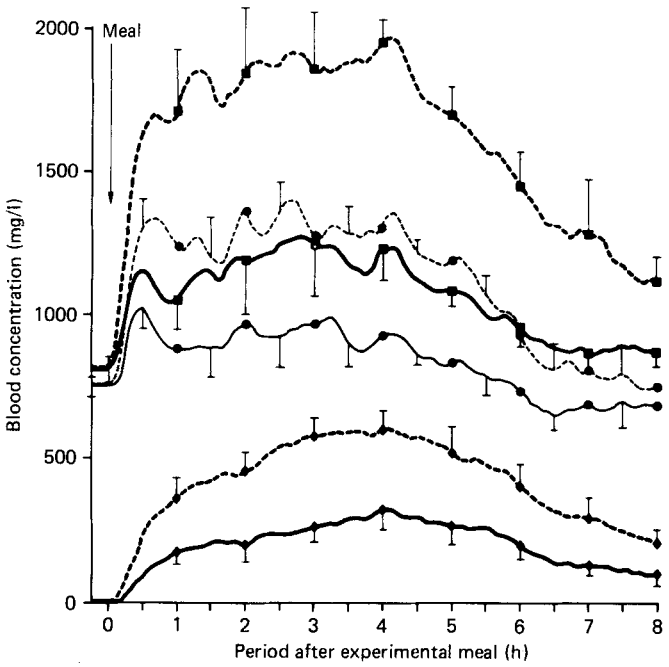

Fig. 3. Variations in blood concentration (mg/l; mean with SEM) of reducing sugars (■), glucose (●) and fructose (◆) in the portal vein (– – – –) and carotid artery (——) after intake of sucrose (800 g). The last non-experimental meal was given 20 h before the experimental meal. Each experimental meal was supplied with 150 g concentrate diet (Table 1). Experimental meals (N 4) in four different animals.

elapsed after the meal. The porto-arterial differences (765 mg/l) represented 106% of the initial concentration 41–42 min after the meal. At 8 h after the meal the concentration of reducing sugars still exceeded the initial values, and the porto-arterial differences were still marked (240 mg/l). Glucose concentrations were slightly lower (86%) than the corresponding levels of reducing sugars and this was also the case for the porto-arterial glycaemia differences.

When the glucose intake was smaller (400 g), the maximum post-prandial concentration rise (change of 167% for the portal blood) and porto-arterial differences were lower (Table 2) and initial levels of reducing sugars and disappearance of porto-arterial differences occurred within less than 8 h except with one animal.

The lactic acid concentration (Fig. 2(*a*)) increased immediately after ingestion of meals at all levels, but this rise was more pronounced in the portal than in the arterial blood. The porto-arterial differences were therefore rather large during the post-prandial period of 8 h.

Sucrose

Intake of a sucrose meal (800 g, Fig. 3) caused an immediate increase in the blood level of reducing sugars, but less marked than that caused by glucose (115% from the initial level in the portal blood; 49% from the initial level in the arterial blood). The maximum change (144% from the initial portal blood level) was only reached 3 h after the meal. An alternation of maxima and minima then occurred with a higher mean level and a smaller difference between maxima and minima than with glucose (Table 2). The porto-arterial differences (568 mg/l at the first peak) were not as large as those observed after glucose intake, but they lasted longer, a marked difference being still apparent 8 h after the meal between the portal and the arterial concentration (291 mg/l) and the reducing sugar con-

