Physiological Chemistry.

The Influence of Various Carbohydrates and Amino-acids on the Blood and Urinary Sugar of the Healthy Organism. A. SCHÄTTI (*Biochem. Z.*, 1923, **143**, 201—220).—In general, the ingestion by normal individuals of 20 g. of the following carbohydrates produces a hyperglycæmia the intensity of which diminishes in the order given: dextrose, lævulose, sucrose, lactose, galactose. The same amounts of starch, glycine, and alanine produce no hyperglycæmia, but after the ingestion of 100 g. of starch the rise in blood-sugar is as great as, but more delayed than, that produced by 100 g. of dextrose. Peptone and coffee give no hyperglycæmia. Lævulose is excreted more quickly and in greater amount than dextrose, and the sugar excreted after ingestion of sucrose consists chiefly of lævulose. Galactose seems to be excreted largely unchanged by the kidneys. Glycosuria following ingestion of dextrose increases with increasing hyperglycæmia, but the former shows a more delayed maximum than the latter. Glycosuria is not dependent on diuresis, but is regarded as a secretory process. J. P.

Blood-clotting as an Agglutination Process. E. HEKMA (*Biochem. Z.*, 1923, **143**, 105—110).—A theoretical paper in which the view is advanced, as a result of the author's previous work, that fibrin formation is a crystallisation process involving the dehydration and agglutination of fibrinogen amicrons. Thrombin is regarded, not as a fibrin ferment, but as an agglutinin, and fibrin consists of fibrinogen plus agglutinin. J. P.

The Influence of Chemicals on Erythrocyte Membranes by Changes in Corpuscular Volume. B. S. NEUHAUSEN and J. E. BRESLIN (*Bull. Johns Hopkins Hosp.*, 1923, **34**, 199—201).—A study of the effects of the salts of the blood and of dextrose on the erythrocyte membranes as shown by a comparison of cell volumes in iso-osmotic solutions. Ions cause swelling in the order, Ca < Na < K. The contracting action of calcium, as opposed to the swelling action of potassium, is specially pointed out. In the case of blood-corpuscles, the membranes are very complex colloidal mixtures, the swelling or precipitation of any component of which will loosen the whole structure.

CHEMICAL ABSTRACTS.

The Rôle of Pancreatic Juice in the Digestion of Proteins. Relative Importance of Trypsin and Erepsin. E. F. TERROINE and ST. J. PRZYLECKI (*Arch. internat. physiol.*, 1923, **20**, 377—396; *Ber. ges. Physiol.*, 1923, **20**, 64; from *Chem. Zentr.*, 1923, iii, 1290).—Pancreatic juice not activated by kinase attacks the normal products of gastric digestion. With activation by kinase, the action is increased. The relative importance of erepsin and trypsin depends on the degree of previous gastric digestion; where this is small, trypsin plays the more important part, but is less important where gastric digestion is more complete. As pancreatic digestion proceeds, the effect of trypsin recedes relative to that of erepsin.

G. W. R.

Effect of Magnesium Sulphate and Metallic Magnesium on Metabolic Exchanges. R. REDING and A. SLOSSE (*Compt. rend. Soc. biol.*, 1923, **88**, 644—646; from *Chem. Zentr.*, 1923, iii, 1108).—Intramuscular injection of 70 c.c. of 10% magnesium sulphate solution decreases the output of total nitrogen, uric acid, and sodium chloride, whilst the output of magnesium is increased. In a rectal carcinoma, injection of 20 c.c. of 10% magnesium sulphate solution was followed by decreased output of total nitrogen, amino-nitrogen, and sodium chloride, and increased output of uric acid and creatine. Similar effects were observed in a rabbit after introduction under the skin of a piece of metallic magnesium.

