

# BRITISH CHEMICAL AND PHYSIOLOGICAL ABSTRACTS

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## A., III.—PHYSIOLOGY & BIOCHEMISTRY (INCLUDING ANATOMY)

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OCTOBER, 1942.

## I.—GENERAL ANATOMY AND MORPHOLOGY.

**Lung lobation in rhesus monkey compared with man.** R. E. Chase (*Amer. J. Phys. Anthropol.*, 1942, 29, 267—286).—The right lung has 4 lobes—upper, middle, lower, and cardiac. The left lung is divided by two fissures into 3 lobes, the upper fissure being incomplete laterally. Normal lobation occurred in over 90%. Variants were mainly in the form of accessory lobes. No lobes referable to abnormal azygos veins were found. The lungs of 458 monkeys were examined. W. F. H.

**New approach to knee joint.** V. O. Mader (*Canad. Med. Assoc. J.*, 1940, 42, 17—18). C. J. C. B.

**Vascularisation in brains of reptiles. Quantitative studies in alligator.** H. Craigie (*J. Anat.*, 1942, 76, 347—355).—A description is given of the capillary bed in the alligator's central nervous system. There is a continuous spongy reticular vascular bed in which there are localised differences in its density. Comparisons are made with the vascular bed in the turtle and in amphibians. W. J. H.

**Periovarial sac in albino rat.** R. H. Alden (*Anat. Rec.*, 1942, 83, 421—435).—The periovarial sac opening is functionally closed at certain periods of the oestrous cycle, probably by the fimbriated tip of the oviduct. When the opening is closed artificially by suture, expansion of the capsule results and the oestrous and gestational periods are affected. The presence of the connexion between the sac and the abdominal cavity is regarded as essential to the normal physiology of the region. W. F. H.

**[Familial incidence of] osteopoikilosis ["speckled bones," Albers-Schönberg].** E. H. McLean (*Northw. Med.*, 1942, 41, 92—93).—Report of a case with widely disseminated patches of osteosclerosis in a man aged 20 whose father at the age of 70 years had a few scattered spots in hands, feet, and pelvis, possibly the end stage of the disease, and whose mother showed no abnormality. 2 living sisters have not been examined. E. M. J.

**Pathology of atrophic arthritis.** C. L. Steinberg (*J. Lab. clin. Med.*, 1942, 27, 435—443).—The histology of the synovial layer in atrophic arthritis varies from polymorphonuclear infiltration to round cell infiltration, both perivascular and non-perivascular. Later there is little round cell infiltration but replacement of the synovial tissue by means of scar tissue. (12 photomicrographs.) C. J. C. B.

**Progress in prematurity care.** R. H. Loder (*Nebraska Sta. Med. J.*, 1941, 26, 177—179). E. M. J.

**Case in which all pulmonary veins drain into superior vena cava.** L. C. Conn, J. Calder, J. W. MacGregor, and R. F. Shaner (*Anat. Rec.*, 1942, 83, 335—340).—The four pulmonary veins drain by a single trunk dorsal to the right bronchus into the superior vena cava. The trunk is probably a hypertrophied right bronchial vein. For the majority of anomalous pulmonary veins, the bronchial vein or some other persisting channel of the early cardinal system is suggested as the significant factor. W. F. H.

**Abnormal venous system in frog.** E. A. Spaul and J. H. Elliott (*Proc. Leeds Phil. Soc., Sci. Sect.*, 1941, 4, 19—24).—Deviations of the venous system from the normal are described in a mature male frog. They were the result of the persistence of larval vessels and their combination with adult vessels. A. S.

**Malformation of hind end of body.** S. Sunderland (*Austral. N.Z. J. Surg.*, 1941, 11, 97—109).—A case (full-term female) is reported in which the gut and genito-urinary ducts opened into a common cloacal chamber from which a canal passed to open at the perineum. The various relevant theories are discussed and the suggestion is put forward that fusion of the Müllerian ducts is responsible for the separation of the hind gut from the ventral cloaca and that further development of the hind end is associated with changes in the distal ends of those ducts. H. L.

**New-born human presenting symphys dipus, anomalous umbilical vein, transposition of viscera, and other anomalies.** E. H. Shryock, J. Janzen, and M. C. Barnard (*Anat. Rec.*, 1942, 82, 347—360). W. F. H.

## II.—DESCRIPTIVE AND EXPERIMENTAL EMBRYOLOGY. HEREDITY.

**Early human foetal movements.** J. E. Fitzgerald and W. F. Windle (*J. comp. Neurol.*, 1942, 76, 159—168).—Movements were elicited in three unanaesthetised, non-narcotised specimens of about 8 weeks' gestation (22—26 mm. crown-rump length). While the foetus was receiving oxygenated blood from the intact placenta its neuro-muscular mechanism was highly excitable and movements which appeared to be individual reflexes of the trunk, arms, and legs were elicited by tapping the amniotic sac lightly. During progressive anoxia these reflexes ceased but responses of trunk musculature to stimulation of mouth and nose regions were obtained for several min. after placental separation. The muscles retained excitability long after all reflexes had been abolished by asphyxia or anaesthesia. J. D. B.

**Experiments on developing rats. III. Induction of artificial pregnancy.** J. S. Nicholas (*Anat. Rec.*, 1942, 83, 457—469).—Two-celled eggs when transplanted to the uterus of virgin rats develop normally when the uterus has been sensitised. Sensitisation was secured by vaginal stimulation applied in late stage three of oestrus and, in addition, stimulation either direct, by mechanical methods, or indirect, through hypophyseal transplantation. W. F. H.

**Duration of fertilising capacity of spermatozoa in female genital tract of rat.** A. L. Soderwall and R. J. Blondau (*J. exp. Zool.*, 1941, 88, 55—64).—The max. duration of fertilising capacity of spermatozoa artificially inseminated into 184 female rats was 14 hr. This is greater than that reported for the mouse sperm (6 hr.) and less than that for either guinea-pig (22 hr.) or the rabbit (30 hr.). Litter size was reduced in the rats inseminated 10—14 hr. prior to ovulation. There were no abnormal pregnancies observed and no significant differences in the sex ratios of the offspring when compared with the controls. J. D. B.

**Neural differentiation without organiser.** L. G. Barth (*J. exp. Zool.*, 1941, 87, 371—383).—Large explants of the gastrular ectoderm of *Amblystoma punctatum* will form neural tubes in the absence of the organiser. The frequency of this differentiation without organiser is increased when the antero-posterior axis of the explant is preserved during healing. J. D. B.

**Development of gastrula ectoderm in regenerating tissue of *Rana pipiens* larvae.** H. S. Emerson (*J. exp. Zool.*, 1941, 87, 403—427).—Gastrula ectoderm in blastema tissue develops into epidermis with superficial and sensory layers, nervous tissue, suckers, cartilage, and possibly dermis. Blastema mesenchyme is therefore a very favourable environment for the development of gastrula ectoderm. J. D. B.

**Ultrastructure of neural plate and tube of early chick embryo.** L. B. Hobson (*J. exp. Zool.*, 1942, 88, 107—134).—The neural tube of living early chick embryos shows measurable birefringence (of the order of  $4 \times 10^{-5}$ ), positive with respect to the long axis of the cells in its walls. Similar birefringence is seen in the neural plate when observed laterally and there are no significant changes during closure. Dehydration experiments suggest that fold closure is the result of a differential inhibition of water by the neural plate. J. D. B.

**Thermal isolation of animal hemisphere of frog embryo. I. Development during isolation.** H. F. Drury (*J. exp. Zool.*, 1941, 88, 219—237).—A temp. differential was applied in such a way that the vegetal hemisphere was subjected to a temp. of 4—5° c. while simultaneously exposing the animal hemisphere to a temp. of 24—25°. Under these conditions gastrulation may be suppressed and in its absence the presumptive ectoderm is not subjected to the induction associated with the process. J. D. B.

**Method of inducing limb regeneration in adult *Anura*.** S. M. Rose (*Proc. Soc. Exp. Biol. Med.*, 1942, 49, 408—410).—Partial regeneration was caused in frog upper limb amputation stumps by treatment twice daily with saturated NaCl solution. V. J. W.

**Morphological effects of denervation and amputation of limbs in urodele larvae.** O. E. Schotté and E. G. Butler (*J. exp. Zool.*, 1941, 87, 279—322).—In larvae of *Amblystoma* and *Triturus* from 16 to 5 mm. in length forelimbs which have been rendered nerveless (by

repeated resections of the brachial plexus) lose their capacity for regeneration. Ability to regenerate is regained as soon as the limb is reinnervated. The experiments are interpreted as demonstrating that the nervous system enables amputated limbs to carry out the complex cellular interactions responsible for regeneration of the urodele limb. J. D. B.

**Rôle of ectoderm in pigment production studied by transplantation and hybridisation.** L. E. deLanney (*J. exp. Zool.*, 1941, **87**, 323—345).—An analysis of the results of experiments in which flank ectoderm was exchanged in various heteroplastic and xenoplastic combinations to test the action of this tissue on the no. and distribution of amphibian melanophores. Hybrids of different species of *Triturus* were also used. J. D. B.

**Influence of adrenal and sex hormones on differentiation of melanophores in chick.** H. L. Hamilton (*J. exp. Zool.*, 1941, **88**, 275—305; cf. A., 1941, III, 268).—Addition of hormones to culture fluid of explants of skin ectoderm has a marked influence on the development of melanophores. The hormones apparently act directly on the melanophores either to catalyse or to inhibit the rate of melanin synthesis within the cell, since the effects on rate of growth are inadequate to explain the effect. J. D. B.

**Origin of melanophores.** C. M. Osborn (*Biol. Bull.*, 1941, **81**, 341—351).—When the normally pale ventral surface of dark-adapted *Paralichthys dentatus* is exposed to light melanophores appear. The positive "dopa" reaction and other data suggest that they are derived from melanophores previously present and not by migration. D. M. SA.

**Temperature change and subsequent rate of development.** F. J. Ryan (*J. exp. Zool.*, 1941, **88**, 25—54).—Experiments are described indicating that the rate of cleavage and later development in *Rana pipiens* can be influenced by previous temp. history. Removal to a high temp. after exposure to a low temp. results in a rate which is faster than that of animals always kept at the same high temp. The converse also holds. There is a crit. review of the literature on temp. changes. J. D. B.

**Collapse of archenteron in embryos of *Amblystoma* and *Rana*.** M. G. Brown (*J. exp. Zool.*, 1941, **88**, 95—106).—The sharp increase in density of neurulae of amphibia is described and its probable causes are discussed. The increased density is associated with a loss of water in the neurula stage accompanying the loss of fluid from the archenteric cavity into the perivitelline space and collapse of the archenteron. There is also an accompanying change in the respiratory rate. J. D. B.

**Mechanics of gastrulation in *Dendrobaena excentricus*.** A. R. Moore (*J. exp. Zool.*, 1941, **87**, 101—112). J. D. B.

**Regeneration in *Perionyx excavatus*.** G. E. Gates (*J. exp. Zool.*, 1941, **88**, 161—185).—An account of regeneration under various conditions in this tropical earthworm. J. D. B.

**Rôle of thyroid and pituitary in anomalous effect of inanition on amphibian metamorphosis.** S. d'Angelo, A. S. Gordan, and H. A. Charipper (*J. exp. Zool.*, 1941, **87**, 259—277).—Histological and physiological evidence is adduced to support the interpretation that the failure to metamorphose in complete starvation of *Rana* tadpoles is directly attributable to a decreased production or release of thyrotropic hormone from the anterior hypophysis. J. D. B.

**Pædogenesis in *Micromalthus*.** A. Scott (*Biol. Bull.*, 1941, **81**, 420—431).—The pædogenetic female gives birth to one male embryo which normally devours the mother. If he is removed the female produces a crop of female eggs which develop into larvæ similar to that which produced the mother. This reversal is environmental rather than intrinsic. D. M. SA.

**Tuberous sclerosis complex.** S. E. Moolten (*Arch. intern. Méd.*, 1942, **69**, 589—623).—A case of tuberous sclerosis complex in a girl aged 20 is described. There were a renal hæmatoma, adenoma sebaceum, phacomia, and clinical signs of cerebral involvement. It is suggested that the condition is due to a defective mechanism of induction by embryonic organisers. C. A. K.

**Electric impedance of frog's egg.** K. S. Cole and R. M. Guttman (*J. Gen. Physiol.*, 1942, **25**, 765—775).—The impedance of fertilised and unfertilised eggs of *Rana pipiens* over a frequency range of 0.05—10 kc. is determined. The resistance of the plasma membrane of the egg is 170  $\Omega$ . cm.<sup>2</sup>, the capacity of the membrane is 2.0  $\mu$ v. per cm.<sup>2</sup>, the phase angle of the membrane is 86°, and sp. resistance of the interior is 570  $\Omega$ . cm. These are average vals. and do not change on fertilisation. There are no spontaneous, rhythmical impedance changes such as occur in the trout's egg (A., 1940, III, 217). J. N. A.

**Cell metabolism and cell division. VI. Glycogen content, carbohydrate consumption, and lactic acid and ammonia production of eggs of *Arbacia punctulata*.** J. O. Hutchens, A. K. Keltch, M. E. Krahl, and G. H. A. Clowes. **VII. Amount and possible function of diphosphoanerin (cocarboxylase) in eggs of *A. punctulata*.** M. E. Krahl, B. J. Jandorf, and G. H. A. Clowes. **VIII. Diphosphopyridine nucleotide (cozymase) content of eggs of *A. punctulata*.**

B. J. Jandorf and M. E. Krahl (*J. Gen. Physiol.*, 1942, **25**, 717—731, 733—747, 749—754; cf. A., 1941, III, 1039).—VI. The eggs contain approx. 110 mg. of acid-hydrolysable carbohydrate (approx. 50% of which is glycogen) per g. of egg protein. This carbohydrate is almost all in the egg proper, little or none occurring in the jelly: no reducing sugar is present. Approx. 7% of the dried egg is carbohydrate, and approx. 65% is protein. Whilst all eggs utilise carbohydrate from the 15th to the 24th hr. of development at 20°, some samples during the 1st to the 6th hr., the period in which cell division proceeds most rapidly, show no utilisation of carbohydrate. During the 1st to the 24th hr. of development, only negligible amounts of lactic acid, but considerable amounts of NH<sub>3</sub>, are formed.

VII. Methods for determining cocarboxylase in the eggs are described. The enzyme is quantitatively extracted when aneurin hydrochloride is present in the extraction fluid and by extracting at  $p_H$  6.3—6.7. Unfertilised eggs contain the equiv. of 2—3  $\mu$ g. of natural yeast-cocarboxylase per g. of wet eggs, whilst eggs fertilised for 30 min. and 10 hr. contain somewhat less. Pyruvic acid is utilised when added to egg cytolysates, and the rate of disappearance is greater under aerobic than under anaerobic conditions. It is also greater for cytolysates from fertilised than from unfertilised eggs.

VIII. Undried eggs contain approx. 25—50 mg.-% of diphosphopyridine nucleotide, and approx. 25—40% of this amount is present in an alkali-stable (probably dihydro-) form. Triose phosphate and glutamic acid dehydrogenases are not present in egg cytolysates. J. N. A.

**Development of minute phenotype in *Drosophila melanogaster*.** K. S. Brehme (*J. exp. Zool.*, 1941, **88**, 135—160).—A comparative study of three minute mutants. J. D. B.

**Flight capacity in relation to phenotypic and genotypic variations in wings of *Drosophila melanogaster*.** M. H. Harnley (*J. exp. Zool.*, 1941, **88**, 263—273). J. D. B.

**Reciprocal crosses between guinea and domestic fowl.** R. D. Owen (*J. exp. Zool.*, 1941, **88**, 187—217).—An analysis of the factors which act to keep the species *Numidia* and *Gallus* separate. J. D. B.

### III.—PHYSICAL ANTHROPOLOGY.

**Method of measuring bone growth in skull.** N. Giblin and A. Alley (*Anat. Rec.*, 1942, **83**, 381—387).—Suture growth was measured by means of inlays of bone wax on the surface of the skull. Accretion of bone on the outer surface and resorption on the cerebral surface were also demonstrated by the method. W. F. H.

### IV.—CYTOLOGY, HISTOLOGY, AND TISSUE CULTURE.

**Porphyrin-excreting Harderian gland of albino rat.** A. L. Grafflin (*Amer. J. Anat.*, 1942, **71**, 43—64).—The secreting cells of the Harderian gland are of two types; "principal" cells, present in large nos., and "clear" cells, relatively few in no. The red fluorescence of both the cytoplasm and pigment accumulations of the glandular lumina is ascribed to the presence of protoporphyrin and coproporphyrin. W. F. H.

**Structure of thyroid gland in whales.** A. L. Grafflin and E. M. K. Geiling (*Anat. Rec.*, 1942, **83**, 367—379).—The gross and microscopic structure of the gland in the adult sperm whale and the blue whale are described. The total wt. of the gland is considerably higher in the blue whale than in the sperm whale. The histological structure shows no significant differences from that in the non-aquatic mammals. "Parafollicular" cells of Nonidez were not observed. W. F. H.

**Development of lymph nodes in fat.** E. S. J. King (*Austral. N.Z. J. Surg.*, 1940, **10**, 126—134).—New development of lymph nodes may occur in adult life; such new nodes almost invariably arise in fatty tissue. H. L.

**Use of serum ultrafiltrate in tissue cultures for studying deposition of fat and for propagation of viruses.** H. S. Simms and M. Sanders (*Arch. Path.*, 1942, **33**, 619—635).—Methods of making serum ultrafiltrate suitable for tissue culture mediums are described. Serum ultrafiltrate is used for preparing cultures free from fat granules. By these cultures fat deposition in cultures of adult arterial tissue is shown to be produced by the B factor (cf. *ibid.*, 1937, **23**, 332) present in serum and tissues and is prevented by an anti-B factor present in serum. (4 photomicrographs.) C. J. C. B.

**Metabolism of tissue cultures. I. Chick embryo.** H. Wilson, E. B. Jackson, and A. M. Brues (*J. Gen. Physiol.*, 1942, **25**, 689—703).—Chick embryo muscle, heart, and liver tissues, 9—13 days old, rapidly utilise glucose: in absence of glucose the tissues soon degenerate. The initial rate of utilisation is increased by increase in the concn. of glucose from 0.1 to 0.5%. Although glucose consumption is necessary for survival of cultures, it can be used at a

rate far greater than that required for life and max. growth. Complete blocking of mitosis by colchicine does not affect the rate of utilisation of glucose, whilst phloridzin increases the no. of mitoses, and probably acts by prolonging the mitotic process rather than by stimulating growth. The cultures show proteolytic activity as indicated by an increase in amino-N in a peptone medium after incubation; in an amino-acid medium, amino-N decreases. Cells that obtain their N from amino-acids proliferate similarly to those grown in a medium containing peptone instead of amino-acids, but the cell type is very different. In media that contain only small amounts of glucose, most of the utilised amino-N is converted into urea and  $\text{NH}_3$ , whilst in presence of glucose only negligible amounts are formed. Lactic acid is formed in presence and absence of glucose, and is increased with increased glucose utilisation. Pyruvate (and perhaps lactate) can be utilised and favour growth in absence of glucose. J. N. A.

**Marchi method.** F. A. Mettler and R. E. Hanada (*Stain Tech.*, 1942, 17, 111—116).—Faulty straining is often due to slices being more than 3 mm. thick, or to a deterioration of the  $\text{OsO}_4$  concn. in the Marchi mixture. The amount of  $\text{OsO}_4$  present can be determined by an application of the Alvarez test. Some helpful remarks on Busch's method are added. E. E. H.

**Rapid silver-on-the-slide method for nervous tissue.** M. L. Silver (*Stain Tech.*, 1942, 17, 123—127).—Formaldehyde fixation is used for nerve fibres and endings, followed by subsequent mordanting with 3%  $\text{K}_2\text{Cr}_2\text{O}_7$  for myelinated fibres and mitochondria. The protargol (0.5%) is mixed with the reducer (1.6% Rochelle salt + traces of  $\text{AgNO}_3$ ,  $\text{MgSO}_4$ , and  $\text{K}_2\text{S}$ ); sections are put in the mixture, warmed to 55°, and stained progressively. E. E. H.

**Polychrome methylene-blue. II. Acid oxidation methods of polychroming.** R. D. Lillie (*Stain Tech.*, 1942, 17, 97—110; cf. A., 1942, III, 570).— $\text{K}_2\text{Cr}_2\text{O}_7$ ,  $\text{Ag}_2\text{O}$ , and  $\text{KMnO}_4$  produce polychroming proportional to the amount of oxidant used, and at 100° it is complete in 15 min. With  $\text{K}_2\text{Cr}_2\text{O}_7$ , subsequent neutralisation of the  $\text{H}_2\text{SO}_4$  with  $\text{BaCO}_3$  removes the salts and prevents alkali polychroming. E. E. H.

**Sectioning and staining refractory materials in paraffin.** T. N. Tahmian and E. H. Slifer (*Science*, 1942, 95, 284).—A new combination of usual methods gave sections of 4—6  $\mu$ . of tissues, e.g., vertebrate eye lens. E. R. S.

**Clarite in embedding paraffin for thin sections.** W. Wehrle (*Stain Tech.*, 1942, 17, 131—132).—The addition of 5% of clarite and 5% of bleached beeswax to 90% (by wt.) of paraffin of m.p. 53° renders the block hard enough to make thin sections in a warm room. E. E. H.

**Staining rack for cover-glass preparations.** T. T. Chen (*Stain Tech.*, 1942, 17, 129—130).—Three parallel rods, one at the bottom and two on the sides, are held together by two end pieces; each rod has 12 slots for holding coverslips apart. The end pieces have holes for inserting the wire tongs or handle. E. E. H.

**Washing bobber.** F. W. Gairns (*Stain Tech.*, 1942, 17, 131).—A rectangular piece of perforated Zn is bent around something cylindrical about 3 in. diameter; the overlapping ends are soldered, and the open base of the tube is closed by another soldered piece of perforated Zn. The tissue is put in, the open end corked, and it is then thrown into a bowl in the sink under a running tap. About 20 bobbers can be used in one bowl. E. E. H.

## V.—BLOOD AND LYMPH.

**Blood.** F. H. Bethell, C. C. Sturgis, R. A. Hettig, and O. T. Mallory (*Arch. intern. Med.*, 1942, 69, 856—926).—Review of recent literature. C. A. K.

**Effect of hæmorrhage on red cell size and distribution.** G. L. Brown, J. A. R. Miles, J. M. Vaughan, and L. E. H. Whitby (*Brit. Med. J.*, 1942, I, 99—102).—Red cell count, hæmoglobin %, red cell vol., red cell diameter, and reticulocyte count were investigated after hæmorrhage in animals and man. Minor increases of red cell size occur after a single large acute hæmorrhage but not after repeated small hæmorrhages. There was no evidence for immobilisation of swollen red cells in muscle capillaries as described by Brennan (A., 1940, III, 789). The cause of the slight swelling is not clear. Hæmodilution is usually complete in about 24 hr. C. A. K.

**Hæmoglobin radioactive iron liberated by erythrocyte destruction acetylphenylhydrazine** promptly neutralised to form new hæmoglobin. W. O. Cruz, P. F. Holm, and W. F. Bale (*Amer. J. Physiol.*, 1942, 135, 595—599).—Fe liberated from hæmoglobin derived from red cells destroyed by acetylphenylhydrazine (pyrocin) is used readily and nearly quantitatively for the regeneration of hæmoglobin in the new red cells during the period of spontaneous recovery in dogs made anæmic by bleeding. In normal dogs, with ample reserve Fe stores, the hæmoglobin-Fe of new red blood cells is

derived from the Fe of old cells broken down in normal wear and tear of the animal's blood, rather than from reserve stores.

M. W. G.

**Radioactive iron used to study red blood cells over long periods. Constancy of total blood volume in dog.** P. F. Holm, W. F. Bale, and W. H. Balfour (*Amer. J. Physiol.*, 1942, 135, 600—605).—When circulating erythrocytes have been tagged by the incorporation of radioactive Fe into the hæmoglobin, they may be followed in the body for many months. Radioactive Fe was given to dogs orally or by vein. Following the establishment of a const. level of isotope and hæmoglobin, Fe in the form of non-radioactive colloidal  $\text{Fe}(\text{OH})_3$  was given by vein. When disintegration of the red cells occurs, either by ageing or by trauma, even in the presence of adequate inert Fe, the labelled Fe from liberated hæmoglobin is immediately re-utilised by new cells such that the total circulating radioactivity is maintained const. Total blood vol. of the dog is maintained at a const. level independent of the state of anæmia. M. W. G.

**Origin and nature of Cabot ring bodies of erythrocytes.** E. M. Schleicher (*J. Lab. clin. Med.*, 1942, 27, 983—999).—By special staining methods and by examination of films of egg white it is concluded that the formation of a Cabot ring body is as follows; a sp. hæmolytic agent, e.g., bile acid, affects certain erythrocytes whether basophilic, polychromatic, or orthochromatic; the lysin injures the envelope, producing changes in the lipoprotein constituents of the surface layer which influence the permeability and may lead to microscopic tears in the envelope. This physicochemical change encourages dissociation of the lipoprotein layers, leading to a separation of the normally fused layers into individual ones, or to the separation of a mass of fused layers. This irregular splitting produces the variability of flatness and thickness of the ring bodies. This segregation of protein layers occurs during the drying process, leading either to the formation of the protein configurations known as Cabot ring bodies, or to globules or bars. Cabot ring bodies are thus neither nuclear remnants nor identical with the nuclear membrane; they are laboratory creations, the expression of cellular degeneration induced by hæmolytic agents; they are the expression of aggregated and denatured colloid protein. C. J. C. B.

**Hæmopoiesis in lead poisoning.** J. E. Kench, A. E. Gillam, and R. E. Lane (*Biochem. J.*, 1942, 36, 384—388).—The physiological action in Pb workers of small amounts of Pb is not due to partial prevention of Fe-porphyrin complex formation, but to restricted formation of protoporphyrin which is incidental to reduced cellular activity. Erythrocytes are hypochromic and reduced in no. according to the degree of inhibition. In the early stages of Pb poisoning increased formation of breakdown products of erythrocytes stimulates the bone marrow. Administration of citrate is recommended to increase mobilisation and excretion of Pb without increasing the concn. of toxic ions, because the citrate complex is the least dissociated of the sol. complexes investigated. P. G. M.

**Formation of phosphorylated glyceric acid in blood cells of various species.** S. Rapoport and G. M. Guest (*J. Biol. Chem.*, 1942, 143, 671—677).—Ox and goose blood was incubated with NaF and Na pyruvate in 0.02N. and 0.01N. solution respectively for 2½ hr. Monophosphoglyceric acid (Ba salt) was isolated in each case, being presumably formed by dephosphorylation from the labile diphosphoglyceric acid found in the blood cells of all mammalian young. P. G. M.

**Blood groups of the Doms.** D. N. Majumdar (*Current Sci.*, 1942, 11, 153—154).—The Hill Doms (an Indian tribe) show a higher % of group O (36.0) than the "criminal" Doms (32.8), whilst the latter have a higher proportion (39.4) of group B than the former (33.8). The incidence of groups B and AB combined is the same (44.0—44.4) for both settlements. P. G. M.

**Filtration of human plasma and serum.** R. G. Macfarlane, J. C. Macsween, B. R. S. Mainwaring, and H. J. Parish (*Brit. Med. J.*, 1942, I, 377—381).—Technical details for the production of human plasma and serum on a large scale are given. C. A. K.

**Faints in blood donors.** H. Brown and P. McCormack (*Brit. Med. J.*, 1942, I, 1—5).—Observations on 48 donors who fainted during withdrawal of blood are reported. The majority showed a fall in pulse rate and blood pressure. The condition is compared with the fainting attacks of effort syndrome and of hyperactive carotid sinus. C. A. K.

**Fainting in blood donors.** C. L. Greenbury (*Brit. Med. J.*, 1942, I, 253—255).—An analysis of 5897 unselected blood donors. The incidence of fainting is higher in the young, in females, and in clerical workers. C. A. K.

**Effect of hypertonic plasma on body fluids in normal animals.** C. T. Ashworth, E. E. Muirhead, and J. M. Hill (*Amer. J. Physiol.*, 1942, 136, 194—199).—Intravenous injection of hypertonic plasma in normal dogs causes a long, sustained increase in plasma vol. proportional to the amount of protein injected; red cell count, hæmoglobin, and hæmatocrit decrease. Total extracellular water vol. increased as shown by dilution of thiocyanate. This water is mainly derived from cellular water; the redistribution depends on

osmosis and diffusion between the cells and their surrounding fluid environment. M. W. G.

**Effects of partial hepatectomy on blood volume in white rat.** P. Lawrence and A. Chanutin (*Amer. J. Physiol.*, 1942, **135**, 606—608).—Plasma, red cell, and total blood vols. decreased markedly in partly hepatectomised rats during the first 48 hr. Plasma vol. reached control level on the 7th day after operation, total blood vol. on the 9th day; red cell vol. determinations were within control range on the 7th day. M. W. G.

**Effects of large intravenous infusions on body fluid.** J. D. Stewart and G. M. Rourke (*J. clin. Invest.*, 1942, **21**, 197—205).—3 patients who had simple surgical procedures involving slight loss of extracellular fluid were given continuous infusion of 0.9% NaCl and of 5% glucose solution. 6.5 l. daily of NaCl solution increased the vol. of extracellular fluid. The daily increments, however, become progressively smaller and at the end of 3—4 days a max. vol. was reached which was followed by a gradual decline. In terms of initial vols. the increase of interstitial fluid was 90% and that of plasma 60%. Plasma-protein is added to maintain a protein concn. of nearly 6 g.-%; oedema does not develop. Continuous infusion of 5% glucose solution (2.4 l. on first day and 6 l. for 24 hr. thereafter) reduces the vol. of extracellular fluid, as evidenced by loss of body wt. and of Na. This dehydrating effect is caused by an incomplete conservation of Na by the kidney. Urine-Na concn. was below 0.01M. and plasma-Na did not fall. Infusion of glucose solution in a single patient at an initial rate of 5.5 l. per 24 hr. caused derangement of renal control of the electrolyte structure of the plasma (water intoxication). This was shown by a failure to conserve Na and a rapid fall of Na concn. in the plasma. With cessation of infusion there was immediate recovery of renal control. The concn. of Na fell in the urine and rose to its usual level in the plasma. C. J. C. B.

**Lymphoid cells of bone marrow and lymph nodes of rabbits and guinea-pigs.** R. D. Sundberg and H. Downey (*Amer. J. Anat.*, 1942, **70**, 455—499).—The bone marrow of rabbits and guinea-pigs contains myeloblasts with a nuclear structure similar to that of human myeloblasts. Evidence of heteroplastic regeneration of lymphocytes from the reticulum of lymph nodes was observed. Leucoblasts of the Rieder type occur in the marrow of normal animals; they resemble the reticular lymphocytes of lymph nodes and differ markedly from leucoblasts of human marrow. In the rabbit and guinea-pig the leucoblast is not a necessary sequence in the development of the granulocyte. The micromyeloblast is a cell type which can be distinguished from the small lymphocyte. W. F. H.

**Measurement and metabolism of thiamin and of pyrimidine stimulating yeast fermentation found in blood cells and urine of (A) normal individuals, (B) patients with leukaemia.** (A) A. T. Gorham, J. C. Abels, A. L. Robins, and C. P. Rhoads. (B) J. C. Abels, A. T. Gorham, L. Craver, and C. P. Rhoads (*J. clin. Invest.*, 1942, **21**, 161—176, 177—189).—(A) Methods for the determination of thiamin and the pyrimidine accelerator of yeast fermentation (PAYF) are adapted for application to the white cells and erythrocytes of normals. The thiamin concn. of leucocytes and platelets is 10 times that of the erythrocytes. In the course of its metabolic activity, thiamin is probably broken down to the PAYF compound.

(B) The average total thiamin levels in the leucocytes and platelets of 33 patients with leukaemia are 3 times the normal average val.; in 82% of the patients they are above the highest normal. The concn. of PAYF in the leukemic leucocytes and platelets forms an abnormally small % of the total thiamin content. The average total thiamin levels in the erythrocytes of leukemic patients are twice the normal average, and are above the highest normal range in 35% of the patients examined. In the erythrocytes, however, the concns. of PAYF formed a normal % of the total thiamin. No correlation exists between the concn. of blood-cell total thiamin and the form, severity, or degree of associated leucocytosis of the leukaemia, the sex or age of the patient, nor between the white cell concn. of thiamin and the apparent youth of the cells. Patients with leukaemia excrete normal amounts of both thiamin and PAYF in the urine. The high concn. of white-cell total thiamin is attributed to impaired utilisation of the thiamin, and not to increased ingestion or faulty excretion of the vitamin, or the youth of the cells involved. Elevated blood-cell concns. of total thiamin were found in patients with Hodgkin's disease and cancer of the gastro-intestinal tract, but not in patients with portal hepatic cirrhosis. C. J. C. B.

**Quantitative and qualitative platelet values of normal newborn infants.** H. N. Sanford and I. Shmigelsky (*Amer. J. Dis. Child.*, 1942, **63**, 729—732).—There is no change in the platelet count of the newborn for the first 10 days of life. The platelets of the newborn show increasing resistance to disintegration until the 4th day of life, after which there is a slow return to normal (adult) coagulation time by the end of 5 days. C. J. C. B.

**Simple method of evaluating blood platelets.** E. W. Pernokis (*J. Lab. clin. Med.*, 1942, **27**, 1069).—Moist films are made on glass slides which have a film of 1% cresyl-blue. C. J. C. B.

**Hæmoglobinometry.** C. Rimington (*Brit. Med. J.*, 1942, **I**, 177—178).—A method for rapid determination of the total blood pigment in samples of whole blood is described. All hæm pigments are converted into pyridine-hæmochromogen and the intensity of absorption of the latter around 550 m $\mu$  is measured with the Hilger-Nutting photometer. C. A. K.

**"Black line" in hæmatocrit determinations.** J. P. Baumberger (*J. Lab. clin. Med.*, 1942, **27**, 1084—1085).—The "black line" appears between the leucocytes and the red cells and is a layer of erythrocytes in which the oxyhæmoglobin has been reduced to hæmoglobin by the metabolic activity of leucocytes. C. J. C. B.

**Shaking device used in collection of blood for transfusion.** L. W. Diggs and H. B. Turner (*J. Lab. clin. Med.*, 1942, **27**, 1070—1071). C. J. C. B.

**Improved pycnometer for blood serum.** R. A. Mortensen (*J. Lab. clin. Med.*, 1942, **27**, 1096). C. J. C. B.

**Relationship of plasma-proteins to corrected sedimentation rate.** I. Kopp (*J. Lab. clin. Med.*, 1942, **27**, 1072—1077).—No abs. correlation was found between plasma-fibrinogen, -albumin, -albumin/-globulin, or -globulin/fibrinogen ratios on the one hand and the corr. sedimentation rate on the other in 5 patients undergoing therapeutic fever. The corr. sedimentation rate was increased in most instances when globulin vals. rose to the upper limit of normal (3 g.-%) and above, regardless of fibrinogen levels. C. J. C. B.

**Plasma-proteins in therapeutic fever.** I. Kopp (*J. Lab. clin. Med.*, 1942, **27**, 1054—1061).—Fever produced by inoculation malaria results in a rapid marked drop in plasma-albumin concn. and fluctuations in -globulin and -fibrinogen. Termination of malarial fever by quinine is followed by a rapid return to normal vals. Fever produced by typhoid vaccine and artificial means (inductotherm) causes only slight fluctuations in the plasma-protein fractions. C. J. C. B.

**Rheology of blood. II. Effect of fibrinogen on fluidity of plasma.** E. C. Bingham and R. R. Roepke (*J. Amer. Chem. Soc.*, 1942, **64**, 1204—1206).—Linear relationships exist between the fluidity and concn. of solutions of fibrinogen from ox plasma. The fluidity-lowering const. of fibrinogen is greater than that of other blood-proteins; that of heparinised blood plasma is less than that of saline solutions of purified fibrinogen. W. R. A.

**Quantitative studies on antithrombin.** S. J. Wilson (*Arch. intern. Med.*, 1942, **69**, 647—661).—A test for the determination of antithrombin in serum and plasma is described. One unit of antithrombin is that amount which inactivates 1 unit of thrombin in 4 min. at 28°. Normal human plasma or serum contains an average of 90 units of antithrombin per c.c. During blood coagulation only a small portion of thrombin is adsorbed on to fibrin, the remainder being neutralised by antithrombin. The hæmorrhagic tendency in hypoprothrombinæmia is therefore not fully explained by the decreased prothrombin and variations in the conversion rate of prothrombin into thrombin. The hæmorrhagic diathesis usually occurs when the prothrombin unitage approximates to or is lower than the antithrombin unitage. C. A. K.

**Purification of thrombin.** H. Milstone (*J. Gen. Physiol.*, 1942, **25**, 679—687).—The prep. of prothrombin from oxalated ox plasma, its conversion into thrombin, and fractionation of the latter are described. Under certain conditions, crude prothrombin changes to thrombin without addition of activators and in absence of Ca<sup>2+</sup>. The essential condition for this "spontaneous" activation may be the concn. of the biological material. The activation is not retarded by 0.02M-oxalate at  $p_H$  7.4, is accelerated by cryst. trypsin, and is unaffected by cryst. trypsin inhibitor. Thrombin is sol., and crude prothrombin is insol., in 45%-saturated (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub>; partly purified thrombin is stable in conc. aq. (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub> at  $p_H$  5.2. A method of prep. based on these properties yields thrombin having an optimum activity 100—175 times the potential sp. activity of whole plasma. J. N. A.

**Explanation of urobilinuria in cases of hæmatoma.** J. W. G. Ten-Boppel Hinnik (*Amer. J. digest. Dis.*, 1942, **9**, 168—172).—88% of the hæmogoblin of blood in natural or artificial hæmatomas was recovered as faecal urobilinogen, but only 25% of the bilirubin of human bile given orally could be so recovered. Therefore only extrahepatic (indirectly reacting) bilirubin is converted into urobilinogen in the intestine. N. F. M.

**Potassium content of whole blood, corpuscles, and serum of wild animals.** A. Urbain and M. A. Pasquier (*Compt. rend.*, 1941, **213**, 83—85).—The K content of the serum of the animals examined was 0.19—0.354, of whole blood 0.29—1.166, and of corpuscles up to 2.284 g. per l. The K content of plasma was slightly less than that of serum. P. G. M.

**Apparatus for distillation of ammonia in determination of urea in blood.** A. B. Anderson (*Analyst*, 1942, **67**, 225—226).—Two aëration tubes with ground-in stoppers are connected so that the distillate from one (A) is carried over by a stream of NH<sub>3</sub>-free air to the bottom of the other (B). A thistle funnel sealed into the stopper of A permits the tungstic acid-cleared solution after incub-

ation with urease to be washed into *A*. Borax is added and a drop of liquid paraffin and the air stream started with HCl in *B*. *A* is heated and 5 ml. of distillate collected and nesslerised. *A* is emptied by suction through a side arm. A wire spring in *A* prevents foaming. As traces of alkaline spray may be carried over, NH<sub>3</sub> cannot be determined by titration. S. B.