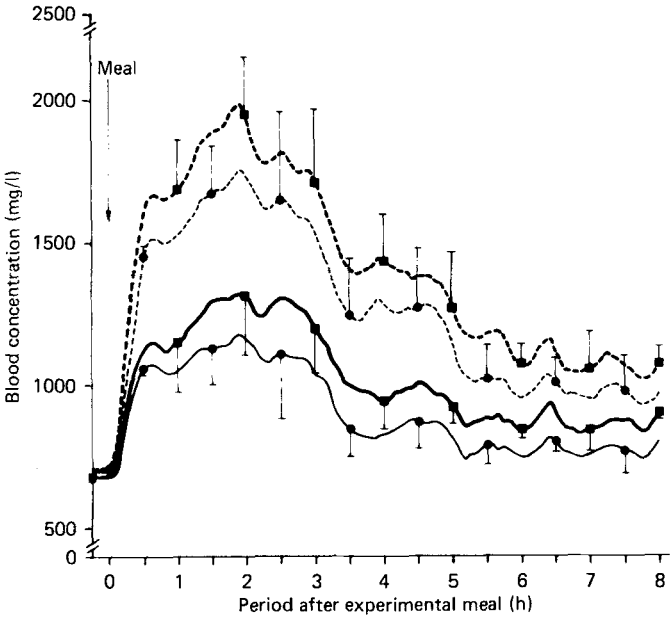

Fig. 4. Variations in blood concentrations (mg/l; mean with SEM) of reducing sugars (■) and glucose (●) in the portal vein (– – – –) and carotid artery (——) after intake of maize starch (800 g). The last non-experimental meal was offered 20 h before the experimental meal. Each experimental meal was supplied with 150 g concentrate diet (Table 1). Experimental meals ($N4$) in four different animals.

centration not returning to the initial value. A prolongation of the experimental period showed that these differences seemed to disappear 10–12 h after the meal. When the level of sucrose intake was lower (400 g) the porto-arterial differences were smaller, but still persisted for longer than 8 h after the meal in six animals out of eight; when the level increased (1200 g), these differences were more marked (527 mg/l at 8 h) and persisted for more than 12 h. Mean blood concentration of glucose represented 75% that of reducing sugars.

The post-prandial pattern of true glycaemia was different from that of fructosaemia (Fig. 3). For an ingestion level of 800 g, the first glucose peak appeared 30 min after the meal after which the level of glucose showed wide variation. Maximum values were observed for 4–5 h, and the same but less marked trend was found for peripheral glycaemia. Fructosaemia undetectable at the time of the meal, increased very slowly, reached a maximum 3 h after the meal and remained at that level for about 2 h. It then decreased very slowly and the initial value was not reached 8 h after the meal. The porto-arterial fructosaemia differences were smaller than those observed for glycaemia, especially during the first 6 h after the meal. Glycaemia and fructosaemia patterns were the same after intake of 400 and 1200 g sucrose, the maxima and differences between maxima and minima being larger and lasting longer with increasing levels of intake.

Variations in lactic acid concentrations (Fig. 2(*b*)) were similar to those described for glucose, but the maximum concentrations observed were higher.

Maize starch

Intake of maize starch (800 g, Fig. 4) led to a rise in the portal blood levels of reducing sugars similar to that observed with glucose except that the maximum value (178% change from the initial value, Table 2) reached 2 h after the meal was shorter-lasting owing to a step-wise decrease in the level of reducing sugars. This was followed by a succession of

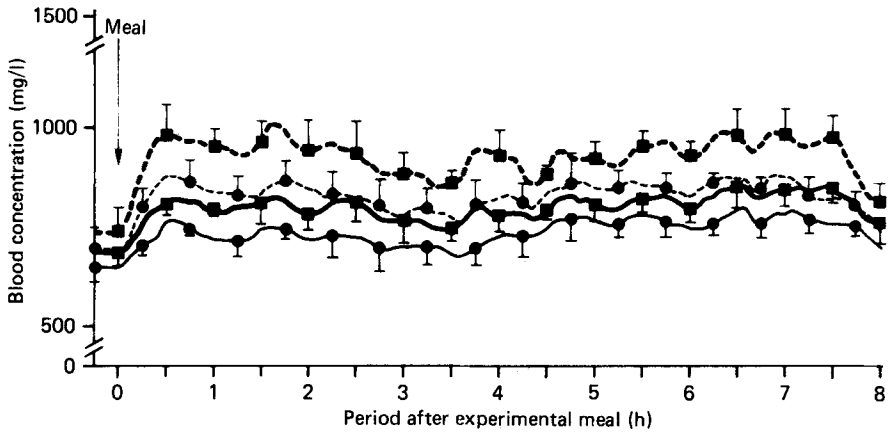

Fig. 5. Variations in blood concentrations (mg/l; mean with SEM) of reducing sugars (■) and glucose (●) in the portal vein (– – – –) and in the carotid artery (——) after intake of lactose (800 g). The last non-experimental meal was offered 20 h before the experimental meal. Each experimental meal was supplied with 150 g concentrate diet (Table 1). Experimental meals (N 4) in four different animals.

concentration waves, but the differences between maxima and minima were very small. The porto-arterial differences were smaller at the first peak (586 mg/l) than those observed after glucose intake and they occurred later. The portal blood level of reducing sugars did not return to its initial value 8 h after the meal and a porto-arterial difference (165 mg/l) persisted, indicating that absorption was not completed. This persistence of porto-arterial differences increasing with the level of intake was also found for lower (101 mg/l) or higher (415 mg/l) dietary supplies. Supply of large amounts led not only to a marked increase in the portal blood level of reducing sugars (three to four times the initial level) but also to a complete overlapping of peaks in the first 4 h after the meal.