G. W. R.

The Internal Secretion of the Genital Organs. I. The Genital Organs and Carbohydrate Metabolism. S. TSUBURA (*Biochem. Z.*, 1923, **143**, 248—290).—Castration has no effect on the blood-sugar of fasting dogs, but in the sexually mature animal the operation produces a lowered sugar tolerance. The same effect is produced by ligature of one spermatic cord and extirpation of the opposite testicle, by ligature of both cords, or by exposing the genital glands to *X*-rays. These operations produce a degeneration of the sperm-cells, but do not affect the interstitial cells. The lowered sugar tolerance may be temporarily raised by transplantation of the sexual organs of another animal, but not by feeding the organs or by injection of extracts. Glycogen formation, glycogenolysis, and glycolysis of the blood-sugar in vitro are somewhat delayed after castration, but the blood amylase is unchanged.

After castration, the reactions of the blood-sugar to adrenaline, diuretin, and pituitrin are increased, but thyroid feeding has no effect, although it raises the low sugar tolerance of the castrated animals. The kidneys of the latter are more permeable to sugar, but the excretion of phenolsulphonephthalein is delayed as compared with the normal condition. In general, parallel experiments on male and female dogs gave similar results. The other endocrine organs show alterations after castration, and the observed results may not be wholly due to disturbances of the internal secretory functions of the genital organs. J. P.

The Internal Secretion of the Genital Organs. II. The Genital Organs and Respiratory Gaseous Metabolism. S. TSUBURA (*Biochem. Z.*, 1923, **143**, 291—322).—Castration produces a lowered gaseous metabolism which becomes apparent about one month after the operation. A similar effect is produced by ligature of one spermatic cord and removal of the opposite testicle. Transplantation of the testes of another animal raises the gaseous exchange whilst feeding experiments are ineffective. Dogs in heat show no increased gaseous metabolism. Thyroid feeding raises the low gaseous exchange of castrated animals. The combined results of thyroid feeding on the sugar tolerance (cf. preceding abstract) and gaseous metabolism of castrated dogs are explained as being due to a moderate stimulation of the sluggish carbohydrate metabolism by the thyroid hormone. J. P.

The Ferments in the Placenta. K. MAEDA (*Biochem. Z.*, 1923, **143**, 347—364).—Extracts of the placenta contain diastase in considerable amount, in part derived from the parental blood. Carboxylase and small quantities of lactase and invertase are present, but glycolytic enzymes are not found. Of proteolytic enzymes, erepsin, a weak trypsin, and traces of pepsin, but no rennin, are found. An asparagine deaminase is present. Mono- and tributyrases are found, and the latter, being stable to both quinine and atoxyl, is thus differentiated from serum lipase. Histozyme, a feeble salicylase, and an oxydase acting on pyrocatechol, adrenaline, and dihydroxyphenylalanine, but not on tyrosine, are present. J. P.

The Site of Origin of Bilirubin. A. R. RICH (*Bull. Johns Hopkins Hosp.*, 1923, **34**, 321—329).—Hæmoglobin introduced into the blood-stream of a dog without a liver, and circulating actively for as long as five and a half hours is not transformed into bilirubin. Since, however, the production of bilirubin from circulating hæmoglobin takes place rapidly and readily in a dog with a liver through which the blood-stream passes, it is concluded that the liver is necessary for the transformation of circulating hæmoglobin into bilirubin. The conclusions are applicable only to hæmoglobin circulating in the blood-stream, not to hæmoglobin liberated into the tissues from extravasated blood; the conversion of hæmorrhage hæmoglobin into bilirubuin (hæmatoidin) is a purely local phenomenon, the exact mechanism of which is unknown.

CHEMICAL ABSTRACTS.

[Fish] Liver Oils. M. TSUJIMOTO (*J. Chem. Ind. Japan*, 1923, **26**, 482—486).—The oil from the Jewfish (*Stereolepis ischinagi* (Hilg.), contains a large amount of peculiar unsaponifiable matter (up to 50%), which is a viscous substance of reddish-orange colour, having a resinous odour, and showing much resemblance to colophony when dried at 100° for several hours. The name "liver-resin" is proposed for it. Liver oil from *Erilepis zonifer* (Lock.) also contains a similar substance (about 4%). These oils and the unsaponifiable matter (especially in carbon disulphide solution) give a deep violet coloration with sulphuric acid. With a sample of *S. ischinagi* liver oil, the coloration was observed to be about one hundred times as strong as that of a cod-liver oil. K. K.