**Collection of blood for determination of lactic and pyruvic acids.** T. E. Friedemann and G. E. Haugen (*J. Biol. Chem.*, 1942, **144**, 67—77).—Blood is withdrawn from a vein by a cool dry syringe and expelled into 5 vols. of trichloroacetic acid. No change in the pyruvic acid content is caused by the use of a tourniquet or by holding in a syringe for up to 3 min. A decrease occurs on the addition of NaHCO<sub>3</sub>, NaF, Na oxalate, or Na<sub>2</sub>SO<sub>4</sub>, whereas Na monoiodoacetate and, in some cases, NaCl cause an increase. H. G. R.

**Photo-electric determination of *dl*- $\alpha$ -tocopherol in serum.** G. G. Mayer and H. Sobotka (*J. Biol. Chem.*, 1942, **143**, 695—699).—The method described is a modification of the 2:2'-dipyridyl-FeCl<sub>3</sub> method (A., 1939, II, 123) using the photo-electric colorimeter. Not more than 10 c.c. of serum are required. Vals. of 0.5—1.92 mg-% were found for fasting human serum. P. G. M.

**Pharmacologically active substances in serum.**—See A., 1942, III, 709.

**Amino-acids. I. Plasma-amino-acid retention in hypoproteinemic dogs as evidence of impaired liver function.** E. Goettsch, J. D. Lyttle, W. M. Grim, and P. Dunbar (*J. Biol. Chem.*, 1942, **144**, 121—134).—Hypoproteinemic dogs, when given a casein hydrolysate, retain more amino-acid in the plasma than do normal dogs. This is probably due to slower deamination caused by liver injury. There is no relation between the concn. of albumin and amino-acid-N in plasma; the latter may remain normal during severe hypoalbuminemia. NH<sub>3</sub> and urea excretion increase after injections of casein hydrolysate. Little amino-acid is excreted. R. L. E.

**Blood-enzymes in aged.** J. Meyer, H. Souther, and H. Necheles (*Amer. J. digest. Dis.*, 1942, **9**, 160—162).—Average vals. for blood pressure, haemoglobin, red cells, and serum-lipase were lower in old age than in youth. Serum-proteins were increased and serum-diastase was unchanged. N. F. M.

**Flow of lymph from lungs of dog.** M. F. Warren and C. K. Drinker (*Amer. J. Physiol.*, 1942, **136**, 207—221).—A const. considerable flow of lymph from the lungs was obtained in nembutalised dogs with opened chests. The anterior mediastinum was exposed by positive pressure and artificial respiration used. When artificial pressure was unduly great or when the lungs were quiescent, the lymph flow was greatly reduced. Ventilation with a mixture low in O<sub>2</sub> increases lymph flow. Increased pressure on the pulmonary veins increased lymph flow greatly, and the lymph soon resembled blood in composition. Drainage of lymph from both lungs was in the main via the right lymphatic duct. The average protein content of lung lymph is 3.66%. M. W. G.

## VI.—VASCULAR SYSTEM.

**Specialised conducting tissue in heart of golden hamster.** E. W. Walls (*J. Anat.*, 1942, **76**, 359—368).—A description is given of the general topography and specialised tissue of the heart of the golden hamster. The sino-atrial node, which is horseshoe-shaped, is large and in relation to the upper part of the sulcus terminalis. The node is comprised almost entirely of muscular fibres. The atrio-ventricular node is in the atrial septum behind the aorta. The sino-atrial node and atrio-ventricular node are not connected directly by any specialised tissue. The Purkinje tissue is confined to the wall of the left ventricle. Nerve elements in relation to the conducting tissue are described. W. J. H.

**Nature of QI and QIII [of electrocardiogram].** H. E. Hoff, L. H. Nahum, and W. Kaufman (*Amer. J. Physiol.*, 1942, **135**, 752—758).—In dogs the downstroke of QI is produced by early electrical activity in the anterior surface of the left ventricle; its upstroke results from excitation of the posterior surface of the right ventricle. The downstroke of QIII is produced by early electrical activity in the posterior surface of the left ventricle, its upstroke by excitation of the anterior surface of the right ventricle. The presence of Q in the ventricular extra systole has the same significance as Q in the normal complex. In lead I it results from primary activity in the anterior surface of the left ventricle and in lead III it arises from primary activity in the posterior surface of the left ventricle. M. W. G.

**Distribution of potential concerned in formation of electrocardiograms.** C. C. Wolferth, M. M. Livezey, and F. C. Wood (*Amer. J. med. Sci.*, 1942, **203**, 641—665).—Observations in man showed that certain definite patterns of variations, with time, of difference of potential can be recognised over considerable areas of the body surface. Some of these patterns obtained between paired areas near the heart undergo decrement as distance of paired areas from the heart increases, but along certain radial lines extending from

the heart little alteration in contour of pattern is demonstrable between paired areas. The results are opposed to Einthoven's equilateral triangle hypothesis. C. J. C. B.

**Electrocardiographic anagrams.** J. E. F. Riseman (*J. Lab. clin. Med.*, 1942, **27**, 1063—1068).—The model described consists of units of e.c.g. complexes prepared in such a fashion that they can be joined together so as to illustrate diagrammatically most of the common e.c.g. patterns. C. J. C. B.

**$\alpha$ -Lobeline measurement of circulation time.** A. Lilienfeld and K. Berliner (*Arch. intern. Med.*, 1942, **69**, 739—745).—100 duplicate measurements of circulation time were made with  $\alpha$ -lobeline hydrochloride, allowing 15—60 min. between the tests. In 3 cases the results were identical, in 85 the average variation was 29%, and in 12 cases the 2nd injection failed to produce cough. Differences were greatest in patients with congestive heart failure. C. A. K.

**Blood viscosity under different experimental conditions and its effect on blood flow.** R. W. Eckstein, D. Book, and D. E. Gregg (*Amer. J. Physiol.*, 1942, **135**, 772—775).—In normal dogs, viscosity varies from 7.1 to 3.7. Blood defibrination, addition of heparin *in vitro* or *in vivo*, and the injection of morphine do not affect the viscosity. Haemorrhage after a short period and barbital anaesthesia cause considerable reduction in viscosity; ether anaesthesia, *in vitro* use of Na citrate, and *in vitro* and *in vivo* use of pantamine-fast-pink (or chlorazol-fast-pink) give large increases in viscosity. These changes are sufficient to alter greatly the ease of blood flow. M. W. G.

**Arm-tongue circulation in chronic asthma.** J. D. Cottrell and D. C. Cuddie (*Brit. Med. J.*, 1942, **1**, 70—71).—The arm-tongue circulation time (decholin) was normal in 21 cases of uncomplicated bronchial asthma both during and between attacks. C. A. K.

**Intrahepatic circulation of blood.** K. G. Wakim and F. C. Mann (*Anat. Rec.*, 1942, **82**, 233—253).—A detailed account of work already noted (A., 1941, III, 652). W. F. H.

**Pressure of blood in right auricle in animals and man, under normal conditions and in right heart failure.** D. W. Richard, jun., A. Cournaud, R. C. Darling, W. H. Gillespie, and E. D. Baldwin (*Amer. J. Physiol.*, 1942, **136**, 115—123).—In a study of venous pressures (one chimpanzee and dogs), the development of right heart failure associated with acute pulmonary oedema was accompanied by a rise of peripheral and right auricular pressures and a disappearance of the normal pressure gradient between peripheral veins and right heart; the right auricular and peripheral venous pressure levels became nearly equal. In human subjects with a normal circulation, right auricular pressure was directly recorded by means of the right heart catheterisation. Average gradient from arm to heart was +41 mm. of water. In some subjects abs. pressure level at the right auricle was determined by locating the position of the catheter by lateral X-ray; the average right auricular pressure (subjects supine) was +37 mm. Three patients in congestive heart failure with high peripheral venous pressures showed decrease in peripheral-central pressure gradient, the pressures in arm vein and in right auricle being almost identical. M. W. G.

**Cardiac massage.** J. C. Nicholson (*Brit. Med. J.*, 1942, **1**, 385—386).—Results in 7 cases showed that cardiac massage should be started within 3—6 min. of cardiac arrest if final recovery is to occur. It is more effective than intracardiac adrenaline, and the technique is described. C. A. K.

**Pulmonocardiac failure from spinal deformity.** A. J. Kerwin (*Arch. intern. Med.*, 1942, **69**, 560—572).—Severe spinal deformity in 5 patients produced pulmonary hypertension leading to right-sided heart failure, apparently the result of mechanical interference with respiratory function. C. A. K.

**Cardiac lesions in rheumatic fever.** A. D. Console (*Arch. intern. Med.*, 1942, **69**, 551—559).—The results of 98 autopsies in patients with previous rheumatic heart disease are reported. Aschoff bodies were found in the myocardium of all cases in which death occurred in the first decade of life, in 64% of those in the second decade, and in 11% of those who died during a later decade. When Aschoff bodies were found the interval between the last attack of polyarthritis and death was always less than 5 months. C. A. K.

**Coronary thrombosis in young diabetic.** N. Reitman, W. R. Greenwood, and J. H. Kler (*Amer. J. med. Sci.*, 1942, **203**, 792—801).—A case report. C. J. C. B.

**Intravenous serum in peripheral vascular disease.** G. W. Howard (*Brit. Med. J.*, 1942, **1**, 285—289).—Intravenous serum injections were successfully used in the treatment of 16 cases of chronic arterial disease, chiefly obstructive in type. Symptomatic relief, healing of ulcerated areas, rise of skin temp., and improvement in oscillometric index were attributed to the increased blood vol. which helps to open up collateral circulation. C. A. K.

**Oxidases, pressor amines, and hypertension.** G. J. Martin, C. T. Ichniowski, W. A. Wisansky, and S. Ansbacher (*Amer. J. Physiol.*, 1942, **136**, 66—69).—The oxidative destruction of adrenaline is promoted in a tyrosinase-adrenaline system by *o*-substituted phenols

(pyrocatechol, *o*-cresol, *o*-aminophenol) and inhibited by aromatic amines and aminobenzoic acids. "Dopa" formed a pressor amine when incubated with minced kidney (dog or cat) tissue and yet is capable of catalysing the oxidative destruction of adrenaline. The effectiveness of tyrosinase in reducing blood pressure in the perinephritic-hypertensive dog (Page) is enhanced by the simultaneous administration of pyrocatechol. M. W. G.

**Renal blood flow in arterial hypertension.** P. P. Foà, W. W. Woods, M. M. Peet, and N. L. Foà (*Arch. intern. Med.*, 1942, 69, 822—835).—Effective renal blood flow, filtration rate, and renal tubular excretory mass were determined in 20 patients with hypertension and in 7 non-hypertensive subjects. In hypertension there is reduced renal blood flow owing to increased resistance of the efferent glomerular vessels, but this may be only a part of the widespread vasoconstriction which raises the blood pressure and does not necessarily imply that renal ischaemia causes arterial hypertension in man. C. A. K.

**Unilateral renal atrophy associated with hypertension.** A. H. Baggenstoss and N. W. Barker (*Proc. Staff Mayo Clin.*, 1941, 16, 833—838). H. H. K.

## VII.—RESPIRATION AND BLOOD GASES.

**Pulmonary ventilation in health and disease.** A. T. Brice (*Amer. J. clin. Path.*, 1942, 12, 144—148).—The ventilation coeff. ( $O_2$  consumed per min ÷  $O_2$  inhaled per min.) normally varies inversely as the basal metabolic rate. In thyroid disease, normal or elevated ventilation was found in cases of simple and non-toxic adenomatous goitres. In other thyroid conditions the normal relationship was disturbed, the ventilation falling as the basal metabolic rate either falls or rises. In cardiac and circulatory conditions the level of ventilation was generally lowered except in hypertension. C. J. C. B.

**Changes in balance of respiratory drives resulting from open pneumothorax.** R. Gesell and C. Moyer (*Amer. J. Physiol.*, 1942, 135, 539—546).—Bilateral open pneumothorax (dog, morphine + urethane) produced systematic breathing changes. First, a marked intensification or prolongation of inspiratory activity (attributed to a combined change in vagal proprioceptive drives) associated with a relatively weak expiratory activity. Secondly, a slowly increasing intensity of both inspiratory and expiratory activity, the latter predominating, then a shortening of the inspiratory phase and an acceleration of breathing, the latter continued up to an equalisation of both activities; expiratory activity then predominated, leading to a marked prolongation of expiratory phase and to a diminution in the respiratory rhythm. M. W. G.

**Concentration of carbon dioxide in expired air.** P. K. Boyer and C. V. Bailey (*Arch. intern. Med.*, 1942, 69, 773—788).—Studies in 2000 subjects not suffering from respiratory disease or thyroid abnormalities showed that pulmonary ventilation was more closely related to  $CO_2$  output than to  $O_2$  consumption. The partial pressure of  $CO_2$  in the expired air in normal subjects ranges from 18 to 22.5 mm. Hg. Changes in disease are discussed. C. A. K.

**Effect of asphyxiation under various tensions of carbon dioxide on swim bladder gases of fresh-water fish.** V. S. Black (*Canad. J. Res.*, 1942, 20, D, 209—219; cf. Safford, A., 1941, III, 84).—The proportions of gases in the swim bladder change, as a result of sealing in bottles of water containing various proportions of  $CO_2$  and  $O_2$ , to approach more nearly to equilibrium with the proportions of these gases in the water,  $CO_2$  being gained and  $O_2$  lost. The effect is more marked in physoclistous (perch, bass, pumpkin-seed, stickleback) than in physostomous fish (dace, sucker, chub, bullhead, trout). Reasons are given for supposing that the  $O_2$  lost from the swim bladder during asphyxiation is of little or no val. for respiration. W. McC.

**Differences in excitability of somatic and autonomic centres in response to anoxia.** E. Gellhorn, N. Cortell, and H. B. Carlson (*Amer. J. Physiol.*, 1942, 135, 641—649).—Effect of anoxia by inhalation of  $O_2-N_2$  mixtures (4.5—8.1%  $O_2$ ) was studied on responses to stimulation of hypothalamus, medulla, and spinal cord in cats (chloralose and/or pentothal). Anoxia increases the contractions of the nictitating membrane produced by stimulation of the hypothalamus; this effect persists after denervation of the carotid sinuses and bilateral vagotomy. Somatic responses are decreased. Blood pressure rise resulting from hypothalamic stimulation increases in the normal animal during anoxia but decreases in the cat deprived of carotid sinuses and vagi. Direct stimulation of the vasomotor centre in the medulla gives similar results. Medullary and spinal stimulation indicate an increased excitability of these centres in anoxia. Somatic movements on stimulation of the spinal cord decline during anoxia. M. W. G.

**Improved equipment for oxygen therapy.** S. L. Cowan and J. V. Mitchell (*Brit. Med. J.*, 1942, I, 118—119).—The injector principle allows simple control of  $O_2$  % delivered to the patient. The disadvantages of partial rebreathing and mechanical resistance of the B.L.B. mask are avoided. C. A. K.

**"Oxygen trough" respiration.** I. F. S. Mackay (*Nature*, 1942, 149, 698).—Cotton's results (A., 1940, III, 388) were confirmed. E. R. S.

**Oxygenation of fish blood.** R. W. Root and L. Irving (*Biol. Bull.*, 1941, 81, 307—323).—In blood of the tautog the  $O_2$  dissociation curve changes from sigmoid to rectangular hyperbolae with increasing pressure of  $CO_2$ . Haemolysis lessens this effect. It is suggested that this is due to the different  $O_2$ -combining components of the haemoglobin. D. M. SA.

**Equilibrium between oxygen and haemoglobin in concentrated urea solution.** J. F. Taylor and A. B. Hastings (*J. Biol. Chem.*, 1942, 144, 1—6).—The equilibrium between  $O_2$  and horse haemoglobin at its isoelectric point in 4M-urea has been investigated. For a given  $O_2$  pressure haemoglobin combines with more  $O_2$  than in absence of urea. The solubility of  $O_2$  in 4M-urea has been determined at 24.5° and 37°. C. R. H.

**Combination of hypoxic and hypercapnic stimulation at carotid body.** C. V. Winder (*Amer. J. Physiol.*, 1942, 136, 200—206).—Isolated carotid body regions (anaesthetised dogs) were perfused by a continuous artificial circuit with continuous gaseous equilibration of the basal perfusion fluid. Brief substitutions of experimental fluids equilibrated with gases high in  $CO_2$ , low in  $O_2$ , or a combination of the two, gave reflex respiratory responses such that the combination effect on min. vol. of  $CO_2$  and low  $O_2$  was approx. equal to the sum of their individual separate effects. Results are interpreted in terms of the theory that intracellular *cH* is a factor in the control of chemoreceptor activity. Chemoreceptors are considered one of several sites for mutual facilitation of hypoxic and hypercapnic stimulation of respiration. M. W. G.

**Effect of respiration of carbon dioxide by the decapitated body having cardiac nerves cut.** A. P. Suñer (*Ciencia*, 1941, 2, 346—350).—Modifications in the respiratory reflexes were studied in a dog's trunk kept alive by artificial respiration, with vagi intact, and receiving blood from a donor dog by means of a carotid anastomosis (Heymans technique). 10% of  $CO_2$  in the inspired air has a stimulating and one of 20% an inhibitory effect. The origin of the reflex changes is discussed. F. R. G.

**Oxidation-reduction potentials of the methaemoglobin-haemoglobin system in concentrated urea solution.** J. F. Taylor (*J. Biol. Chem.*, 1942, 144, 7—13).—Oxidation-reduction potentials of horse and dog methaemoglobin-haemoglobin mixtures in 4M-urea containing  $PO_4^{''}$  buffers are consistent with the transfer of two electrons during oxidation. C. R. H.

**Oxidation-reduction potentials of the metmyoglobin-myoglobin system.** J. F. Taylor and V. E. Morgan (*J. Biol. Chem.*, 1942, 144, 15—20).—Oxidation-reduction potentials of horse metmyoglobin-myoglobin mixtures in presence of  $PO_4^{''}$  buffers are consistent with the transfer of one electron during oxidation. The potentials are less than those for corresponding methaemoglobin-haemoglobin mixtures in absence of urea. C. R. H.

**Salmon blood during migration.** E. Benditt, P. Morrison, and L. Irving (*Biol. Bull.*, 1941, 80, 429—440).—Fishes from fresh water have blood of 30% less  $O_2$  capacity and cell vol. than those from brackish estuary waters (probably due to dilution). Haemoglobin affinity for  $O_2$  is greater in fresh water but the  $CO_2$  dissociation curves are little different. D. M. SA.

**Radiological [pulmonary] manifestations of erythema nodosum.** P. Kerley (*Brit. J. Radiol.*, 1942, 15, 155—165).—Tracheo-bronchial and hilar glandular enlargement with or without miliary nodular infiltration of the lung parenchyma was seen before, during, or after the appearance of erythema nodosum in 21 of 23 cases. E. M. J.

## VIII.—MUSCLE.

**X-Ray diagram of dehydrated muscle.**—See A., 1942, III, 715.

**Electron-microscopic investigation of muscle-protein myosin.**—See A., 1942, I, 297.

**Determination of creatine in muscle by dinitrobenzoate method.** E. Lehnartz (*Z. physiol. Chem.*, 1941, 271, 265—274).—The determination is improved by rigid control of the alkalinity of the solution. The method is applied to the determination of creatine in muscle. H. W.

**Origin of muscle-creatine.** I. E. Lehnartz and R. Jensen (*Z. physiol. Chem.*, 1941, 271, 275—288).—Arginine is converted into creatine in frog's muscle, the  $pH$  optimum of the change being at 6.6—7.0. Transformation takes place only with  $PO_4^{''}$  buffer. Buffering with borate, veronal, or glycine completely inhibits the transformation. There is no evidence of the formation of creatine from glycine in muscle. H. W.

**Is muscle contraction essentially an enzyme-substrate combination?** J. Needham, A. Kleinzeller, M. Miall, M. Dainty, D. M. Needham, and A. S. C. Lawrence (*Nature*, 1942, 150, 46—49).—Adenyl pyrophosphate reduces the slow-birefringence (Needham *et al.*, A., 1942, I, 207) and viscosity of a myosin solution, inorg.

PO<sub>4</sub>''' is produced, and the vals. slowly return to their originals. The significance and specificity of this reaction are discussed.

E. R. S.

**Transmission fatigue and contraction fatigue [in skeletal muscle].** E. C. del Pozo (*Amer. J. Physiol.*, 1942, 135, 763—771).—Indirect max. stimulation at different frequencies of the gastrocnemius-plantaris of the cat (dial) shows 2 modes of fatigue: transmission fatigue caused by frequencies of stimulation above 30 per sec., and contraction fatigue, below 20 per sec. Recovery of transmission fatigue is prompt, that of contraction fatigue is slow. The two phenomena are independent. Transmission fatigue is discussed from the viewpoint of decreased acetylcholine output, and contraction fatigue is attributed to metabolic changes at the muscles. At frequencies between 20 and 30 per sec. "subliminal transmission fatigue" is present.

M. W. G.

**Choline-esterase activity of serum and neuromuscular transmission.** H. Salvetrini, J. V. Luco, and F. Huidobro (*Ciencia*, 1941, 2, 351—352).—Muscular stimulation (cat) is accompanied by increase in serum-choline-esterase concn.

F. R. G.

**Influence of prostigmine, atropine, and other substances on fibrillation and atrophy in denervated skeletal muscle (rat).** R. Levine, J. Goodfriend, and S. Soskin (*Amer. J. Physiol.*, 1942, 135, 747—751).—The acute effects of various agents on the fibrillation of paralysed muscle (rats) were studied. Materials which showed acute effects on fibrillation were tested as to their influence on chronic muscular atrophy. Of the substances influencing fibrillation, only prostigmine and atropine had any influence on the rate of atrophy. Prostigmine, which increased fibrillation, increased the rate of atrophy by 47%. Atropine, which diminished fibrillation, decreased the rate of atrophy by 39%.

M. W. G.

**Action potentials of skeletal muscles of frog.** H. Goldberg and J. A. E. Eyster (*Amer. J. Physiol.*, 1942, 135, 676—689).—The potential distribution on the surface of the skeletal muscle during contraction brought about by a single stimulus to its motor nerve differs from that on the surface of the normally contracting heart, in that movement of potential max. occurs during only a part of the action potential period. In the fundamental aspect of the polar nature of the potential distribution, the heart and the skeletal muscle are the same: regions of positive and negative potentials, with respect to the potential of resting muscle, developing coincidentally, undergoing growth and decline, and certain displacements and reversals of polarities.

M. W. G.

**Potassium and muscular disorders.** J. N. Cumings (*J. Neurol. Psychiat.*, London, 1941, 4, 226—234).—Vals. are given for the K content of muscles in various muscular disorders. 2 cases of myasthenia gravis were in positive K balance; after prostigmine injection they showed increased K content in red blood corpuscles and serum but not in urine and faeces. Muscular K content was not affected by spinal anaesthesia or motor nerve section. Extracts from American and Chinese gelsemium roots did not produce muscular disorder in rabbits.

H. L.

**Cure of repeated attacks of nutritional muscular dystrophy in rabbit by  $\alpha$ -tocopherol.** C. G. Mackenzie (*Proc. Soc. Exp. Biol. Med.*, 1942, 49, 313—317).

V. J. W.

**$\alpha$ -Tocopherol in neuromuscular disorders.** A. J. Lubin (*Arch. intern. Med.*, 1942, 69, 836—855).— $\alpha$ -Tocopherol was given in large doses to 35 patients with amyotrophic lateral sclerosis, muscular dystrophy, and other neuromuscular diseases. Measurements of muscular strength by dynamometer, electric tests, and creatinine and creative excretion in the urine showed that the substance had no beneficial therapeutic actions.

C. A. K.

## IX.—NERVOUS SYSTEM.

**Living nerves. VII. Growth adjustments of cutaneous terminal arborisations.** C. C. Speidel (*J. comp. Neurol.*, 1942, 76, 57—73).—A description of the changes, during growth of the animal, of myelinated nerve endings in three amphibian species (*Pseudacris ferriarum*, *Hyla crucifer*, and *Rana clamitans*). Among the features observed were multiplication of endings by branching, loss of some endings by retraction or degeneration, growth in length of new endings of as much as 200  $\mu$ ., and variations from day to day in exact position of end-bulbs.

J. D. B.

**Local extension of nerve fibres into denervated areas of skin.** G. Weddell, L. Guttman, and E. Gutmann (*J. Neurol. Psychiat.*, London, 1941, 4, 206—225).—The relation of the cutaneous territory of the sural nerve in the rabbit to those of the adjacent nerves (tibial and peroneal) and the shrinking of the analgesic area after resection of either tibial and peroneal nerves together, or of the sural nerve only, were studied by nociceptive sensory tests and by staining nerve fibres throughout large preps. of skin by local injection of methylene-blue. Both the autonomous zone of the sural nerve (analgesic area after sural nerve section) and its max. zone (area of remaining sensibility after section of adjacent nerves) corresponded almost exactly with the neurohistological boundaries. The inter-

mediate zone (difference in extent of autonomous and max. zones) was characterised by fewer, diminished, or delayed responses and by a diminished no. of nerve fibres which gave rise to isolated endings. The shrinkage of the area of sensory loss after sural nerve section and the extension of the max. zone following tibial and peroneal section are due to an ingrowth of nerve fibres from surrounding normal nerves; the process is far slower than would be expected in a process of regeneration from the central stump and moreover begins before regeneration from above could have occurred. The majority of the fibres extending from intermediate zones are non-medullated and originate from fibres which give rise to nerve nets or which supply hair follicles at the periphery of the innervated skin area. The no. of fibres from other than intermediate zones is far smaller and most of them can only be followed proximally into nerve bundles of the surrounding normal nerves.

H. L.

**Respiration of neurones.** J. Pearce and R. W. Gerard (*Amer. J. Physiol.*, 1942, 136, 49—65).—Pieces of frog brain tissue (1 mg.) were dissected from anterior olfactory nucleus, primordium pallii, hippocampus, ventral hypothalamus, and cerebellum. Q<sub>o</sub>, vals. up to 700 are higher than those previously reported for frog and average 40% of those for mammalian brain. In order of descending Q<sub>o</sub>, the series is cerebellum, hippocampus, anterior olfactory nucleus, primordium pallii, ventral hypothalamus at first, with cerebellum falling off faster than other regions. From the data obtained it is possible to express O<sub>2</sub> consumption as a function of the total neurones assuming the morphologic relations described by Bok of the total neurite surface, perikaryon surface, process surface, nuclear surface, nuclear vol., perikaryon vol., and total protoplasm vol. Respiration of various brain regions is constantly related to the mass of protoplasm, but not to the surface area, of the neural elements present.

M. W. G.

**Organisation and termination of fibres of posterior columns in *Macaca mulatta*.** A. E. Walker and T. A. Weaver (*J. comp. Neurol.*, 1942, 76, 145—158).—The relationships within the posterior columns and posterior column nuclei were studied with the Marchi technique following myelotomy and posterior root section. Fibres up to the mid-thoracic level terminate in the gracile nucleus, the lower fibres ending caudo-medially. Fibres above this level terminate in the cuneate nucleus and in the external cuneate nucleus. As the latter does not contribute to the medial lemniscus, but to the cerebellum, it is suggested that support is given to the conception of somatotopic cerebellar representation.

J. D. B.

**Decerebrate rigidity in infant aged 6½ months.** D. T. Dimitrijevic and L. Vulovic (*M Schr. Psychiat. Neurol.*, 1941, 104, 171—178).—Extreme reversible rigidity of trunk and limbs in a case of severe alimentary disturbance is attributed to mesencephalic lesions of pyramidal and extra-pyramidal tracts.

H. L.

**Anatomical and clinical studies of complex hyperkinetic syndromes. III. Gilles de la Tourette's disease.** A. Dewulf and L. van Bogaert (*M Schr. Psychiat. Neurol.*, 1941, 104, 53—61).—A case is reported showing no cerebral lesions post mortem.

H. L.

**Anatomical and clinical studies of complex hyperkinetic syndromes. IV. Syndrome of red nucleus.** G. de Morsier and L. van Bogaert (*M Schr. Psychiat. Neurol.*, 1941, 104, 228—279).—Clinical and post-mortem findings are reported in detail of a case showing the dystonic form of Benedikt's syndrome. The lesion of the red nucleus causes retrograde degeneration of superior cerebellar peduncle and medial lemniscus with their cells of origin, and transneuronal degeneration of the olive with its efferent paths, of nucleus arcuatus and nucleus reticularis bulbi. There was no degeneration of the rubro-spinal bundle but marked degeneration of the fasciculus centralis tegmenti.

H. L.

**Surgical lesions of spinal cord and nerve-roots.** G. L. Alexander (*Edinb. Med. J.*, 1942, [iv], 49, 409—424).—A lecture containing a resumé of modern methods of treatment.

H. S.

**Influence of carbon dioxide and oxygen in varying percentages on spinal reflexes of *Leptodactylus ocellatus* at different temperatures.** M. Ozorio de Almeida, H. Moussatché, and M. Vianna Dias (*Rev. Brazil. Biol.*, 1941, 1, 293—300).—Inflation of the lungs of *L. ocellatus* with mixtures of air containing varying CO<sub>2</sub> and O<sub>2</sub> % affects the curves of duration of spinal reflexes in relation to temp.

I. C.

**Pyramidal muscle reflex.** L. Benedek and L. von Angyal (*M Schr. Psychiat. Neurol.*, 1942, 105, 109—114).—In cases of disseminated sclerosis with absent abdominal reflexes contraction of the pyramidal muscles was elicited by percussion of the symphysis pubis. The reflex is regarded as sign of a pyramidal lesion.

H. L.

**Evolution and anatomy of cerebellum.** R. S. Dow (*Biol. Rev.*, 1942, 17, 179—220).—A review.

J. D. B.

**In vitro formation of phospholipin by brain and nerve with radioactive phosphorus as indicator.** B. A. Fries, H. Schachner, and I. L. Chaikoff (*J. Biol. Chem.*, 1942, 144, 59—66).—The conversion of inorg. P into phospholipin is greater by brain from young (15 g.) than from old (200 g.) rats and is 3—5 times as great by brain slices as by homogenate or shreds. No significant variation with age is observed in the breakdown of phospholipin by brain and, assum-



ing a slow and equal penetration of inorg. P, it is assumed that young brain does not form more total phospholipin than old brain. The formation of phospholipin by the sciatic nerve of the dog is of the same order as that of young rat brain homogenate. H. G. R.

**Cells of Meynert in visual cortex of monkey.** W. E. Le G. Clark (*J. Anat.*, 1942, 76, 369—376).—Solitary cells of Meynert are conspicuous elements in lamina VI in the visual cortex of macaque monkey. It is estimated that there are approx. 1300 Meynert cells in the area striata. Experimental evidence is adduced in favour of the conclusion that the axons of these cells contribute to cortico-mesencephalic connexions. W. J. H.

**Cerebral cortex in groundhog (*Marmota monax*) and deer mouse (*Peromyscus maniculatus*).** H. C. Elliott (*J. Comp. Neurol.*, 1942, 76, 75—89).—Gurewitsch's method for preparing maps of cerebral cortex in two dimensions was applied to the hemispheres of the two rodents. Attention is drawn to inaccuracies in the method but it is shown to be of val. in comparative studies. J. D. B.

**Cytoarchitecture of sheep cortex.** J. E. Rose (*J. comp. Neurol.*, 1942, 76, 1—55).—A detailed account of the cytoarchitecture is given, using the terminology of M. Rose, and a summary of the latter's analysis of cortical cytoarchitectonics is included. J. D. B.

**Functions of various parts of human cerebral cortex.** E. H. Strasburger (*M Schr. Psychiat. Neurol.*, 1941, 104, 78—125).—A survey. H. L.

**Effects of prostigmine and acetylcholine on cortical potentials.** P. O. Chatfield and E. W. Dempsey (*Amer. J. Physiol.*, 1942, 135, 633—640).—Acetylcholine applied locally to cerebral cortex of cats did not change the electrocorticogram. Prostigmine caused a transient depression of spontaneous activity both at the site of application and elsewhere. Prostigmine followed by acetylcholine applied to the somæsthetic, auditory, and motor cortex causes a series of characteristic changes, consisting of increased spontaneous activity, appearance of characteristic 5—10 per sec. spikes, and later development of rapid (20—30 per sec.) lower-voltage potentials. Atropine abolished the spikes occurring in the intervals between bursts, but had no effect on other changes produced by prostigmine and acetylcholine. Section of thalamic radiations abolished all the effects of treatment except the rapid low-voltage activity. M. W. G.

**Unusual lesions of cerebral cortex due to insulin.** E. Grünthal (*M Schr. Psychiat. Neurol.*, 1941, 104, 301—311).—A schizophrenic patient progressed from an induced hypoglycæmic coma immediately into a condition characterised by complete absence of movement (except eyelids), speech, and deep, cutaneous, and blinking reflexes; swallowing, corneal and pupillary light reflexes were normal. After 7 weeks she showed sluggish muscular reflexes and slight extra-pyramidal rigidity in upper limbs. The latter increased gradually, involving in the 18th week the whole body. Spontaneous movements never returned. Widespread massive cortical destruction of vascular origin was found post mortem with retrograde atrophy of part of the thalamus. H. L.

**Audiogenic seizures in rats.** D. B. Lindsley, F. W. Finger, and C. E. Henry (*J. Neurophysiol.*, 1942, 5, 185—198).—High-pitched tonal stimulation induced abnormal, seizure-like behaviour in about 50% of unrestrained rats but no attacks occurred in mechanically restrained, curarised, or vagotomised animals. Various phases of an attack were associated with convulsive-like electro-cortical discharges; in the absence of seizures in unrestrained animals heart rate changes were often accompanied by some "substitute behaviour." Marked tachycardia and/or bradycardia occurred during onset of stimulation and attack; they are attributed to dual action of the autonomic system with sympathetic predominance. H. L.

**The olfactory-parotid reflex.** C. A. Elsberg, H. Spotnitz, and E. I. Strongin (*Arch. Neurol. Psychiat.*, 1942, 47, 707—717).—Olfactory stimulation of the nasal mucosa produced increased secretion of the ipsilateral parotid; it is thought that the afferent pathway of the reflex involves the 5th and 7th cranial nerves and that the cerebral pathways cross in their course from cortex to brainstem. H. L.

**Effect of hypoglycæmia on electroencephalogram at varying degrees of oxygenation of the blood.** E. Gellhorn and M. Kessler (*Amer. J. Physiol.*, 1942, 136, 1—6).—The effect of anoxia on brain potentials in chloralose-anæsthetised cats and unanæsthetised rats is aggravated during insulin hypoglycæmia. The effects of hypoglycæmia on the brain potentials can be annulled by inhalation of pure O<sub>2</sub>; if, however, the hypoglycæmia is so severe and prolonged as to lead to almost complete disappearance of brain potentials (prior to the convulsive state) no restoration of normal brain potentials results from pure O<sub>2</sub>, although they are restored by the injection of sugar. The assumption that the rate of oxidation of the brain depends on the sugar level as well as O<sub>2</sub> tension of the blood is supported by these experiments. M. W. G.

**Effect of choline-like substances on cerebral electrical discharges in epilepsy.** D. Williams (*J. Neurol. Psychiat.*, London, 1941, 4, 32—

47).—Injection of eserine (large dose), prostigmine, carbamylcholine chloride, and acetylcholine in patients with petit mal was followed by increase in the recorded epileptic activity; the effect was inhibited by atropine. Histamine, pilocarpine, and apomorphine did not alter incidence or duration of the attacks. Several of the peripheral effects of acetylcholine (respiratory, blood-pH, sugar changes, or cerebral vascular dilatation) could be excluded as factors modifying the attacks. H. L.

**Electroencephalogram in acute head injuries.** D. Williams (*J. Neurol. Psychiat.*, London, 1941, 4, 107—130).—74 cases were examined within 20 days of the injury. Widespread abnormally slow waves, suppression of normal frequencies, and outbursts of high-voltage sine waves with a frequency of 2—3 per sec. were observed in the acute stage. The degree of the electrical changes was closely correlated with the clinical state but in the later stages of recovery the encephalogram sometimes remained abnormal in cases with complete clinical recovery. Focal areas of max. abnormality became evident as the general disturbance subsided, corresponding with localising clinical signs, but were also seen in silent areas of the cerebral cortex. The initial epileptic type of disturbance had no prognostic significance in regard to post-traumatic epilepsy. H. L.

**Electroencephalogram in chronic post-traumatic states.** D. Williams (*J. Neurol. Psychiat.*, London, 1941, 4, 131—146).—600 cases were examined at intervals after injury from a few hr. to 12 years. 50% had abnormal encephalograms; this high % persisted for many years after injury. There was positive correlation between it, the severity of injury, persistence of symptoms, and presence of dural penetration. An abnormal encephalogram was found in 55% of 207 cases with organic symptoms, in 37% of 50 constitutionally inferior subjects, and in 20% of 58 neurotic cases. Association of a normal encephalogram with persistence of symptoms may point to a bad ultimate prognosis (complete destruction of abnormal cerebral tissue). No difference was apparent between traumatic and idiopathic epilepsy. H. L.

**Significance of an abnormal electroencephalogram.** D. Williams (*J. Neurol. Psychiat.*, London, 1941, 4, 257—268).—% figures are given for abnormal electroencephalograms at rest and after over-breathing in groups of normal, psychoneurotic, epileptic, and post-traumatic cases (900 subjects). An abnormal encephalogram based on a norm which excludes 10% of the healthy population indicates a genotypical defect which may find expression as epilepsy or behaviour disorder in the subject or his offspring. H. L.

**Cephaloglycic reactions of non-labyrinthine origin.** E. A. Spiegel (*Amer. J. Physiol.*, 1942, 135, 628—632).—Bilateral section of the 8th nerves in decerebrate cats reverses the direction of the rotation of the head produced by a d.c. flowing transversely through the skull, the rotation being directed towards the anode before and towards the cathode after section of the 8th nerves. After unilateral section of the 8th nerve the rotation to the anode is preserved when the cathode lies on the normal side, while the rotation to the cathode appears if the latter lies on the operated side. On bipolar monaural stimulation rotation to the opposite side occurs as long as the 8th nerves are intact, and to the same side after the corresponding nerve has been cut. M. W. G.

**Head injuries and meningitis.** E. A. Linell and W. L. Robinson (*J. Neurol. Psychiat.*, London, 1941, 4, 23—31).—In 3 cases meningitis occurred 14, 5, and 2½ years respectively after injury. H. L.