In all cases, glycaemia was slightly lower (about 10%) than the blood level of reducing sugars; this was also true for the porto-arterial glycaemia difference.

Variations in the portal and arterial concentrations of lactic acid were analogous to those found with glucose (Fig. 2(c)).

Lactose

Intake of lactose (800 g, Fig. 5) caused a small but immediate increase (40 min) in the concentration of reducing sugars (41% change from the initial level in the portal blood, 23% in the arterial blood, Table 2). A succession of maxima and minima with small differences between them was observed after the first peak. At the end of the 8 h post-prandial period, the portal and arterial blood levels of reducing sugars exceeded the initial levels, but the porto-arterial differences, always very small during the first hour (222 mg/l, i.e. 32% of the initial level) did not persist beyond 13 h. Reduction in the level of intake (400 g, Table 2) did not change the over-all concentration trends, the porto-arterial differences remaining almost identical to those recorded after an intake of 800 g. Mean blood concentration of glucose represented 89% that of reducing sugars. The instantaneous variations in the porto-arterial differences of true glycaemia (Fig. 5) followed those of the reducing sugars and the fact that these differences represented more than half the porto-arterial differences of reducing sugars, must be underlined.

The post-prandial increase in the blood level of lactic acid (Fig. 2(d)) was not as high as that recorded for the other sugars.

DISCUSSION

Methodology

For determining the enrichment of portal blood during digestion, variations due to the intestinal afferent blood concentration of nutrients must be eliminated. To that end, the venous systemic route has sometimes been used to sample peripheral reference blood (Denton & Elvehjem, 1954; Annison *et al.* 1957; Prewitt *et al.* 1975). This method is supported by studies made in the pig (Nordstrom *et al.* 1970; Stockland *et al.* 1971) according to which concentrations of nutrients are almost the same in the vena cava, the aorta and the jugular vein. Therefore, before any study of nutrient absorption was undertaken, it seemed worthwhile to determine whether the peripheral venous blood was representative of the arterial blood as suggested by the investigations cited previously. A systematic study of the concentrations of total reducing substances in the blood of the anterior vena cava and the aorta showed that the concentration was always higher in the arterial blood than in the systemic venous blood during the 20 h observation periods (Rérat *et al.* 1980). Similar observations were made about the levels of free amino acids in a pig receiving a protein-free diet (Rérat *et al.* 1980). This is due to the fact that in the systemic venous blood, nutrients were taken up by the tissues, which was not the case in the arterial blood. Consequently, the porto-arterial differences will always be smaller than the differences between the portal vein and the vena cava. Thus, calculated absorption values will depend on whether the arterial or the peripheral venous blood is used as a systemic blood reference (Rérat *et al.* 1980).

It is interesting to consider the role of the length of pre-experimental fasting. The porto-arterial differences are almost zero 20 h after a maize starch meal (Rérat *et al.* 1980). Thus, it seems advisable to let the animals fast for 20 h to eliminate interference due to absorption of nutrients from the former meal if it contained starch. This is confirmed by the kinetics of gastric emptying of such a meal which is finished 20–22 h after its ingestion (Laplace, 1979). The disadvantage of a prolonged fasting period before the assay is the accelerated gastric emptying of a fraction of the experimental meal. This is especially the case during the first hour after its ingestion since the presence of food in the small intestine inhibits stomach emptying (Auffray *et al.* 1967). However, the consequences of this acceleration are small in terms of amounts emptied, as shown by gastro-duodenal transit values (Cuber & Laplace, 1979).

For practical reasons, our experiments generally did not exceed 8 h. However, it is evident that, when using high levels of intake of slowly-digested carbohydrates, digestion is not finished 12–15 h after the meal. It is therefore difficult to establish total digestion balances and to extrapolate the quantities absorbed after 8 h from those absorbed during the first 8 h.