The Highly Unsaturated Acids in Cod-liver Oil. M. TSUJIMOTO and K. KIMURA (*J. Chem. Ind. Japan*, 1923, **26**, 1162).—By the potassium salt–ether method, and the lithium salt–acetone method, a mixture of highly unsaturated acids was obtained. By converting it into the methyl ester, a fraction (about 4% of the oil) boiling at 220—226°/5 mm. was isolated. The analysis of the ester and the fatty acid from the ester gave $C_{22}H_{33}O_2Me$ and $C_{22}H_{34}O_2$ respectively, and behenic acid was produced from the latter by hydrogenation. Cod-liver oil therefore contains a large amount of clupanodonic acid, $C_{22}H_{34}O_2$. The part of the methyl ester boiling below 220°/5 mm. is supposed to contain a large amount of a highly unsaturated acid containing eighteen or twenty carbon atoms. K. K.

Further Studies on Autoxidations and Oxido-reduction Processes. V. E. ABDERHALDEN and E. WERTHEIMER (*Pflüger's Archiv*, 1923, **200**, 176—193; from *Chem. Zentr.*, 1923, iii, 1290).—The reducing power of muscle in which thermal rigor has been induced is three or four times that of normal muscle. In chemical rigor and rigor mortis, there is a similar increase in reducing power. An increase also occurs during normal or tetanic contraction. It is supposed that such increases are associated with the presence of a thiol group in the muscle fibres. Cysteine serves as a hydrogen carrier and acceptor. A scheme for a stable oxido-reduction system is given. G. W. R.

A Methylation Function of the Thyroids and the Biological Significance of Iodine. B. STUBER, A. RUSSMAN, and E. A. PROEBSTING (*Biochem. Z.*, 1923, **143**, 221—234).—If guanidine-acetic acid be administered intravenously to young dogs, an increase of blood creatine–creatinine follows. No such increase occurs in thyroidectomised animals, but if these be given dried thyroid or potassium iodide per os, before the injection of guanidineacetic acid, they react like the normal animals. The blood of normal dogs, but not of thyroidectomised dogs, is also effective in this respect. It is concluded that the capacity of the organism to methylate guanidineacetic acid, and so form creatine and creatinine, is dependent on the integrity of the thyroids, and that iodine compounds are necessary for this methylation process. J. P.

The Oxytocic–Pressor–Diuretic Principle of the Infundibular Portion of the Pituitary Gland. J. J. ABEL, C. A. ROUILLER, and E. M. K. GEILING (*J. Pharm. exp. Ther.*, 1923, **22**, 289—316).—After a purification of the gland material by precipitation with mercuric chloride, phosphotungstic acid, and tannic acid in succession, the residue is dissolved in an alcoholic solution of tartaric acid and re-precipitated with ether. The resulting precipitate is called the tartrate of the active principle. Its action on the isolated uterus of the guinea-pig may be as much as 1,250 times as great as that of histamine phosphate, whilst it also retains the diuretic and pressor action characteristic of the pituitary extract. C. R. H.

Anaërobic Processes Involved in Muscular Activity. W. HARTREE and A. V. HILL (*J. Physiol.*, 1923, **58**, 127—137).—In muscle, 0·1% of lactic acid can be produced so rapidly by exercise or by stimulation that no appreciable escape of carbon dioxide or of acid is possible. Hence if the hydrogen-ion concentration inside a muscle is not to rise to an excessive degree during exercise, there must be some buffer in it, much more effective than a bicarbonate solution. Meyerhof (*Pflüger's Archiv*, 1922, **195**, 22) has emphasised that in a frog's muscle the absolute amount of bicarbonate present, as determined from the carbon dioxide driven out from the muscle by excess of acid, is quite inadequate to neutralise the lactic acid liberated in severe stimulation. Even if all the carbon dioxide were driven out, only one-seventh to one-tenth of the lactic acid could be neutralised.