**Coup-contrecoup mechanism of cranio-cerebral injuries.** C. B. Courville (*Arch. Surg.*, Chicago, 1942, 45, 19—43).—The similarity of coup and contrecoup lesions and the fact that gross lesions of either type never occur in the occipital region (diffuse contusion excepted) suggest that the anatomical relation of the brain and the portion of the skull proximate to it is essentially responsible for the nature and the distribution of the lesion. The contrecoup lesion is produced by a wave of force which is transmitted directly through the nervous tissue and thrusts the cortex of the opposite diameter of the brain against the resisting irregularities and contours of the skull. F. S.

**Mechanism of contrecoup injury.** A. F. Goggio (*J. Neurol. Psychiat.*, London, 1941, 4, 11—22).—The "compression wave" and "elastic body" theories and the significance of differences in sp. gr. of the intracranial contents are discussed; a hydrodynamic pressure gradient hypothesis is presented implying a momentary pressure imbalance by which the small blood vessels in the area of contrecoup are distended and ruptured by a sudden release of external pressure which is not completely met by a fall in intravascular pressure due to circulatory inflow from an outside pressure source (heart). A rotatory component of a blow or fall may also be a factor causing cerebral damage in a region with intracranial projections. H. L.

**Psychosis associated with vitamin-B deficiency.** E. Slater (*Brit. Med. J.*, 1942, I, 257—258).—Case report. C. A. K.

**Periodic somnolence and morbid hunger (Kleine-Levin syndrome).** M. Critchley and H. L. Hoffman (*Brit. Med. J.*, 1942, I, 137—139).—2 cases are reported. C. A. K.

**Emotional aspects of speech and language development.** J. L. Despert (*M Schr. Psychiat. Neurol.*, 1941, 104, 193—227). H. L.

**Alzheimer's disease with predominating crossed cerebrotellar hemiatrophy.** R. E. Hemphill and E. Stengel (*J. Neurol. Psychiat.*, London, 1941, 4, 97—106).—A case is reported. H. L.

**Dementia in middle age.** W. H. McMenemy (*J. Neurol. Psychiat.*, London, 1941, 4, 48—79).—A crit. review. H. L.

**Congenital form of amaurotic family idiocy.** R. M. Norman and N. Wood (*J. Neurol. Psychiat.*, London, 1941, 4, 175—190).—An 18-days-old female infant showed post mortem a considerably reduced brain (87 g.) with retarded frontal convolitional development. The nerve cells showed swelling of the amaurotic family idiocy type with intracellular lipin resistant to solvents (cerebral and cerebellar cortices). Further findings included lipid abnormalities in glial cells and visceral reticulo-endothelial system, retarded myelinisation, heavy deposits of extracellular doubly refractive cholesteryl ester crystals in white matter of brain and cerebellum, and severe olivo-cerebellar atrophy. H. L.

**Imperception for position of eyelids on one side.** L. H. Rubinstein (*J. Neurol. Psychiat.*, London, 1941, 4, 191—205).—4 cases are reported. H. L.

**Micro-determination of bromine in cerebrospinal fluid.** P. J. Hardwick (*Analyst*, 1942, 67, 223—225).—1 or 2 ml. of c.s.f. is saponified, in a sealed tube, with *N*-sodium ethoxide and the product is alkaline ashed (A., 1933, 1317). Last traces of org. matter are removed without  $K_2Cr_2O_7$ . The residue is taken up in water, neutralised to methyl-red with 2*N*-HCl, and made up to 10 ml. Determination of small amounts of Br<sup>-</sup> in presence of a large excess of Cl<sup>-</sup> is not possible by the fuchsin method (A., 1934, 1188) or the iodometric method of Dixon (A., 1934, 338) but is satisfactory by both the fluorescein method (A., 1933, 920; 1935, 835; 1936, 914) and the iodometric method of Doering (A., 1937, I, 260). Details of each procedure are given. S. B.

**Choline-esterase in normal and pathological cerebrospinal fluid in man.** H. Birkhäuser (*Schweiz. Arch. Neurol. Psychiat.*, 1941, 46, 185—190).—Choline-esterase was estimated by measuring the CO<sub>2</sub> liberated from acetic acid obtained during hydrolysis of acetylcholine. The mean val. for 23 normal cases was 14 cu. mm. of CO<sub>2</sub> per 0.5 c.c. of c.s.f. per 120 min. (mean deviation:  $\pm 1.1$ ). Choline-esterase was increased in meningococcal and tuberculous meningitis (not in correlation to increased cell content) and slightly also in schizophrenia. H. L.

**Ventriculometry.** F. Morel and R. de Montmollier (*M Schr. Psychiat. Neurol.*, 1941, 104, 1—14, 150—169).—Data are given on capacity of lateral ventricles and wt. of cerebral hemispheres, both measured in the undeformed, fixed brain, of cases of oligophrenia, schizophrenia, general paralysis of the insane, and of degenerative and circulatory cerebral disorders. H. L.

**Chronic polyradiculoneuritis with high protein and normal cell content in cerebrospinal fluid.** M. André (*M Schr. Psychiat. Neurol.*, 1941, 104, 34—52).—2 cases are reported. H. L.

**Blood supply of nerves.** W. E. Adams (*J. Anat.*, 1942, 76, 323—339).—A review. W. J. H.

**Sympathectomy of upper extremity: only the second thoracic ganglion need be removed for complete sympathectomy.** O. R. Hyndman and J. Wolkin (*Arch. Surg., Chicago*, 1942, 45, 145—155).—Five cases are described in which removal of only the 2nd thoracic sympathetic ganglion resulted in as complete a sympathectomy insofar as central connexions are concerned as does removal of the inferior cervical and upper two thoracic ganglia. Hence no sympathetic fibres which supply the skin structures of the face and the upper extremity travel in the first anterior thoracic root in man. F. S.

**Sympathetics of brachial region of *Triturus viridescens*.** M. Singer (*J. comp. Neurol.*, 1942, 76, 119—143).—The sympathetic supply to the forelimb was studied in intact and operated specimens of this urodele. It is mainly by way of large subclavian nerves which leave the sympathetic chain at the subclavian plexus. The fifth spinal nerve is the only nerve of the brachial plexus receiving a ramus communicans and, as in selachii, it is composed entirely of preganglionic and visceral afferent fibres. Some details of the vertebral nerve and of the nerve supply to the heart are given. J. D. B.

**Differences in argyrophilia of sympathetic and other nerve fibres.** J. F. Nonidez and K. Hare (*J. comp. Neurol.*, 1942, 76, 91—117).—Experiments in dogs in which preganglionic and afferent connexions were destroyed resulted in the disappearance of fibres in sympathetic trunks and ganglia which stain darkly with Ag. The intact postganglionic fibres are always pale-staining. Cholinergia and strong affinity for Ag are not correlated. J. D. B.

**Studies on pain: measurement of effect of ethyl alcohol on pain threshold and on "alarm" reaction.** H. G. Wolff, J. D. Hardy, and H. Goodell (*J. Pharm. Exp. Ther.*, 1942, 75, 38—49).—Quant. measurements were made of the pain threshold by irradiating 3.5 sq. cm. of skin surface for 3 sec. with different intensities of heat; the intensity of reaction which barely evoked pain was taken as pain threshold. The smallest amount of ethyl alcohol with which the highest threshold-raising effect was attained was 30 c.c. The max. pain threshold-raising effect was elevated about 45% above the control level. The duration of the effect was comparatively short (1 hr. with 15 c.c. and 4 hr. with 90 c.c.). The rate of rise of pain threshold after alcohol was, however, swifter than with other agents investigated with the exception of inhaled trichloroethylene. The max. elevation of pain threshold after 90 c.c. was attained within 15 min. and after 30 c.c. within 40 min. Addition of 0.3 g. of acetylsalicylic acid to 30 c.c. of 95% alcohol prolonged the threshold-raising effect. When 95% alcohol was administered during a 40-min. period of induced pain, the pain-threshold-raising action of alcohol was not diminished. Alcohol accentuated the ability to dissociate pain perception from the fight-flight-anxiety reaction pattern of pain. Measurements of electrical skin resistance showed that, in contrast to uniformity for pain threshold, threshold for "alarm" reaction (induced by heat) varied widely both individually and in the same individual. H. L.

## X.—SENSE ORGANS.

**Industrial eye injuries.** A. MacNalty (*Brit. Med. J.*, 1942, I, 173—177).—A lecture. C. A. K.

**Paralytic squint.** E. T. Smith (*Med. J. Austral.*, 1942, 29, I, 619—620).—In a traumatic case of complete abducens paralysis, pedicle grafting of slips from superior and inferior rectus tendons to the insertion of the lateral rectus with zig-zag lengthening of the medial rectus tendon gave a very good result. H. L.

**Slit-lamp and corneal microscope.** L. S. Bhave (*Ind. J. Ophthalm.*, 1942, 3, 35—46).—A survey. H. L.

**The cornea.** D. Cogan and V. E. Kinsey (*Science*, 1942, 95, 607—608).—Corneae immersed in various aq. solutions could be maintained in a state of normal dehydration only by maintaining an osmotic gradient across the intact epithelium and/or endothelium so that water was abstracted from the cornea as rapidly as it became available in the stroma. A similar mechanism is postulated for the conditions *in vivo*; it also serves to transport O<sub>2</sub> and other dissolved materials from the blood to the corneal tissues. The optical difference between cornea and sclera is due to the absence of a dehydrating mechanism in the latter. H. L.

**Effect of benzene and its derivatives on pupil of isolated frog eye.** K. Yoshimura (*Japan. J. Med. Sci.*, 1941, IV, 13, 149—206).—Data are given on the effect of benzene and related compounds, of mono-, di-, and tri-hydric phenols, benzoic acid and derivatives, and of amines (aniline, benzylamine, etc.) on the pupillary size. Combinations of the tested substance with BaCl<sub>2</sub>, adrenaline, pilocarpine, or atropine indicated that the mydriatic effect of benzene and benzylamine is mainly due to parasympathetic paresis, that phenols act directly on pupillary muscles, and benzoic acid (miosis) by parasympathetic excitation. The mydriatic effect of alkyl compounds was especially marked when the alkyl group was not directly attached to the benzene ring. A directly attached halogen only increased the effect of the mother nucleus, a directly attached carboxyl group had parasympathetic effect, whilst compounds with a directly attached amino-group stimulated sympathetic fibres. H. L.

**Pupillary and other responses from stimulation of frontal cortex and basal telencephalon of cat.** R. Hodes and H. W. Magoun (*J. comp. Neurol.*, 1942, 76, 461—474).—Stimulation of the rostral portion of the cerebral hemisphere with the Horsley-Clarke technique showed a dilator field located chiefly in the gyri proreus and genuis of the frontal cortex with a caudal continuation extending through the basal telencephalon to the hypothalamus. The dilator responses were effected entirely by oculomotor inhibition, and reactions of the parasympathetomised iris, nictitating membrane, or pilomotor system could not be elicited. Pupilloconstriction was obtained from the anterior portion of the gyrus cinguli and adjacent part of gyrus genuis overlapping to some extent the dilator field. Stimulation of forebrain dilator fields also elicited inhibition of respiration. H. L.

**Cataract and other manifestations of tryptophan deficiency in rats.** A. A. Albanese and W. Buschke (*Science*, 1942, 95, 584—586).—Acute and chronic cataracts developed in growing rats fed on a tryptophan-deficient diet. Associated lesions were enamel defects of incisor teeth, corneal vascularisation, alopecia, and testicular atrophy with aspermiogenesis, the last 3 lesions developing also in adult animals; there was also retardation of growth and loss of skin turgor. On the basis of morphological characteristics, progress, and associated lesions the cataract belongs to the epitheliodystrophic group which

includes cataract due to ariboflavinosis, chronic TI poisoning, X-rays, scleroderma, atopic eczema, and atrophic myotonia.  
H. L.

**Posterior subcapsular opacities of lens following recurrent attacks of uveitis.** B. K. Dasgupta (*J. Ind. Med. Assoc.*, 1942, 11, 103—105).—3 cases are reported.  
H. L.

**Effect of infra-red light on retinal pigment migration.** K. Sai (*Japan. J. Med. Sci.*, 1941, IV, 13, 65—73).—Infra-red light causes forward migration of retinal pigment in the frog *in vivo* and in the isolated frog eye. The perfusate of hindlimbs irradiated with infra-red rays contained a substance producing forward migration of retinal pigment in frogs kept in the dark; this effect was absent after ligation of the adrenal blood vessels. The infra-red effect was delayed in hypophysectomised animals.  
H. L.

**Effect of splenectomy on retinal pigment migration and its seasonal variations.** I. Shima (*Japan. J. Med. Sci.*, 1941, IV, 13, 57—64).—In frogs splenectomised in spring or autumn, the retrogression of retinal pigment (in the dark) was delayed in the first and accelerated in later stages; forward migration (in daylight) was slightly delayed. In summer, pigment retrogression after splenectomy was initially accelerated while forward migration showed a late delay; experiments in winter showed no effect on pigment migration. The findings are attributed to increased adrenaline secretion as a sequel to a rise in blood-histamine following splenectomy (absent in winter).  
H. L.

**Aniseikonia.** H. B. Field (*J. Amer. Med. Assoc.*, 1942, 119, 132—133, and *Sight Saving Rev.*, 1942, 12, 98—102).  
H. L.

**Bishop Harman's night vision test.** J. W. Barrett (*Med. J. Austral.*, 1942, 29, I, 541).—This test was considered simplest and most suitable for routine clinical purposes, for which the precise time of dark adaptation is not necessary.  
D. M. S.

**Visual sensitivities to colour differences in daylight.** D. L. MacAdam (*J. Opt. Soc. Amer.*, 1942, 32, 247—274).—An apparatus is described in which colours obtained by filters are mixed in any proportions by a polarising system and the resultant colour is compared with another filter-colour; the brightness of the mixture can be automatically const., irrespective of proportions of component colours, or variable. The sensitivity to colour differences was measured by the standard deviation of colour matches totalling over 25,000 on one observer; a series of ellipses at various points in the colour triangle was thus obtained.  
K. J. W. C.

**Vitamin-A for colour-blindness.** K. Dunlap and R. D. Loken (*Science*, 1942, 95, 554).—Cases of colour-blindness were "cleared up" in 3—8 days by doses of vitamin-A, 25,000 i.u. daily. The effect was accelerated by larger doses.  
D. M. S.

**Alternate approach to mathematical biophysics of perception of combinations of musical tones.** N. Rashevsky (*Bull. Math. Biophysics*, 1942, 4, 89—90).—Mathematical treatment of the assumption that the intensities of excitation of the centres corresponding to non-overlapping harmonics are the same while excitation is twice as great for a centre corresponding with 2 overlapping harmonics leads to an expression for pleasure vals. of different binary combinations of musical sounds.  
H. L.

**Audiogenic seizures in rats. Olfactory-parotid reflex.**—See A., 1942, III, 739.

**Sensation of vibration. II.** M. Brown and G. K. Yacorzynski (*Arch. Neurol. Psychiat.*, 1942, 47, 813—820).—Findings in 9 cases of unilateral retrogasserian neurectomy indicated that vibratory sensibility depends on stimulation of both touch and deep pressure receptors.  
H. L.

**Sense of taste.**—See A., 1942, III, 673.

**Cerebral activity including conscious sensation as physico-chemical process.**—See A., 1942, III, 674.

## XI.—DUCTLESS GLANDS, EXCLUDING GONADS.

**Co-ordination of vertebrate melanophore responses.** H. Waring (*Biol. Rev.*, 1942, 17, 120—150).—A review of melanophore responses to light and their nervous and humoral control.  
W. T. A.

**Peripheral blood flow in myxœdema.** H. J. Stewart and W. F. Evans (*Arch. intern. Med.*, 1942, 69, 808—821).—The peripheral blood flow was measured by a method previously described (*Amer. Heart J.*, 1940, 20, 715) in 6 patients with myxœdema. It was decreased in proportion to the basal metabolic rate, and when the latter was raised to normal by thyroid therapy peripheral and cardiac blood flow were simultaneously increased. It is suggested that the decrease in peripheral blood flow helps in the conservation of body heat.  
C. A. K.

**Carotinæmia in myxœdema.** R. F. Escamilla (*J. clin. Endocrinol.*, 1942, 2, 33—35).—Carotinæmia was found in 7 consecutive cases of myxœdema. The condition gradually cleared up during thyroid treatment. Carotinæmia was also seen in a case of Simmonds'

disease. It is suggested that it caused by a failure to convert carotene into vitamin-A in cases with lowered basal metabolic rate.  
P. C. W.

**Thyroxine in myxœdema.** W. R. Trotter and N. Wallace (*Brit. Med. J.*, 1942, I, 183—184).—A patient with myxœdema could not tolerate oral thyroid extract, which produced persistent vomiting. Subsequently 7.5 mg. of thyroxine intravenously every 4 weeks produced satisfactory results.  
C. A. K.

**Sexual infantilism of hypothyroid origin.** H. Lissner (*J. clin. Endocrinol.*, 1942, 2, 29—32).—A case is reported.  
P. C. W.

**Action of pathological thyroid from rabbits and sheep on metabolism and heart rate in thyroidectomised rats.** A. E. Meyer and D. Marine (*Endocrinol.*, 1942, 30, 558—563; cf. A., 1939, III, 908).—When goitrous thyroids produced in rabbits by a special low-I diet, and occurring spontaneously in sheep, are fed to thyroidectomised rats, little effect is produced on heart rate by glands containing colloid. Glands without colloid caused heart stimulation but no metabolic effect, but a single dose of KI caused an appearance of metabolic hormone with no change in heart effect.  
V. J. W.

**Response of blood-lipins to thyroidectomy.** C. Entenman, I. L. Chaikoff, and F. L. Reichert (*Endocrinol.*, 1942, 30, 794—801).—The rise in blood-lipins normally caused by thyroidectomy in the dog does not occur in states of under-feeding.  
V. J. W.

**Effects of thyroidectomy on excretion and retention of creatine and creatinine in male rat.** C. Glaser (*Endocrinol.*, 1942, 30, 564—570).—Thyroidectomised rats excrete less creatine than controls, whether or not creatine injections are given to them. Creatinine excretion is not altered either by thyroidectomy or by creatine injections, and creatinine injections do not alter creatine excretion. There is no increased storage of creatine after thyroidectomy.  
V. J. W.

**Liver damage in thyrotoxicosis.** N. Wyndham (*Austral. N.Z. J. Surg.*, 1940, 9, 385—392).—No constancy was found post mortem in presence or degree of liver damage in 43 cases of fulminating thyrotoxicosis.  
H. L.

**Essential biochemical derangements in hyperthyroidism.** W. Bartlett (*Arch. Surg., Chicago.*, 1942, 45, 103—110).—The CO<sub>2</sub>-combining power of the plasma is within normal limits even in severe exacerbations if the patient is at rest and the physiological needs for water and nourishment are met; a prompt decrease in alkali reserve occurs on exertion. During improvement on treatment preliminary to thyroidectomy and subsequent to operation, a profound fall in the total titratable acid of the urine, of the org. acids, and of NH<sub>3</sub> occurs, and the pH of the urine rises if the intake of food is kept const. quantitatively and qualitatively. In nearly 50% of the patients who show prompt improvement after thyroidectomy, a rise in the alkali reserve of the plasma occurs. The acidosis probably causes the tendency in hyperthyroidism to water retention by the tissues through impairment of capillary integrity.  
F. S.

**Behaviour of iodine content of the thyroid in course of experimental scurvy.** I. Mihályfi (*Z. physiol. Chem.*, 1941, 271, 289—292).—From the end of the 1st to about the end of the 4th week of scurvy there is an accumulation of I in the thyroid. In the final stages of the disease the I content diminishes. These variations appear to be related to variations in the activity of the gland.  
H. W.

**Influence of vitamin-E deficiency on thyroid and basal metabolism of male rats.** C. Biddulph and R. K. Meyer (*Endocrinol.*, 1942, 30, 551—557).—Rats maintained on the vitamin-E-deficient diet of Evans *et al.* (cf. A., 1935, 548) had an increased basal metabolic rate and histological signs of activity in the thyroid. These were found to be due to the low I content of the diet and were abolished when I was added.  
V. J. W.

**Influence of stilbœstrol and thyroxine on galactose absorption and liver function.** R. C. Grauer, W. F. Starkey, and E. Saier (*Endocrinol.*, 1942, 30, 474—482).—Daily injections of 0.5 mg. per kg. of thyroxine in dogs cause intestinal absorption of galactose to be increased for 7 days, decreased after 14 days, returning to normal in 20 days. When it is given for 16—22 days liver damage, with increase in blood-galactose, is produced. Stilbœstrol (5—10 mg. daily) increases galactose absorption and disappearance from the blood by glycogen formation.  
V. J. W.

**Parathyroid of Virginia deer at different times of year.** A. L. Grafflin (*Endocrinol.*, 1942, 30, 571—573).—The basophil granules previously described (A., 1940, III, 280) show no seasonal variations.  
V. J. W.

**Behaviour and mood cycles related to parathyroid deficiency.** C. P. Richter, W. M. Honeyman, and H. Hunter (*J. Neurol. Psychiat.*, London, 1940, 3, 19—26).—Parathyroidectomised monkeys kept on a low-Ca diet drank in fairly regular cycles of about 40 days large amounts of a 2.4% Ca lactate solution which alleviated their symptoms of insufficiency. A woman aged 56 suffering from parathyroid deficiency showed cyclic variations (about 40 days) in mood and behaviour which disappeared on Ca therapy.  
H. L.

**Involution and regeneration of thymus in rats on choline-deficient diets.** K. Christensen and W. H. Griffith (*Endocrinol.*, 1942, 30,

574—580).—These changes (A., 1940, III, 149) are described in greater detail and wts. tabulated. V. J. W.

**Adrenal gland innervation.** H. A. Teitelbaum (*Quart. Rev. Biol.*, 1942, 17, 135—148).—A review. J. D. B.

**Age and sex variations in fat of adrenal cortex of the white rat.** C. E. Tobin and R. Whitehead (*J. Anat.*, 1942, 76, 342—346).—The histological distribution of fat and cholesterol was studied in the adrenal cortex of normal rats. The fat has a regional distribution which shows variations with age but was similar in both sexes. Cholesterol has a similar distribution to the fat. There is no relation between the fat in the cortex and the oestrous cycle. W. J. H.

**Physiological significance of compensatory adrenal atrophy.** H. Selye and C. Dosne (*Endocrinol.*, 1942, 30, 581—584).—Pre-treatment of rats with high doses (10 mg. daily for 7—13 days) of deoxycorticosterone acetate lessens the adrenal cortical response to the "alarm" reaction, so that the resistance of such animals to noxious agents is subnormal, and the usual hyperglycæmic response is diminished. V. J. W.

**Response of melanophores of skin to injections of adrenaline with special reference to body weight of animal.** M. E. Pierre (*J. exp. Zool.*, 1941, 86, 189—203). J. D. B.

**Circulatory response of unanæsthetised dog to small physiological quantities of adrenaline.** H. C. Wiggers, A. M. Duschatko, and R. C. Kory (*Amer. J. Physiol.*, 1942, 136, 87—94).—Small intravenous injections of adrenaline in the unanæsthetised dog raise or lower blood pressure according to the dose. The depressor reaction is elicited by 0.1 µg. per kg. and is reproducible in the same dog at 20-min intervals. Higher concns. may be pressor. The depressor response may be due to initial dilatation of pulmonary vessels. A transient reduction of left ventricular filling occurs, accentuated by the simultaneous tachycardia. Adrenaline may augment the capacity of the aorta and its immediate large branches. M. W. G.

**Effect of Röntgen rays on action of adrenalone on blood-sugar of rabbits.** E. Sai (*Japan. J. Med. Sci.*, 1941, IV, 14, 1—12).—The hyperglycæmic action of adrenalone is increased after exposure of the drug to X-rays. The increase becomes less if the amount of Röntgen rays exceeds 4800 r. Erythrosin, trypanflavine, or methylene-blue acts as photocatalyst for the action of the X-rays. H. H. K.

**Diagnosis of Addison's disease.** J. M. Rogoff (*J. clin. Endocrinol.*, 1942, 2, 36—42).—Clinical signs and symptoms in 2 cases of Addison's disease are described in detail. Diagnostic signs are discussed. P. C. W.

**Treatment of Addison's disease with interrenalin (adrenal cortex extract).** J. M. Rogoff (*J. clin. Endocrinol.*, 1942, 2, 43—48).—Prolongation of life is the only satisfactory criterion of beneficial treatment in Addison's disease. 20 patients treated with adrenal cortex extract survived 1—2 years in 8 cases and 2½—7½ years in 12 cases. In 12 cases not so treated the average survival was 0.8 (max. 1½) years. P. C. W.

**Effect of adrenal cortical extract, deoxycorticosterone, and added potassium on electrolyte balance in normals and Addison's disease.** J. A. Greene, A. David, and G. W. Johnston (*J. clin. Endocrinol.*, 1942, 2, 49—52).—3 normal subjects and 3 cases of Addison's disease were studied; all were maintained on a diet containing 12 g. of Na and 2 g. of K per day. Adrenal cortex extract (2500 dog units) caused Na retention in the normal subjects but not in the patients with Addison's disease. Both groups showed Na retention after 25 mg. of deoxycorticosterone. Addition of 6 g. of K to the diet produced a reduction in Na storage in normals and in those patients with high Na storage. The patients stored more of the added K than the normals. When adrenal cortex extract was given as well as the increased K in the diet the results were inconst. P. C. W.

**Preoperative administration of deoxycorticosterone acetate in prevention of surgical shock.** F. R. Keating, E. H. Rynearson, and M. H. Power (*J. clin. Endocrinol.*, 1942, 2, 53—58).—9 of 19 women undergoing radical mastectomy for carcinoma of the breast were treated pre-operatively with deoxycorticosterone acetate (20—40 mg.). Extensive blood examinations were made before and at intervals after the operation. There was no evidence that the pre-operative treatment had any beneficial effects. The blood pressure fell to shock levels in 6 cases in both control and experimental groups; of these 12 cases 2 in each group showed a prolonged period of low blood pressure after the operation. P. C. W.

**Changes in blood chemistry associated with circulatory failure in adrenalectomised dog.** J. W. Remington, V. A. Drill, W. Kleinberg, and W. W. Swingle (*Endocrinol.*, 1942, 30, 692—701).—Circulatory failure following trauma or hæmorrhage is followed by hæmo-concn. only when trauma leads to local loss of fluid. Hæmorrhage does but trauma does not cause a rise in blood-urea-N. This can be corr. by deoxycorticosterone. Fall in serum-Na<sup>+</sup> and -Cl<sup>-</sup> follows on surgical shock. This is corr. by giving deoxycorticosterone, but accompanying circulatory failure is not. Trauma, but not hæmorrhage, causes a fall in blood-sugar. Effects of cortical extract,

deoxycorticosterone, corticosterone, and 17-hydroxy-11-dehydrocorticosterone are compared. V. J. W.

**Syndrome simulating diabetes insipidus in dogs induced by deoxycorticosterone acetate.** R. C. Moehlig and L. Jaffe (*J. Lab. clin. Med.*, 1942, 27, 1009—1012).—A syndrome of polydipsia and polyuria was induced in 2 dogs by 5—10 mg. of deoxycorticosterone acetate daily over 4 months. Sections of the hypothalamus showed a localised encephalitic reaction with a plasma-cell infiltration of the leptomeninges and adjacent intracerebral blood vessels. A patient with myasthenia gravis, who had an overdose of the drug (1300 mg. by implantation of pellets of this material and the crushing of 1 pellet), showed the syndrome of polydipsia, polyuria, and muscular weakness with signs and symptoms of tetany. C. J. C. B.

**Influence of hormones on carbohydrate levels of chick.** W. R. C. Golden and C. N. H. Long (*Endocrinol.*, 1942, 30, 675—686).—Blood-sugar is 200 mg.-%, (pectoral) muscle-glycogen 1%, and liver-glycogen 2%. Adrenaline and insulin have the same effects as in mammals, but the convulsive dose of insulin was irregular. Adrenal cortical extract causes hyperglycæmia; in fasting chicks it increases liver glycogen but not in fed birds. Pituitary extract had no effect on blood-sugar. V. J. W.

**Opposite effects of adrenal and thyroid hormones on glycogen metabolism of liver.** I. Abelin and U. Althaus (*Helv. Chim. Acta*, 1942, 25, 205—215).—Lack of insulin and also an excess of thyroid hormone lead to a persistent glycogen impoverishment of the liver (rat). Lack of glycogen does not depend entirely on increased combustion of sugar since the liver is poor in, or free from, glycogen at a time when the increase of energy production is scarcely observable. The adrenal glands appear to play a significant part in the deposition of glycogen. Rats which receive adrenal extracts in addition to thyroid hormone store nearly normal amounts of glycogen in the liver in spite of marked thyroid poisoning. The glycogen metabolism of muscle is not affected by adrenal extracts. The adrenals are recognised as particularly sensitive, secretory organs that become involved when the activity of the thyroid is increased. Thus extracts of the adrenals of hyperthyroidised animals cannot inhibit the glycogen-impoverishing action of thyroxine. Corticosterone and deoxycorticosterone and its acetate abolish the effect of mild (but not severe) hyperthyroidism on liver-glycogen. H. W.

**17-Ketosteroid excretion in adrenal virilism.** J. Patterson, I. M. McPhee, and A. W. Greenwood (*Brit. Med. J.*, 1942, I, 35—39).—17-Ketosteroid output in the urine was estimated in cases of adrenal tumour, primary and secondary virilism. Adrenal tumours and primary virilism cases showed very high excretion rates, and it was impossible before puberty to distinguish between adrenal hyperplasia and tumours. Over 50% of cases of secondary virilism showed normal ketosteroid vals., but some had raised ketosteroid/creatinine ratios. 3 cases of feminism showed low ketosteroid excretion rates. C. A. K.

**Effect of sympathomimetic amines on pancreatic secretion in dogs.** H. Greengard, R. A. Roback, and A. C. Ivy (*J. Pharm. Exp. Ther.*, 1942, 74, 309—318).—The following compounds inhibited the secretion evoked by continuous secretin stimulation: β-phenylethylamine, β- and γ-amino-α-phenylpropane, N-methyl-β-phenylethylamine, pervitin, deoxyephedrine, propadrine, d-ephedrine, ephedonal, tyramine, veritol, sympathol, arterenol, epinephrine, corbasil, 3:4-dihydroxyphenylethyldimethylamine, corynein chloride, dihydroxyephedrine, ethylnorsuprarenin, adrenalone, 6:7-dihydroxy-2-methyltetrahydroisoquinoline. The mechanism whereby this took place is probably on a vasoconstrictor basis. m-Hydroxyphenylethylamine, hydroxytyramine, epinine, m-hydroxy-, m- and p-methoxy-, and 3:4-dimethoxy-phenylethylamine, and 3:4-dihydroxyphenylalanine stimulated pancreatic secretion; they are structurally similar to the extent of hydroxylation of the benzene ring in sp. positions, an unsubstituted C atom in juxtaposition to the ring, and a primary or secondary amino-nitrogen. Methylation of the OH groups leaves the secretory potency attenuated but not abolished. H. H. K.

**Increased glucose appetite of normal rats treated with insulin.** C. P. Richter (*Amer. J. Physiol.*, 1942, 135, 781—788).—Adult rats treated daily with progressively increasing doses of insulin (2—16 units per day for 26—54 days) show a markedly increased appetite for a 40% solution of glucose. Daily glucose intake increased from 14.6 c.c. for the last 10 days before treatment to 31.5 c.c. for the last 10 days of insulin injections. Intake of food increased only from 10.1 to 12.1 g. Discontinuing treatment causes a sharp but temporary decrease in glucose appetite; food intake showed a less sharp decrease. Several weeks after discontinuing insulin the glucose appetite was greater than during pre-treatment and food intake was lower. M. W. G.

**Influence of pituitary and adrenal cortex on resistance to low environmental temperatures.** R. Tyslowitz and E. B. Astwood (*Amer. J. Physiol.*, 1942, 136, 22—31).—Young rats could not maintain their body temp. when exposed to an environment of 0° after hypophysectomy; they became progressively more sensitive to cold over the first 4 post-operative days and thereafter exhibited a const.

response to cold. Crude pituitary extracts or purified corticotrophin increased the cold-resistance of such animals before or after thyroidectomy but not after adrenalectomy. When administered 1 hr.—14 days prior to exposure adrenal cortical extracts increased the resistance of hypophysectomised rats and of similar animals without thyroids or adrenals. The protection against cold afforded hypophysectomised rats by pituitary extracts paralleled their corticotropic activity and was attributable to it. T. F. D.

**Water intoxication of frog [rôle of hypophysis].** E. C. Schneider and W. C. Grant (*Amer. J. Physiol.*, 1942, 136, 42—48).—Water administered subcutaneously or by stomach tube to normal frogs at intervals of 30—60 min. in doses of 2—4 c.c. for 3 days failed to kill. Hypophysectomy renders the frog susceptible to water intoxication; blood-Ca is then  $\frac{1}{2}$  less than normal. The water content of body and organs of the intact or hypophysectomised frogs rose approx. equally. The brain showed no hydration. Cl<sup>-</sup> excretion first rose and later decreased. M. W. G.

**Influence of hypophysectomy on ability of rat to remove fructose from its blood.** R. M. Reinecke and L. T. Samuels (*Endocrinol.*, 1942, 30, 687—691).—Such ability is very slightly reduced by hypophysectomy. V. J. W.

**Blood-lipins of hypophysectomised thyroidectomised dog.** C. Entenman, I. L. Chaikoff, and F. L. Reichert (*Endocrinol.*, 1942, 30, 802—815).—The rise in blood-lipins after thyroidectomy is not modified by previous hypophysectomy. V. J. W.

**Effect of purified pituitary preparations on liver weights of hypophysectomised rats.** H. L. Fraenkel-Conrat, M. E. Simpson, and H. M. Evans (*Amer. J. Physiol.*, 1942, 135, 398—403).—Putrified thyrotropic hormone causes an abs. and relative increase in liver vol. of young hypophysectomised rats, when given for 10—15 days. Thyroxine causes similar liver growth effects. Growth hormone causes only a slight abs. increase, far less than the body wt. increase; thus expressed in % of body wt. growth hormone causes a relative decrease in liver wt. Determination of N, fat, water, and glycogen contents of livers of rats treated with various fractions revealed no difference in their composition. M. W. G.

**Anterior pituitary in carbohydrate metabolism of eviscerated rat.** J. A. Russell (*Amer. J. Physiol.*, 1942, 136, 95—104).—Hypophysectomised and adrenal demedullated rats after evisceration survived half as long as normal demedullated rats and utilised blood-glucose at 2—3 times the normal rate. There was no increase in blood lactic acid but muscle-glycogen fell. This fall was prevented, and the survival time was prolonged, by previous treatment of the rats with saline anterior pituitary extract. Hypophysectomised rats required glucose administration at the rate of 25—30 mg. per 100 g. per hr. to maintain normal blood-sugar levels; control rats required only 13.5 mg. These increased requirements of glucose were not due to deposition of muscle-glycogen or to liberation of lactic acid but presumably to increased utilisation of carbohydrate in the peripheral tissues in the absence of the anterior pituitary. Nephrectomy increased moderately the need for glucose in rats as in rabbits. Kidney damage is not an important factor in producing the increased utilisation of carbohydrate in hypophysectomised rats. T. F. D.

**Effects of pancreas and adrenals on production of nitrogen storage with pituitary preparations.** O. H. Gaebler and A. R. Robinson (*Endocrinol.*, 1942, 30, 627—634).—In insulin-maintained depancreatised dogs, single doses of growth hormone (Antuitrin-G) cause loss of N and increased glycosuria. If insulin dosage is increased sufficiently, N is stored and diabetic symptoms disappear when growth hormone is given. Neither insulin nor growth hormone alone will cause N storage, but some of each is necessary. V. J. W.

**Influence of anterior pituitary extract on protein and carbohydrate metabolism.** K. E. Paschke (*Amer. J. Physiol.*, 1942, 136, 128—135).—An anterior pituitary extract containing growth hormone (Antuitrin-G) produced a decrease of blood-urea-N, indicative of protein anabolism, and sometimes hypoglycaemia, in the normal fasted rat; in the adrenalectomised fasting rat maintained with NaCl, and in the partly pancreatectomised diabetic rat. The enhancing effect of anterior pituitary extracts on protein anabolism is thus not mediated through either the adrenals or the pancreas. T. F. D.

**Prolactin.** A. White, R. W. Bonsnes, and C. N. H. Long (*J. Biol. Chem.*, 1942, 143, 447—464).—A homogeneous, cryst. protein may be prepared from highly purified amorphous prolactin by the acetic acid-pyridine method or by pptn. from dil. aq. acetone; this has an activity (30—35 i.u. per mg.) equiv. to that of the starting material. The protein has a mol. wt. of 32,000, contains 5.51, 1.30, and 3.36% of tyrosine, tryptophan, and cystine, respectively, and has an isoelectric point of  $p_H$  5.56—5.70. It is heat-stable at  $p_H$  1—9, but at higher  $p_H$  there is considerable loss of potency. The activity is destroyed by acid hydrolysis and destruction occurs in peptic and tryptic digests prior to complete hydrolysis of the protein. H. G. R.

**Correlative cyclical changes in hypophysis and gonads of male *Necturus maculosus*.** H. W. Aplington (*Amer. J. Anat.*, 1942, 70,

201—249).—The histology of the hypophysis, including the Golgi material of its cells, is detailed for the various seasons. During the growth period of the testis (April to August) marked alterations occur in the granular basophils and agranular basophils of type 1, and it is on the basis of these changes, primarily an increase in the no. of granular basophils, that correlations between hypophysis and testis may be detected. W. F. H.

**Biological assays of male chicken pituitary for gonadotropic potency.** G. M. Riley and R. M. Fraps (*Endocrinol.*, 1942, 30, 529—536).—1 mouse uterine unit (Levin and Tyndale, *Physiol. Abs.*, 1937, 22, 965) is contained in 0.3—0.5 mg. of dried gland of various varieties of chicken. The same wt. of gland contained 10 weaver finch units (Witschi, A., 1940, III, 899) of luteinising hormone. V. J. W.

**Relationship of gonad-stimulating activity of hen anterior pituitary to reproductive conditions.** G. M. Riley and R. M. Fraps (*Endocrinol.*, 1942, 30, 537—541).—1 mouse uterine unit was contained in 4.5, 3.5, and 2.9 mg. of pituitary powder from birds with follicles in mature, regressing, and quiescent conditions respectively. V. J. W.

**Swine pituitary. I. Effects of purified follicle-stimulating (thylakentrin) and interstitial cell-stimulating (metakentrin) hormones.** R. O. Greep, H. B. van Dyke, and B. F. Chow. **II. Preparation of a protein apparently identical with metakentrin.** B. F. Chow, H. B. van Dyke, R. O. Greep, A. Rothen, and T. Shedlovsky. **III. Immunological specificity of metakentrin.** B. F. Chow (*Endocrinol.*, 1942, 30, 635—649, 650—656, 657—661).—Metakentrin causes growth of sex organs in hypophysectomised male rats and maintains interstitial cells in hypophysectomised females. It is ineffective in castrates. It has no destructive action on corpora lutea persisting after hypophysectomy, nor does it maintain their secretory activity. Gonadotropic action of pregnant mare serum is greatly reduced. Thylakentrin stimulates seminiferous tubules and Graafian follicles but has no effect on other organs. The method previously described (*Science*, 1940, 92, 178) for purification of metakentrin is modified and evidence given for its homogeneity; its anti-serum from the rabbit is species-sp. and also fails to react with other pituitary hormones. V. J. W.