Another point of the methodology concerns the blood indices used. Comparison of the curves describing the variations in concentrations of reducing sugars with those of glycaemia after intake of glucose or maize starch (carbohydrates formed of glucose chains) shows that measurement of reducing sugars includes substances other than sugars. The pattern of variation of these substances (e.g. urea) is not necessarily the same as that of absorbed sugars. Thus, urea, at its usual blood concentration, is generally secreted in the digestive tract during digestion (Rérat *et al.* 1979*a*); accordingly the absorption of sugars is underestimated if the reducing substances only are taken into account. On the other hand, reducing substances other than sugars may be absorbed and may therefore lead to an over-estimation of carbohydrate absorption. However, this error may be evaluated by quantitative comparisons between reducing sugars and absorbed glucose.

Variations in sugar concentration, length of time of digestion and metabolism of peripheral tissues

When blood concentration curves of animals which have ingested the same amount of sugars are pooled together, some of the instantaneous variations unfortunately disappear. The general shape of the curves accounting for post-prandial values after ingestion of an equal amount of the same carbohydrate is the same, but the time of appearance of maxima and minima may be very different. A maximum on one curve may correspond to a minimum on another curve. These differences can be observed from one meal to another for each carbohydrate and each level of intake. Thus, the curves representing reducing sugar concentrations in the portal vein and systemtic blood are sinusoidal and the magnitude of the differences between maxima and minima decreases with the time elapsed after the meal (Aumaitre *et al.* 1973; Rérat *et al.* 1977). These carbohydrate appearance waves may be related to the kinetics of gastric emptying although it has been shown in the case of maize starch that the amounts present in the duodenum always greatly exceed those appearing in the portal vein (Rérat, 1981*a*). The variations in the total blood concentrations of carbohydrates may be due in part to an insulin-like, hormonal control.

The duration of all the processes of digestion and absorption varied from one carbohydrate to another and from one level of intake to another as shown by the time when the porto-arterial differences disappeared, but it must be underlined that very few observations were made over periods lasting more than 8 h. Digestion of the four carbohydrates was generally not finished within the 8 h post-prandial period, except for the lowest level of intake (400 g) of glucose and sometimes sucrose. With regard to maize starch, studies over 24 h showed that disappearance of the porto-arterial differences generally occurred 18 h after a meal, especially a large one (Rérat *et al.* 1980). This confirms the findings from the absorption curves of reducing sugars after ingestion of 1200 g barley or wheat: the porto-arterial differences only disappear after 20 and 22 h respectively for barley and wheat (Rérat, 1981*a*). It may therefore be assumed that the length of time of digestion as well as the initial time of appearance and importance of reducing sugars in the portal vein are highly variable depending on the sugars ingested and their levels of intake. These phenomena should be discussed further in connection with gastric emptying and possible enzymic degradation times.

Absorption of sugars leads to an increase in the systemic blood levels of reducing sugars and glucose. This has also been demonstrated in the rat after ingestion of glucose (Strubbe & Steffens, 1977). The increase grows with increasing portal blood levels, which depend on the type and amount of sugar ingested. These variations show that the liver is not able to take up all sugars absorbed, at least during the largest absorption period so that the systemic blood is enriched with a fraction of sugars. This has been shown previously with amino acids (Ostrowski, 1969; Rérat *et al.* 1979*b*). In addition, the rise is higher in arterial than in venous blood, owing to the metabolic utilization of reducing sugars by the peripheral tissues.

It may be concluded that, after intake of various carbohydrates, sugars appear very early in the portal blood in successive waves of minima and maxima, the range of differences of which decrease with the time elapsed after the meal. The porto-arterial differences in the concentration of sugars, accounting for the appearance of sugars in the animal beyond the intestinal cell wall, varied greatly according to the nature of the carbohydrate ingested and its level of intake. At each level of intake studied, these differences occurred earlier and were higher and shorter-lasting for glucose and sucrose than for maize starch. For each of the three sugars, it was noticed that the higher the level of intake the more long-lasting the porto-arterial differences. Lactose represented a particular case since the porto-arterial

differences seemed to be lower than those recorded with the other sugars and independent of the level of intake.

All these findings, however, should be considered only as over-all trends. The difference in the rate of digestion of sugars and in the rate of absorption of their hydrolysis products can only be assessed after a simultaneous recording of the porto-arterial differences and the variations in the blood flow-rate of the portal vein (Rérat *et al.* 1984).