The authors further calculate that the 0·3% inorganic phosphate in muscle (a mixture of KH_2PO_4 and K_2HPO_4) is also quite inadequate to keep the P_H within the extreme limits observed, viz., a change from 7·5 to 6·9). Bicarbonate and phosphate together are insufficient to neutralise half the lactic acid formed. There must be, as Meyerhof assumes, some other powerful buffer, doubtless an alkali–protein salt. The addition of lactic acid to shed blood also produces only a fraction of the change in the hydrogen-ion, which would result in a bicarbonate solution of the same concentration; here also alkali protein must play a considerable part.

The authors confirm the existence of a secondary maximum in the production of heat in a muscle in the absence of oxygen; this maximum occurs after about two and a half minutes (see the following abstract for its interpretation). A "balance sheet" is given of the heat evolved in the different phases of muscular contraction; it shows that during the recovery process something between 1/4·7 and 1/6 of the lactic acid removed is oxidised, the rest being reconverted into glycogen. G. B.

Heat of Combustion of Glycogen in Relation to Muscular Contraction. W. K. SLATER (*J. Physiol.*, 1923, **53**, 163—167).—Glycogen (from *Mytilus edulis*, a convenient source) only becomes anhydrous at 100° in a vacuum over phosphoric oxide (cf. Harden and Young, T., 1902, **81**, 1224). Dried by the method of Atkins and Wilson (T., 1915, **107**, 916), it has the composition $(C_6H_{10}O_5,H_2O)_n$.

The heat of combustion seems previously to have been determined with slightly hydrated material. The author finds the heat of combustion of hydrated glycogen to be 3,883 cal. per gram, which is about 100 cal. higher than that calculated from Stohmann and Schmidt's value, 4,190 (A., 1895, ii, 102), for (presumably imperfectly) dehydrated glycogen, for this would give for the hydrated form $162/180 \times 4{,}190 = 3{,}771$ cal. The heat of wetting is found to be about 9 cal., hence the heat of combustion of hydrated glycogen in solution is 3,874 cal. The heat of combustion of lactic acid is, according to Meyerhof, 3,601 cal., leaving 273 cal. for the conversion of 1 g. of glycogen into 1 g. of lactic acid. The heat of neutralisation of lactic acid by acid salts is, according to Meyerhof (*loc. cit.*), 19 cal. per g., which leads to the value $273 + 19 = 292$ cal. for the heat liberated during contraction and relaxation of a muscle, per g. of lactic acid formed. The total initial heat, given by Hartree and Hill in their balance-sheet (as the result of physical measurement, see preceding abstract) is 296 cal., in close agreement with that calculated above. The salt buffering of the lactic acid is, however, probably only of a temporary character and is replaced by the more efficient buffering by alkali-protein. Meyerhof has shown that the neutralisation of 1 g. of lactic acid by alkali-protein produces 138 cal., or 119 more than that by salts. The whole of these 119 cal. are, however, not produced; according to Hartree and Hill the delayed anaërobic heat production is 74 cal. per g. of lactic acid, so that only about 60% of the lactic acid formed is neutralised by alkali protein (the rest remains neutralised by salts). The delayed heat production, presumably due to a transfer of alkali from protein to acid phosphate and carbonic acid, is the cause of the secondary maximum in the heat production occurring after two and a half minutes (preceding abstract). The velocity of this reaction is independent of the temperature, and thus probably depends on a physical process (rate of diffusion of acid phosphate- and carbonic acid-ions) through the muscle-tissue. This theory demands some special distribution of phosphate and carbonate molecules in the muscle substance, and may be represented by the following scheme :

Contraction (*a*) Glycogen $\longrightarrow$ lactic acid; (*b*) lactic acid and contractile mechanism produce mechanical response.