**Increase in gonadotropic content of pituitary glands of female rats treated with anti-gonadotropic serum.** R. K. Meyer, H. S. Kupperman, and J. C. Finerty (*Endocrinol.*, 1942, 30, 662—666).—Daily treatment for 8 weeks with anti-serum markedly increased ovary-stimulating power of pituitaries of female rats, their ovaries were greatly diminished in wt., and basophil pituitary cells were increased. They became normal in all respects 19 days after cessation of treatment. V. J. W.

## XII.—REPRODUCTION.

**Influence of moisture on eggs of *Austroicetes cruciata*, Sauss. (Orthoptera), with reference to their ability to survive desiccation.** L. C. Birch and H. G. Andrewartha (*Austral. J. Exp. Biol.*, 1942, 20, 1—8).—The amount of water in turgid grasshopper eggs increases throughout development; the increase is slight in diapause and greater in post-diapause eggs. The passage of water into the eggs is explained in terms of physical laws. A water-absorbing area occurs at the posterior pole of the egg. The susceptibility of eggs to desiccation varies according to the stage of embryonic development and is greatest for new-laid and least for diapause eggs. The difference between prediapause and diapause eggs is connected with the absence of yellow and white cuticle in prediapause eggs. There is a sudden increase in susceptibility to desiccation in eggs in the field at the end of May and this is connected with elimination of diapause and increase in water content. Post-diapause are more easily killed by desiccation than are diapause eggs. Eggs with embryos in advanced state of development will hatch at R.H. as low as 22% at 20°. J. N. A.

**Influence of temperatures above developmental zero on development of eggs of *Austroicetes cruciata*, Sauss. (Orthoptera).** L. C. Birch (*Austral. J. Exp. Biol.*, 1942, 20, 17—25).—In the field diapause is eliminated slowly from Feb. onwards, and during this time the embryo grows slowly and growth almost ceases during the few weeks before katatrepsis. Eggs collected in mid-May hatch after incubation at 18.9—27°, and during late diapause this range of temp. has some effect in eliminating diapause. Eggs collected in June hatch at 30°. In the field diapause is finally eliminated as katatrepsis is completed, but not necessarily under artificial conditions. Eggs develop slowly at 13.5°, and the true zero of development is approx. 8°. When post-diapause eggs are incubated on alternate days at two temp. between 16.1° to 30.5° the rate of development at each temp. is the same as when incubation occurs at these temp. all the time. The rates of development of eggs incubated at R.H. 70—100% are practically the same. J. N. A.

**Cross-fertilisation of echinoderms.** E. B. Harvey (*Science*, 1941, 94, 90).—A higher proportion of cross-fertilisation was noted when the eggs were unwashed, and 10% of *Strongylocentrotus purpuratus*

eggs were fertilised by *Dendroaster excentricus* (sand-dollar) sperm, though none of the washed eggs. E. R. S.

**Influence of age and rate of breeding on ability of female rat to reproduce and raise young.** S. A. Asdell, R. Bogart, and G. Sperling (*Cornell Univ. Agric. Exp. Sta. Mem.*, 1941, No. 238, 26 pp.).—Female rats were allowed to breed as soon after puberty as possible, at 100 days old (normal), or at 270 days. The early and late breeding produced smaller litters than normal and at subsequent longer intervals. In rats bred at 100 days but with their young removed to prevent lactation, the subsequent litters were larger than those from normally lactating rats. Individual birth wts. of young from early or late breeding rats were larger, and those from non-lactating rats smaller, than normal. A. W. M.

**Metabolism of steroid hormones. III. Isolation of pregnane-3(a):20(a)-diol from urine of pregnant chimpanzees.** W. R. Fish, R. I. Dorfman, and W. C. Young (*J. Biol. Chem.*, 1942, 143, 715—720).—The total pregnenediol isolated from urine of pregnant chimpanzees represents 0.79 mg. per l., or a min. quantity of 2 mg. per animal per day. The possible source of this is discussed. P. G. M.

**Comparison between gravimetric and reduction methods for determination of pregnenediol glycuronide.** O. Hechter (*Proc. Soc. Exp. Biol. Med.*, 1942, 49, 299—302).—Hydrolysis by  $H_2SO_4$  and colorimetric determination of Cu reduction by the method of Polin and Wu gives more accurate results than Venning's gravimetric method, which in some cases gives an apparently high result, in absence of any luteal activity, due to pptd. impurities. V. J. W.

**Modification of sex development in marbled salamander by administration of synthetic sex hormones.** C. L. Foote (*J. exp. Zool.*, 1941, 86, 291—319). J. D. B.

**Progesterone-like activity of some steroid compounds and of diethylstilbestrol in stimulating mammary lobule-alveolar growth.** J. P. Mixner and C. W. Turner (*Endocrinol.*, 1942, 30, 706—710).—In the spayed mouse, various steroids have activities as follows: progesterone 1, pregnenolone  $\frac{1}{2}$ , deoxycorticosterone acetate and dehydroandrosterone  $\frac{1}{4}$ , diethylstilbestrol  $\frac{1}{4}$ , acetoxypregnenolone  $\frac{1}{8}$ , methyltestosterone  $\frac{1}{2}$ . Testosterone has no effect. V. J. W.

**Relative absorption rates of pellets of crystalline compounds implanted subcutaneously in rats.** T. R. Forbes (*Endocrinol.*, 1942, 30, 761—764).—90% absorption took for deoxycorticosterone acetate 64 days, oestradiol 93 days, androsterone 120 days,  $\alpha$ -oestradiol 180 days, its dipropionate 220 days, and benzoate 415 days. Cholesterol was not absorbed. V. J. W.

**Influence of steroids on diffusion of chlorides into peritoneal spaces.** A. E. Rakoff and A. Cantarow (*Endocrinol.*, 1942, 30, 816—818).—Administration of  $\alpha$ -oestradiol benzoate, diethylstilbestrol, or testosterone propionate increases rate of diffusion of  $Cl^-$  into the peritoneal cavity of dogs but not of rabbits (cf. A., 1940, III, 897). V. J. W.

**Synthetic oestrogen with prolonged action when given orally.** J. M. Robson and A. Schönberg (*Nature*, 1942, 150, 22—23).— $\alpha$ -Bromo-*pp'*-diethoxystilbene, injected subcutaneously into mice, has a relatively high threshold; given orally to mice its threshold is similar to that of oestradiol and much higher than that of stilbestrol. In both cases a small increase in the dose produces a marked increase in the duration of its action. A. A. E.

**Clinical experiences with sublingual administration of  $\alpha$ -oestradiol.** G. J. Hall (*J. clin. Endocrinol.*, 1942, 2, 26—28).—Study of vaginal smears in 41 patients showed that the sublingual administration of  $\alpha$ -oestradiol in propylene glycol was as effective (wt. for wt.) as subcutaneous injection of oestradiol benzoate. P. C. W.

**Mechanism of oestron production in ovary.** B. Zondek (*J. Endocrinol., Lond.*, 1942, 3, 1—4).—Previous work has shown that oestrogen passes into the circulation during the 26th hr. after chorionic gonadotrophin injection in immature rats. Oestrus may be prevented by the injection of antigonadotrophin into the rats 18 hr. after the gonadotrophin injection but if a 2nd dose of chorionic gonadotrophin is injected 2 hr. after the antihormone, oestrus occurs at the same time as if the first injection alone had been given. It is concluded that an inactive pro-oestrogen is formed in the ovary during the 18 hr. after the injection of chorionic gonadotrophin. P. C. W.

**Effect of oestron on mice of three inbred strains, with special reference to the mammary glands.** E. W. Miller and F. C. Pybus (*J. Path. Bact.*, 1942, 54, 155—167).—Mice of 3 inbred strains differing in their incidence of spontaneous mammary carcinoma were divided into 4 classes in each strain—ovariectomised and non-ovariectomised females, castrated and non-castrated males—and given weekly subcutaneous injections of 300 i.u. of ketohydroxy-oestrin from the age of 5 weeks until death. Oestron treatment produced 44 palpable mammary tumours in 135 Simpson mice, 3 in 75 Edinburgh mice, and none in 112 CBA mice, although these last had the longest life-span and therefore received the greatest total amount of oestron. The mammary glands of ovariectomised females responded more slowly and to a smaller degree than those of non-ovariectomised females. Ovariectomy prolonged life. The

mammary glands of castrated males responded more rapidly, and to a greater degree, than those of non-castrated males, and to a degree similar to that found in ovariectomised females. Castration shortened life. The action of the male hormone is antagonistic to that of oestron on the mammary glands; naturally-occurring female hormone increases the effect of treatment. Instead of producing mammary carcinoma in the 54 CBA females, oestron treatment produced many (30) uterine tumours, mainly fibromata or fibrosarcomata of the cervix. 4 tumours of this type occurred in the Edinburgh and only 2 in the Simpson strain. Oestron treatment did not affect the incidence of lung tumours but increased the incidence of various lymph-adenopathies. C. J. C. B.

**Influence of diethylstilbestrol on carbohydrate metabolism.** R. G. James and W. O. Nelson (*Amer. J. Physiol.*, 1942, 136, 136—139).—Fasting blood-sugar levels and urinary N vals. tend to increase after 20 days' stilbestrol treatment in normal or castrated rats, and adrenals and pituitaries enlarge. Liver-glycogen increased after treatment more in males than in females but muscle-glycogen was unaltered. T. F. D.

**Clinical use of stilbestrol monomethyl ether.** C. F. Geschickter and E. W. Byrnes (*J. clin. Endocrinol.*, 1942, 2, 19—25).—A series of mono- and di-alkyl ethers of stilbestrol and hexoestrol were assayed in rats. Lengthening of the alkyl substituent decreased oestrogenic activity in all cases. Stilbestrol monomethyl ether was used in the treatment of 49 patients with menopausal symptoms or menstrual disorders. All received benefit with doses of about 1 mg. every 1 or 2 days. Toxic symptoms were much less than with stilbestrol. In susceptible rats the carcinogenic activity of stilbestrol monomethyl ether was similar to that of the natural oestrogens in relation to oestrogenic activity. P. C. W.

**Distribution of fat in endometrium of castrate rabbit after treatment with oestradiol benzoate and progesterone.** C. Gilbert (*Endocrinol.*, 1942, 30, 773—781).—Either hormone alone causes a decrease of endometrial fat, but both together usually increase it. V. J. W.

**Effects of oestrogens on young of injected lactating rats.** C. K. Weichert and S. Kerrigan (*Endocrinol.*, 1942, 30, 741—752).—The young of rats injected from day of parturition with theelin or diethylstilbestrol show retarded growth by the 5th day with histological abnormalities of the reproductive system in both sexes. V. J. W.

**Effect of vitamin-B deficiency on inactivation of ovarian oestrogen in liver.** M. S. Biskind and M. C. Shelesnyak (*Endocrinol.*, 1942, 30, 819—820).—Spayed rats in which one ovary has been transplanted into the spleen remain anaestrous, but develop oestrous reactions after 3—25 days on a vitamin-B-free diet. V. J. W.

**Response of fish (*Lebistes reticulatus*) to mammalian gonadotropins.** P. Berkowitz (*J. exp. Zool.*, 1941, 86, 247—255).—Four different preps. of mammalian gonadotropins (extracts of whole sheep pituitaries, pregnant mare serum, human pregnancy urine, and normal male urine) gave positive results in male fish. There were no effects on the secondary sex characters on gonads of immature female fish. J. D. B.

**Effect of gonadotropic substance of pregnant mare serum on blood-ascorbic acid of castrate bovine.** F. N. Andrews and R. E. Erb (*Endocrinol.*, 1942, 30, 671—674).—Blood-ascorbic acid decreased by 50% 5 hr. after injections of 1000—2000 r.u. of "Gonadin" and returned to normal in 44—68 hr. V. J. W.

**Assay of prolactin by ovarian weight increase.** F. Bischoff (*Endocrinol.*, 1942, 30, 667—670).—If adequate doses are given to littermates of suitable strain this method is as satisfactory as that employing uterine wt. V. J. W.

**Percutaneous potency of progesterone.** R. R. Greene and S. C. Harris (*J. Lab. clin. Med.*, 1942, 27, 746—748).—In virgin immature rabbits primed with oestron progesterone is less effective by the percutaneous route than by the parenteral method. With oil as the solvent it is less than  $\frac{1}{4}$  as potent. With alcohol as the solvent it is less than  $\frac{1}{2}$ , but more than  $\frac{1}{4}$ , as potent percutaneously as parenterally. C. J. C. B.

**Effects of progesterone on male and female mice.** W. F. Starkey and J. H. Leatham (*Amer. J. Physiol.*, 1942, 135, 567—571).—Injections of progesterone caused in immature male mice a retardation of seminal vesicle wt. gain without influence on spermatogenesis, in immature female mice a suppression of corpus luteum formation without impairment of ovarian follicular growth, and in mature female mice ovarian atrophy and involution of the corpora lutea. Ovarian response to pregnant mare serum was uninfluenced by simultaneous injections of progesterone. T. F. D.

**Effect of progesterone on ovaries and embryos of mice in early pregnancy.** H. O. Burdick (*Endocrinol.*, 1942, 30, 619—622).—1 mg. daily of progesterone, begun on day of mating, prevents implantation. If administration is delayed till the 2nd day implantation occurs, but, as with deoxycorticosterone (A., 1941, III, 572), the embryos die when injections are stopped. V. J. W.

**Cyclic variations in lipins of corpus luteum.** S. Weinhouse and J. I. Brewer (*J. Biol. Chem.*, 1942, 143, 617—623).—Free cholesterol

is const. throughout all stages but cholesteryl esters and glycerides decrease with increasing activity of the gland and exhibit a marked increase during regression. Phospholipins reach a max. at max. activity and decline slightly during regression; no changes occur in the relative proportions of lecithin, kephalin, or sphingomyelin.

H. G. R.

**Patterns of uterine motility.** W. Bickers and R. J. Main (*J. clin. Endocrinol.*, 1941, 1, 992—995).—Uterine motility was assessed in women by records from an intrauterine balloon. The normal uterus shows contractions of low amplitude, high frequency, and tetanic in appearance during the follicular phase of the cycle. During the luteal phase the contractions are of higher amplitude and lower frequency without the tetanic appearance. Follicular type of contractions persist throughout the cycle in cases of anovulatory bleeding. The uteri of a castrate woman and of a case of missed abortion were quiescent and unresponsive to pituitrin; motility and responsiveness were restored by oestrogen therapy. Coitus temporarily abolished the intrinsic motility of the uterus.

P. C. W.

**Study with radioactive phosphorus on permeability of rat placenta to phospholipin.** P. E. Nielson (*Amer. J. Physiol.*, 1942, 135, 670—675).—With radioactive P as indicator, the tagged phospholipin content of the foetus in pregnant rats was studied in the last 8 days of gestation. Foetal phospholipin had a much higher ratio when inorg. <sup>32</sup>P was injected into the mother than when tagged phospholipin was injected. The placental transfer of the phospholipin mol. as such is extremely slow.

M. W. G.

**Hormonal control of deciduomata and metrial glands.** H. Selye, A. Borduas, and G. Masson (*Anat. Rec.*, 1942, 82, 199—209).—Deciduomata produced by uterine trauma in spayed animals pretreated with progesterone are not maintained even when large doses of the hormone are given. Degeneration of the deciduoma continues until only a narrow band of cells in the vicinity of the mesometrial side of the uterine wall remains. During involution the metrial gland forms in the uterine wall. Progesterone produces proliferation of metrial gland tissue and considerably prolongs its life span. Oestradiol hastens involution of deciduomata and metrial gland tissue. The development of the mammary gland in the spayed rat is greatly stimulated by progesterone but this hormone elicits no milk secretion. Small doses of oestradiol in combination with progesterone have a lactagogue effect.

W. F. H.

**Effects of hypophysectomy at mid-pregnancy in mouse.** W. U. Gardner and E. Allen (*Anat. Rec.*, 1942, 83, 75—97).—The mice recovered quickly, increased in wt., and the majority delivered normal litters. Corpora lutea compared favourably with those of normal mice at the end of gestation; the presence of a pituitary luteotrophin is therefore not essential. The adrenal glands exhibited marked cortical atrophy but there was no evidence of cortical deficiency during pregnancy. Ova which would have ovulated normally after parturition underwent early maturation stages. Ovarian interstitial tissue regressed. The onset of lactation was as in the normal animal. Resorption of the medial ends of pubic rami and replacement of the symphysis by an interpubic ligament proceeded as in normal controls.

W. F. H.

**Effects of testosterone propionate and stilbœstrol on mammary gland postpartum.** E. M. Jeppson, H. Y. Kasabach, and A. E. Kanter (*J. clin. Endocrinol.*, 1942, 2, 16—18).—Results of treatment in 124 patients are analysed. Testosterone propionate had no beneficial effects in suppressing painful engorgement of the breasts during the puerperium and often delayed the onset of lactation. Both compounds were effective in suppressing lactation.

P. C. W.

**Experimental oestrogen control of prolonged pregnancy in lactating rat.** C. K. Weichert (*Anat. Rec.*, 1942, 83, 1—17).—When oestrogen (theelin in oil) was injected into inseminated lactating rats suckling young, the blastocysts implanted and developed in 68% just as in normal non-lactating pregnant rats. It is suggested that in prolonged pregnancy, oestrogens essential for the prep. of the endometrium are removed through the mother's milk.

W. F. H.

**Initiation of lactation at parturition. I. Can oestrogen suppress the pituitary lactogenic hormone? II. Why lactation is not initiated during pregnancy. III. Can oestrogen account for sudden increase in lactogen content of pituitary following parturition?** J. Meites and C. W. Turner (*Endocrinol.*, 1942, 30, 711—718, 719—725, 726—733).—Pituitary lactogen is increased in rats and guinea-pigs by large or small doses of diethylstilbœstrol. In parturient rats which do not suckle their young it is diminished by 50% as compared with controls. Pituitary lactogen is also increased by œstrone, but if progesterone is given at the same time no such increase takes place. Oestron injections cause in male guinea-pigs an increase in pituitary lactogen, max. if injections are spread over 5 days. Lactogen increase at parturition is due to the presence of large amounts of oestrogen + disappearance of progesterone. Lactogen of rabbit pituitary is increased 83% by hysterectomy on the 20th day of pregnancy, with resulting degeneration of corpora lutea.

V. J. W.

**Inhibition of ovary or testis in immature *Amblystoma* by another gonad homotypic as to sex.** R. R. Humphrey (*Amer. J. Anat.*, 1942, 70, 345—358).—*A. punctatum* hosts carrying large gonads derived from *A. tigrinum* preprimordia exhibit marked reduction in size of both testis and ovary in the presence of a homotypic gonad. It is concluded that the growth of one gonad in the immature animal is inhibited by the presence of the other gonad. The inhibition of gonads by their homotypes may be brought about by the same inductor substances or hormones which cause inhibition of a heterotypic gonad.

W. F. H.

**Amount and distribution of cytochrome oxidase in bull spermatozoa.** C. A. Zittle and B. Zitin (*J. Biol. Chem.*, 1942, 144, 99—104).—The average O<sub>2</sub> consumption of spermatozoa disintegrated by sonic treatment in a *p*-phenylenediamine or Na succinate substrate in presence of excess of cytochrome-*c* is 25 cu. mm. per mg. per hr., the heads, midpieces, and tails giving vals. of 1.2, 14.4, and 29.1, respectively. With quinol as substrate the O<sub>2</sub> consumption is lower, being negligible with ground spermatozoa. 5.1 cu. mm. per mg. per hr. is consumed by sonically treated spermatozoa with *p*-phenylenediamine but no added cytochrome-*c*.

H. G. R.

**Non-haemin and total iron in bull spermatozoa.** C. A. Zittle and B. Zitin (*J. Biol. Chem.*, 1942, 144, 105—112).—Dried, lipin-free spermatozoa contain approx. 0.0071% of Fe, the greatest concn. being found in the midpieces and tails. Non-haemin-Fe accounts for 60% of the total Fe and cytochrome-*c* could not be detected spectroscopically.

H. G. R.

**Creatine retention capacity of boys in relation to androgen function.** D. A. Duckworth (*J. clin. Endocrinol.*, 1942, 2, 13—15).—Tests were performed on 18 boys 5—15 years of age. 24-hr. samples of urine were analysed for creatinine and creatine under normal conditions and after the ingestion of 2 g. of creatine hydrochloride. The creatine retention test was repeated after a course of thrice-weekly injections of 200 r.u. of chorionic gonadotropin (total dose 1800—8800 r.u.) or of 25 mg. of testosterone propionate thrice weekly (total dose 300—585 mg.). Under control conditions 67% of the ingested creatine was excreted in 24 hr. and 99% in 48 hr. Neither course of injections affected these figures nor did they alter the normal excretion of creatinine.

P. C. W.

**Effects of testosterone propionate in spayed female rats.** G. L. Laqueur (*Proc. Soc. Exp. Biol. Med.*, 1942, 49, 425—426).—In spayed rats the genital tissues react to testosterone as they do in normal rats in late diœstrus.

V. J. W.

**Effects of postnatal androgenic treatment in female rat.** R. R. Greene, M. W. Burrill, and A. C. Ivy (*Anat. Rec.*, 1942, 83, 19—29).—The ovaries were small and the oviducts had fewer convolutions than normal and were markedly increased in diameter. The uteri in all animals were much enlarged and coiled. Increase in diameter of the uteri was due in part to distension by contained fluid; the latter was clear in some, in others turbid, and in a few pus occurred. The ovaries contained only primordial and secondary follicles, and ova in the latter were usually degenerated. Rete and tubules were more prominent than in normal ovaries of the same age. No epididymal structures were found.

W. F. H.

**Effect of esterified androgen on sex eminence of chick.** I. L. Kossin (*Endocrinol.*, 1942, 30, 767—772).—Intramuscular injections totaling 4—6 mg. stimulated growth of the sex eminence in both sexes.

V. J. W.

**Factor of age in rate of absorption of, and mammary stimulation by, testosterone monopropionate pellets in rats.** T. R. Forbes (*Endocrinol.*, 1942, 30, 765—766).—Absorption during 12 days decreased from 33% when implant was made at the 16th day to 26% when implant was at the 47th day. Mammary stimulation only took place when the pellet was present after puberty.

V. J. W.

**Effect of œstrous cycle on action of testosterone propionate on organ and body weights of female rats.** W. Schilling and G. L. Laqueur (*Endocrinol.*, 1942, 30, 753—760).—In œstrus testosterone propionate increases wt. of kidneys, ovaries, thyroid, and uterus and decreases wt. of adrenals. Liver is not affected. In diœstrus kidney wt. is increased as in œstrus, but wts. of other organs are all decreased. Body wt. was increased more in diœstrus.

V. J. W.

**Conditions modifying effectiveness of testosterone, testosterone propionate, and methyltestosterone.** R. R. Greene, M. W. Burrill, E. Oppenheimer, and D. Nelson (*Endocrinol.*, 1942, 30, 734—740).—All 3 steroids were most effective as implanted pellets, and, if painted on the skin, were more effective in alcoholic than in oily solution. Results of subcutaneous injection in these solvents varied with the hormone used and are tabulated. (Cf. A., 1942, III, 390.)

V. J. W.

### XIII.—DIGESTIVE SYSTEM.

**Mechanism of deglutition.** A. R. Thomas (*Brit. J. Radiol.*, 1942, 15, 209—210).—Progress of a Ba-swallow was observed in a man standing on his head and showed no difference from that in the

normal position: the meal "rose" rapidly up to the cardia followed by a column of air, suggesting a mechanism of suction. E. M. J.

**Reaction and neutralising ability of contents of the pyloric antrum and first part of duodenum in normal dogs fed an Ewald meal.** J. E. Berk, J. E. Thomas, and M. E. Rehfuess (*Amer. J. Physiol.*, 1942, **136**, 157—166).—There is a difference in acidity, as indicated by  $p_H$  measurement, of samples collected simultaneously from just below or above the pylorus in normal dogs fed an Ewald meal (stale bread and 250 c.c. of water). Acidity of intestinal contents is less after this meal than after meat. Although titratable total acidities follow a parallel course in the antrum and duodenal bulb,  $p_H$  vals. in both regions vary independently of these and each other; variations in titratable free acid in the stomach are not regularly accompanied by corresponding changes in duodenal  $p_H$ . The acidity of the duodenal contents in the normal dog bears little relation to the acidity (in  $p_H$  units) of the gastric contents. Free acid is usually absent in the contents of the 1st part of the duodenum; acid produced in the stomach is quantitatively buffered in the pyloric antrum and as it enters the duodenum it is quickly diluted and partly neutralised. The 1st part of the duodenum is equipped with a remarkable neutralising, buffering, and diluting capacity in excess of its physiological needs. M. W. G.

**Gastric inhibition caused by amino-acids in small intestine.** J. E. Thomas (*Amer. J. Physiol.*, 1942, **135**, 609—613).—Monoamino-monocarboxylic acids caused gastric inhibition regularly when administered in neutral solution in the small intestine of unanesthetised fistula dogs. The inhibitory effect of these acids was roughly proportional to their mol. wts., but it was also influenced by other factors. Phenylalanine, tyrosine, and tryptophan showed the most pronounced inhibitory effect of these acids. Dicarboxylic and diamino-acids caused gastric inhibition when administered as free acids without neutralisation; they were ineffective in neutral solution. M. W. G.

**Experimental peptic ulceration by vasomotor episodes [from pitressin] (pitressin episodes) and autonomic disturbances.** M. Berg (*Arch. Path.*, 1942, **33**, 636—645).—A single injection of pitressin (4 units per kg.) produces in the stomach vascular contraction and relaxation with scattered superficial erosions or localised areas of oedema which occur with about the same extent and frequency in normal and in vagotomised dogs. In sympathetomised animals, the reaction is diminished, and erosions or localised areas of oedema are infrequent. Injections of pitressin produce, first, inhibition of gastric and duodenal peristalsis. This period of inhibition is shortest in vagotomised dogs and longest in sympathetomised dogs, normal dogs occupying an intermediate position. This is followed by severe gastric peristalsis in the normal and the vagotomised dogs. In the normal dogs occasional reverse peristalsis is seen but not in dogs with vagus nerves sectioned. With frequent injections of pitressin, lesions of the stomach are obtained in normal and to a greater extent in vagotomised animals but not in sympathetomised animals. C. J. C. B.

**[Treatment of] bleeding peptic ulcer.** N. W. Chaikin and O. Tannenbaum (*Amer. J. digest. Dis.*, 1942, **9**, 150—151).—56 cases of bleeding peptic ulcer were treated by starvation and supportive measures and 6.9% died. In 30 similar cases on the Meulengracht regime there was only one death. N. F. M.

**Alkalosis in peptic ulcer therapy.** J. B. Kirsner and W. L. Palmer (*Arch. intern. Med.*, 1942, **69**, 789—807).—Alkalosis, diagnosed by electrolytic changes in the serum, was found in 111 patients with peptic ulcer treated by the Sippy diet, sometimes without symptoms. It may occur in patients with normal renal function but is more liable to develop if renal function is impaired, or after excessive loss of gastric juice. The decreased renal function, estimated by the urea clearance test, which alkalosis produces is usually only temporary but may persist for several months. C. A. K.

**Relationship between blood-sugar and general complaints following subtotal gastric resection.** A. Schwarz, I. Reingold, and H. Necheles (*Amer. J. digest. Dis.*, 1942, **9**, 151—154).—There was no relationship and the symptoms are ascribed to distension of the upper small intestine. N. F. M.

**Gastric mucosa in benign adenomas.** R. Schindler (*Amer. J. digest. Dis.*, 1942, **9**, 149—150).—Analysis of 2167 gastroscopies, 36 being cases of benign gastric polyps, showed that the condition was 7 times as frequent in stomachs with atrophic gastritis as in stomachs without atrophic gastritis. N. F. M.

**Total gastrectomy.** R. C. Lynch and J. T. Priestley (*Proc. Staff Mayo Clin.*, 1941, **16**, 653—656).—Case report. H. H. K.

**Is beneficial effect of urine extracts on Mann-Williamson ulcers due to gastric secretory depressant in urine?** D. J. Sandweiss, M. H. F. Friedman (*Amer. J. digest.*, 1942, **9**, 166—168).—No; the effect is due to stimulation of fibroblastic proliferation. N. F. M.

**Prolongation of survival time in Mann-Williamson dogs by supplementing diet with amino-acids.** D. Shock and S. J. Fogelson (*Amer.*

*J. digest. Dis.*, 1942, **9**, 173—176).—Feeding pre-digested protein prolonged survival and delayed the appearance of the ulcers. N. F. M.

**Active pancreatic secretion in the aged.** H. Necheles, F. Plotke, and J. Meyer (*Amer. J. digest. Dis.*, 1942, **9**, 157—159).—The  $HCO_3^-$  and the amylase in pancreatic juice were increased in the aged, trypsin and vol. of juice were normal, and lipase was 21% lower than in the control (young) group. N. F. M.

**Effect of magnesium sulphate on sphincter of Oddi in man.** G. S. Bergh and J. A. Layne (*Amer. J. digest. Dis.*, 1942, **9**, 162—165).—The effects were very variable and relaxation was less const. than with amyl nitrite or a fatty meal. N. F. M.

**Effects of trauma and traumatic shock on gastro-intestinal motility and secretions.** H. Necheles and W. H. Olson (*Amer. J. Physiol.*, 1942, **136**, 32—37).—Dogs (pentobarbital or ether) were used and blows administered to one or both hind legs. Stimulated salivary and pancreatic secretions were not much affected; biliary secretion decreased. Gastric secretion was not affected. Effects on gastric motility, intra-abdominal pressure, and respiration were slight and variable. The effects of mechanical and thermal trauma were very different. M. W. G.

**Nervous pathways for reflex regulation of intestinal pressure.** W. B. Youmans, A. I. Kerstens, and R. W. Aumann (*Amer. J. Physiol.*, 1942, **135**, 619—627).—The immediate nervous inhibition of one segment of intestine following the sudden production of a pressure of 40 mm. Hg or more in the other segment in unanesthetised dogs each having 2 high jejunal fistulae is abolished by sympathectomy; this intesto-intestinal inhibitory reflex is not altered by vagotomy and is not mediated through the decentralised pre-aortic ganglia. A powerful contractile response is induced by sudden distension of the sympathetically denervated intestine, whether the vagi are intact or not. The irregularity of the motility of the sympathetically denervated intestine is discussed. The regulation of intestinal motility by its extrinsic nerves consists, in part, of reflex inhibition of the intestine as a result of stimuli arising from excessively strong intestinal contractions. M. W. G.

**Utilisation of blood-oxygen by distended intestine.** H. Lawson and A. M. Ambrose (*Amer. J. Physiol.*, 1942, **135**, 650—659).—Moderate distension (distending pressure 30 cm. water) of barbiturised dog's ileum or jejunum raises the  $O_2$  content of venous blood returning from the loop; arterio-venous  $O_2$  difference falls to one half. Vol. flow of blood (after initial brief reduction) is little changed.  $O_2$  consumption of intestine is reduced. Treating intestine with cocaine during distension reduced  $O_2$  content of mesenteric venous blood.  $O_2$  consumption is unchanged or reduced slightly. After section of mesenteric nerves, distension of the untreated intestine has no consistent effect on  $O_2$  content of mesenteric venous blood or  $O_2$  consumption. M. W. G.

**Sudden compression injuries of abdomen.** N. P. Breden, A. L. d'Abreu, and D. P. King (*Brit. Med. J.*, 1942, **1**, 144—146).—Sub-peritoneal and submucous haemorrhages and lacerations of the intestine occurred in 10 patients after exposure to sudden compression waves from torpedo or depth-charge explosions. C. A. K.

**Effects of bile and bile salts on absorption of sodium oleate from jejunal loops of dogs.** R. W. Virtue, M. E. Doster-Virtue, D. I. Smith, and J. Greenblatt (*Amer. J. Physiol.*, 1942, **135**, 776—780).—Bile (but not Na taurocholate and/or Na glycocholate) increased absorption of Na oleate from the small intestine. M. W. G.

**Familial occurrence of chronic ulcerative colitis (thrombo-ulcerative colitis).** R. J. Jackman and J. A. Barger (*Amer. J. digest. Dis.*, 1942, **9**, 147—149).—In 1.8% of 900 cases the disease occurred in more than one member of a family. N. F. M.

**Familial polyposis of colon.** V. S. Falk (*Arch. Surg., Chicago*, 1942, **45**, 123—128).—In a family of 7 children 3 males and 3 females had polyposis; their ages were from 10 to 30 years (average 19.7). The unaffected male was 18 years old. The two eldest, males of 27 and 30 years, had already developed rectal carcinoma. F. S.

**Occult blood: use of carmine for marking stools.** M. Kirschen, H. Sorter, and H. Necheles (*Amer. J. digest. Dis.*, 1942, **9**, 154—156). 146 "normal" persons were given 1.5—2 g. of haemoglobin by mouth. 41% had positive occult blood in the faeces (benzidine slide method) and 59% gave negative results. Carmine may yield false positives. N. F. M.

**Food remnants as cause of confusion in diagnosis of intestinal parasites.** N. Denison (*J. Lab. clin. Med.*, 1942, **27**, 1036—1042). C. J. C. B.

#### XIV.—LIVER AND BILE.

**Liver and biliary tract.** C. H. Greene (*Arch. intern. Med.*, 1942, **69**, 691—714).—A review of literature for 1941. C. A. K.

**Influence of detergents on physiological phenomena, especially stellate cells of frog liver.** R. Höber and J. Höber (*J. Gen. Physiol.*,



1942, 25, 705—715).—The cytolytic properties of detergents are discussed. The cytolytic activity of some Na alkyl sulphates and sulphosuccinates and bile salts is determined by liberation of hæmoglobin from erythrocytes or by the development of an injury potential in muscle. The hæmolytic and myolytic activities of the detergents follow fairly closely their surface activities. The stellate cells of frog liver perfused with Ringer's solution do not take up colloidal dyes except after addition of a small amount of serum to the perfusing solution, and under these conditions uptake of dye is increased in presence of a detergent. This is probably due to a combined action of protein and detergent. Relatively high concns. of detergents cause cytolysis of stellate cells, whilst small concns. increase the functional activity. J. N. A.

**Effect of dietary choline, ethanolamine, serine, cystine, homocysteine, and guanidoacetic acid on liver-lipins of rats.** D. Stetten, jun., and G. F. Graff (*J. Biol. Chem.*, 1942, 144, 175—181).—The amounts of liver-lipin and its contents of N, P, and choline have been determined in rats on various levels of choline nutrition and with addition of serine, ethanolamine, cystine, homocysteine, and guanidoacetic acid to the diet. With deficiency of dietary choline, the liver-fat is poor in lecithin, whilst after feeding cystine and homocysteine the fat is extremely rich in lecithin. Ethanolamine and serine produce no great increase in liver-lipin, but cause a rise in monoaminophosphatides in this organ. Feeding guanidoacetic acid produces very fatty livers the lipins of which contain only very small amounts of choline. A hypothesis is suggested relating these latter findings to the irreversible biological methylation of guanidoacetic acid. J. N. A.

**Unsaponifiable matter. II. From foetal livers and from the livers of adults.** A. Dimter (*Z. physiol. Chem.*, 1941, 271, 293—312).—*Hepene*, which in many respects resembles squalene, is invariably present in the livers of adult mammals but not in foetal livers, which are characterised by a much higher cholesterol content. It gives a hydrochloride,  $C_{45}H_{76} \cdot 8HCl$ , m.p. 127°, and a dodecylbromide,  $C_{45}H_{76}Br_{12}$ . It is found solely in the liver and is very probably an intermediate in the production of cholesterol; this rôle cannot be ascribed to squalene, which is not present in liver. The lipochrome and, almost invariably, the vitamin-A content is greater in the adult than in the foetal liver. H. W.

**Liver fats and glycogen of hypophysectomised rats on high-carbohydrate and high-fat diets.**—See A., 1942, III, 686.

**Hepatic function in thyrotoxicosis.**—See A., 1942, III, 684.

**Liver function in hepatolenticular degeneration.**—See A., 1942, III, 673.

**Value of hippuric acid test and Takata-Ara reaction in investigation of hepatic deficiency.** M. Henderson and B. Splatt (*Med. J. Austral.*, 1942, I, 185—195).—From 313 tests in 240 adult subjects it is concluded that Quick's hippuric acid excretion test is a reliable and sensitive index of liver function. From tests on the serum of 143 patients and 50 normal subjects it is concluded that the Takata-Ara reaction is practically valueless as a test of liver function, but is a useful confirmatory test for parenchymatous liver disease. F. S.

**Coproporphyrin excretion in hepatic disease.** S. Nesbitt and A. M. Snell (*Arch. intern. Med.*, 1942, 69, 573—588).—In patients with severe parenchymatous liver damage there was an increased excretion of coproporphyrin in the urine and a diminished excretion in the faeces. The urinary excretion was raised from normal vals. of 100—200 µg. per 24 hr. to 300—600 µg. in severe cases. Similar changes occurred in cases of biliary obstruction and normal vals. were then restored after operation. C. A. K.

**Histogenesis and repair of hepatic cirrhosis in rats.** E. D. Lillie, L. L. Ashburn, W. H. Sebrell, F. S. Daft, and J. V. Lowry (*U.S. Publ. Health Repts.*, 1942, 57, 502—508).—In rats low-protein, choline-poor diets produce hepatic cirrhosis characterised by fatty infiltration and by the appearance of a hyaline substance in liver cells and various phagocytic cells in the liver and in other viscera, notably spleen and lungs. Correction of the major dietary defects results in prompt regression of the fatty changes and increases in size and no. of liver cells. C. G. W.

**Liver-catalase activity of tumour-bearing mice.**—See A., 1942, III, 615.

**Pathogenesis of cholecystitis.** N. A. Womack (*Arch. Surg.*, Chicago, 1942, 44, 658—676).—In dogs, complete obstruction of the cystic duct produces no inflammation if the imprisoned bile is replaced by saline. If the bile is left there ensues inflammation, the severity and type of which are in proportion to the amount and concn. of the bile obstructed. (14 photomicrographs.) F. S.

**Bile acid metabolism. I. Fate of cholic acid in the guinea-pig.** L. H. Schmidt and H. B. Hughes (*J. Biol. Chem.*, 1942, 143, 771—783).—Cholic acid injected intravenously is excreted via the bile. It was recovered from the functionally inactive caecum, and its destruction was observed in the isolated caecum. This is attributed to *Alcaligenes faecalis* or a similar organism. R. L. E.