Acknowledgement is given to Mrs K. Rérat for the English translation of the manuscript.

REFERENCES

Aherne, F., Hays, V. W., Ewan, R. C. & Speer, V. C. (1969). *Journal of Animal Science* **29**, 444–450.
Annison, E. F., Hill, K. J. & Lewis, D. (1957). *Biochemical Journal* **66**, 592–599.
Auffray, P., Martinet, J. & Rérat, A. (1967). *Annales de Biologie Animale, Biochimie, Biophysique* **7**, 262–279.
Aumaitre, A., Rérat, A., Vaissade, P. & Vaugelade, P. (1973). *Annales de Biologie Animale, Biochimie, Biophysique* **13**, 784–787.
Aumaitre, A., Rérat, A., Vaissade, P. & Vaugelade, P. (1975). *Annales de Biologie Animale, Biochimie, Biophysique* **15**, 547–558.
Brooks, C. C. & Iwanaga, I. I. (1967). *Journal of Animal Science* **26**, 741–745.
Cuber, J. C. & Laplace, J. P. (1979). *Annales de Biologie Animale, Biochimie, Biophysique* **19**, 899–905.
Denton, A. E. & Elvehjem, C. A. (1954). *Journal of Biological Chemistry* **206**, 449–454.
Ekstrom, K. E., Benevenga, N. J. & Grummer, R. H. (1975). *Journal of Nutrition* **105**, 1032–1038.
Elman, R. (1953). *Journal of Clinical Nutrition* **1**, 287–294.
Février, C. (1969). *Journées de la Recherche Porcine en France*, pp. 91–98. Paris: Institut National de la Recherche Agronomique, Institut Technique du Porc.
Hill, J. B. & Kessler, G. (1961). *Journal of Laboratory and Clinical Medicine* **57**, 970–980.
Hoffman, W. S. (1937). *Journal of Biological Chemistry* **120**, 51–55.
Laplace, J. P. (1979). *Medecine et Nutrition* **15**, 17–23.
Ling, E. R. (1951). *Journal of the Science of Food and Agriculture* **2**, 279–288.
Nordstrom, J. W., Windels, H. F., Typpo, J. T., Meade, R. J. & Stockland, W. L. (1970). *Journal of Animal Science* **31**, 874–884.
Ostrowski, H. (1969). *Animal Production* **11**, 521–532.
Prewitt, L. R., Jacobson, D. R., Hemken, R. W. & Hatton, R. H. (1975). *Journal of Animal Science* **41**, 596–600.
Rérat, A. (1971). *Annales de Biologie Animale, Biochimie, Biophysique* **11**, 277 Abstr.
Rérat, A. (1981*a*). *World Review of Nutrition and Dietetics* **37**, 229–287.
Rérat, A. (1981*b*). *Bulletin de l'Académie Nationale de Médecine* **8**, 59–65.
Rérat, A., Aumaitre, A., Vaissade, P. & Vaugelade, P. (1977). *Annales de Biologie Animale, Biochimie, Biophysique* **17**, 589–596.
Rérat, A., Lisoprawski, C., Vaissade, P. & Vaugelade, P. (1979*a*). *Bulletin de l'Académie Vétérinaire de France* **52**, 333–346.
Rérat, A., Vaissade, P. & Vaugelade, P. (1979*b*). *Annales de Biologie Animale, Biochimie, Biophysique* **19**, 739–747.
Rérat, A., Vaissade, P. & Vaugelade, P. (1984). *British Journal of Nutrition* **51**, 517–529.
Rérat, A., Vaugelade, P. & Villiers, P. A. (1980). In *Current Concepts of Digestion and Absorption in Pigs. Technical Bulletin* no. 3, pp. 177–214 [A. G. Low and I. G. Partridge, editors]. Reading and Ayr: National Institute for Research in Dairying and Hannah Research Institute.
Roe, J. H. (1934). *Journal of Biological Chemistry* **107**, 15–22.
Snedecor, G. W. & Cochran, W. G. (1967). *Statistical Methods*, pp. 593. Ames, Iowa: Iowa State University Press.
Stockland, W. L., Meade, R. J., Tumbleson, M. E. & Palm, B. W. (1971). *Journal of Animal Science* **32**, 1143–1152.
Strubbe, J. H. & Steffens, A. B. (1977). *Physiology and Behaviour* **19**, 303–307.

Printed in Great Britain