Relaxation (*a*) Lactic acid + K_2HPO_4 and $KHCO_3 \longrightarrow$ K lactate + KH_2PO_4 and H_2CO_3.

Anaërobic recovery (*a*) KH_2PO_4 and H_2CO_3 + K protein $\longrightarrow$ $K_2HPO_4 + KHCO_3$ + H protein. G. B.

Products of the Catalytic Hydrolysis of Horse Hair. V. S. Sadikov (*Biochem. Z.*, 1923, **143**, 504—511).—Fractionation of the product obtained by the catalytic hydrolysis of horse hair under pressure yielded the following substances : (1) A peptide anhydride (m. p. 258°) of alanine and leucine to which the formula $C_{21}H_{36}O_4N_4$ or $C_{21}H_{38}O_4N_4$ is ascribed. (2) A peptide anhydride of alanine and leucine (m. p. 260°) of the formula $C_9H_{16}O_2N_2$. (3) A peptide anhydride of leucine and valine (m. p. 246°) of the formula

$C_{11}H_{20}O_2N_2$. (4) Butylalanyl-leucine anhydride, $C_{10}H_{18}O_2N_2$, or its unsaturated derivative, $C_{10}H_{16}O_2N_2$ (m. p. 224°). (5) Diketomethylpiperidine, $C_6H_9O_2N$ (m. p. 196°). (6) A cyclic nitrogen substance, $C_{15}H_{30}N$, of an alkaloidal nature. (7) A substance of the formula $C_{13}H_{20}O_3N_2$ (m. p. 170°). Various other less well characterised derivatives of the nature of diketopiperazines and keto- and diketo-piperidines were obtained. J. P.

Calcium Fixation by Animal Tissues. IX. E. FREUDENBERG and P. GYÖRGY (*Biochem. Z.*, 1923, **142**, 407—416).—A study has been made of the extent to which calcium phosphate and carbonate are formed, when solutions of casein and egg-albumin are treated with sodium phosphate or carbonate at different p_H, and dialysed against calcium chloride. After dialysis the distribution of calcium, phosphate, and fixed carbon dioxide was determined both externally and internally, and the bearing of the results on the process of calcification occurring in animal tissues is discussed. The conclusion is drawn that a reversible fixation of calcium by the tissue proteins takes place. With diminishing metabolic activity, the fixed calcium reacts with phosphate and carbonate to give rise to complex protein–calcium–phosphate–carbonate compounds from which the carbonate is gradually eliminated by the acid nature of the proteins under tissue conditions. The excess of calcium phosphate is then split off, leaving the original active group of the protein free to combine with more calcium (cf. also György, this vol., i, 120). J. P.

The Basis of the Oxidation Theory of Wieland. O. WARBURG (*Biochem. Z.*, 1923, **142**, 518—523).—A criticism of Wieland's views on the mechanism of oxidation. In place of the theory that organic oxidations proceed in two phases, first hydration followed by abstraction of hydrogen by interaction with molecular oxygen, the theory advanced by the author supposes that molecular oxygen is first activated by combination with a catalyst (iron, platinum, or organic catalyst) and then reacts directly with the oxidisable substance. It is argued that Methylene-blue, quinone, etc., do not react like molecular oxygen when substituted for the latter (Wieland, A., 1913, i, 1304), but as activated oxygen, *i.e.*, molecular oxygen + catalyst. The action of hydrocyanic acid in inhibiting oxidations is regarded as opposing Wieland's theory, whilst it receives a ready interpretation on the author's views, since it combines with the catalyst (*e.g.*, iron) and prevents the activation of the oxygen. J. P.

Carnisapidine in Animal Tissues. F. BATTELLI and L. STERN (*Compt. rend. Soc. Biol.*, 1923, **88**, 575—577; from *Chem. Zentr.*, 1923, iii, 1037).—The constituent of animal tissues to which the taste is due is named by the author *carnisapidin*. The amount present in different tissues was determined approximately by dilution of extracts until the taste was just perceptible. The highest content was found in muscles, liver, spleen, and kidneys, lower amounts were found in the thymus, brain, and lungs. G. W. R.