## XV.—KIDNEY AND URINE.

**Site of renin formation in the kidney.**—See A., 1942, III, 580.

**Blood-pressure-reducing property of extracts of kidneys.**—See A., 1942, III, 668.

**[Renal changes following] blood transfusion.**—See A., 1942, III, 662.

**Relation of kidney to cardiovascular disease.**—See A., 1942, III, 666.

**Hyperparathyroidism in experimental nephritis.**—See A., 1942, III, 595.

**Kidney damage correlated with inadequacy of pantothenic acid.**—See A., 1942, III, 621.

**Renal lesions following intravenous hypertonic sucrose.** R. H. Rigdon and E. S. Cardwell (*Arch. intern. Med.*, 1942, 69, 670—690).—The kidneys were studied histologically in 16 patients who died of various diseases after hypertonic solution had been given intravenously. Dehydration and the quantity of sucrose injected were the most important factors in producing the swelling of the epithelium of the convoluted tubules. Similar renal lesions may be produced in rabbits. C. A. K.

**Crush injury with renal failure.** E. J. Bradley (*Brit. Med. J.*, 1942, I, 294).—Case report, with recovery. C. A. K.

**Effect of denervation on filtration rate and blood flow in dog kidneys rendered hyperæmic by administration of pyrogen.** E. P. Hiatt (*Amer. J. Physiol.*, 1942, 136, 38—41).—The action of pyrogenic insulin in causing renal hyperæmia in dogs (ureters explanted) is not affected by sympathetic denervation of the kidney. M. W. G.

**Essential fructosuria.** B. Sachs, L. Sternfeld, and G. Kraus (*Amer. J. Dis. Child.*, 1942, 63, 252—269).—2 Jewish brothers, 6 and 9 years old, with essential fructosuria are described. Following 50 g. of fructose by mouth there was a rise in blood-lactic acid in 2 controls and none in the cases. The fructosuria may result from ingested fructose not being broken down to lactic acid, as it is in normal subjects. C. J. C. B.

**Micturition in crayfish.** N. S. R. Maluf (*Biol. Bull.*, 1941, 81, 134—148).—Ureteral fibres are described as a probable sphincter. Urine is discharged by a local rise in the hæmocœlic pressure. Secretion from the coelomosac by the shedding of complete cells is negated by the absence of mitoses. D. M. SA.

**Creatinine excretion in women.** O. W. Smith (*J. clin. Endocrinol.*, 1942, 2, 1—12).—The 24-hr. urinary excretion of preformed creatinine was determined frequently in 35 women; the patients were normal, abnormal undergoing sex hormone treatment, pregnant, in labour, or had pre-eclampsia. Simultaneously oestrogen excretion was determined and occasionally in the pregnant cases urinary gonadotropin and pregnanediol were assayed. There was a rise in urinary creatinine excretion during the second half of the normal menstrual cycle and a fall preceded or accompanied the onset of menstrual flow. The preformed creatinine in the urine rose during the latter fall. Since these changes were also seen during anovulatory cycles they are attributed to changes in water metabolism and vascularity accompanying menstruation rather than as a direct result of ovarian secretion. A post-menstrual rise in creatinine excretion was found in some normal cases. There was a rise in creatinine excretion during labour but otherwise there were no consistent changes under any of the conditions or treatments examined. P. C. W.

**Determination of urinary cholesterol.** M. Bruger and S. B. Ehrlich (*J. Lab. clin. Med.*, 1942, 27, 1093—1095).—A colorimetric procedure employing ovalbumin to bind the cholesterol is described. C. J. C. B.

**Sugar of normal urine.**—See A., 1942, III, 645.

## XVI.—OTHER ORGANS, TISSUES, AND BODY-FLUIDS.

**Phenol studies.** W. Deichman and L. J. Schafer (*Amer. J. clin. Path.*, 1942, 12, 129—143).—The phenol content (in mg.-%) of normal human tissues and fluids (spectrophotometric determination) was: for blood: 0.0—0.08 free phenol; 0.00—0.08 conjugated, 0.0—0.08 total; for sweat the corresponding vals. were 0.09—0.44, 0.04—0.11, 0.16—0.55; saliva: 0.11—0.23, 0.07—0.15, 0.28—0.37; urine: 0.0—3.8, 10—39, 13—42 for a 24-hr. specimen; faeces 1.9—4.2 total phenol. C. J. C. B.

**Echinoderm pigments.** D. L. Fox and B. T. Scheer (*Biol. Bull.*, 1941, 80, 441—455).—Various species were tested and carnivorous species yielded three times as much carotenoids as did herbivorous. Echinochromes are discussed. D. M. SA.

**Macromolecular substances (proteins, carbohydrates, lipins) as pathogenic agents.** W. C. Hueper (*Arch. Path.*, 1942, 33, 267—290).—A general review. C. J. C. B.

**Tryptophan-containing acid hydrolysates of proteins suitable for intravenous administration.** A. White and R. Elman (*J. Biol. Chem.*, 1942, 143, 797—798).—Hydrolysis for 6 hr. with 2.6N-H<sub>2</sub>SO<sub>4</sub> liberates 60% of the casein-amino-N and 55% of the globulin-amino-N given by 24 hr. hydrolysis with 8N-H<sub>2</sub>SO<sub>4</sub>, with 65% yield of tryptophan.  
R. L. E.

## XVII.—TUMOURS.

**Examination of dimethylanthracenes for carcinogenic properties.** E. L. Kennaway, N. M. Kennaway, and F. L. Warren (*Cancer Res.*, 1942, 2, 157—159).—Five dimethylanthracenes (1:2-, 1:3-, 1:4-, 2:3-, and 9:10-) were tested for carcinogenic activity by application to the skin, and by subcutaneous injection and implantation, in mice. The 9:10-compound alone produced epithelioma of the skin, and adenomas of the lung were most abundant in the mice receiving this compound. None of the 5 compounds produced tumours of the subcutaneous tissue.  
F. L. W.

**Effect of solvents in methylcholanthrene epidermal carcinogenesis.** R. E. Stowell and W. Cramer (*Cancer Res.*, 1942, 2, 193—197).—Epidermal carcinogenesis was obtained somewhat more rapidly with acetone than with benzene as a solvent for 0.3% methylcholanthrene. Mice painted with 0.3% methylcholanthrene in acetone thrice weekly and with pure benzene on alternate days developed cancer even more rapidly. 56% of the 123 mice thus painted had multiple malignant neoplasms of the skin.  
F. L. W.

**Additive effects of carcinogenic hydrocarbons.** P. S. Lavik, P. R. Moore, H. P. Rusch, and C. A. Baumann (*Cancer Res.*, 1942, 2, 189—192).—Of 586 mice which developed tumours as a result of the application of methylcholanthrene for 2 months, 92% did not develop them until after the carcinogenic treatment had been discontinued. Methylcholanthrene was applied to the skin of mice in amounts insufficient to produce tumours in the majority of the animals, and methylcholanthrene, benzopyrene, dibenzanthracene, or benzantracene was then applied for relatively short periods. The hydrocarbons exerted additive effects equiv. to their carcinogenic potencies. When methylcholanthrene was applied for 2 months and then re-painted for 1 month after an intermission of 4 months many tumours developed.  
F. L. W.

**Non-additive effect of ultra-violet light and other carcinogenic procedures.** H. P. Rusch, B. E. Kline, and C. A. Baumann (*Cancer Res.*, 1942, 2, 183—188).—The carcinogenic process initiated by the application of benzopyrene, methylcholanthrene, or 9:10-dimethyl-1:2-benzanthracene to the backs of mice can be completed by either of the other two hydrocarbons. Ultra-violet light did not increase the no. of tumours produced in mice rendered precarcinogenic with methylcholanthrene or 9:10-dimethyl-1:2-benzanthracene, and the hydrocarbons did not increase the no. of tumours in animals previously irradiated with ultra-violet light. Ultra-violet irradiation and X-rays failed to increase the carcinogenicity of the Shope papilloma virus.  
F. L. W.

**Absence of carcinogenicity of cod-liver oil concentrate.** P. E. Steiner (*Cancer Res.*, 1942, 2, 181—182).—A cod-liver oil concentrate prepared by saponification and extraction with ethylene dichloride was not carcinogenic when injected into mice.  
F. L. W.

**Choline in tumour-bearing animals and a choline-like effect of butter-yellow.** H. P. Jacobi and C. A. Baumann (*Cancer Res.*, 1942, 2, 175—180).—Less than half of rat tumour phospholipins are present as lecithin. Tissues of tumour-bearing animals contain normal amounts of free and bound choline. Neither deficiency nor excess of choline affected the development of the tumours. Addition of butter-yellow to a choline-free diet prevented kidney lesions in young rats.  
F. L. W.

**Effect of carcinogens on hepatic vitamin-A stores of mice and rats.** C. Carruthers (*Cancer Res.*, 1942, 2, 168—174).—Repeated biweekly intraperitoneal injections of lard solution of methylcholanthrene for 18 weeks in mice did not affect hepatic stores of vitamin-A. Sarcomas and ascites were induced. In rats methylcholanthrene and 3:4-benzopyrene reduced hepatic -A. Phenanthrene produced some reduction. With the carcinogens the depletion of -A was greater than the inhibition of growth. The storage or assimilation of the vitamin in mice receiving -A orally was not affected by simultaneous injection of phenanthrene, 3:4-benzopyrene, methylcholanthrene, or 1:2:5:6-dibenzanthracene.  
F. L. W.

**Influence of toxic amounts of oestrin on intact and castrated male Marsh-Buffalo mice.** F. Bischoff, M. L. Long, J. J. Rupp, and G. J. Clarke (*Cancer Res.*, 1942, 2, 198—199).—Oestradiol in sesame oil was administered in a total dose of 3.5 mg. per mouse intermittently for 12 months to castrated and intact male Marsh-Buffalo mice. In the castrated mice the incidence of lymphosarcoma was greater than that occurring in oestrin-treated or control intact mice. No mammary tumours occurred in intact males and only 6% (2 tumours) in the castrated group.  
F. L. W.

**Quantitative aspects of antifibromatogenic action of synthetic deoxycorticosterone acetate.** A. Lipschutz, J. V. Luco, and J. Zanartu (*Cancer Res.*, 1942, 2, 200—203).—When tablets or pellets of

oestradiol and synthetic deoxycorticosterone acetate are implanted simultaneously under the skin of the female guinea-pig, the fibromatogenic action of the oestrogen can be inhibited by equimol. quantities of the adrenal cortical steroid.  
F. L. W.

**Relation of the antifibromatogenic activity of certain steroids to their molecular structure and to various actions of these hormones.** A. Lipschutz, O. Vera, and S. Gonzalez (*Cancer Res.*, 1942, 2, 204—209).—The antifibromatogenic threshold was lowest with progesterone, deoxycorticosterone was next, testosterone was highly active but less than progesterone, and deoxycorticosterone and androstenediol had no antifibromatogenic action.  
F. L. W.

**Occurrence of crystalline material in lungs of normal and cancerous Swiss mice.** E. U. Green (*Cancer Res.*, 1942, 2, 210—217).—The crystals in the lung tissue of Swiss mice are of two main types, large extracellular plates and small plates within the cytoplasm of the "dust cells." Measurement of angles indicates the crystal nature of the plates, which consist of protein. Incidence of crystals is correlated with age and with vol. of lung tumour. Injection of 1:2:5:6-dibenzanthracene induces lung tumours and high crystal counts at an earlier age in control mice. Incidence of crystals in other strains of mice does not show a positive correlation with incidence of lung tumours.  
F. L. W.

**Dusts and lung tumours in mice.** J. A. Campbell (*Brit. Med. J.*, 1942, I, 217—221).—The incidence of primary lung tumours in mice was observed after intermittent exposure, for up to 1 year, to inhalation of pptd. SiO<sub>2</sub> mixture containing methylcholanthrene, steel grindings, a mixture of Al<sub>2</sub>O<sub>3</sub> + pptd. SiO<sub>2</sub> + brown Fe oxide or the same + CaCO<sub>3</sub>, a radioactive dust from Czechoslovakia, or a sample of tarred road dust. All dusts increased the incidence of tumours by about three times (average). Attempts to inhibit lung tumours by Al<sub>2</sub>O<sub>3</sub> or CaCO<sub>3</sub> were unsuccessful. The dusts increased the incidence of hyperplasia of lymph tissue in the lungs and in the tracheo-bronchial glands. Silicotic nodules were produced in these glands by inhalation of amorphous SiO<sub>2</sub>.  
C. A. K.

**Stimulating action of nucleic acids on mouse heart fibroblasts.** R. Tennant, K. G. Stern, and A. A. Liebow (*Cancer Res.*, 1942, 2, 218—222).—Na nucleates from various sources increase the expansion rates of mouse heart fibroblasts at concn. in the medium of about 1 in 50,000.  
F. L. W.

**Hæmoglobin level and tumour growth.** A. Taylor and M. A. Pollack (*Cancer Res.*, 1942, 2, 223—227).—In mice implanted with tumours the hæmoglobin level was depressed by the time the implant had reached measurable size and thereafter became progressively lower until death occurred. The precancerous condition induced by methylcholanthrene was associated with a gradual fall of hæmoglobin. Ingestion of butter-yellow by rats induced a sudden decrease in the hæmoglobin by the 30th day of treatment. Thereafter the hæmoglobin concn. remained const. until hepatomas developed, after which a further decline occurred. The hæmoglobin of these rats on a rice-carrot diet was lower (76%) than on a Purina dog Chow diet (83%).  
F. L. W.

**Endogenous production of carcinogenic substances in man.** W. Friedrich and N. Koyenuma (*Naturwiss.*, 1942, 30, 145—146).—Examination by the fluorescence-spectroscopic method of extracts of cultures of *B. coli* from the intestines of healthy persons and of persons suffering from cancer of the intestine shows that added deoxycholic acid and dehydronorcholene are not converted by the cultures into methylcholanthrene. The method permits the detection of less than 0.15 µg. of methylcholanthrene per c.c. of extract.  
W. McC.

**Deterrent effect of light on incidence of spontaneous breast cancer in strain A mice.** F. L. Apperly and M. K. Cary (*Brit. J. exp. Path.*, 1942, 23, 133—135).—By exposing strain A mice to ultra-violet light the cancer rate in breeding females was reduced from 80 to 41%.  
F. S.

**Influence of one growing tumour on another.** S. Russ and G. M. Scott (*Brit. J. exp. Path.*, 1942, 23, 127—133).—The growth of Jensen's rat sarcoma in rats in one flank was accelerated by inoculating in the other flank, grafts taken from a much more rapidly growing Jensen's rat sarcoma. An opposite effect was obtained by inoculating a slow growing tumour in the other flank.  
F. S.

**Tracer studies with radioactive phosphorus in malignant disease.** J. M. Kennedy, L. D. Marinelli, and H. Q. Woodard (*Radiology*, 1941, 37, 683—687).—About 0.5 microcurie (µc.) of <sup>32</sup>P (as 1.5—3.0% Na<sub>2</sub>HPO<sub>4</sub>) was administered preoperatively to patients with mammary carcinoma, osteogenic and lympho-sarcoma. Differential absorption ratios (µc. of <sup>32</sup>P per kg. tissue/µc. of <sup>32</sup>P given per kg. body-wt.) of malignant tissues were higher than in the corresponding normal tissue.  
E. M. J.

**Action of different dosage rates of X-radiation on growth factors of mouse sarcoma 180 grown in vivo following irradiation in vitro.** A. Goldfelder (*Radiology*, 1941, 37, 705—716).—Subcutaneous implants of mouse sarcoma 180 were excised after 8—10 days, fragmented, irradiated with X-rays at 200 and 45 kv. (½-val. layers of 0.85 mm. Cu and 0.28 mm. Al) and dosage rates of 40 and 640 and 365 and

5750 r. per min. respectively, and reimplanted by injection into other mice. Const. effects were seen above 1000 and 800 r. respectively; all tumours regressed if 2000 r. had been given by any means. Takes were lower and regression rates higher at the lower dosage rates. E. M. J.

**Macro- and micro-diagnosis of cancer.** E. K. Dawson and W. F. Harvey (*Edinb. Med. J.*, 1942, [iv], 49, 401—408).—In a series of 264 cases including all types of breast lesion removed at operation a correct macro-diagnosis was made in from 92% (local excision) to 100% (simple mastectomy). In 181 cases of breast tumour there was only one failure to make a correct pathological diagnosis. A method for the removal of suitable tissue is suggested. (7 photographs.) H. S.

**Tumour of sympathetic cells of the ovary causing virilism.** L. Berger (*Rev. Canad. Biol.*, 1942, 1, 539—566).—Sympathicotropic cells are morphologically identical with testicular Leydig cells. In the case reported removal of the tumour was followed by reduction of the virilism. (6 figures.) E. B.

**Simpler classification of mammary tumours.** N. C. Foot (*Arch. Path.*, 1942, 33, 905—916).—A general discussion. C. J. C. B.

**Effect of raised intracranial pressure on cerebral blood flow.**—See A., 1942, III, 579.

**Tumours of ciliary retina. Histogenesis of glioma retinae.**—See A., 1942, III, 681.

## XVIII.—NUTRITION AND VITAMINS.

**Growth curve of albino rat in relation to diet.** T. F. Zucker, L. Hall, M. Young, and L. Zucker (*J. Nutrition*, 1941, 22, 123—138).—The growth rate of rats on a stock diet was represented by the formula  $\log W = -(k/t) + \log A$  ( $t$  = time from weaning and  $A$  = wt. approached asymptotically in adults). Neither natural variation in size of animals nor artificially stimulated growth caused deviation from this formula. The val. of  $k$  is substantially the same for both sexes. A. G. P.

**Effect of diminishing the proportion of starch in the feed of laying hens.** G. D. Buckner, W. M. Insko, jun., and A. Harms (*Kentucky Agric. Exp. Sta. Bull.*, 1942, No. 423, 18 pp.).—Hens were fed a ration in which yellow maize was partly or completely replaced by distillers' maize dried grains, wheat feed, soya-bean oilmeal, dried skim milk, and meat scrap all containing little or no starch. The amount of feed consumed varied but the wt. of eggs, dried shell, white, wet and dried yolk, and of fat in the egg was approx. the same for each lb. of food consumed. Most fat was deposited in the bodies of the hens with the high-starch diet. Relations of diet with egg- and body-fat are discussed. A. W. M.

**Resistance to infection as affected by variations in the proportions of protein, fat, and carbohydrate in diet.** W. S. Sako (*J. Pediat.*, 1942, 20, 475—483).—Young mice fed for 6 weeks on special diets were inoculated with a standard multiple-lethal dose of virulent pneumococci. The diets represented all combinations of protein, fat, and carbohydrate. Animals on a very low protein intake showed decreased post-inoculation survival time; those with an excessively high protein intake showed lengthening of the survival time. High-carbohydrate feeding *per se* failed to reduce resistance if the protein content of the diet was 10% or over. C. J. C. B.

**Nutritive value of yeast-protein. Yeast-protein and caseinogen as supplements to maize-protein in diet of pig.** T. F. Macrae, M. M. El-Sadr, and K. C. Sellers (*Biochem. J.*, 1942, 36, 460—477).—The capacity of the non-autolysed proteins of pure cultures of *Torula utilis* to supplement the proteins of maize is approx. equal to that of caseinogen, the addition of yeast-protein or caseinogen in amounts equal to approx. 20% of the protein in the maize increasing the val. of the total protein to more than double that of the unsupplemented maize-protein. Since the inadequacy of a diet of maize and yeast extract to maintain growth is corr. by adding caseinogen or commercial pig feed but not by adding gelatin and tryptophan, lysine and tryptophan are not the only constituents of caseinogen and yeast-protein required to supplement the deficiencies of maize-protein. The "apparent" digestibility coeff. of maize-N is approx. 79 and the "true" digestibility coeff. of yeast-N approx. 90.

[E. H. Callow.] The carcass data are approx. the same for pigs given yeast-protein and those given caseinogen. In both cases the I val. of the fat (80.4—83.1) is excessively high.

[J. O. Irwin and J. C. D. Hutchinson.] The experimental data are examined statistically. W. McC.

**Value of urea in the synthesis of proteins in the paunch of the ruminant. (I) In maintenance. (II) In growth.** L. E. Harris and H. H. Mitchell (*J. Nutrition*, 1941, 22, 167—182, 183—196).—I. In sheep of 15—18 months (approx. 34 kg.) the endogenous N excretion averaged 0.033 g. per kg. body-wt. or 1.23 g. per sq. m. body surface. Metabolic N in faeces averaged 0.55 g. per 100 g. of dry matter consumed. Urea fed to sheep at the maintenance level was not excreted through the skin. When added to a low-N ration 88% of

the urea was digested and the digestibility of cellulosic matter was improved. Rations containing urea + a min. amount of protein maintained sheep in body-wt. and N equilibria for over 100 days, N equilibrium being attained by feeding 202 mg. of urea- and 161 mg. of casein-N per kg. body-wt. At N equilibrium the biological vals. of urea- and casein-N were 62 and 79, respectively.

II. A low-N diet inadequate to support growth or consistently to maintain N equilibrium in lambs produced substantially normal growth when supplemented with urea. The biological val. of the N of a ration decreased with increase in the amount of urea given. Rations containing 3-16% of urea have no toxic effects on lambs. A. G. P.

**Effect of substituting peanut meal in part for the animal protein in laying mash on egg production, hatchability, and viability of chicks.** R. S. Dearstyne, C. O. Bollinger, and H. P. Brigman (*N. Carolina Agric. Exp. Sta. Bull.*, 1940, No. 326, 10 pp.).—Replacing 62—94% of the animal protein in a laying mash with peanut meal had no significant effect on egg production, hatchability, or viability of the chicks. A. W. M.

**Amino-acids and man.** W. E. Gaunt (*Nature*, 1942, 149, 666—667). E. R. S.

**Effect of added cystine in purified rations for the chick.** G. M. Briggs, jun., R. C. Mills, C. A. Elvehjem, and E. B. Hart (*J. Biol. Chem.*, 1942, 144, 47—52).—Chicks on a diet containing 18% of casein require at least 0.3% of cystine (or its equiv. of methionine) together with supplements of arginine and glycine for max. growth. Prevention of gizzard erosion is aided by the cystine-arginine-glycine supplement and also by chondroitin, the latter exhibiting no growth activity. A combination of cystine, arginine, glycine, and chondroitin exhibits the properties of cartilage growth factor, both as regards growth and production of gizzard erosion. H. G. R.

**Improved diets for nutritional and pathologic studies of choline deficiency in young rats.** R. W. Engel and W. D. Salmon (*J. Nutrition*, 1941, 22, 109—121).—A choline-deficient diet produced fatal toxicity in 6—10 days, symptoms consisting of inactivity, abdominal distention, enlarged kidneys, laboured breathing, coma, and, in some cases, eye hæmorrhage and vertigo. Administration of 10—20 mg. of choline chloride per rat per day prevented these symptoms. A. G. P.

**Effect of heat on availability of iron of beef muscle.** H. G. Oldham (*J. Nutrition*, 1941, 22, 197—203).—Fe given to anæmic rats as oven-dried meat and as  $\text{FeCl}_3$  induced similar increases in hæmoglobin. Vac.-dried meat was somewhat less effective. At least 50% of the Fe in beef exists in org. forms; heat renders this org. Fe as available as that in inorg. Fe salts. A. G. P.

**Seasonal calcium and phosphorus requirements of range cattle as shown by blood analyses.** J. H. Knox, J. W. Benner, and W. E. Watkins (*New Mexico Agric. Exp. Sta. Bull.*, 1941, No. 282, 28 pp.).—The amount of Ca, but not of P, in the forage consumed by cows and steers was adequate. Blood-Ca was of no val. in measuring Ca intake but the inorg. P in plasma was related to P intake. Amounts of Ca and P in the forage eaten were higher in the spring and summer than in winter. Breeding cows receiving a P supplement produced more living, and heavier, calves than did cows receiving no extra P. A. W. M.

**Vitamins and foods.** R. Kuhn (*Angew. Chem.*, 1942, 55, 1—6).—A lecture.

**Vitamins.** C. R. Addinall (*J. Chem. Educ.*, 1942, 19, 203—214). L. S. T.

**Relation of adrenals to vitamins.** J. von Kup (*Z. Vitaminforsch.*, 1942, 12, 251—259).—A discussion of previous work on the relation between the adrenals and the vitamin-B complex, ascorbic acid, and lactoflavin. P. G. M.

**Avitaminosis in apparently healthy Trinidadians.** K. V. Earle (*Brit. Med. J.*, 1942, I, 255—257).—Signs of various vitamin deficiencies were found in 1011 apparently healthy men in Trinidad. C. A. K.

**Prevention of tumour-growth by injection of yeast and vitamins.**—See A., 1942, III, 698.

**Rôle of vitamins in otolaryngology.**—See A., 1942, III, 682.

**Carotene in relation to animal nutrition. III. Stability of carotene in plant material with special reference to hay making and storage.**

**IV. Carotene balance experiments with cows and bullocks.** P. A. Seshan and K. C. Sen (*J. Agric. Sci.*, 1942, 32, 275—285, 286—293).—III.  $\text{O}_2$  is the principal and essential factor in carotene destruction; heat, light, and moisture are accelerating factors. Dried grass is of doubtful val. as a source of carotene in India, owing to heavy losses under unfavourable storage conditions.

IV. Cows may secrete milk containing vitamin-A and carotene while in negative carotene balance. Addition of carotene to this diet causes an immediate rise in milk-carotene. Bullocks in negative carotene balance on low-carotene diets excrete a const. amount of carotene in the faeces. Feeding carotene produces a positive balance and a rise in blood-carotene. R. L. E.

**Effect of certain fats and unsaturated fatty acids on utilisation of carotene.** W. C. Sherman (*J. Nutrition*, 1941, **22**, 153—165).—On a low-fat diet the response of vitamin-A-deficient rats to carotene feeding is influenced by the nature of the fat given. Of the fats examined soya-bean oil gave best results, cottonseed, linseed, maize, and wheat-germ oils were beneficial, but butter fat and coconut oil had no appreciable effect. Methyl linoleate and methyl linolenate produced antagonistic effects when added to a low-carotene diet. With sufficient carotene the antagonism was overcome. The varied effects of fat are not due to their influence on carotene absorption. A. G. P.

**Vitamin therapy in ophthalmology. Early detection of avitaminosis-A by examination of conjunctiva.**—See A., 1942, III, 676.

**Absorption spectra of whale-liver oils.** Z. Nakamiya, K. Koizumi, and I. Kawakami (*Bull. Inst. Phys. Chem. Res. Japan*, 1941, **20**, 576—578).—The absorption max of the liver oils of *Balanoptera physalus*, *B. musculus*, *Megaptera nodosa*, and *Physeter macrocephalus* are between 290 and 305  $\mu$ . Hence, a vitamin-A other than  $A_1$  or  $A_2$  may be present. F. O. H.

**Excretion of vitamin-A in urine.** W. Tomaszewski (*Edinb. Med. J.*, 1942, [iv], **49**, 375—383).—Examination of urines of 104 miscellaneous patients shows that healthy subjects excrete no vitamin-A in urine. Positive results were found in cases of renal and hepatic disease and with certain febrile infections. Saturation tests in 7 cases were "negative." H. S.

**Modified vitameter-A.** R. J. Taylor (*Analyst*, 1942, **67**, 248—254).—The Hilger Vitameter-A has been modified so that the overall error is reduced from  $\pm 10\%$  to  $\pm 3\%$ . For calibration, a standard dye solution, benzeneazo-*p*-cresol in oil and cyclohexane, permits greater accuracy and compensates for errors in cell thickness. An electrode holder with horizontal pointed electrodes and a pendulum type photographic shutter reduce the effective variations of the image intensity and a light-tight photographic paper holder permits the use of the instrument in an undarkened room. S. B.

**Evaluation of preparations of vitamin-B complex.** L. B. Pett, J. A. McKirdy, and M. M. Cantor (*Canad. Med. Assoc. J.*, 1942, **46**, 413—416).—6 commercial preps. of vitamin-B complex were compared by feeding groups of rats a -B-free diet supplemented by the -B complex preps. so that the thiamin intake was uniform in all groups. The best results were given by a prep. containing thiamin 1.0, riboflavin 0.8, pyridoxine 0.4, pantothenic acid 1.0, nicotinic acid 5.0. C. J. C. B.

**Microbiological assay method for six B vitamins using *Lactobacillus casei* and medium of essentially known composition.** M. Landy and D. M. Dicken (*J. Lab. Clin. Med.*, 1942, **27**, 1086—1092).—Max. growth and acid production of *L. casei* have been obtained in a medium in which all constituents, except casein hydrolysate, were chemically defined. Using this medium with *L. casei* as the assay agent, a general assay procedure for the vitamins essential for growth and acid production by this organism (pantothenic acid, riboflavin, nicotinic acid, pyridoxine, folic acid, and biotin) has been detailed. C. J. C. B.

**Vitamin-B<sub>6</sub> deficiency and nervous disease.**—See A., 1942, III, 586.

**Action of thiamin and cocarboxylase on frog ventricle.**—See A., 1942, III, 665.

**Vitamin-B<sub>1</sub> and bacterial oxidations.**—See A., 1942, III, 645.

**Myocardial dysfunction due to vitamin-B<sub>1</sub> deficiency and its treatment.**—See A., 1942, III, 578.

**Lesions of optic nerve in vitamin-B<sub>1</sub> deficiency.**—See A., 1942, III, 682.

**Avian thiamin deficiency.**—See A., 1942, III, 673.

**Induced thiamin deficiency in man.** R. D. Williams, H. L. Mason, B. F. Smith, and R. M. Wilder (*Arch. intern. Med.*, 1942, **69**, 721—738).—A diet adequate in all other respects, but containing only 0.54 mg. of thiamin, was given to 11 physically healthy white women aged 23—46 years for 132 days. States of emotional instability, restriction of activity, and numerous somatic symptoms were produced (cf. A., 1941, III, 209) before objective signs of thiamin deficiency occurred. The "minimal" daily requirement of thiamin was 0.22—0.5 mg. per 1000 cal. of diet and the "optimal" intake was 0.5—1.0 mg. per 1000 cal. C. A. K.

**Vitamin-B<sub>1</sub>-sparing action of fat and protein. III. Oxidation of pyruvate by tissues of symptom-free rats on vitamin-B<sub>1</sub>-deficient diets.** G. G. Banerji and J. Yudkin (*Biochem. J.*, 1942, **36**, 530—541; cf. A., 1942, III, 328).—Unlike the usual signs of vitamin-B<sub>1</sub> deficiency, e.g., loss of wt., polyneuritis, and bradycardia, which do not occur even on a -B<sub>1</sub>-deficient diet if it contains a large proportion of fat or protein, the sp. defect in carbohydrate metabolism is not abolished whatever the composition of the diet. The mode of action of the vitamin is discussed in the light of its effect on the *in-vitro* respiration of rat kidney slices. P. G. M.

**Vitamin-B<sub>1</sub> content of urine of *Trichosurus vulpecula*.** A. Bolliger and C. R. Austin (*J. Proc. Roy. Soc. New South Wales*, 1941, **75**, 118—

121).—The average urinary excretion of vitamin-B<sub>1</sub> by the phalanger is about 0.5 mg. per 24 hr.; and the faecal excretion is small. The animal appears to be able to synthesise -B<sub>1</sub>. F. O. H.

**Urinary excretion of thiamin after test dose.** H. L. Mason and R. D. Williams (*J. clin. Invest.*, 1942, **21**, 247—255).—The diet prior to and during the test period should contain 800—900  $\mu$ g. of thiamin daily. The test dose consists of 1 mg. of thiamin (0.4 mg. per 1000 cal. of diet). Excretion of over 100  $\mu$ g. in the urine in 24 hr. and recovery ultimately of at least 20% of the test dose is evidence of adequate thiamin nutrition. C. J. C. B.

**Determination of the thiamin (vitamin-B<sub>1</sub>) content of diet of service personnel in training.** M. V. Bainton and E. C. Slater (*Med. J. Austral.*, 1942, I, 135—139).—Analysis of the food served gave the average daily intake per man as: thiamin, 0.9 mg.; protein, 106 g.; carbohydrate, 286 g.; fat, 119 g.; calories, 2704. The ratio of thiamin to non-fat calories was 0.56. The agreement between these vals. and those calc. from the food purchases was close. F. S.

**Riboflavin for corneal disease.**—See A., 1942, III, 678.

**Effect of lactoflavin on intestinal sugar absorption.**—See A., 1942, III, 608.

**Riboflavin content of liver.** G. C. Supplee, O. G. Jensen, R. C. Bender, and O. J. Kahlenberg (*J. Biol. Chem.*, 1942, **144**, 79—85).—Injection of riboflavin into the blood stream causes an immediate increase in concn. in the liver. It is also increased during digestion and assimilation, even after a prolonged deficiency of riboflavin. This mobilisation is prevented by a deficiency of thiamin or pantothenic acid, but vitamin-B<sub>6</sub> has a very slight influence. H. G. R.

**Pellagra in Northern Ireland.** J. Deeny (*Brit. Med. J.*, 1942, I, 157—158).—16 cases are reported. C. A. K.

**Nicotinamide-containing nutrients for *Haemophilus parainfluenzae*.**—See A., 1942, III, 647.

**Nicotinic acid content of cereal products.**—See B., 1942, III, 199.

**Nicotinamide content of vegetables.** M. Morel (*Compt. rend.*, 1941, **213**, 530—533).—The nicotinamide content of a no. of vegetables has been determined, acid giving a higher val. than aq. hydrolysis; Human requirements are of the order of 0.1—0.2 mg. per kg. daily. H. G. R.

**Green pigment-producing compound in urine of pyridoxine-deficient rats.** S. Lepkovsky and E. Nielsen (*J. Biol. Chem.*, 1942, **144**, 135—138).—Pyridoxine-deficient rats excrete a substance which forms a green pigment with Fe salts. The excretion stops within a few hr. on feeding pyridoxine. R. L. E.

**Lesions produced in rats by diets free from vitamin-B<sub>6</sub> and response to -B<sub>6</sub>.** W. Antopol and K. Unna (*Arch. Path.*, 1942, **33**, 241—258).—Hyperkeratosis and acanthosis of the ears, paws, and snout, together with oedema of the corium, are considered characteristic of vitamin-B<sub>6</sub> deficiency in rats and respond to -B<sub>6</sub> therapy. (12 photomicrographs.) C. J. C. B.

**Effect of vitamin-B<sub>6</sub> on blood and bone marrow.**—See A., 1942, III, 571.

**Vitamin-B<sub>6</sub> in treatment of pseudohypertrophic muscular dystrophy among children.**—See A., 1942, III, 671.

**Relief of muscular weakness by pyridoxine hydrochloride.**—See A., 1942, III, 583.

**Increased requirements of pantothenic acid and vitamin-B<sub>6</sub> during experimental hyperthyroidism.**—See A., 1942, III, 594.

**Physiologically active metabolite of pyridoxine.**—See A., 1942, III, 648.

**Pantothenic acid absorption in pernicious anaemia.**—See A., 1942, III, 662.

**Procarcinogenic effect of biotin.**—See A., 1942, III, 697.

**Use of avidin in studies on biotin requirement of micro-organisms.**—See A., 1942, III, 718.

**Stability of avidin.** P. György, C. S. Rose, and R. Tomarelli (*J. Biol. Chem.*, 1942, **144**, 169—173).—Avidin activity is destroyed by light, particularly in presence of riboflavin, or by keeping in solution, and partly by HCl at  $p_H$  1.8. Biotin is liberated by the action of light on avidin, but not by treatment with acid. R. L. E.

**Analysis of tobacco mosaic virus for biotin, riboflavin, and pantothenic acid.**—See A., 1942, III, 720.

**Toxicity of *p*-aminobenzoic acid.**—See A., 1942, III, 636.

**Factors affecting synthesis of ascorbic acid in rats.** T. S. Sutton, H. E. Kaeser, and S. L. Hansard (*J. Biol. Chem.*, 1942, **144**, 183—191).—When the vitamin-A intake of rats is below the optimum amount, there is a significant decrease in the plasma-ascorbic acid, and in the amount of ascorbic acid excreted in the urine. Daily administration of 0.1 mg. of stilbesterol increases the excretion of ascorbic acid in normal and castrated female rats, the response being slightly greater with the latter. Chloretone-fed rats with gonads, pituitary, and adrenal glands removed are still capable of synthesis-

ing ascorbic acid. It is concluded that no particular organ or gland is involved but that synthesis of ascorbic acid is a general metabolic function. J. N. A.

**Influence of diet on ascorbic acid requirement of premature infants.** M. Dann (*J. clin. Invest.*, 1942, 21, 139—144).—Premature infants fed on human milk retain a "saturation dose" of ascorbic acid better than infants fed on cow's milk. C. J. C. B.

**Effect of ascorbic acid on survival of traumatised animals.** G. Ungar (*Nature*, 1942, 149, 637—638).—Anæsthetised guinea-pigs were injured by wts. dropped from different heights. Death does not result when the energy of the blow is 2.75 kg.-m. or less, but always results at 3.7 kg.-m. or more. When more than 100 mg. of ascorbic acid per kg. of body wt. was injected immediately after injury death does not result from 3.7 kg.-m. 5—15 mg. of vitamin-B<sub>1</sub> per kg. body wt. was less effective. Ascorbic acid had a protective effect on rats also. E. R. S.

**Influence of vitamin-C as controlling factor in incidence of dental caries in already calcified teeth.** W. B. Grandison, L. B. Stott, and D. B. Cruickshank (*Brit. Dental J.*, 1942, 72, 237—239).—Children to whom 200 mg. of ascorbic acid were administered daily for 2 years were in better health than controls, but there was no apparent improvement in their dental condition. P. G. M.

**Excretion of ascorbic acid in urine after intake of natural and synthetic vitamin.** I. J. Hangartner and T. Gordonoff (*Z. Vitaminforsch.*, 1942, 12, 226—238).—No difference could be established. P. G. M.

**Isolation of ascorbic acid from urine after intake of natural and synthetic vitamin.** II. T. Gordonoff and J. Hangartner (*Z. Vitaminforsch.*, 1942, 12, 238—250).—The vitamin isolated (as dinitrophenyl-oxazone) from urine after administration of both forms of ascorbic acid is the same. P. G. M.

**Can hypovitaminosis-C be demonstrated in urine?** E. Ramel and J. J. Schenk (*Z. Vitaminforsch.*, 1942, 12, 223—226).—200 mg. of ascorbic acid are administered intravenously, and the excretion in urine determined by the dichlorophenol-indophenol method during 3 hr. A total excretion of over 18—20 mg. indicates absence of hypovitaminosis. P. G. M.