Sarcochromogen in Animal Tissues. F. Battelli and L. Stern (*Compt. rend. Soc. Biol.*, 1923, **88**, 679—681; from *Chem. Zentr.*, 1923, iii, 1037; cf. preceding abstract).—Aqueous extracts of animal tissues contain a sarcochromogen which differs from sarcochrome in not being precipitated by metaphosphoric acid. Sarcochromogen is not changed into sarcochrome on evaporation to dryness if the temperature remains below 80°. G. W. R.

The Rôle of Carnisapidin and Sarcochromogen in Animal Tissues. L. Stern and F. Battelli (*Compt. rend. Soc. Biol.*, 1923, **88**, 681—683; from *Chem. Zentr.*, 1923, iii, 1037; cf. preceding abstracts).—The occurrence of carnisapidin and sarcochromogen in animal tissues appears to have no direct correlation with metabolic exchange. Certain proteins such as casein give, on treatment with pepsin, substances analogous to sarcochromogen. Carnisapidin and sarcochromogen are easily dialysable, and are not attacked by digestive ferments. Carnisapidin, administered intravenously, is eliminated in the urine, but when administered orally or subcutaneously it is retained or decomposed, probably in the liver. G. W. R.

The Extractives of *Actinia equina*. D. Ackermann, F. Holtz, and H. Reinwein (*Z. Biol.*, 1924, **80**, 131—136; cf. A., 1923, i, 1155).—The extractives of *Actinia equina* were subjected to fractionation by Kossel and Kutscher's method. The histidine and arginine fractions have not yet been worked up. In the purine fraction there was obtained adenine, isolated in the form of its picrate. From the lysine fraction there was obtained (1) tetramethylammonium hydroxide (as picrate); (2) a base, identified as pyridylmethylammonium hydroxide (*chloroaurate*, m. p. 252—253°, and *chloroplatinate*); (3) a base of unknown constitution, *actinine*, $C_{13}H_{24}O_5N_2$; from it there were prepared a *chloroaurate*, m. p. 169°, a *chloroplatinate*, dark red, crystalline nodules, decomp. 209°, and a *hydrochloride*, m. p. 207—208°. C. R. H.

The Chemical Differential Diagnosis of Transudates and Exudates. K. Hiruma (*Biochem. Z.*, 1923, **142**, 506—517).—Exudates contain an albumin precipitable by an acetate mixture at C_H $0{\cdot}36 \times 10^{-4}$, by a phosphate mixture at C_H $0{\cdot}214 \times 10^{-2}$, and by a citrate mixture at C_H $1{\cdot}13 \times 10^{-4}$. Inflammatory cerebrospinal fluids contain the same type of albumin as the exudates formed in pleurisy and in peritonitis. The average sugar content of transudates is 0·115% and of exudates 0·07%, the former being in general above the plasma sugar value of the patient, and the latter somewhat lower. The residual nitrogen of transudates, and the amino-nitrogen and ammonia of both transudates and exudates, are approximately the same as those of the blood, whilst the residual nitrogen of exudates is higher than that of transudates. In nephritis, the residual nitrogen is greater the more marked is the œdematous condition. J. P.

The Distribution of Nitrogen in the Urine of Young Dogs and its Dependence on Diet. F. Serio (*Biochem. Z.*, 1923, **142**, 440—453).—The distribution of nitrogen in the urine of young dogs

kept on a diet rich in fat but poor in, or free from, nitrogen is similar to that of starved animals. The urea is diminished and the ammonia increased due to starvation acidosis. The excretion of amino-acids is not influenced by a low nitrogen diet, but it is lowered on a milk diet. The "urease" method for estimating urea gives lower results than the Mörner-Sjöquist method, a difference which is more marked the smaller the ratio of urea nitrogen to total nitrogen becomes. During the period of pre-mortal increase in the nitrogen excretion, the difference disappears, and it is less apparent on a milk diet. The suggestion is made that the difference is due to allantoin and creatinine being estimated along with urea in the Mörner-Sjöquist method, and that the excretion of these urinary constituents varies under the conditions studied, ceasing during the pre-mortal period. J. P.

Sulphatase. III. The Enzymatic Fission of the Ethereal Sulphates in the Urine of the Horse, Camel, and Dog. J. Noguchi (*Biochem. Z.*, 1923, **143**, 186—189).—By treating horse and camel urine with preparations of sulphatase at 37°, hydrolysis of the ethereal sulphates varying from 60 to 90% was obtained. Similar extensive hydrolysis of the organic sulphates occurred in the urine of a dog to which phenol had been administered. J. P.

Acetonuria and Acidosis. D. Adlersberg (*Biochem. Z.*, 1923, **143**, 527—532).—From a study of the effect of orally administered ammonium dihydrogen phosphate on normal subjects kept on a diet free from carbohydrate, and on diabetic subjects, it is concluded that acidosis diminishes the urinary excretion of ketone substances in these conditions. J. P.

Thymolglycuronic Acid. K. Takao (*Z. physiol. Chem.*, 1923, **131**, 304—306).—According to Blum (A., 1892, 1116), thymol is not excreted by the dog as thymolglycuronic acid, although it is so in man. It is now shown, however, that a very small fraction of it is so excreted by the dog. By treatment with sodium hypochlorite and fuming hydrochloric acid, thymolglycuronic acid in the urine was converted into *dichlorothymolglycuronic acid*, $C_{16}H_{22}O_8Cl_2$, colourless needles, m. p. 118—119°, $[\alpha]_D^{20}$ −66·46° (c = 3·9497%) (*barium* salt, $[C_{16}H_{21}O_8Cl_2]_2Ba$). W. O. K.

Phosphorus Metabolism in Avitaminosis. K. Morinaka (*Biochem. Z.*, 1923, **142**, 381—384).—The phosphorus content of the livers of avitaminosed rats to which sodium phosphate, lecithin, or phosphoprotein has been administered does not differ appreciably from that of the livers of similarly fed normal rats. The conclusion is drawn that in avitaminosis the soft tissues do not lose their power of phosphorus retention. J. P.

The Kephalin and Lecithin Content of the Brain in Avitaminosis. H. Naito (*Biochem. Z.*, 1923, **142**, 385—392).—The percentage content of kephalin and lecithin in the brains of avitaminosed rats and guinea-pigs does not depart appreciably from normal, although there is a diminution in the total mass of brain-tissue. J. P.

The Lecithin Content of the Brain and Liver of Normal and Avitaminosed Pigeons after Forced Lecithin Feeding. H. NAITO (*Biochem. Z.*, 1923, **142**, 393—397).—Lecithin feeding slightly increases the amount of the lipoid found in the livers of normal, but not in the livers of avitaminosed pigeons. The lecithin of the brain is not affected in either case. It is concluded that in the avitaminosed liver, phosphorus compounds other than lecithin increase at the expense of the latter (cf. Morinaka, above). J. P.

Fat Metabolism in Avitaminosis. IV. The Gaseous Metabolism of Starved Avitaminosed Rats during Digestion and after Adrenaline Injection. K. ASADA (*Biochem. Z.*, 1923, **143**, 387—398).—The oxygen consumption of partly and completely avitaminosed rats is sub-normal, both during a period of starvation and while a vitamin-free meal is being digested. The extent of the increase observed under the latter conditions compared with the requirements of the starved animals varies inversely with the severity of the avitaminosis. Adrenaline injections produce a greater increase in oxygen consumption in avitaminosed than in normal rats. A discussion is appended of the earlier results obtained in the study of carbohydrate and fat metabolism in avitaminosis. J. P.

Uric Acid and Allantoin Metabolism in Avitaminosis. A. ADACHI (*Biochem. Z.*, 1923, **143**, 408—422).—In avitaminosis, the excretion of allantoin in dogs shows no notable variations, whilst that of uric acid is variable and may show transitory increases. In long-continued avitaminosis, the uric acid excretion is markedly increased and may reach values three times those obtained in normal animals. It is concluded that an increased consumption of nucleins occurs in the later stages of avitaminosis. J. P.