**Vitamin-C deficiency in South African native.** H. O. Hofmeyr (*Proc. Staff Mayo Clin.*, 1941, 16, 644—652). H. H. K.

**Detoxifying action of vitamin-C in arsenical therapy.**—See A., 1942, III, 711.

**Redox systems of retina.** I. Ascorbic acid and glutathione.—See A., 1942, III, 681.

**Effect of l-ascorbic acid on isolated frog heart.**—See A., 1942, III, 665.

**Causes of high vitamin-C content of aqueous humour and lens.**—See A., 1942, III, 679.

**Reduction of methæmoglobin by ascorbic acid.**—See A., 1942, III, 581.

**Arsenical sensitivity and vitamin-C.**—See A., 1942, III, 634.

**Effect of vitamin-C and other substances on growth of microorganisms.**—See A., 1942, III, 717.

**Photochemical decomposition of ascorbic acid.** M. Vacher and Y. Lortie (*Compt. rend.*, 1941, 213, 726—728).—A refinement of the photochemical method for determining ascorbic acid by measuring the intensity of the absorption band of aq. solutions in the far ultra-violet has been developed and the effect of the  $pH$  of the solution has been studied. J. L. E.

**Oxidation of ascorbic acid in presence of copper.** E. M. Mystkowski (*Biochem. J.*, 1942, 36, 494—500).—Oxidation of ascorbic acid by O<sub>2</sub> in presence of CuSO<sub>4</sub> is inhibited by NaCl, amino-acids, and proteins. In contrast, NaCl activates the catalytic decomp. of H<sub>2</sub>O<sub>2</sub> and the oxidation of methyl-red and tyrosine; it has no inhibiting effect on the ascorbic acid oxidase of cucumber juice, and only a slight action on one of the two mechanisms of oxidation of ascorbic acid in potato extracts. P. G. M.

**Vitamin-C in walnuts.** M. Pyke, R. Melville, and H. Sarson (*Nature*, 1942, 150, 267—268).—Unripe walnuts (*Juglans regia*) contain 1500—1800 mg. of ascorbic acid per 100 g.; vals. for other *Juglans* species are 410—1120. Certain methods of pickling etc. retain the vitamin-C. A. A. E.

**Kohlrabi as source of vitamin-C.** L. G. G. Warne (*Brit. Med. J.*, 1942, I, 387).—Kohlrabi contains 47—66 mg. of ascorbic acid per 100 g., which is over twice the amount found in root vegetables such as turnips. C. A. K.

**Rickets in black races.** J. Millot (*Compt. rend.*, 1941, 213, 370—372).—The incidence of rickets in various black races living in the open and in forests is briefly indicated. It is suggested that a relationship between the pigmentation of the skin and the formation of vitamin-D in it partly explains the differences found. W. F. H.

**Metabolism of calcium and phosphorus as influenced by various activated sterols.** E. W. McChesney and F. Messer (*Amer. J.*

*Physiol.*, 1942, 135, 577—586).—Dogs were given massive doses of vitamins -D<sub>2</sub> and -D<sub>3</sub> (5 mg. per kg.), A.T. 10 (0.1 c.c. of a 10% solution per kg.), or Ertrou (240,000 units per kg.). All the preps. raised serum-Ca within 24 hr. A.T. 10 caused an increase of 3 mg.-% on each of the first 2 days. The responses to -D<sub>2</sub> and Ertrou are essentially the same. -D<sub>3</sub> is characterised by long persistence of moderate hypercalcaemia. All the preps. except -D<sub>3</sub> improved Ca balance. Ertrou improved P balance slightly. They also cause a rise in serum-P (Ertrou not studied). The preps. decrease faecal and increase urinary output of Ca, decrease faecal output of P, and either increase urinary output or maintain it at a const level. M. W. G.

**Growth and calcification on a diet deficient in phosphate but otherwise adequate.** T. F. Zucker, L. Hall, and M. Young (*J. Nutrition*, 1941, 22, 139—151).—A diet similar to a standard rachitogenic ration became adequate for normal rat growth when PO<sub>4</sub><sup>'''</sup> but not when vitamin-D was added. Addition of -D to low-P rachitogenic diets prevented anatomical symptoms of rickets but did not increase the % of ash in bones to the level of that in animals receiving a normal diet; supplementary feeding of PO<sub>4</sub><sup>'''</sup> produced normal bone composition. A. G. P.

**Antirachitic effect of fat on rats receiving high-calcium-low-phosphorus, rachitogenic diets.** R. G. Booth, K. M. Henry, and S. K. Kon (*Biochem. J.*, 1942, 36, 445—455; cf. A., 1934, 459).—Sufficient proportions of vegetable fats and triglycerides resynthesised from the fatty acids of butter, lard, coconut, olive, linseed, soya-bean, and arachis oil from which the unsaponifiable residue has been removed have antirachitic effects, as determined by the bone-ash method, on rats on high-Ca-low-P rachitogenic diets, even when these contain all their P in inorg. combination. The fats have no antirachitic properties when they are constituents of a high-P-low-Ca rachitogenic diet. Compared with an equalcaloric addition of starch, addition of fat to the high-Ca-low-P rachitogenic diet diminishes faecal P and urinary Ca excretion. The antirachitic action of fat is qualitatively different from that of vitamin-D. W. McC.

**Activated sterols and calcium salts in treatment of parathyroid tetany.**—See A., 1942, III, 685.

**Effect of vitamin-D on prothrombin deficiency in rat.**—See A., 1942, III, 664.

**Vitamin-D content of English butter fat.** K. M. Henry and S. K. Kon (*Biochem. J.*, 1942, 36, 456—459).—During the period March, 1940, to March, 1941, the vitamin-D content of the butter fat from the Shinfield (Nat. Inst. Res. Dairying) herd of cattle ranged from 0.07 to 0.1 i.u. per g. in winter to 0.55 i.u. in July and to approx. 1 i.u. in August, there being good correlation between the content and the amount of sunshine to which the cows were exposed. The prophylactic bone-ash method of assay was used, rats being given the untreated fat and the unsaponifiable residue. Vals. for untreated fat were always higher than those for the residue, the difference being much more marked in winter than in summer. W. McC.

**Vitamin-D assay technique with radioactive strontium.** L. H. Weissberger and P. L. Harris (*J. Biol. Chem.*, 1942, 144, 287—288).—Radioactive Sr and vitamin-D are fed to rachitic rats and the activity of the ash of the excreta is determined: -D acts rapidly in promoting retention of Sr, and the amount of Sr excreted varies inversely as the amount of -D administered and is approx. proportional to the length of time that elapses between administration of -D and Sr. J. N. A.

**Spectrographic determination of vitamin-D.** IV. Z. Nakamiya and K. Koizumi (*Bull. Inst. Phys. Chem. Res. Japan*, 1941, 20, 569—575).—The val. of E<sub>1 cm.</sub><sup>1%</sup> at 265 m $\mu$ . of olive and liver oils decreases on ultra-violet irradiation of the oil. Vitamin-A and -D are adsorbed by acid clay or Al<sub>2</sub>O<sub>3</sub> from their solutions in benzene, but not from those in alcohol. F. O. H.

**Enamel organ of rat's incisor tooth in vitamin-E deficiency.** J. T. Irving (*Nature*, 1942, 150, 122—123).—Rats on a diet adequate in vitamin-A but deficient in -E developed a typical atrophy of the enamel organ in the incisor teeth. E. R. S.

**Vitamin-E in sarcoma.**—See A., 1942, III, 615.

**Treatment of muscular atrophies with vitamin-E.**—See A., 1942, III, 583.

**Prevention by  $\alpha$ -tocopherol of "cod-liver oil muscular dystrophy" in rabbit.**—See A., 1942, III, 672.

**Tocopherol in blood. Possible test for avitaminosis-E.** A. Vinet and P. Meunier (*Compt. rend.*, 1941, 213, 709—711).—CHCl<sub>3</sub> extraction of blood serum, dried with Na<sub>2</sub>SO<sub>4</sub>, removes the tocopherol, which can be determined by the Fe<sup>III</sup> ferri cyanide method, allowance being made for the small amount of carotenoids also extracted. It is suggested that a blood concn. below 3 mg. per 100 c.c. (rabbit) and 1.5 mg. per 100 c.c. (man) indicates avitaminosis. W. C. J. R.

**Prophylactic use of vitamin-K in obstetrics.**—See A., 1942, III, 664.

Dietary requirements for lactation. XII. Prevention of vitamin- $L_2$  deficiency by dextrin diet. XIII. Deficiency of saké yeast in vitamin- $L_1$ . XIV. Separation of vitamin- $L$  complex from filtrate factors. W. Nakahara, F. Inukai, and S. Ugami (*Sci. Papers Inst. Phys. Chem. Res. Tokyo*, 1941, **38**, 415—419, 420—423, 424—432; cf. A., 1941, III, 210, 1038).—XII. Fairly satisfactory lactation is obtained in rats on a vitamin- $L_2$ -deficient diet when dextrin is used as source of carbohydrate:  $-L_2$  is possibly produced from dextrin by intestinal yeasts.

XIII. *Saccharomyces saké* is a good source of  $-L_2$ , but not of  $-L_1$ .

XIV. The  $-L$  complex is shown not to be identical with the filtrate factors pantothenic acid and anti-achromotrichia factor (*p*-amino-benzoic acid). Factors other than those necessary for growth and maintenance are specifically required for lactation. F. O. H.

Biological test for vitamin- $P$ . A. L. Bacharach, M. E. Coates, and T. R. Middleton (*Biochem. J.*, 1942, **36**, 407—412).—A modification of the method of Zacho (*Acta Path. Microbiol. Scand.*, 1939, **16**, 144) is described. Results of tests with guinea-pigs to which hesperidin and other water-sol., vitamin- $P$  preps. are administered show that the variance of response to dose is small and apparently independent of the level of response and that, at higher levels of response, the slope of the response curves is satisfactory. Accuracy is achieved by comparing test substances with a provisional standard, using groups of approx. 10 guinea-pigs, employing doses in the ratio of 2:1, and choosing these so that the pressure at which petechiae are first seen is 70—90 mm. of Hg. W. McC.

## XIX.—METABOLISM, GENERAL AND SPECIAL.

Calorigenic effect produced by various mixtures of foodstuffs. J. C. Ring (*Amer. J. Physiol.*, 1942, **135**, 742—746).—In rats, the sp. dynamic action (S.D.A.) of 1.5 c.c. of oleic acid + 3 c.c. of 50% glucose ingested together is less than that of 1.5 c.c. of oleic acid alone. S.D.A. of 3 c.c. of 50% peptone + 3 c.c. of 50% glucose is less than that of 3 c.c. of 50% peptone alone, or of the sum of the individual calorigenic effects. The S.D.A. of 3 c.c. of 50% peptone + 1.5 c.c. of oleic acid is almost equal to the sum of the S.D.A. of peptone ingested alone + that of oleic acid. M. W. G.

Influence of temperature on the stimulation of oxygen consumption of isolated brain and kidney of rats by 2:4-dinitrophenol. F. A. Fuhrman and Y. Field (*J. Pharm. Exp. Ther.*, 1942, **75**, 58—63).—The intensity of the acceleration of  $O_2$  consumption of excised rat cerebral cortex and kidney cortex by a given concn. of dinitrophenol ( $3.35 \times 10^{-3}M$ ) varies with temp. The max. % increase in respiration occurred at 32° in cerebral cortex and at 22° in kidney cortex. Vals. of the temp. coeff.,  $Q_{10}$ , for the respiration of excised cerebral and kidney cortex were calc. for small intervals over the range 10—42°. A progressive decrease in  $Q_{10}$  from 10° to 30° followed by a definite rise in the range 30—35°, with diminution on further rise in temp., was found. H. H. K.

Metabolism of brain.—See A., 1942, III, 585.

Effect of lactoflavin on utilisation of glucose by living cell in respiration.—See A., 1942, III, 620.

Respiratory metabolism of lens and vitreous body.—See A., 1942, III, 680.

Metabolism of phenylalanine. K. Closs and K. Braaten (*Z. physiol. Chem.*, 1941, **271**, 221—245).—Relationships in the alimentary canal are not responsible for the incidence of alkaptonuria after administration of *l*-phenylalanine as Na salt to the white rat since it is also observed after subcutaneous administration.  $p_H$  of the solution is not in itself decisive but it must be so high that part at any rate of the phenylalanine is present as anion. Possibly the alkali introduced simultaneously is the main factor.  $p_H$  in the kidneys (urine) is unimportant. The amount and concn. of the administered solutions do not influence the nature of the reaction products but only the amount and duration of the separations. In white rats alkaptonuria is caused by amounts of *l*-phenylalanine (as Na salt) which are unable to cause pptn. of phenylpyruvic acid. *d*-Phenylalanine and *d*- or *l*-phenyl-lactic acid are not intermediates in the formation of alkapton from *l*-phenylalanine. Tyrosine is not involved since the alkaptonuria observed after administration of *l*-tyrosine, dissolved in the equiv. amount of aq. NaOH, is less pronounced and more slowly incident than that due to *l*-phenylalanine. Alkaptonuria is observable about 2 hr. after subcutaneous injection of *l*-phenylalanine as Na salt and is immediately max. Its duration depends on the amount used. Its cessation is as sudden as its incidence in contrast to the separation of phenylpyruvic acid which gradually increases after use of *l*-phenylalanine, attains its max. after about 4 hr., and then slowly diminishes. With rats from which the kidney has been removed subcutaneous injection of the Na salt of *l*-phenylalanine causes concn. of *l*-phenylalanine in the blood lower than that observed with use of neutral or HCl solutions. Evidence is adduced in favour of the view that "non-natural" *d*-phenylalanine is formed in the body from the *l*-isomeride and is the intermediate product of the conversion of *l*-phenylalanine into phenylpyruvic acid. H. W.

Intermediary metabolism of histidine. V. S. Edlbacher and H. von Bidder (*Z. physiol. Chem.*, 1942, **273**, 163—176; cf. A., 1934, 920).—Urocanic acid could not be isolated as an intermediate product during the enzymic decomp. of histidine by liver extract. When large amounts of histidine were administered orally and parenterally to dogs and rabbits urocanic acid was not detected in the urine. The acid was decomposed by extracts of guinea-pig, rat, rabbit, and pigeon liver with formation of a labile compound which yielded  $NH_3$  when treated with NaOH. *d*-Histidine and pyruvic acid inhibited hydrolytic deamination of histidine by rat liver histidase but not decomp. of urocanic acid. If the breakdown of histidine in the liver involves formation of urocanic acid, at least two enzymes, histidase and a urocanase, must be present. J. N. A.

Amino-acid metabolism. VIII. Metabolism of *l*(-)-histidine in the normal rat. L. F. Remmert and J. S. Butts (*J. Biol. Chem.*, 1942, **144**, 41—46).—Liver-glycogen formation occurs 8 hr. after feeding of *l*(-)-histidine, the max. deposition occurring at 16 hr., and in some cases hæmaturia develops. Ketonuria produced by a high-fat diet is decreased by *l*(-)-histidine proportionally to the glycogen formation. N excretion shows the expected increase and no appreciable amount of the  $NH_3$ -acid is excreted. H. G. R.

Glutathione. II. Metabolism of glutathione studied with isotopic ammonia and glutamic acid. H. Waelsch and D. Rittenberg (*J. Biol. Chem.*, 1942, **144**, 53—58).—Glutathione is rapidly metabolised and interacts more rapidly with dietary N than the protein of the same tissue since appreciable amounts of the isotope are found in the glutathione of the liver and intestine 2½ and 2 hr. after administration of isotopic  $NH_3$  to rabbits and labelled *dl*-glutamic acid to rats, respectively. The half lifetime of glutathione in rat and rabbit liver is 2—2½ hr. H. G. R.

Metabolism of dimethylaminoazobenzene (butter-yellow) in rats. E. S. Stevenson, K. Dobriner, and C. P. Rhoads (*Cancer Res.*, 1942, **2**, 160—167).—*p*-Aminophenol, *p*-acetamidophenol, *p*-phenylenediamine, and *NN'*-diacetyl-*p*-phenylenediamine were isolated from the urine of rats fed on rice containing *NN'*-dimethyl-*p*-aminoazobenzene. An additional metabolite was isolated but not identified. F. L. W.

Deamination, amination, and trans-amination. T. Wieland (*Angew. Chem.*, 1942, **55**, 147—151).—A review.

Creatine metabolism in hypothyroid infants and children.—See A., 1942, III, 684.

Cholesterol metabolism. R. P. Cook (*Nutr. Abs. Rev.*, 1942, **12**, 1—11).—A review.

Obesity in childhood. I. P. Bronstein, S. Wexler, A. W. Brown, and L. J. Halpern (*Amer. J. Dis. Child.*, 1942, **63**, 238—251).—No endocrine disturbance was found in physical studies of 35 obese children. The mean intelligence was above normal. Only 2 of 24 boys showed a tendency toward femininity. There is no known history of obesity in most of the parents or siblings of these children. C. J. C. B.

Rôle of lipophilia in ætiology of obesity. M. Block (*Proc. Soc. Exp. Biol. Med.*, 1942, **49**, 496—499).—3 obese subjects remained in N equilibrium during periods of under-nutrition and their blood-lipins were the same as in controls. The view of Hetenyi that their fat-cells have an abnormal capacity to remove fat from the circulation is therefore unfounded. V. J. W.

Metabolism of adipose tissue *in vitro*.—See A., 1942, III, 642.

Carbohydrate storage and mobilisation in rats. M. M. Guest (*J. Nutrition*, 1941, **22**, 205—221).—By an improved technique liver- and muscle-glycogen in rats are shown to exceed vals. previously accepted. Liver-glycogen may be maintained at any desired level (0—8% of fresh wt.) by nutritional control. At the end of an experimental period liver-glycogen was somewhat greater when a high-carbohydrate than when a high-protein diet was used. The reverse was true after food had been withheld for 24 hr. Effects of pre- inanition diets on liver-glycogen and blood-sugar were accentuated by maintaining rats at 0.5 atm. pressure. Positive physiological relationships are established between blood-sugar and log liver-glycogen in fasted rats and between liver- and muscle-glycogen in fed and fasted rats. Blood-sugar and muscle-glycogen are probably related in fasting rats. A. G. P.

Metabolism of fructose of eviscerated rat. R. M. Reinecke (*Amer. J. Physiol.*, 1942, **136**, 167—172).—Injected fructose disappears from the tissues of adult eviscerated nephrectomised rats.

M. W. G.

Significance of glucose-nitrogen ratio and its bearing on mechanism of diabetes mellitus. D. R. Drury, H. Edelbrock, and L. Mill (*J. clin. Invest.*, 1942, **21**, 153—158).—For the classical glucose:N ratio of 3.65 or glucose:protein ratio of 0.58 in the phloridzinised fasting dog, the urinary sugar must be considered as only part of the total sugar produced from the protein represented by N of the urine. The other portion of this sugar is used by the tissues of the animal. On feeding the animal protein, there is an increase in this tissue utilisation so that the urinary glucose:N ratio decreases. The

depancreatised dog shows less increase in tissue-glucose utilisation on feeding. The extra sugar resulting from fed protein can be estimated since the amount used by the tissues is small and is easily corr. for. When this correction is applied the glucose:N ratio is between 5 and 6 and then the glucose:protein ratio approaches 1. With this higher glucose:N ratio both the glucose excreted in the urine and that used by the tissues in fasting diabetics can be taken as coming from body-protein; conversion of fat into glucose need not be assumed. C. J. C. B.

**Thyroid and parathyroid hormone effects on calcium and phosphorus metabolism.**—See A., 1942, III, 595.

**Relation of heat production to water metabolism during administration of synthetic thyroxine.**—See A., 1942, III, 594.

**Metabolic water and desiccation.** K. Mellanby (*Nature*, 1942, 150, 21).—Theoretical. E. R. S.

**Fate of morphine [in addicts].**—See A., 1942, III, 633.

## XX.—PHARMACOLOGY AND TOXICOLOGY.

**Long-chain sulphonamides and their therapeutic properties.**—See A., 1942, II, 307.

**Sulphanilamide and related chemotherapeutic agents.** L. H. Amundsen (*J. Chem. Educ.*, 1942, 19, 167—171). L. S. T.

**Derivatives of aminoisoquinolines [sulphanilamides]. 2-Phenyl-oxazole and *o*-substituted derivatives thereof [sulphanilamines].**—See A., 1942, II, 330, 335.

**Sulphanilamide and sulphapyridine in experimental cerebral wounds.** E. H. Botterell, E. A. Carmichael, and W. V. Cone (*J. Neurol. Psychiat.*, London, 1941, 4, 163—174).—500 mg. of either drug, placed in brain defects (cats), disappeared completely in 18—36 or 44—58 days respectively; a comparable quantity of drug applied to cerebral injury in man would be 25—50 g. Impairment to general health, liver, bone marrow, kidney, or bladder was never observed, and the highest blood levels resulting from local absorption were never sufficient to be of therapeutic val. The drugs provoked a local tissue reaction and were eventually encapsulated; polymorphonuclear cells were especially marked in the early stages in the sulphanilamide series, whilst connective tissue encapsulation was thicker with sulphapyridine. No undesirable effects were observed on nerve cells, neuroglia, or myelin at a distance from the drugs. Scarring was normal. For human cases a thin coating of the walls of the wound with sulphapyridine is sufficient. H. L.

**Pathological tissue changes produced by sulphathiazole and sulphathiazoline in rabbits.** J. A. Kohler (*J. Lab. clin. Med.*, 1942, 27, 1043—1046).—Neither compound damaged the meninges, brain, spinal cord, kidneys, liver, or spleen in doses of 0.05 g. per kg. twice daily for 20 doses. Both compounds in doses of 0.1 and 0.2 g. per kg. twice daily for 20 doses produced equal renal damage (due to obstruction of the tubules by crystals of acetylated compounds, sometimes associated with tubular necrosis). Both were equal in causing liver damage (cloudy swelling, slight fatty degeneration, and necrosis of the peripheral cells of the lobules) and equal spleen damage (slightly hyperplastic changes in the germinal centres of the Malpighian corpuscles). C. J. C. B.

**Sulphonamides in pneumococcal endocarditis.** R. N. Tattersall (*Brit. Med. J.*, 1942, I, 293—294).—Sulphapyridine and sulphathiazole were ineffective in a fatal case of type 4 pneumococcus ulcerative endocarditis. C. A. K.

**Local sodium sulphacetamide in experimental corneal ulcers.** J. M. Robson and G. I. Scott (*Brit. Med. J.*, 1942, I, 5—8).—30% solution of Na sulphacetamide was applied to rabbits' eyes after experimental infection of the cornea with *B. pyocyaneus*. All control eyes developed severe corneal ulceration. When the drug was applied 1 hr. after inoculation ulceration occurred in 6 of 17 eyes, when applied after 5 hr., in 7 of 12 eyes, and when applied after 12 hr., in 10 of 12 eyes, though the treated eyes were less severely affected than controls. C. A. K.

**Sulphapyridine in ophthalmia neonatorum.** A. Sorsby, E. L. Hoffa, and E. W. Smellie (*Brit. Med. J.*, 1942, I, 323—325).—The results of sulphapyridine therapy (oral) in 273 cases of ophthalmia neonatorum were compared with those in 46 cases treated by the older local methods. In all cases, whether gonococcal or not, the drug was much more effective than local therapy, 62% being cured with the former and 15% with the latter within 8 days. C. A. K.

**Sulphathiazole ointment in impetigo.** A. J. Steigman (*Brit. Med. J.*, 1942, I, 12—13).—5% sulphathiazole ointment healed impetigo in about half the time taken by ung. hydrarg. amm. dil. in 51 cases. C. A. K.

**Sulphanilamide paste in impetigo.** E. L. Cohen (*Brit. Med. J.*, 1942, I, 359).—Successful use in 69 cases is reported. C. A. K.

**Chemotherapy and chemosotherapy of experimental staphylococcal infections.** J. A. Kolmer, H. Brown, and A. M. Rule (*Arch. intern.*

*Med.*, 1942, 69, 636—646).—Sulphathiazole and sulphathiazoline (2-sulphanilyl-3:5-dihydrothiazole) were more effective than sulphapyridine and sulphadimethylpyrimidine against experimental *Staph. aureus* infections in mice and rabbits. Survival is the only criterion of cure. Simultaneous administration of antitoxin + drug enhanced the activity of the drug slightly. C. A. K.

***Staphylococcus aureus* septicæmia successfully treated with sulphathiazole.** P. W. Mathew and A. G. Shera (*Brit. Med. J.*, 1942, I, 258).—Case report. C. A. K.

**Treatment of gonorrhœal urethritis in male with sulphonamide derivatives.** L. W. LaTowsky, F. Knight, C. A. W. Uhle, and R. B. Baker (*J. Lab. clin. Med.*, 1942, 27, 1001—1006).—The rate of cure was 92% of 87 cases for sulphapyridine, 96% of 55 cases for sulphathiazole, and 93% of 57 cases for sulphadiazine. The average dose required was: sulphapyridine 23.5 g. in 8 days, sulphathiazole 28 g. in 8 days, and sulphadiazine 17.5 g. in 8 days. Toxic reactions from the drugs occurred in 75% of the cases treated with sulphapyridine, 11.5% with sulphathiazole, and 8.8% with sulphadiazine. The average times for the discharge to cease were 3 days with sulphapyridine or sulphathiazole, and 4 days with sulphadiazine. The average times for a bacteriological cure were 51 days for sulphapyridine, 28 days for sulphathiazole, and 13 days for sulphadiazine. The "carrier state" (period from disappearance of symptoms to last culture of prostatic fluid known to contain living gonococci) averaged 17 days for sulphapyridine, 17 days for sulphathiazole, and 3 days for sulphadiazine. C. J. C. B.

**Sulphanilamide for treatment of gonorrhœa of anal canal.** R. Turell (*J. Lab. clin. Med.*, 1942, 27, 1046—1048).—2—4 g. daily, to a total of 50 g., was successful in 2 cases. C. J. C. B.

**Molluscum contagiosum cured with sulphapyridine.** W. R. Hill and J. G. Downing (*Arch. Dermat. Syphilol.*, 1942, 46, 139—140).—A case report. C. J. C. B.

**Effect of sulphonamide drugs on *V. cholerae*.** J. J. Griffiths (*U.S. Publ. Health Repts.*, 1942, 57, 814—818).—Sulphathiazole, sulphadiazine, and sulphanilamide inhibited the growth of *V. cholerae* *in vitro*. Sulphathiazole and sulphadiazine given subcutaneously or intragastrically were effective in the treatment of mice previously inoculated with lethal doses of cholera vibrios in mucin. Succinylsulphathiazole and sulphaguanidine given intragastrically were effective in the treatment of mice experimentally infected with *V. cholerae*. C. G. W.

**Clinical use of succinylsulphathiazole.** E. J. Poth and F. L. Knotts (*Arch. Surg.*, Chicago, 1942, 44, 208—222).—5% of the drug administered orally in therapeutic doses is excreted in the urine. It can be maintained in high concn. in the diseased gastro-intestinal tract with low concn. in the blood without toxic effects. There is a profound change in the physical characters and bacterial flora of the faeces after treatment. F. S.

**Sulphonamide therapy of malaria in ducks.** E. K. Marshall, jun., J. T. Litchfield, jun., and H. J. White (*J. Pharm. Exp. Ther.*, 1942, 75, 89—104).—The antimalarial activity of a no. of sulphonamides and sulphones has been tested. *p*-Aminobenzoic acid has a slight antimalarial activity, and has a definite antagonistic effect on the antimalarial action of sulphonamide drugs. H. H. K.

**Therapeutic and prophylactic detoxication of sulphonamides.** R. J. Martin, C. V. Fisher, and M. R. Thompson (*Arch. intern. Med.*, 1942, 69, 662—669).—The acute toxicities of sulphanilamide, sulphathiazole, and sulphapyridine in mice were reduced by up to 50% by the prophylactic and therapeutic use of detoxifying agents, e.g., cystine, glycine, Ca glucuronate, and ascorbic acid, and at the same time their chemotherapeutic efficiency was maintained or enhanced. Sulphanilamide absorption in dogs was speeded up by detoxifying substances. C. A. K.

**Influence of sulphanilamide on development of eggs of sea urchin.** J. A. Thomas (*Compt. rend.*, 1941, 213, 890—892).—Concn. of 1—5% in sea-water arrest segmentation of sea-urchin eggs. W. C. J. R.

**Cinchona alkaloids in pneumonia.** X. apoCupreine 6- $\beta$ -alkylthioethyl ethers.—See A., 1942, II, 336.

**Antiplasmodial action and chemical constitution. VI. Compounds related to lepidylamine.**—See A., 1942, II, 288.

**Chemotherapeutic pyroplasmocidal compounds. I. Alkylamino-phenylcarbamides.**—See A., 1942, II, 307.

**Effect of quinine derivatives on growth rate of Fujinawa's rat sarcoma in tissue culture.** N. Sofue (*Japan. J. Med. Sci.*, 1941, IV, 13, 139—147).—Quinine and 22 derivatives were examined. Growth-inhibiting power increases with the no. of C atoms in the side-chain at position 6 of the quinoline ring, and again with changes in the vinyl radical of quinine in such a way that vinyl and ethyl < halogenoethyl < dihalogenoethyl, where Cl < Br < I. H. H. K.

**Recent advances in chemotherapy.** W. O. Kermack (*Edinb. Med. J.*, 1942, [iv], 49, 429—457).—A lecture containing an excellent account of the subject to date. H. S.

**Serum therapy in acute bacillary dysentery.** H. H. Macumber (*Arch. intern. Med.*, 1942, **69**, 624—635).—Multivalent serum therapy was of no val. in 263 cases (Flexner 91%, Sonne 6.5%).

C. A. K.

**Infectious entero-toxaemia of sheep in Western Australia.** C. R. Toop (*J. Dept. Agric. W. Australia*, 1941, **18**, 35—43).—A single injection of 5 c.c. of alum-pptd. entero-toxaemia vaccine prevents this disease (also known as "Braxy-like disease" or "pulpy kidney"), which is caused by *C. Welchii* type D.

A. W. M.

**Selective bacteriostatic action of gentian-violet.** L. P. Garrod (*Brit. Med. J.*, 1942, **I**, 290—291).—The influence of various concns. of gentian-violet on growth of Gram-positive bacteria in 5% blood agar was studied, and it is shown that 1/500,000 concn. inhibited the growth of staphylococci, diphtheroids, and aerobic spore-bearing bacilli, but allowed free growth of streptococci.

C. A. K.

**Harmful effects of liquid paraffin purgatives.** J. W. Morgan (*J. Amer. Med. Assoc.*, 1941, **117**, 1335—1339).—Review and discussion. Digestive symptoms, diminished absorption of vitamins-A and -D, increased motility of the small intestine, and anal leakage may all result from administration of liquid paraffin.

C. A. K.

**Anti-histamine activity of thymoxyethyl-diethylamine and N'-ethyl-N-diethylaminoethylaniline as judged by the gastric response to histamine.** H. B. Burchell and R. L. Varco (*J. Pharm. Exp. Ther.*, 1942, **75**, 1—5).—The drugs showed no sp. histamine antagonism, as tested by the gastric secretory response of Heidenhain pouch dogs to histamine stimulation. Some lowered pain sensitivity was observed if large doses of thymoxyethyl-diethylamine were given subcutaneously in dogs.

H. H. K.

**Morpholinoethyl ethers and amides possessing antispasmodic activity.**—See A., 1942, II, 334.

**Colour reactions of sympathomimetic amines with diazonium compounds.**—See A., 1942, II, 305.

**Effect of sympathomimetic amines on pancreatic secretion.**—See A., 1942, III, 746.

[Action of acetylcholine.] S. O. Alcalde (*Ciencia*, 1941, **2**, 337—344).—A review.

F. R. G.

**Action of drugs on *Daphnia magna*.** V. Obreshkove (*Biol. Bull.*, 1941, **81**, 105—113).—Acetylcholine produces vigorous intestinal contractions observable directly under the microscope. The latent period is dependent on the dilution; it may be up to 2 hr. Atropine blocks this action; eserine shortens the latent period and intensifies the action.

D. M. Sa.

**Influence of esmodil on adrenaline hyperglycaemia.** N. Izaki (*Japan. J. Med. Sci.*, 1941, **IV**, **13**, 127—137).—Esmodil in large doses produces hyperglycaemia in rabbits and summates with acetylcholine in producing it. Adrenaline hyperglycaemia is decreased by small and increased or not affected by larger doses.

H. H. K.

**Analysis of action of acetylcholine on cardiac ganglion of *Limulus polyphemus*.** W. E. Garrey (*Amer. J. Physiol.*, 1942, **136**, 182—193).—Acetylcholine, even with eserine, is without effect in facilitating transmission of nerve impulses to the musculature of the *Limulus* heart. This is true for choline esters, lentin and mecholyl, and for choline (bromide) itself. Acetylcholine (1 in 10,000) stimulates the ganglion, increasing the rate and amplitude of muscular contraction. Eserine potentiates this effect 20—50 times for some hr. but not with lentin or mecholyl. Atropine stimulates the ganglion but does not annul the action of acetylcholine before or after eserine. Acetylcholine restores function to the depressed cardiac ganglion of the *Limulus* heart.

M. W. G.

**Effect of papaverine, adrenaline, and quinidine on fibrillation threshold of the mammalian ventricles.** R. Węgria and N. D. Nickerson (*J. Pharm. Exp. Ther.*, 1942, **75**, 50—57).—Papaverine hydrochloride and quinidine sulphate raise the fibrillation threshold of dog's ventricle significantly and for a significant length of time. Adrenaline raises the fibrillation threshold very significantly for a short time. If quinidine and papaverine are given in such doses that they produce a significant fall of blood pressure, the raising of the fibrillation threshold is neutralised or decreased.

H. H. K.

**Chemical and pharmacological studies on rhodeatoxin, a glucoside extracted from the leaf of *Rhodea japonica*.** S. Nakaya and Y. Tanno (*Japan. J. Med. Sci.*, 1941, **IV**, **13**, 121—126).—*Rhodeatoxin*, m.p. 178, is chemically related to but not identical with rhodein, m.p. 193°, occurring in the root of the same plant. The drug acts mainly on the circulation.

H. H. K.

**Scilliroside.**—See A., 1942, II, 218.

**Effect of some anaesthetic agents on volume of body fluid in dogs.** D. D. Bonnycastle (*J. Pharm. Exp. Ther.*, 1942, **75**, 18—29).—Administration of ether, morphine, and atropine produced a decrease of plasma vol. and in interstitial fluid space in dogs. Pentobarbital and pentothal Na caused a significant increase in plasma vol. and a decrease in cell vol. No significant alterations were observed

in the total blood vol., total available fluid, or interstitial fluid space.  $N_2O$  and cyclopropane showed no significant changes in plasma vol., total available fluid, or interstitial fluid space. Significant increases were observed in the % of cell vol., red cell vol., and in blood vol.

H. H. K.

**Optically active phenylurethane anaesthetics.**—See A., 1942, II, 301.

**Anaesthetic action of cyclopropyl vinyl ether (cyprethylene ether).** J. C. Krantz, jun., W. E. Evans, jun., S. E. Forman, and H. L. Wollenweber (*J. Pharm. Exp. Ther.*, 1942, **75**, 30—37).—The drug, b.p. 67°, has approx. the same potency as  $CHCl_3$  and an anaesthetic index of more than twice that of ether. In the monkey it produces no functional liver damage. No histopathological changes were seen in lung, liver, and kidney of mice, rats, dog, and monkey after the administration. The drug depressed the rate of beat and amplitude of the perfused frog's heart. The explosive range of concns. of cyprethylene ether with  $O_2$  is approx. the same as that of ether. The oil/water coeff. is 16 times that of ether. Anaesthetic concns. in the blood are one fifth of those found with ether. Blood pressure is slightly lowered by anaesthetic concns. in the dog.

H. H. K.

**Heavy percaïne spinal anaesthesia.** W. W. Mushin (*Brit. Med. J.*, 1942, **I**, 139—143).—Clinical results in 420 cases are discussed.

C. A. K.

**Procaine infiltration in fibrositis.** E. J. Moynahan and E. S. Nicholson (*Brit. Med. J.*, 1942, **I**, 65—68).—Procaine infiltration is of val. in diagnosis and treatment of fibrositis.

C. A. K.

**Measurement of effect of alcohol on pain threshold and on the "alarm" reaction.**—See A., 1942, III, 742.

**Drug addiction.** C. K. Himmelsbach (*Arch. intern. Med.*, 1942, **69**, 766—772).—Measurements of wt., calorie intake, sleep, basal metabolic rate, temp., respiration, blood pressure, and blood studies were made in 21 drug addicts (to morphine or its derivatives) during the period of recovery after abrupt withdrawal. Physical recovery takes about 6 months and is assessed by an increase in body wt. The basal metabolic rate of the recovered addict is subnormal.

C. A. K.

**Addiction liability of demerol (D-140).** C. K. Himmelsbach (*J. Pharm. Exp. Ther.*, 1942, **75**, 64—68).—The drug (ethyl 4-phenyl-1-methylpiperidine-4-carboxylate) possesses analgesic and spasmolytic properties together with a mild atropine-like action. When substituted for morphine demerol partly satisfied the physical dependence on morphine. On withdrawal, after 10 days of substitution, a mild abstinence syndrome occurred. Physical dependence on demerol resulted from its regular administration to post-addicts over a period of 10 weeks. The abstinence syndrome which occurred following its withdrawal was milder than the morphine abstinence syndrome but otherwise quite typical. The duration of the physical dependence action was considerably shorter than for morphine.

H. H. K.

[Magnesium chloride as] narcotic for marine invertebrates. I. C. Ledingham and G. P. Wells (*Nature*, 1942, **150**, 121—123).—Animals were narcotised by immersion in 8%  $MgCl_2$  for 1—4 hr.

E. R. S.

**Antagonism between anaesthetic steroid hormones and pentamethylenetetrazole (metrazol).** H. Selye (*J. Lab. clin. Med.*, 1942, **27**, 1051—1053).—In young rats the anaesthesia produced by intraperitoneal administration of steroid hormones (progesterone or deoxycorticosterone acetate) can be interrupted by metrazol. Conversely, fatal doses of metrazol are readily tolerated without causing either convulsions or lung oedema by rats receiving suitable doses of anaesthetic steroid hormones.

C. J. C. B.

**Neorsphenamine in bacterial infections.** E. E. Osgood (*Arch. intern. Med.*, 1942, **69**, 746—765).—Neorsphenamine in concns. which do not kill living human cells (bone-marrow) is effective against some strains of *Strep. viridans*, most staphylococci, and some other bacteria. Effective blood concns. of the drug can be maintained by suitable dosage but toxic effects are rather frequent; 3 out of 7 patients with subacute bacterial endocarditis were free of symptoms 8, 13, and 12 months after treatment began. Neorsphenamine + sulphathiazole may be more effective than either drug alone.

C. A. K.

**Thrombocytopenic purpura after arsphenamines.** S. M. Laird (*Brit. Med. J.*, 1942, **I**, 381—382).—A case report.

C. A. K.

**Chemical specificity of skin hypersensitiveness of guinea-pigs to old arsphenamine.** W. Frei (*J. invest. Dermatol.*, 1942, **5**, 29—38).—The skin hypersensitiveness of sensitised guinea-pigs to As is towards aromatic arsenicals in general.

C. J. C. B.

**Arsenic in hair and bone in arsenical poisoning.** E. G. Young and R. P. Smith (*Brit. Med. J.*, 1942, **I**, 251—253).—The As content of hair, bone, and adipocere of the remains of 2 individuals exhumed after 9½ years was determined for medico-legal purposes. The hair-As content was 0.44 and 0.32 mg.-% dry tissue, and in bone 0.05 and 0.02 mg.-%. In one case, where death occurred 30 hr. after poisoning, the proximal portion of scalp hair contained 0.77 mg.-%.

C. A. K.



**Acute toxicity for mice of phthalic acid and certain derivatives.** H. C. Hodge, M. R. Goldstein, and M. Wrightington (*Proc. Soc. Exp. Biol. Med.*, 1942, **49**, 471—473).—LD<sub>50</sub> doses per kg. are for phthalic acid 0.55 g., for Na phthalate 2.1 g., for dimethyl phthalate 2.4 ml., for di-*n*-butyl phthalate 5.5 ml., and for di- $\beta$ -octyl phthalate 46 ml. V. J. W.

**Effect on small laboratory animals of injection of crystalline hydrochloride of a sulphur protein from wheat flour.** E. J. Coulson, T. H. Harris, and B. Axelrod (*Cereal Chem.*, 1942, **19**, 301—307).—When injected intraperitoneally or intravenously into mice, guinea-pigs, and rabbits, the protein was toxic, producing laboured respiration, coma, and death. Taken orally it was harmless. Contraction of isolated guinea-pig uteri, using the Schulz-Dale technique, was induced by less than 1 p.p.m., but the muscle was not desensitized to subsequent doses of the protein. N. L. K.

**Analytical classes of cannabinol compounds in marihuana resin.** C. C. Fulton (*Ind. Eng. Chem. [Anal.]*, 1942, **14**, 407—412).—The cannabinol compounds are divided into four main classes: I-RAB which are reactive in the alkaline Beam test and are extracted from light petroleum by alkali; I-NRAB which are non-reactive and extracted by alkali; II-RAB which are reactive and not extracted by alkali; II-NRAB which are non-reactive and not extracted by alkali. Directions are given for performing colour tests on each of these classes. J. D. R.

**Chronic toxicity of *Derris*.** A. M. Ambrose, F. DeEds, and J. B. McNaught (*Ind. Eng. Chem.*, 1942, **34**, 684—689).—The relative chronic toxicity of *Derris* root (4 samples), fed in very small doses to albino rats, is in accordance with the rotenone contents, but the differences may not be significant and *Lonchocarpus* root is much less toxic. The extent of liver damage (evident after doses of 75 p.p.m. of diet for 100 days), the only notable post mortem symptom, does not accord with rotenone content. Constituents other than rotenone are thus of importance. *Derris* root extracted with acetone is not toxic. R. S. C.

**Relative increase in chloride excretion in dog after graduated doses of mercurial diuretics.** C. C. Roby and C. Pfeiffer (*Amer. J. Physiol.*, 1942, **135**, 591—594).—In trained unanesthetized dogs with bladder fistulae the range of dosage between beginning tubular inhibition and the max. obtainable Cl<sup>-</sup> excretion varies for each diuretic. With 0.5—2.5 mg. of Hg per kg. salyrgan produced a max. Cl<sup>-</sup> excretion of 200 mg. of NaCl per 10 min. per 10-kg. dog. Esidrone (Na salt of pyridinedicarboxymercurihydroxypropylamide, 0.5—3.0 mg. Hg per kg.) produced a max. excretion of 300 mg. of NaCl. Mercupurin (0.5—6.0 mg. of Hg per kg.) a max. excretion of 500 mg. of NaCl. Cardiac death occurred with esidrone after doses of 4 mg. of Hg per kg. M. W. G.

**Effect of prostigmine on urinary excretion of potassium in normal subject.** J. N. Cumings (*J. Neurol. Psychiat.*, London, 1941, **4**, 235—236).—Prostigmine had no effect on urinary excretion of K. H. L.

**Stimulating influence of sodium citrate on cellular regeneration and repair in the kidney injured by uranium nitrate.** G. L. Donnelly and R. L. Holman (*J. Pharm. Exp. Ther.*, 1942, **75**, 11—17).—The use of Na citrate facilitated repair and regeneration of the injured tubular epithelium which was damaged by U nitrate. 92% of the animals receiving a lethal dose of U nitrate recovered. H. H. K.

**Electrolytic [preparation of a] solution of sodium hypochlorite.**—See A., 1942, I, 333.

**Arrow poisons.** B. Witkop (*Angew. Chem.*, 1942, **55**, 85—90).—A review.

**Action of senecionine, integerrimine, jacobine, longilobine, and spartioidine, especially on liver.** P. N. Harris, R. C. Anderson, and K. K. Chen (*J. Pharm. Exp. Ther.*, 1942, **75**, 69—77).—Large doses caused rapid respiration, clonic convulsions, and death within a few min. in mice. The median lethal doses are given. The drugs produced necrosis of the liver, chiefly central, associated with sinusoidal congestion and hemorrhage into cell cords. Repeated administration of small sublethal doses was followed by hypertrophy of liver cells and their nuclei. The drugs lowered blood pressure of etherised cats, and inhibited peristaltic movements of isolated rabbit's intestines. Integerrimine, longilobine, and jacobine caused contractions of isolated guinea-pig's uterus. H. H. K.

**Action of monocrotaline and retronecine.** P. N. Harris, R. C. Anderson, and K. K. Chen (*J. Pharm. Exp. Ther.*, 1942, **75**, 78—82).—Median lethal doses were determined. Mice receiving near lethal doses of retronecine developed clonic convulsions, and then died immediately or recovered completely. Monocrotaline produced liver necrosis associated with sinusoidal congestion and hemorrhage into necrotic cell cords. Repeated administration of monocrotaline produced hypertrophy of liver cells. Monocrotaline inhibited isolated rabbit's intestines and lowered cat's blood pressure. Large doses of retronecine lowered cat's blood pressure and stimulated isolated guinea-pig's uterus. Small doses of retronecine inhibited isolated rabbit's intestines but large doses stimulated them. H. H. K.

**Toad poisons. Constitution of bufotalin etc.**—See A., 1942, II, 322.

**Action of isatidine, pterophine, and scleratine.** P. N. Harris, R. C. Anderson, and K. K. Chen (*J. Pharm. Exp. Ther.*, 1942, **75**, 83—88).—The drugs were obtained from various species of *Senecio*. Acute and chronic toxicity were studied in mice. Lethal doses produce liver necrosis in mice. Pterophine lowers cat's blood pressure, inhibits isolated rabbit's intestine, and contracts isolated guinea-pig's uterus. Scleratine and isatidine raise cat's blood pressure and stimulate isolated guinea-pig's uterus. H. H. K.

**Toxicology of sodium nitroprusside. I. Decomposition and determination of sodium nitroprusside.** H. E. Hill. **II. Toxicity of sodium nitroprusside for guinea-pigs.** L. W. Mahaffey (*J. Proc. Austral. Chem. Inst.*, 1942, **9**, 89—93, 93—94).—I. Only slight decomp. of Na nitroprusside occurs on steam-distillation with dil. H<sub>2</sub>SO<sub>4</sub> or tartaric acid. The CN group may be determined by digestion with NaOH, removal of Fe(OH)<sub>3</sub> by filtration, acidification with H<sub>2</sub>SO<sub>4</sub>, and steam-distillation with the addition of urea to remove liberated HNO<sub>2</sub>. Animal tissue and blood decompose Na nitroprusside with liberation of HCN, which may be recovered (98.8% with blood) by steam-distillation from acid solution. It is similarly decomposed by the animal system when given orally.

**II.** The toxicity (m.l.d.  $\frac{1}{4}$  grain) is approx. the same as that of KCN. The symptoms are similar but their onset and death are delayed. H. G. R.

**Acute hæmolytic anæmia following phenothiazine.** R. D. C. Johnstone (*Brit. Med. J.*, 1942, I, 259).—Non-fatal case report. C. A. K.

**Acute relapsing dermatitis due to nail varnish.** H. C. Semon (*Brit. Med. J.*, 1942, I, 146—147).—Case report. C. A. K.

**Excretion of selenium by rats on a seleniferous wheat ration.** H. D. Anderson and A. L. Moxon (*J. Nutrition*, 1941, **22**, 103—108).—Rats receiving seleniferous wheat excreted most of the Se in 2 weeks when transferred to a Se-free diet. A small portion of the Se was retained for some months. Storage of Se in the liver was similar in old and in young rats but in other parts of the body young animals stored relatively more. A ration containing 25 p.p.m. of Se depressed the body wt. A. G. P.

**Action of mustard gas on skin.** G. B. Frost and H. M. Gelly (*Pharm. J.*, 1942, **149**, 70—71).—The rate of formation of erythema following contamination with liquid mustard gas is const. for the individual, the rate of penetration for fair skin being approx 4 times that for dark, sallow skin. The max. rate of healing occurs in healthy types which tan easily. Healing is aided by an alkaline diet before and during vesiculation. H. G. R.

**Toxicity of sodium pentachlorophenoxide and pentachlorophenol to fish.** C. J. Goodnight (*Ind. Eng. Chem.*, 1942, **34**, 868—872).—Sodium pentachlorophenoxide (and the phenol) are toxic to 19 species of fish, 0.2—0.6 p.p.m. being fatal (threshold toxicities 0.94—3.0 according to the species). Small variations of temp. or  $p_H$  have little effect. Death occurs by capillary rupture and is preceded by excitement; if the initial concn. is low, fish migrate to clean water. Eggs of lake trout are resistant; these fish are most sensitive in the yolk sac stage immediately after hatching. Invertebrates are not affected. R. S. C.

**Pharmacological action of Korean *Inula helenium*, L.** C. Go (*Japan. J. Med. Sci.*, 1941, IV, **13**, 75—93).—*I. helenium* contains alanto-isoalanto-, and dihydroisoalanto-lactone. Their actions were studied in mice and rabbits, on isolated frog's heart, isolated rabbit's intestine, and earthworm muscle. H. H. K.

**Pharmacological action of *Sargassum thunbergii*, J. Ag.** C. Go (*Japan. J. Med. Sci.*, 1941, IV, **13**, 95—107).—Subcutaneous and oral administration of an aq. extract into mice and rabbits produced diminished movements, paralysis, and finally death without convulsions. 0.1—0.2% solution of the aq. extract contracted isolated rabbit's intestine. Oral administration of 0.2 g. of an aq. extract and 0.5 g. of an alcoholic extract had an anthelmintic effect in infected cats. H. H. K.

**Toxicity of cacao husks and their influence on toxicity of caffeine.** L. Millet (*Compt. rend.*, 1941, **213**, 591—593).—Aq. extracts of roasted cacao husks are only slightly toxic to guinea-pigs and increase the animals' tolerance to caffeine. W. C. J. R.

**Fumigants for disinfection of bedding and clothing.** G. C. Sherrard (*U.S. Publ. Health Repts.*, 1942, **57**, 753—759).—Fumigants tested were HCN, chloropicrin, methyl bromide, ethylene oxide-CO<sub>2</sub> mixture (1:9), and ethylene dichloride-CCl<sub>4</sub> mixture (3:1). The properties of each are discussed. C. G. W.

**Fern poisoning in sheep, goats, and cattle due to *Notholaena sinuata*, var. *crenata*.** F. P. Mathews (*Texas Agric. Exp. Sta. Bull.*, 1942, No. 611, 15 pp.).—A disease occurs in sheep, to a small extent in goats, and probably not at all in cattle and horses, called "jimmies" which was reproduced by feeding *N. sinuata*, var. *crenata*. The symptoms of trembling and sudden death, which are induced by walking the animal, are described. The toxic principle apparently

acts on the respiratory centre. It is transmitted by milk to suckling lambs and resists drying for 8 months. A. W. M.

**Preparation of acetylsalicyl and salicyl disulphides.**—See A., 1942, II, 312.

**New base for poison ivy protective ointment.** L. Schwartz, J. E. Dunn, and F. H. Goldman (*U.S. Publ. Health Repts.*, 1942, 57, 578—588).—Na perborate can detoxify the active principle of poison ivy in an acetone-aq. medium. Two new formulæ are presented for Na perborate ointment which retains its O for several weeks in a closed container, but liberates it when exposed to perspiration or water. C. G. W.

**Inhibition of aggravating action of olive oil on guinea-pig tuberculosis by ethyl succinate.** A. Berthelot, L. Nègre, and J. Bretey (*Compt. rend.*, 1941, 213, 90—91).—Subcutaneous injection of mixtures of olive oil and ethyl succinate prevents the aggravation of tubercular infection in the guinea-pig which is produced by olive oil and, in smaller degree, by ethyl succinate alone. Acidosis appears to play a part in the action. P. G. M.

## XXI.—PHYSIOLOGY OF WORK, AND INDUSTRIAL HYGIENE.

**Use of double work periods in study of fatigue and influence of caffeine on recovery.** E. Fodtz, A. C. Ivy, and C. J. Barborika (*Amer. J. Physiol.*, 1942, 136, 79—86).—Using the bicycle ergometer, the subjects worked to complete fatigue, rested 10 min., and worked to complete fatigue again. Double work periods yielded data less variable than single work periods. % recovery (under conditions described) is inversely related to the work output of the 1st period, correlation coeff. being 0.95. Caffeine—Na benzoate (0.5 g.) intravenously at the end of 1st work period increases % recovery or the output of work during the 2nd period. M. W. G.

**Heat exchanges during recovery from experimental deficit of body heat.** E. A. Pinson and E. F. Adolph (*Amer. J. Physiol.*, 1942, 136, 105—114).—Heat exchanges in 4 subjects (male) who sat unclothed in air at 31°, 25% R.H., were measured after injection of 1.4—1.7 l. of water at 1—3°, at short intervals, for 150—210 min. The body became cooler by 0.72 cal. per kg. when 1.5 l. of ice water were drunk. In 200 min. 85% of the initial heat debt was paid off. Half the heat regained came from reduction in loss by vaporisation of water, the other half from reduction in loss by variation and convection, concomitant with the diminution of surface temp. Heat produced by oxidation was not augmented. At 28° recovery was slower. Heart frequency was diminished after the water and ensuing water diuresis was sometimes delayed. M. W. G.

**Findings from major studies of fatigue.** R. R. Sayers (*U.S. Bur. Mines*, 1942, *Inf. Circ.* 7209, 46 pp.).—A review. The physical manifestations and physiological basis of fatigue are examined and the rôle of fatigue in causing accidents and lowered efficiency in industry is discussed. Shorter hr. of work, rest periods, and improvement in working conditions are of val. in preventing fatigue. J. H. B.

**Silicon in non-silicotic lungs: its relation to apical scars and to nodules.** G. J. McHefey (*J. Lab. clin. Med.*, 1942, 27, 1023—1035).—Anthracotic and siliceous dust deposits in the lung tissue increase with age; anthracosis in a lung gives a rough index to the relative amount of SiO<sub>2</sub> deposits present in the same lung. The average total SiO<sub>2</sub> content in the lungs of men is higher than that of women; the % of cases of apical scars increases with age and with the grade of the total SiO<sub>2</sub> content of the lung. Of 133 cases of apical scars, 97% had healed tuberculosis elsewhere in the lung or hilus lymph nodes. Of 200 consecutive cases 88.5% showed healed tuberculosis. The incidence of the silicotic nodule in the non-silicotic lung is 10.5%. (8 photomicrographs.) C. J. C. B.

**Occupational deafness: audiometric observations on aural fatigue and recovery.**—See A., 1942, III, 683.

## XXII.—RADIATIONS.

**Effect of Roentgen rays on action of tyramine, phenylethanolamine, and sympathol on blood-sugar.**—See A., 1942, III, 708.

**Effect of X-irradiation on formation and storage of bile and glycogen in liver cells of rats.**—See A., 1942, III, 610.

**Susceptibility of amphibian red cells to X-rays.**—See A., 1942, III, 571.

**Effect of X-rays on *Arbacia* egg.**—See A., 1942, III, 567.

**Radiation effects on nervous system and Roentgen pigmentation of gold-fish.**—See A., 1942, III, 672.

**Survival of mice of different strains and ages after a single, total irradiation by X-rays.** N. Dobrovol'skaia-Zavad'skaia, S. Verétnikov, and M. Rodzévitch (*Compt. rend.*, 1941, 213, 704—706).—Male animals are less resistant than females and the time of survival

is not proportional to the dose, a time-lag occurring before the fatal effects are observed. Age and strain have a considerable influence, females of middle age exhibiting the longest survival. H. G. R.

**Measurement of radiation for medical purposes.** W. V. Mayneord (*Proc. Physical Soc.*, 1942, 54, 405—421).—A survey of the ionisation method of measuring X-rays and  $\gamma$ -rays, the fundamental nature of the röntgen, the distribution of radiation, the variation of dosage-rate in typical cases along the axis of a beam sent into soft tissues, and hence the study of "isodose surfaces" and simple geometrical methods of obtaining them. The "integral dose" is obtained by integrating, throughout the whole mass, the product of the dose and element of mass, and simple geometrical and analytical methods of deducing the quantities are described. N. M. B.

**Continuous dosage measurement in deep X-ray therapy.** F. T. Farmer (*Brit. J. Radiol.*, 1942, 15, 203—208).—Errors of 5—10% are made by using a dosimeter such as the Mekapion in the base of applicators due to the variation possible in instrument-skin distance. The use of a dosimeter fixed to the window of the tube with calibrations for different focus-skin distances and applicators is advocated and such an instrument is described. E. M. J.

**Volume dosage in deep X-ray therapy.** F. Ellis (*Brit. J. Radiol.*, 1942, 15, 174—177, 194—201).—Vol. dose in r. c.c. or ergs per cm.<sup>2</sup> per r. is measured by the product of the surface dose in r. with back scatter, the max. skin dosage in r. per min. obtainable under standard conditions divided by the skin dosage rate for the field size used, the area of the field in sq. cm., and a graph relating vol. dose per r. at skin surface to thickness of tissue through which the beam passes. Vol. dose during the course of various treatments is correlated with clinical findings and blood counts at a certain stage. Histaminase was found useful in the treatment of general radiation reactions. E. M. J.

**Representation of deep X-ray therapy beams by isodose charts.** D. E. A. Jones (*Brit. J. Radiol.*, 1942, 15, 178—184).—Retention of the identity of the primary beam and the existence of a sharp delimiting edge in a wax phantom is shown by radiographs and ionisation measurements. At the edge there is a sharp drop in dosage rate. E. M. J.

**Angiomatosis retinae. Results of X-ray treatment.**—See A., 1942, III, 681.

**Treatment of deafness by irradiation (radium).** See A., 1942, III, 683.

**Iodinated organic compounds as contrast media for radiographic diagnoses. I. Iodinated aracyl esters.**—See A., 1942, II, 311.

**Use of slow neutrons for cancer therapy. Radioactive phosphorus in malignant neoplastic disease. Results of radiotherapy in benign giant-cell tumour of bone.**—See A., 1942, III, 616.

**Penetration of ultra-violet radiation into skin as a factor in carcinogenesis.**—See A., 1942, III, 696.

**Extra strong heliotropic effect of neon lights.** C. N. Ray (*Science*, 1941, 94, 585—586).—Insects which previously swarmed around street lights now swarm around Ne lighting, recently installed. The use of Ne lights in eradicating crop pests is suggested. E. R. S.

**Ultra-violet radiation intensities in high latitudes.**—See A., 1942, I, 310.

## XXIII.—PHYSICAL AND COLLOIDAL CHEMISTRY.

**Heats of organic reactions. XII. Reaction between methæmoglobin and salicylate.** R. M. Roberts (*J. Amer. Chem. Soc.*, 1942, 64, 1472—1475).—Heats of reaction between methæmoglobin and Na salicylate at  $p_H$  7.3 increase with salicylate concn. up to 0.8M., suggesting a stoichiometric reaction in which H-bond formation between salicylate and side-chains of methæmoglobin contributes to the heat of reaction. The heat of reaction between Na salicylate and carboxy-hæmoglobin at  $p_H$  7.3 has also been measured. The state of oxidation of the hæmoglobin-Fe does not affect the val. of heat of reaction at a given salicylate concn. No relationship exists between heats of reaction and absorption spectra. W. R. A.

**Oxidation-reduction potential of the system methæmoglobin-ethyl hydrogen peroxide.** M. Polonovskii, M. F. Jayle and G. Fraudet (*Compt. rend.*, 1941, 113, 740—742).—Curves plotted from results of experiments with methæmoglobin and ethyl H peroxide at 1° and varied  $p_H$  indicate that the Fe<sup>III</sup> of methæmoglobin is converted by the peroxide into Fe<sup>IV</sup> in an unstable methæmoglobin derivative, the peroxidase activity being due to the change in valency. The Fe of the co-enzyme of plant peroxidases probably undergoes the same change. W. McC.

**Hydrogen-ion dissociation curve of  $\beta$ -lactoglobulin.**—See A., 1942, III, 606.

**Absorption measurements with biological liquids (blood) in the wave-length range 50—100 cm.** G. Gsell (*Physikal. Z.*, 1942, 43, 101—107).—A method of determining the electrical conductivity

of liquids which are good conductors is described. It employs a Lecher system and can be used in the  $\lambda$  range 50—100 cm. The method has been used with blood. A. J. M.

**Light absorption by bilirubin solutions.** M. Roy and A. Boutaric (*Compt. rend.*, 1941, 213, 189—191).—Spectroscopic examination at  $\lambda$  440—520 of solutions of bilirubin in  $\text{CHCl}_3$  and aq.  $\text{NaOH} + \text{KCl} + \text{H}_3\text{BO}_3$  + serum-albumin (as protective colloid for stabilisation) and at  $\lambda$  440—500 of dil. buffered ( $p_{\text{H}}$  7.4) horse serum show that bilirubin solutions do not obey Beer's law even approx. since the ratio of optical density to concn. first increases to a max. and then decreases but that solutions of serum obey the law very approx. since the optical density varies approx. inversely with the dilution coeff. W. McC.

**Electrophoretic properties of serum-proteins. II. Fractions of crystalline horse serum-albumin.** D. G. Sharp, G. R. Cooper, J. O. Erickson, and H. Neurath (*J. Biol. Chem.*, 1942, 144, 139—147; cf. A., 1942, III, 416).—Electrophoretic examination of Kekwick's fractions A and B of serum-albumin shows that the albumin is complex, and the fractionation is not due to changes during purification. The mobility of fractions A and B is the same at all  $p_{\text{H}}$ . Denaturation of serum-albumin is irreversible. R. L. E.

**Effect of urea on electrophoretic patterns of serum-proteins.**—See A., 1942, I, 297.

**Calcium-protein relationship studied with the ultracentrifuge. I. Calcium caseinogenate solutions.** A. Chanutin, S. Ludewig, and A. V. Masket. **II. Serum.** S. Ludewig, A. Chanutin, and A. V. Masket. **III. Augmentation of serum with calcium and phosphate.** A. V. Masket, A. Chanutin, and S. Ludewig (*J. Biol. Chem.*, 1942, 143, 737—751, 753—761, 763—769).—I. Ca caseinogenate has a dissociation const. independent of concn. or of  $p_{\text{H}}$  between 6.3 and 8.5, but varying with different preps. of caseinogen, and increased in aq.  $\text{NaCl}$ . Addition of citrate increases diffusible Ca and decreases  $[\text{Ca}^{++}]$ , confirming the applicability of the law of mass action to this system.

II. The relation between Ca and total serum-protein is irregular, and is not represented by a single mass law equation. Total diffusible Ca is const. in all centrifuged fractions of a single serum, is increased by Na citrate, which also decreases the Ca bound to protein, and is decreased irregularly by a rise in  $p_{\text{H}}$ .

III. Addition of Ca causes irregular variations in the distribution of protein on centrifuging. Horse serum normally contains a phosphate-protein complex. Addition of Ca or  $\text{PO}_4^{---}$  causes sedimentation of a  $\text{Ca-PO}_4^{---}$  complex. R. L. E.

**Denaturation of proteins and its apparent reversal. IV. Enzymic hydrolysis of native, denatured, and apparently reversibly denatured proteins.** F. Bernheim, H. Neurath, and J. O. Erickson (*J. Biol. Chem.*, 1942, 144, 259—264; cf. A., 1942, III, 416).—The rates of hydrolysis of native, irreversibly denatured, and so-called "regenerated" horse serum-albumin and -pseudoglobulin by trypsin are determined. With both proteins, proteolysis proceeds more slowly with native than with irreversibly denatured material, and the extent of proteolysis is greater with the albumin than with the globulin fraction. The rates of proteolysis of irreversibly denatured and regenerated serum-albumin are the same, indicating that the latter is essentially in a denatured state. With the pseudoglobulin, the rate of digestion of regenerated protein is less than that of the irreversibly denatured, and only somewhat greater than that of native protein. The results are discussed in relation to structural differences between serum-albumin and -pseudoglobulin. J. N. A.

**Influence of hydrotropic substances on the colloidal properties of serum.** C. Wunderly (*Kolloid-Z.*, 1942, 98, 76—82).—The peptisation of prontosil rubrum by normal serum and by various hydrotropic substances was studied by colorimetric examination of a treated prontosil sol after passing through a filter with pore diameter  $1.5 \mu$ , which completely retains untreated prontosil. Serum alone peptises prontosil more effectively than does any of the other agents, and when used in conjunction with the latter the effect is additive, except when the auxiliary agent is *l*-tyrosine, with which the effect is more than additive. The peptising action of serum on acridine-red 3B, trypanflavine, and acridine-orange NO increased in that order. The coagulating concn. of  $\text{CaCl}_2$  for serum is not changed by the addition of physiological amino-acids or the Na salts of some aromatic acids, but the dispersity of the serum-proteins, as revealed by nephelograms, is characteristically altered, especially by Na benzenedisulphonate. F. L. U.

**Native proteins, flexible frameworks, and cytoplasmic organisation.** D. Wrinch (*Nature*, 1942, 150, 270—271).—A discussion of the nature of the association of individual protein units in the protein framework, the units lying at various distances apart up to a fixed max. and in a variety of orientations. The nature of biologically active membranes is considered from this viewpoint. A. A. E.

**Critical peptisation temperatures of zein.**—See A., 1942, I, 327.

**Origin and reversal of certain surface structures of alga zoöspores.** E. C. Teodoresco and E. Angelescu (*Kolloid-Z.*, 1941, 97, 216—223).—

The formation of definite patterns of alga zoöspores on the surface of and under water is ascribed to the presence of cybotactic groups. Substances (e.g.,  $\text{O}_2$ ) liberated by the zoöspores change the surface tension of water and lead to the ultimate break-up of the patterns. C. R. H.

## XXIV.—ENZYMES.

**Effect of deuterium oxide on action of some enzymes.** K. P. Jacobsohn, J. Tapadinhas, and M. Soares (*Arch. Port. Sci. Biol.*, 1938, 4, 111—125).— $\text{D}_2\text{O}$  decreases the reaction velocity in the following enzymic systems: phosphoric ester-phosphoesterase; fumaric acid-fumaric hydratase; *l*-aspartate-fumaric aminase. The reaction velocity in the system K *l*-malate-malic dehydrogenase is increased in the presence of  $\text{D}_2\text{O}$ . The final equilibrium of the enzymic reactions is not altered by  $\text{D}_2\text{O}$ . I. C.

**Localisation of enzymes in nerve fibres.**—See A., 1942, III, 672.

**Hydrolysis of *d*-tripeptides by serum enzymes.** H. Herken, R. Merten, and A. Schmitz (*Naturwiss.*, 1942, 30, 226—227).—Relative amounts of hydrolysis of the *d*-components of *dl*-alanyl-, aminobutyryl-, and leucyl-glycylglycine by serum enzymes are determined. A. T. P.

**Hydratases and vitamins.** K. P. Jacobsohn and A. da Cruz (*Bull. Soc. Port. Sci. Nat.*, 1940, 13, 131—133).—Aneurin, lactoflavin, and vitamin-C do not influence the enzymic activities of  $\alpha$ - and  $\beta$ -aconitases and of fumarico-aminase. I. C.

**Mechanism of the ascorbic acid-ascorbic acid oxidase reaction. Hydrogen peroxide question.** H. G. Steinman and C. R. Dawson (*J. Amer. Chem. Soc.*, 1942, 64, 1212—1219).—Highly purified  $\text{P}_2\text{O}_7^{---}$ , and less well  $\text{P}_2\text{O}_7^{---}$ -citrate, buffers inhibit, atm. oxidation of ascorbic acid, even in presence of  $\text{Cu}^{++}$ , probably by formation of complex Cu ions, but oxidation is rapid in  $\text{PO}_4^{---}$  or acetate buffers.  $\text{P}_2\text{O}_7^{---}$  is, therefore, used in studies recorded below.  $\text{H}_2\text{O}_2$  does not react with ascorbic acid with or without peroxidase; it does react with dehydroascorbic acid, faster at  $p_{\text{H}}$  7 than at  $p_{\text{H}}$  5, but the acid disappears the faster so that absence of  $\text{H}_2\text{O}_2$  when dehydroascorbic acid is formed is evidence against simultaneous formation of  $\text{H}_2\text{O}_2$ ; the reaction rate is the same whether the dehydroascorbic acid is formed by *p*-benzoquinone or enzymically, proving that  $\text{H}_2\text{O}_2$  is not formed in the oxidations. Non-formation of  $\text{H}_2\text{O}_2$  is confirmed by identity of the rate of oxidation, consumption of  $\text{O}_2$  (1 mol.), and amount of dehydro-acid formed when ascorbic acid is oxidised in presence of catalase-free ascorbic acid oxidase (optimum  $p_{\text{H}}$  5.6—7.0) in presence or absence of catalase (sufficient to survive reaction); all three criteria are, however, altered by presence of catalase when oxidation is effected by  $\text{Cu}^{++}$  catalysis, which is known (and thus confirmed) to produce  $\text{H}_2\text{O}_2$ . Methæmoglobin inhibits inactivation of ascorbic acid oxidase during the reaction (but not the initial rate of oxidation), the effect not being enzymic (unaffected by boiling) or due to coupled oxidation (no catalytic effect). Quercitin is without effect on the enzymic oxidations. R. S. C.

**Effect of amine oxidase on hypertension.**—See A., 1942, III, 669.

**Titration of liver-catalase activity of normal and tumour-bearing rats and mice.**—See A., 1942, III, 698.

**Carbonic anhydrase. II. Zinc in relation to carbonic anhydrase activation and inactivation.** E. R. Main and A. Locke (*J. Biol. Chem.*, 1942, 143, 729—736).—Carbonic anhydrase is activated by adrenaline, pyrocatechol, and pyrogallol, but not by various related substances, and also by  $\text{P}_2\text{O}_7^{---}$ , cysteine, and diethyldithiocarbamate.  $\text{PO}_3^{---}$  is slightly active. These effects are inhibited by Zn, sulphide, dithione, and sulphanilamide. The activation by pyrocatechol and histamine is additive, but neither increases the effect of  $\text{P}_2\text{O}_7^{---}$ . The intensifying activity of these substances is paralleled by their protective effect against Zn pptn. of the enzyme at  $p_{\text{H}}$  7.4. R. L. E.

**Carbonic anhydrase in mammalian ocular tissues.**—See A., 1942, III, 680.

**Regional and seasonal variations in serum-choline-esterase of man and dogs.**—See A., 1942, III, 576.

**Choline-esterase of choroid plexus and ciliary processes. Distribution of enzymes in choroid plexus.**—See A., 1942, III, 680.

**Enzymic conversion of radioactive sulphide-sulphur into cysteine-sulphur.** C. V. Smythe and D. Halliday (*J. Biol. Chem.*, 1942, 144, 237—242).—When cysteine and  $\text{H}_2^{35}\text{S}$  are added to an enzyme prep. that converts cysteine into pyruvic acid,  $\text{NH}_3$ , and  $\text{H}_2\text{S}$ , the cysteine that remains after enzymic reaction has proceeded for a time contains appreciable amounts of  $^{35}\text{S}$ . The mechanism of the reaction is unknown. J. N. A.

**Adenosine deaminase.** T. Brady (*Biochem. J.*, 1942, 36, 478—484; cf. A., 1941, III, 1022; Conway, A., 1939, III, 518).—The extraction and purification of the enzyme from a rich source (superficial mucosa of calf's intestine) are described. The dry material retains its activity in the cold for at least 1 year.  $\text{Cu}^{++}$ ,  $\text{Ag}^{++}$ ,  $\text{Cr}^{+++}$ , I, and  $\text{KMnO}_4$  inactivate the enzyme, but other org. (e.g., iodoacetate, urethane, chloral hydrate) and inorg. (e.g.,  $\text{Co}^{++}$ ,  $\text{Ni}^{++}$ ,  $\text{NaCN}$ ,  $\text{NaF}$ ,  $\text{Na}_2\text{S}_2\text{O}_8$ ,



Other analogues (e.g.,  $\gamma$ -hydroxy-butryl- and -valeryl- $\beta$ -alanine) inhibit the growth of those micro-organisms which synthesise and hence do not require added pantothenate. Possibly the inhibitions result from displacement, by the analogues, of pantothenate from essential enzymes which are consequently prevented from functioning.

W. McC.

**Chemical reactions between inhibitors of bacterial growth and constituents of bacteriological media.** A. E. Oxford (*Biochem. J.*, 1942, **36**, 438—444).—Incubation with sterile broth or 1% aq. peptone at  $p_H$  7 and 37° for 1—3 days diminishes the bacteriostatic power of penicillic acid and 4-methoxytoluquinone. Simple amines (e.g., methylamine) and amino-acids (e.g., glycine, alanine, *p*-aminobenzoic acid) also diminish the power. These compounds react with *p*-methoxytoluquinone also, producing coloured compounds that are not less anti-bacterial than is the quinone itself. The concn. of compounds which decrease the anti-bacterial activity of penicillic acid is diminished when heart broth containing peptone is autoclaved with 2% glucose at  $p_H$  7.2. (Cf. A., 1942, III, 490).

W. McC.

**Bacterial inhibition by metabolite analogues. III. Pantoyltaurine. Antibacterial index of inhibitors.** H. McIlwain (*Brit. J. exp. Path.*, 1942, **23**, 95—102).—Pantoyltaurine (R-SO<sub>3</sub>H) is related to pantothenic acid (R-CO<sub>2</sub>H), a substance essential to many pathogenic bacteria, in the same way as sulphanilamide is related to *p*-aminobenzoic acid. Pantoyltaurine inhibits the *in vitro* growth of hæmolytic streptococci, pneumococci, and *C. diphtheria* and apparently acts by becoming attached to the bacteria where pantothenate must react for normal growth.

F. S.

**Mechanism of *p*-aminobenzoic acid action and parallel effects of ethyl carbamate (urethane).** F. H. Johnson (*Science*, 1942, **95**, 104—105).—The stimulatory and inhibitory effects of *p*-aminobenzoic acid, sulphanilamide, and urethane on bacterial growth and luminescence are related to the action of narcotics, and do not involve structural similarity between mols. of the inhibitor and the intermediary of normal metabolism in the cell.

E. R. S.

**Failure of certain growth factors to inhibit sulphonamide bacteriostasis. Action of sulphonamides on bacterial cell.**—See A., 1942, III, 625.

**Synergistic action of sulphamido-compounds and azochloroamide on pathogens *in vitro*.**—See A., 1942, III, 626.

**Surface behaviour of antibacterial substances. I. Sulphanilamide and related substances.**—See A., 1942, III, 639.

**Mode of action of sulphonamides *in vitro*.**—See A., 1942, III, 704.

**Growth stimulation by sulphanilamide in low concentration.**—See A., 1942, III, 705.

**Trypsin as digestant of sputum and other body fluids preliminary to examination for acid-fast bacilli.** E. Haynes (*J. Lab. clin. Med.*, 1942, **27**, 806—809).—A satisfactory method of digesting sputum and other body fluids with trypsin preliminary to microscopic examination, culturing, and guinea-pig inoculation is described. In a comparison of this method and the flocculation method of Hanks (*ibid.*, 1938, **23**, 736) on 100 positive specimens, 11% more positives were found by the trypsin method. In 71% of the smears made from the trypsin-digested portion of the specimens, more acid-fast bacilli were found than in smears made from the flocculation concn. and in 30% of these at least twice as many bacilli were found. Only 28% of the smears made from the flocculation concn. showed more bacilli than those made from the trypsin-digested portion of the specimen, and of these, only 7% showed twice as many or more acid-fast bacilli.

C. J. C. B.

**Simplified medium for pathogenic organisms.** N. Grossowicz and I. J. Kligler (*Amer. J. Publ. Health*, 1942, **32**, 745—747).—The medium consists of a peptone-salt solution and tomato juice mixture to which agar or blood may be added. It may be used for all the usual bacteriological examinations and is more efficient than ordinary agar.

C. J. C. B.

**Sneezing and disinfection by hypochlorites.** R. B. Bourdillon, O. M. Lidwell, and J. E. Lovelock (*Brit. Med. J.*, 1942, I, 42—44).—Tests in a closed room showed that a sneeze may liberate about 100,000 bacteria-carrying particles of which 4% remain suspended in still air for 30 min. Almost all the bacteria can be killed in 3—4 min. by a spray of 1% NaOCl solution (2.1 c.c. per 1000 cu. ft. of air), though the activity is reduced by low R.H. or high content of org. matter in the air.

C. A. K.

**Sterilisation of rubber gloves.** D. A. Sandford and H. A. Cookson (*Brit. Med. J.*, 1942, I, 412—413).—Autoclave experiments on sterilisation of rubber gloves suggest that, with elimination of air from the chamber, steam might sterilise at atm. pressure with less deterioration of rubber.

C. A. K.

**Action of high-frequency sound waves on bacteria.**—See A., 1942, III, 639.

**Symbiosis of leguminous plants and nodule bacteria. II. Excretion of nitrogenous substances from nodules.**—See A., 1942, III, 653.

**Mechanism of biological nitrogen fixation. IX. Properties of hydrogenase in *Azotobacter*.** J. B. Wilson, S. B. Lee, and P. W. Wilson. **X. Hydrogenase in cell-free extracts and intact cells of *Azotobacter*.** S. B. Lee, J. B. Wilson, and P. W. Wilson (*J. Biol. Chem.*, 1942, **144**, 265—271, 273—281; cf. A., 1942, III, 421).—IX. Using O<sub>2</sub> as H acceptor, the concn. of washed cells of *Azotobacter* is an important factor in the determination of hydrogenase activity, and must be carefully controlled. The greater is the cell concn., the higher is the optimum partial pressure of O<sub>2</sub>. There is no difference in the rate of uptake of H<sub>2</sub> within the range of H<sub>2</sub> partial pressure from 0.1 to 0.95 atm.; below 0.1 atm., the activity decreases sharply. The optimum temp. is 40° and  $p_H$  7.3—7.8, the activity decreasing much more rapidly at alkaline than at acid reactions. Although the optimum  $p_H$  region is the same as that for nitrogenase, the function as a whole is quite different, for at  $p_H$  6.0 the activity of nitrogenase decreases suddenly to zero, whilst that for hydrogenase decreases only by approx. 30%. A method for study of hydrogenase in *Azotobacter* and probably other N-fixing organisms is described.