The Pharmacology of the Rare Earths. I. Cerium. S. HARA (*Arch. expt. Path. Pharm.*, 1923, **100**, 217—253).—In general, cerium bears a close resemblance to aluminium and to the heavy metals in its pharmacological action. Proteins are precipitated by moderate but not by strong concentrations of its salts. C. R. H.

The Pharmacology and Toxicology of Carbon Tetrachloride. P. D. LAMSON, G. H. GARDNER, R. K. GUSTAFSON, E. D. MAIRE, A. J. MCLEAN, and H. S. WELLS (*J. Pharm. expt. Ther.*, 1923, **22**, 215—288).—A comprehensive study of the pathological effects following administration to dogs of carefully purified carbon tetrachloride. Oral administration produced only slight and transitory symptoms unless accompanied or preceded by administration of fats or alcohol. When given intravenously, it is lethal in doses of 0·154 c.c. per kg. body-weight.

The pathology of carbon tetrachloride poisoning consists essentially (apart from local irritant effects such as bronchitis following inhalation) in damage done to the liver, one of the first indications of which is the appearance of abnormal amounts of bilirubin in the blood. C. R. H.

The Rôle of certain Carbohydrates in the Organism. E. O. FOLKMAR (*Bibliotek laeger*, 1923, **115**, 120—125; from *Chem. Zentr.*, 1923, iii, 1291).—With continued intravenous injection of pentoses in small amounts at a rate comparable to intestinal absorption, approximately one-half is excreted in the urine. Galactose can be assimilated at the daily rate of 1—2 g. per kg. of live weight without glycosuria or appreciable increase in blood-sugar. Maltose is well utilised, after becoming hydrolysed by maltase. There is an appreciable retention of sucrose in certain circumstances; invertase is, however, not present in blood. Raffinose is completely excreted. The small amounts of ethyl alcohol normally present in blood are attributed to casual decomposition of carbohydrates and not to the action of an alcoholase. G. W. R.

The Influence of Oral Administration of Dextrose on the Blood-sugar and on Glycosuria in Healthy Individuals. G. CONSTAM (*Biochem. Z.*, 1923, **143**, 75—104).—The elimination of sugar in the urine is best followed by estimating the amount excreted in unit time. Observations based purely on changes in concentration do not give dependable results. In normal individuals a mixed meal is followed by a definite glycosuria. Tap water produces a dilution glycosuria which reaches a maximum in about one hour. The assimilation limit for sugar in the same individual is variable and is higher after five hours' than after fourteen hours' abstention from food, whilst the assimilation capacity varies in the opposite sense. In the cases quoted, the tolerance lay between 100 and 150 g. of dextrose in 500 c.c. of water after five hours, and between 20 and 30 g. in the same volume after fourteen hours' starvation; less concentrated solutions caused dilution glycosuria which diminished with increasing concentration of dextrose until the assimilation limit was reached, when a true glycosuria supervened. The blood-sugar reaches its maximum about half an hour before the urinary sugar. The suggestion is made that the power of the organism to raise the sugar tolerance is a protective mechanism which prevents loss of sugar during periods of diminished rate of assimilation. J. P.

A New Method of Preparing *s*-Diphenylguanidine. Its Pharmacological Effects. O. RIESSER (*Z. physiol. Chem.*, 1923, **131**, 204—213).—Diphenylguanidine is formed together with a small amount of triphenylmelamine when dicyanodiamine is heated with aniline hydrochloride at 190—200°. It crystallises from alcohol in white needles, m. p. 145—148°, and forms a chloroaurate. The pharmacological action of this substance has been investigated. It increases the reflex irritability and also causes central paralysis. In warm-blooded animals, it causes death by arrest of the respiration. It produces progressive paralysis when applied to isolated frog's muscle. W. O. K.