X. The prep. of a cell-free enzyme extract of *Azotobacter* that contains an active hydrogenase capable of transferring H<sub>2</sub> to methylene-blue or O<sub>2</sub> is described. The uptake of gas in a mixture of H<sub>2</sub> and O<sub>2</sub> by the cell-free extract or a resting suspension consists primarily in the oxidation of H<sub>2</sub> to water. CN<sup>-</sup> and NaN<sub>3</sub> inhibit the oxidation. Hydrogenase activity in *Azotobacter* growing on a medium containing NH<sub>4</sub>NO<sub>3</sub> is considerably less than that of cells which are fixing N. N<sub>2</sub> does not inhibit oxidation of H<sub>2</sub> by cell-free extracts or cell suspensions. The probable relationship between hydrogenase and biological N-fixing systems is discussed.

J. N. A.

**Dissociation constant in nitrogen by *Azotobacter*.** P. W. Wilson, R. H. Burris, and C. J. Lind (*Proc. Nat. Acad. Sci.*, 1942, **28**, 243—250).—Four methods for determining the dissociation (Michaelis) const. of N fixation by *Azotobacter vinelandii* are compared. The microspirometer method gives on the whole the most reliable results. The probable val. of  $K_{N_2}$  based on these experiments is 0.02 ± 0.005 atm. The corresponding val. for the dissociation const. of the enzyme inhibitor complex  $K_{H_2}$  is 0.11 ± 0.028 atm.

H. M. J.

**Growth-stimulating substances from *Lactobacillus casei*.** R. E. Feeny and F. M. Strong (*J. Amer. Chem. Soc.*, 1942, **64**, 881—884).—Measurement of growth of *L. casei* during 24 hr. permits detection of effects masked later by pantothenic acid. The active ingredients in yeast are *l*-asparagine, adenine, and guanine. Glutamine and glutamic acid are also effective, *l*-asparagine + glutamic acid showing an enhanced effect. Min. and max. effective concns. per 100 c.c. of medium are: glutamine 1, 100—200, glutamic acid 1000, 5000, *l*-asparagine 100, 5000, adenine = guanine more than 10, 100  $\mu$ g. Glutamine produces its effect very rapidly. Aspartic acid is inhibitory. Numerous substances have no effect. During assay of pantothenic acid, 2.5 mg. of *l*-asparagine or 10<sup>-4</sup> g. of glutamine replaces 25 mg. of yeast, but adenine and guanine then act as inhibitors. During determination of riboflavin, adenine, guanine, and pantothenic acid have no effect. Published methods for microbiological assay of pantothenic acid and riboflavin remain valid, as the above-named effects require high concns. of addenda.

R. S. C.

**Glutamine and glutamic acid as growth factors for lactic acid bacteria.** M. A. Pollack and M. Lindner (*J. Biol. Chem.*, 1942, **143**, 655—661).—The bacteria, some strains of which require much more acid than amide although others respond equally well to both substances, probably require them for construction of cell proteins. It is unlikely that the acid is utilised through the amide, since the addition of NH<sub>4</sub> has little effect and none of the bacteria can produce glutamine from glucose and NH<sub>4</sub> salts.

H. G. R.

**Dissimilation of phosphoglyceric acid by *Escherichia coli*.** M. F. Utter and C. H. Werkman (*Biochem. J.*, 1942, **36**, 485—493).—Cell-free extracts of *E. coli* contain the enzymes that convert phosphoglyceric into phosphopyruvic acid, and transfer PO<sub>4</sub><sup>'''</sup> from the latter to added adenylic acid, or partly to glucose via adenylic acid with formation of an unidentified hexose mono- or di-phosphate. Hexose monophosphate cannot replace glucose as PO<sub>4</sub><sup>'''</sup> acceptor. 0.005M-MgCl<sub>2</sub> or -MnSO<sub>4</sub> stimulates the conversion of (-)3-phosphoglyceric acid into (+)2-phosphoglyceric acid (by phosphoglyceromutase) and subsequent formation of phosphopyruvic acid (by the enolase). NaF and, to a smaller extent, Na oxalate (0.04M.) inhibit the enolase.

P. G. M.

**Metabolism of strict anaerobes. VIII. Metabolism of amino-acids by *Cl. welchii*.** D. D. Woods and A. R. Trim (*Biochem. J.*, 1942, **36**, 501—512).—Only 5 out of 21 amino-acids examined are attacked by *Cl. welchii*, viz., serine, threonine, cysteine, cystine, and arginine. The first four are broken down with formation of NH<sub>3</sub>, CO<sub>2</sub>, and (except cystine) H<sub>2</sub>; co-enzymes other than co-enzymes I and II and flavine-adenine dinucleotide are involved. Arginine is decomposed with formation of NH<sub>3</sub> and CO<sub>2</sub>, but no H<sub>2</sub> is formed and no co-enzyme is involved; it is not attacked by *Cl. tetanomorphum*.

P. G. M.

**Immunisation against diphtheria with alum-precipitated toxoid.** F. Fulton, B. Moore, J. Taylor, A. Q. Wells, and G. S. Wilson (*Brit. Med. J.*, 1942, I, 315—320, 349—352).—Inoculations of 0.1 and

0.3 c.c. of alum-pptd. toxoid (A.P.T.) against diphtheria were given at an interval of 4–6 weeks to 30,000 children during a 2-year period. Schick tests done 8–16 weeks after the 2nd injection showed a conversion rate of 75–80% positive to 70–99.5% negative (varying with the batch of toxoid used). 1 year later 966 children still showed 91% Schick-negative. It is recommended that the initial dose of A.P.T. be increased to 0.2 c.c. and the 2nd dose to 0.5 c.c. Initial immunisation should be done at 1 year, and further single doses of 0.3–0.5 c.c. of A.P.T. given at 5 years and 10 years to maintain immunity. C. A. K.

**Initial response to immunisation with diphtheria and tetanus alum toxoid.** W. C. Deamer, G. Bates, and F. S. Smyth (*J. Pediat.*, 1942, 20, 169–181).—Over 600 infants were given combined diphtheria and tetanus alum toxoid. The majority received 3 injections at intervals of 1 month each. Where the interval was longer, a higher tetanus antitoxin response occurred. The variation in response was very great, but all of the 240 cases in which it was determined had over 1/100 of a unit of antitoxin following this primary immunisation procedure. 96% negative Schick tests were obtained. The infants tolerated the antigen well. C. J. C. B.

**Combined immunisation of infants against diphtheria, tetanus, and whooping cough.** J. H. Lapin (*Amer. J. Dis. Child.*, 1942, 63, 225–237).—78 infants, 6–9 months of age, were given a combined immunisation with diphtheria toxoid, alum-pptd. tetanus toxoid, whooping cough vaccine, and whooping cough toxin in 5 monthly injections. Diphtheria immunisation (1 c.c., 1 c.c., and 0.5 c.c.) resulted in negative Schick reactions for 100% of the subjects, 97% having above 1/250 unit of antitoxin per c.c. of blood and 91% having above 1/10 unit per c.c. Tetanus immunisation (0.5, 0.5, 0.5 c.c.) resulted in an increase of the average antitoxic level to 0.50 unit, with 93% of the subjects having 3 months after immunisation an antitoxic level greater than is attained by the usual prophylactic injection of tetanus antitoxin. Whooping cough immunisation (10<sup>11</sup> in all and 5 doses of 0.1 c.c. of toxin) resulted in positive reactions to agglutination tests for 100% of the subjects, complement fixation of 4 plus for 87 and positive for 97%, and a positive mouse protection titre for 87%. C. J. C. B.

**Toxins of dysentery bacillus.** T. Wagner-Jauregg and E. Helmert (*Angew. Chem.*, 1942, 55, 21–24).—After dried Shiga-Kruse bacilli have been extracted with 0.85% aq. NaCl containing Na<sub>2</sub>CO<sub>3</sub> at pH approx. 7.2 successive treatment with dil. trichloroacetic acid and, after dialysis, alcohol results in elimination of toxin and separation of two endotoxins, one containing N 4.8–5.0 and a more toxic one containing 6.0–9.5%. Possibly the endotoxins are derived from a native endotoxin, the less toxic being the more denatured of the two. Slight increase in toxicity of the less toxic substance is achieved by adsorption on Al<sub>2</sub>O<sub>3</sub>, elution with aq. Na citrate, and dialysis. The endotoxin is a phospholipin-carbohydrate-poly-peptide complex but the phospholipin component is not essential for activity. The toxin, which is a protein, after pptn. with trichloroacetic acid is best purified by fractional pptn. with (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub>. W. McC.

**Use of SS (*Shigella-Salmonella*) agar for isolation of Flexner dysentery bacilli from faeces.** H. M. Rose and M. H. Kolodny (*J. Lab. Clin. Med.*, 1942, 27, 1081–1083).—SS agar is as efficient as deoxycholate-citrate agar for the isolation of Flexner dysentery bacilli. The medium is superior to deoxycholate-citrate agar for inhibition of coliform bacteria, colony differentiation, and growth-promoting qualities for the pathogens. The combination of SS agar and a suitable non-selective medium furnishes a reliable method for the isolation of the intestinal pathogens. C. J. C. B.

**Recovery of antibody from immune precipitate of type B Friedländer bacillus.** S. Liu and H. Wu (*Proc. Soc. Exp. Biol. Med.*, 1942, 49, 381–383).—The method used for pneumococcus (A., 1940, III, 771) can also be applied to Friedländer bacillus. V. J. W.

**Technique for isolation of *Bact. granulosis* (*Noguchia granulosis*).** C. Weiss (*Amer. J. Clin. Path. Tech. Suppl.*, 1942, 6, 19–21).—*Staphylococcus albus* or *aureus* can kill many *Noguchia granulosis* varying in age from 6 to 72 hr. Aq. solutions of gentian-violet or Victoria-blue in concns. of 1:10,000 will, after 15 min., inhibit the growth of staphylococci without affecting proliferation of cultures of *N. granulosis*. C. J. C. B.

**C factor as requirement for growth of *Haemophilus gallinarum*.** J. P. Delaplane and H. O. Stuart (*J. Agric. Res.*, 1941, 63, 29–30).—*H. gallinarum* grew well in boiled but not in autoclaved chick serum and plasma preps. In boiled media the growth factors are associated with the coagulated proteins. *H. influenzae* grew in boiled citrated serum and plasma preps. but not in bacterial filtrates from these materials. The C factor (Kessens) is probably of a physical rather than a chemical nature. A. G. P.

**Action of germicides on meningococcus.**—See A., 1942, III, 629.

**Nuclear apparatus and sexual mechanism in a micrococcus.** C. C. Lindgren (*Iowa State Coll. J. Sci.*, 1942, 16, 307–318).—A method employed in staining and microscopically examining the internal structures of a non-acid-fast diplococcus-tetrad is described and

reasons are given for supposing that these structures play a part in a sexual mechanism. W. McC.

**Paratyphoid of pigeons.** H. C. Gauger, R. E. Greaves, and F. W. Cook (*N. Carolina Agric. Exp. Sta. Tech. Bull.*, 1940, No. 62, 71 pp.).—Paratyphoid in pigeons is caused by *S. typhi-murium*, var. *binus*, which could be recovered from the faeces and mouth fluids of all serologically positive birds and also from the blood of squabs. The tube agglutination test was unreliable in detecting infected birds. Live autogenous antigens were superior to autogenous but treated antigens in detecting agglutinins. Haematological studies could not distinguish chronic carriers from healthy non-infected birds. The pathogen could apparently be transmitted through the ovum, since eggs from serologically positive mated birds contained the organism. A. W. M.

**Immunity to whooping cough judged by skin test on rabbits.** J. H. Lapin (*J. Pediat.*, 1942, 20, 161–168).—Rabbits injected with 200–800 billion of *H. pertussis* vaccine within 1 month are protected from the skin-necrotising effect of the intradermal injection of 0.2 c.c. of a 2 billion to the c.c. "unwashed" saline suspension of live *H. pertussis* organisms. Of animal immune sera, horse serum gives no protection against the living bacteria on injection into a rabbit; rabbit serum very slight. Human immune serum, prepared by injecting a human being with a previous history of whooping cough with a trillion of *H. pertussis* vaccine within a period of 1 month, gives complete protection. Human convalescent serum gives moderate protection. Haemorrhagic areas with marked infiltration form, but the typical end-point of necrosis, ulceration, and scab formation is missing. C. J. C. B.

**Use of alum-treated pertussis vaccine, and of alum-precipitated combined pertussis vaccine and diphtheria toxoid, for active immunisation.** P. L. Kendrick (*Amer. J. Publ. Health*, 1942, 32, 615–626).—The results of both methods were good as shown by antitoxin production or opsonocytaphagic tests. C. J. C. B.

**Slide agglutination in detecting pertussis antibodies.** L. Mishulow (*J. Lab. Clin. Med.*, 1942, 27, 792–796).—More positives are obtained on slide than on tube agglutination in detecting pertussis antibodies. An antigen preserved with merthiolate and stored at 8–10° gave as good results as live antigens and retained its agglutinability for at least 7 months. The results obtained on slide agglutination were as sp. as on tube agglutination. C. J. C. B.

**Velocity of combination of antibody with specific polysaccharides of pneumococcus.** M. Mayer and M. Heidelberger (*J. Biol. Chem.*, 1942, 143, 567–574).—Combination of types III and VIII sp. polysaccharides (S-III and S-VIII) with type VIII anti-pneumococcus serum is 90% complete in less than 3 sec. at 0°, although the formation of insol. aggregates takes place with diminishing velocity. In presence of homologous polysaccharide, S-III may be liberated from combinations with antibody, the velocity decreasing as aggregation proceeds and complete elimination occurring only if S-VIII is added during the early stages. H. G. R.

**Delayed typing of pneumococcus.** G. M. Mood and E. H. Fowler (*J. Lab. Clin. Med.*, 1942, 27, 805–806).—Dried smears can still be successfully typed even weeks after prep. C. J. C. B.

**In-vitro susceptibility of pneumococci to sulphonamide.**—See A., 1942, III, 705.

**Type-specific antibodies in blood in pneumococcal pneumonia.**—See A., 1942, III, 626.

**Diffusing factor in aerobic micro-organisms.** J. Ungar and A. L. Bacharach (*Brit. Med. J.*, 1932, I, 409–412).—The amounts of diffusing factor present in bacterial cultures of various micro-organisms were estimated semiquantitatively by measurement of the area of bleb produced after intradermal injection of the fluid into rabbits. Among pathogenic aerobes the factor was found only in staphylococcus, pneumococcus, and streptococcus. There was no correlation between general invasiveness or toxigenicity and production of diffusing factor in many bacteria studied. Production of diffusing factor is independent of rate of toxin production. Heat at 45° for ½ hr. destroys diffusing factor. C. A. K.

**Production of ammonia by bacteria.** P. G. Ashworth (*Med. J. Austral.*, 1942, I, 125–131).—The ability to produce NH<sub>3</sub> from urea in 200 strains of staphylococci was inversely associated with pigment formation, production of coagulase (pathogenicity), and fermentation of mannitol. The three latter properties were directly associated. F. S.

**Use of dried plasma for coagulase test.** E. J. Foley (*Science*, 1942, 95, 416).—The coagulase test (for virulent staphylococci) gave the same results using fresh or dried rabbit plasma. Dried plasma is more convenient for storage and transport. E. R. S.

**Agglutination of rabbit leucocytes by *Staph. aureus* toxin.**—See A., 1942, III, 662.

**Leucocidin of group A haemolytic streptococci.** E. W. Todd (*Brit. J. Exp. Path.*, 1942, 23, 136–145).—Streptolysin O has a powerful lytic action on leucocytes when tested at a low O<sub>2</sub> tension in the absence of red blood cells and may be responsible for the leucocidin

action of virulent group A hæmolytic streptococci, for attempts to prepare an independent leucocidin were unsuccessful. F. S.

**Serological typing of hæmolytic streptococci of Lancefield group A.** L. R. Kantz (*J. clin. Invest.*, 1942, 21, 217—226).—Hæmolytic streptococci of the Lancefield group A may be classified by the agglutination method of Griffith. From 260 cases it was found that hæmolytic streptococci of a few types are responsible for nearly all of the infections in any season in San Francisco. The same types do not predominate in every season. Type 25 was isolated more than twice as frequently from cases of otitis media as any other type. C. J. C. B.

**Bacteriostatic effect of sulphonamide drugs on growth of 25 strains of *Strep. viridans*.**—See A., 1942, III, 625.

**Congenital syphilis acquired by fœtus before appearance of chancre in mother.** W. Bickers (*Arch. Dermat. Syphilol.*, 1942, 46, 135—136).—A case report. C. J. C. B.

**Serological diagnosis of syphilis.** J. A. Kolmer (*Arch. Dermat. Syphilol.*, 1942, 45, 455—477).—Spirochætal complement-fixation tests in syphilis of human beings and rabbits are not as sp. as the Wassermann and flocculation tests, because of the presence in normal serum of natural spirochætal antibody, which reacts not only with cultivated *S. pallida* but with *S. macrodentium*, *S. microdentium*, and other spirochætes. Spirochætal complement-fixing antibody is present in most cases of malaria and in leprosy. C. J. C. B.

**Rôle of auto-antibodies in serodiagnosis of syphilis.** A. W. Ratcliffe (*J. Lab. clin. Med.*, 1942, 27, 729—735).—By tests in rabbits the reagin was shown to be a closely related group of antibodies and not a single chemically const. compound. While the primary serologic change in syphilis may be the formation of antibodies to *T. pallidum*, the potential or actual occurrence of positive or doubtful reactions with lipin antigens in the absence of syphilis results from the formation of antibodies to the "ubiquitous lipin" either from some other infectious agent or from the tissues of the host, dependent on the liberation of lipid haptens and protein derivatives capable of activating them. C. J. C. B.

**Specificity in serodiagnosis of syphilis.** F. Rytz (*Amer. J. clin. Path.*, 1942, 12, 166—173; cf. A., 1941, III, 805).—Further satisfactory results of the author's differential test are given. C. J. C. B.

**Association of Wassermann antigen with heavy materials present in tissues.** J. Furth and E. A. Kabat (*Science*, 1941, 94, 46—47).—The Wassermann hapten is associated with materials sedimentable at high speed present in normal and neoplastic tissues. Although these heavy materials are strongly antigenic, the immune sera produced by them in rabbits react strongly with homologous heavy materials but do not give a positive Wassermann reaction. E. R. S.

**Bacterial reduction of tetrathionate.** M. R. Pollock, R. Knox, and P. G. H. Gell (*Nature*, 1942, 150, 94).— $S_4O_6^{2-}$  was reduced quantitatively to  $S_2O_3^{2-}$  by *Bact. typhosum*, *paratyphosum B* and *C*, *typhimurium*, *Thompson*, *Reading*, and *Dublin*, and by members of the *Proteus* group, but not by most other fecal organisms. The reaction proceeds more rapidly in the presence of a H donator such as mannitol or glucose. E. R. S.

**Test for Vi agglutinative properties for *Eberthella typhosa*.** M. B. Coleman (*Amer. J. Publ. Health*, 1942, 32, 843—847).—Of 82 known typhoid carriers, 75% showed Vi agglutination, of 37 patients with typhoid fever 37%, of 46 persons who had received typhoid vaccine 15%, and of 157 patients with other conditions 5%. C. J. C. B.

**Mucin in experimental cholera infections in mice.** J. J. Griffiths (*U.S. Publ. Health Repts.*, 1942, 57, 707—710).—Recently isolated strains of *Vibrio cholera* are capable of proliferating and killing mice when relatively small nos. of organisms suspended in mucin are injected intraperitoneally. C. G. W.

**Macro-molecular component of chick embryo tissue [infected with encephalomyelitis].** A. R. Taylor, D. G. Sharp, D. Beard, and J. W. Beard (*Science*, 1941, 94, 613—615).—The two heavy components of chick embryo tissue infected with equine encephalomyelitis have been separated by extraction with water for 42—96 hr. and filtration through Celite before centrifugation, all at  $p_H$  7.2—7.4. The lighter component had a much lower infectivity and was shown, by analyses and qual. tests, to be identical with that obtained from normal embryo tissue and not specifically associated with the virus. E. R. S.

**Serological studies in infectious mononucleosis.** R. Straus and M. T. Bernstein (*Amer. J. clin. Path.*, 1942, 12, 174—182).—The "Davidsohn differential test" (A., 1938, III, 537) was performed in 110 cases with and without infectious mononucleosis. The differential absorption test is of diagnostic val. especially in cases of infectious mononucleosis with "border-line" titres of heterophile agglutinins and those complicated by a history of therapeutic injections of horse serum. In cases with low titres of heterophile agglutinins, a negative differential test does not exclude the disease. In cases with a higher titre, the procedure is of val. as another confirmatory

test. In most of 76 cases, the titre of the antibodies was 1:64 or less within 150 days. C. J. C. B.

**Detection of "masked" virus (Shope papilloma virus) by means of immunisation.**—See A., 1942, III, 615.

**Factor in domestic rabbit papilloma tissue hydrolysing the papilloma virus protein.** Enduring partnership of neoplastic virus and carcinoma cells.—See A., 1942, III, 697.

**Serological studies on plant viruses.** R. Mushin (*Austral. J. Exp. Biol.*, 1942, 20, 59—63).—Several plant viruses were tested using the precipitin titration technique. The following viruses gave positive flocculation reactions: ordinary tobacco mosaic, tobacco acubca, tomato bushy stunt, potato-virus X and X + B, -acubca mosaic, and tuber blotch, whilst negative results were obtained with potato spindle tuber, tomato spotted wilt, rose wilt, and strawberry crinkle. Cross-pptn. tests showed that only the two strains of tobacco mosaic virus are serologically related. Chester's field method (A., 1937, III, 358) of precipitin testing gives reliable results when the optimum antigen-antibody flocculation ratio is applied, whilst the drop method of Dunin and Popova does not give consistent results. J. N. A.

**Valency of antibodies and structure of antigen-antibody precipitate.** F. Haurowitz and P. Schwerin (*Brit. J. exp. Path.*, 1942, 23, 146—150).—Rabbits were immunised with arsanil-sheep globulin, an antigen containing two different determinant factors, the phenyl-arsinic acid group and the species-sp. sheep group. From fractional pptn. of the rabbit antisera to arsanilazosheep-serum-globulin it was concluded that such sera contain mainly univalent antibody mols. The antigen-antibody ppt. would then be an aggregate of complex particles, each consisting of a multivalent antigen mol. to which several univalent antibody mols. are bound. F. S.

**Chicken embryos as sensitising agents.** T. O. Berge and M. V. Hargett (*U.S. Publ. Health Repts.*, 1942, 57, 652—667).—Chick-embryo protein derived from embryos of different ages possesses the power to produce anaphylactic sensitisation in young guinea-pigs in direct ratio to the age of the embryos employed. When doses of comparable N content are employed, chick-embryo extract from 10-day or younger embryos is found to be weakly anaphylactogenic, while that from 14-day embryos is highly so. C. G. W.

**Histamine release in allergy.** G. Katz and S. Cohen (*J. Amer. Med. Assoc.*, 1941, 117, 1782—1783).—Venous blood from a patient sensitive to an allergen (giant ragweed, timothy, or horse dust), when incubated at 37° for 15 min. with extract of allergen, showed a marked increase of plasma-histamine, which did not occur when normal venous blood was mixed with the extract. The reaction was sp. to the allergen to which the patient was sensitive. The source of histamine was the cells and not the plasma. C. A. K.

**Allergy as a contributing factor to biologic deafness.**—See A., 1942, III, 683.

**Cerebral allergic oedema.** W. R. Crowe (*J. Allergy*, 1942, 13, 173—176).—A case report. C. J. C. B.

**Dermatitis of eyelids due to philodendron (*Scandens cardatum*) plants.** J. H. Harris (*Arch. Dermat. Syphilol.*, 1942, 45, 1066—1068).—A case report. C. J. C. B.

**Dermatitis due to blossom of *Grevillea banksii*.** H. L. Arnold, jun. (*Arch. Dermat. Syphilol.*, 1942, 45, 1037—1051).—Report of 30 cases. C. J. C. B.

**Chemical studies on allergens in pollens.** J. M. Newell (*J. Allergy*, 1942, 13, 177—203).—A review. C. J. C. B.

**Immunology of ragweed pollen proteins.** M. Mosko, R. Hecht, and H. Weil (*J. Allergy*, 1942, 13, 149—152).—Short ragweed pollen extracts as usually prepared contain 2 main types of mol. aggregates, large and small, which may be separated by means of heat at the isoelectric point. The smaller elicits skin reactions in patients sensitive to ragweed pollen more readily than the larger mol. aggregate. The former alone gives precipitin reactions with ragweed precipitin antiserum and cannot elicit anaphylactic shock in guinea-pigs sensitised to whole ragweed pollen. C. J. C. B.

**Relationship of maternal diet to intrauterine sensitisation.** B. Zohn (*J. Allergy*, 1942, 13, 153—155).—In 21 cases, excessive consumption of foods, such as chocolate, milk, egg, strawberry, potato, buckwheat, banana, plum, and peach, by mothers during their pregnancies had no demonstrable effect on the offspring from a viewpoint of sensitisation. In the newborn infant no positive reactions to the foods consumed in excess by the mother during pregnancy were obtained by direct or indirect methods of testing. C. J. C. B.

**Cereal-free elimination diets and soybean emulsion for study and control of infantile eczema.** A. H. Rowe and C. L. Mauser (*J. Allergy*, 1942, 13, 166—169). C. J. C. B.

**Contact dermatitis due to cod-liver oil.** E. P. Cope (*Arch. Dermat. Syphilol.*, 1942, 46, 140).—Report of a case. C. J. C. B.

**Dermatitis from imitation leather.** S. J. White (*Arch. Dermat. Syphilol.*, 1942, 45, 1164—1165).—Case report. C. J. C. B.

**Nail polish dermatitis in a man.** W. Garbe (*Arch. Dermat. Syphilol.*, 1942, 46, 141). C. J. C. B.

**New technique for patch tests: use of Pyrex cups.** J. H. Sterner (*Arch. Dermat. Syphilol.*, 1942, 45, 761—762).—A moderately firm 10-mm. ball of cotton is moistened with the test solution, or impregnated by being rolled in the powdered test material, and put in the Pyrex cup which is bound to the arm by adhesive tape. C. J. C. B.

## XXVI.—PLANT PHYSIOLOGY.

**Production of life in sea.** H. W. Harvey (*Biol. Rev.*, 1942, 17, 221—246).—A review of the factors concerned in the production of plants in the sea. J. D. B.

**Photoperiodic behaviour of some mints (Labiatae).** H. A. Allard (*J. Agric. Res.*, 1941, 63, 55—64).—In most species examined (*Mentha* and *Monarda* spp.) long-day conditions favoured profuse flowering. Species producing long stolons showed accentuation of the stoloniferous habit under short-day conditions. A. G. P.

**Responses of some plants to equal and unequal ratios of light and darkness in cycles ranging from one to seventy-two hours.** H. A. Allard and W. W. Garner (*J. Agric. Res.*, 1941, 63, 305—330).—Effects of ratios of light: darkness ranging from 1:3 to 3:1 and of cycles of 1—72 hr. on the growth of 5 plant species are recorded. Increased growth and dry matter production are associated with increase in light interval (*a*) when the total light received during the experimental period is increased, *i.e.*, with unequal ratios of light and darkness in the same cycle, or (*b*) when equal light ratios were maintained in cycles of increasing length, the total light received being unchanged. A. G. P.

**Progress of germination of seed of *Digitaria* as influenced by germination temperature and other factors.** E. H. Toole and V. K. Toole (*J. Agric. Res.*, 1941, 63, 85—90).—Effects of temp., of germination, storage, and pre-chilling on the rate of germination of *D. ischaemum* and *D. sanguinalis* are recorded. Treatment of seeds with H<sub>2</sub>SO<sub>4</sub> shortened the incubation period. Addition of 0.2% aq. KNO<sub>3</sub> accelerated germination of *D. ischaemum* under certain but not under all temp. conditions. A. G. P.

**Distribution of potassium in bright leaf cigarette tobacco and its influence on the quality of the leaf.** M. F. Gribbins, J. J. Reid, and D. E. Haley (*J. Agric. Res.*, 1941, 63, 31—39).—Inadequate K supply to tobacco plants is associated with low contents of reducing substances (including glucose) in all leaves and a relatively higher proportion of K in the upper leaves. With adequate K supplies the proportion of reducing substances in leaves reaches a max. and K is uniformly distributed between the top and bottom leaves. Excessive K supplies lead to increased K absorption and relatively greater proportions of K in lower than in upper leaves but without any corresponding increase in their glucose contents. The most satisfactory N/K ratio in the nutrient for tobacco plants is 0.8—1.1, the corresponding ratio in leaves being 0.5—3.9. Neither extreme was associated with best leaf quality. A. G. P.

**Effects of prolonged rotation of plants on a horizontal klinostat.** III. Physiological reactions in the hypocotyl of *Lupinus albus*. E. D. Brain (*New Phytol.*, 1942, 41, 81—90).—Prolonged rotation of seedlings of *L. albus* on a horizontal klinostat does not affect the geotropic response or the  $p_H$  of the cell sap of the hypocotyl. The suction pressure of the cells, the extensibility of the cell walls, and the amount of growth hormone that will diffuse out of the hypocotyl segments is increased by klinostat treatment. L. G. G. W.

**Causes of regeneration after longitudinal splits.** R. Snow (*New Phytol.*, 1942, 41, 101—107).—In young stems and roots split longitudinally the half-rings of cambium regenerate on their inner sides so that complete rings result. This is not due to a wound or other hormone. The causal factor is the interruption of cambial continuity which interrupts some process that normally takes place in a transverse direction and needs continuity of protoplasm. L. G. G. W.

**Whorled phyllotaxis.** R. Snow (*New Phytol.*, 1942, 41, 108—124).—Stem apices of species of Labiatae split longitudinally develop in each half a new apex which exhibits a phyllotaxy that is decussate or nearly so. Two regulating factors are probably involved. One tends to equalise the levels at which leaves arise and the other to equalise the divergence angles between leaves of a whorl so that a decussate phyllotaxy with whorls of two leaves develops. The position, shapes, and sizes of the leaves immediately below the stem apex also affect the phyllotaxy. In *Epilobium* split decussate apices show less tendency to develop decussate phyllotaxy in the regenerated apices. L. G. G. W.

**Proliferation-promoting intercellular hormones.** III. Relation of aeration to activity of proliferation-promoting factors from injured

cells. J. R. Loofbourow, A. M. Webb, D. G. Loofbourow, and R. K. Abramowitz (*Biochem. J.*, 1942, 36, 513—518; cf. A., 1942, III, 433).—Wound hormones from yeast under aerated conditions show 32 times the activity of uninjured cell products, but no difference exists under non-aerated conditions. The ultra-violet absorbing part (max. at 2600 Å) does not contain pyridine and is not identical with co-enzymes I and II, but it does constitute a large part of the proliferation-promoting substances. Other adenine complexes (adenylic acid, adenosine, etc.) may contribute materially to the activity. P. G. M.

**Release of auxin from isolated leaf proteins of spinach by enzymes.** S. G. Wildman and S. A. Gordon (*Proc. Nat. Acad. Sci.*, 1942, 28, 217—228).—Auxin is associated with the protein mol., and can be released by enzyme action. Trypsin, chymotrypsin, papain, and commercial tryptic extract was used, the last being the most active, probably because it contained a mixture of proteolytic enzymes. Auxin can be obtained from both cytoplasmic and chloroplastic proteins. The cytoplasmic protein has been fractionated by an isoelectric method, and the two fractions yield different amounts of auxin. The diffusion rates of leaf auxin and that obtained by hydrolysis are comparable, and indicate that auxin has a lower mol. wt. than indolylacetic acid. The results are not affected by bacterial contamination. H. M. J.

**Effect of human cerebrospinal fluid on plant growth.**—See A., 1942, III, 586.

**Ammonia production by root nodules of Leguminosae.** S. Winogradsky and H. Winogradsky (*Compt. rend.*, 1941, 113, 713—717).—Nodules dried at 35—50° retain for years their power to give off NH<sub>3</sub>. Evolution of NH<sub>3</sub> occurs only in presence of moisture; it is very slight or absent at 20° and is probably max. at 50°. Complete removal of moisture suspends but does not prevent evolution of NH<sub>3</sub>. The only deaminating enzyme in the nodules is urease, which, however, diminishes NH<sub>3</sub> production. The agent or system responsible for NH<sub>3</sub> production is not destroyed in 30 min. at 120°; it appears to be a biocatalyst of unknown type. W. McC.

**Factors affecting the pathogenicity of *Fomes lignosus*, Klotzsch.** J. G. Harrar (*Minnesota Agric. Exp. Sta. Tech. Bull.*, 1937, No. 123, 28).—*F. lignosus*, which causes white rot of rubber trees, has a temp. range of 2—36°, will grow in media of  $p_H$  4—10, in daylight, darkness, or blue, red, or yellow rays. Direct sunlight or ultra-violet light adversely affect growth; fertilisers have no effect on growth in the soil but org. Hg dusts are extremely toxic. A. W. M.

**Histology of snap bean tissues affected with black rot.** W. A. Jenkins (*J. Agric. Res.*, 1941, 62, 683—690).—Necrosis and rapid wilting of diseased plants are associated with suberisation and plugging of cells of the conducting tissues of roots and stems, and consequent interference with the intake of water and nutrients and the transference of elaborated materials within the plant system. A. G. P.

**Distribution by the sap stream of spores of three fungi that induce vascular wilt diseases of elm.** W. M. Banfield (*J. Agric. Res.*, 1941, 62, 637—681).—The sap stream in the large vessels of the new annular ring probably conveys the fungal spores throughout the bole and crown of the elm. A. G. P.

**Environmental and nutritional factors affecting *Aphanomyces*, root-rot of garden pea.** P. G. Smith and J. C. Walker (*J. Agric. Res.*, 1941, 63, 1—20).—Radial growth of *A. euteiches* on potato-glucose-agar was optimum at 28°; none occurred at 8° or 36°. In buffered (PO<sub>4</sub>'') media the  $p_H$  limits of growth were 3.8 and 8.0 and the optimum was  $p_H$  4.5—6.5. Max. infection of sand-cultured pea plants occurred at 28—24°, and in soils containing 75% of their water-holding capacity; none was apparent in soils having 45% of their water capacity. In artificial cultures high nutrient concn. in the nutrient was associated with low % of infection and vice versa. The ratio of N, P, and K in dil. or conc. nutrient media had no influence on infection. Increase in nutrient concn. after plants were infected did not affect the course of development of the disease. Concns. of nutrients in media which prevented infection of plants permitted growth of the organism in agar cultures. A. G. P.

**Biochemistry of wood-rotting fungi. III. Production of methyl mercaptan by *Schizophyllum commune*, Fr.** J. H. Birkinshaw, W. P. K. Findlay, and R. A. Webb (*Biochem. J.*, 1942, 36, 526—529).—*S. commune* produces methyl mercaptan and (?) H<sub>2</sub>S from synthetic media containing SO<sub>4</sub>''. *Penicillium brevicaulis*, which has a similar methylating mechanism, does not reduce oxy-acids of S to sulphide. P. G. M.

## XXVII.—PLANT CONSTITUENTS.

**Spectroscopic analysis. II. Minor elements in a sea-weed (*Macrocyctis pyrifera*).** S. H. Wilson and M. Fieldes (*New Zealand J. Sci. Tech.*, 1942, 23, B, 47—48; cf. A., 1940, I, 330).—Spectrographic analysis shows that the Mn content is much less and the As and Sr contents are much greater than those of land plants. The minor



elements are not present in sufficient quantity to render this a useful agricultural source of the elements. When used as a K fertiliser the As content is too low to increase appreciably the amount normally present in the soil. J. W. S.

**Schedule for chromosome counts in plants with small chromosomes.** B. L. Hancock (*Stain Tech.*, 1942, 17, 79—83).—Fixation with Belling's Navashin type fixative is followed by a modification of the card-mount method for mounting a no. of small root-tips together (Randolph, A., 1940, III, 702). Mordanting with 1% aq. chromic acid intensifies staining of small chromosomes with 0.1% crystal-violet. E. E. H.

**Structure and staining of starch grains of potato tuber.** J. C. Bates (*Stain Tech.*, 1942, 17, 49—56).—The structure of starch grains is shown by differential staining made possible by using formaldehyde as a swelling agent. Stains used are hæmatoxylin or safranin with fast-green FCF. E. E. H.

**Localisation of lignin in plant membranes.** A. Dauphiné (*Compt. rend.*, 1941, 213, 739—740).—Colour tests show the existence of concentric layers of cellulose and lignin in sections of the lignified parenchyma of the pith of *Clematis vitalba* and in the supporting elements of the secondary vascular ring of *Euonymus japonicus* and *Pinus sylvestris*. After treatment with NaOCl, pectin but no lignin is found. Lignin and pectin occur in an intermediate layer and in certain concentric layers. During growth, pectin is probably produced before lignin and acts as intermediate between cellulose and lignin. W. McC.

**Vanillin from lignin materials.**—See A., 1942, II, 314.

**Crystalline protein obtained from a lipoprotein of wheat flour.**—See B., 1942, III, 199.

**Active principles of *Embelia robusta*, Roxb., *Myrsine semiserrata*, Wall, and *M. capitellata*, Wall.** S. Krishna and B. S. Varma (*Forest Res. Inst. Dehra Dun*, 1941, *Forest Bull.*, 102, 3 pp.; cf. A., 1936, 910).—The dried berries of *E. robusta*, *M. semiserrata*, and *M. capitellata*, extracted successively with light petroleum, CHCl<sub>3</sub>, and alcohol, yield respectively: fat 7.5, 1.8, 3.6; embelic acid 1.6, 0.4, 1.6; quercitol 0.0, 0.8, 0.0; and K H oxalate 0.15, 0.00, 0.00%. W. McC.

**Constituents of *Caucalis scabra*, Makino.**—See A., 1942, II, 338.

**Neolarin, a new glucoside from *Linaria vulgaris*, L.**—See A., 1942, II, 303.

**Sophorabioside, a new glucoside from *Sophora japonica*, L.**—See A., 1942, II, 302.

**New active constituent of pyrethrum flower.** D. N. Roy and S. M. Ghosh (*Nature*, 1942, 150, 153).—Mortality curves suggest that a toxic principle X accompanies pyrethrin I and II. Moreover, the kerosene extract prepared from the sun-dried residue after removal of pyrethrin I and II from pyrethrum flowers was still active. A. A. E.

**Light petroleum-soluble constituents of pasture fractions prepared for feeding experiments in facial-eczema investigations.** F. B. Shorland (*New Zealand J. Sci. Tech.*, 1941, 23, A, 112—120).—A suitable scheme of extracting both "juice" and "fibre" in frozen samples of pasture is given. Total lipins constituted 5.59% of the dry matter, and were divided into phosphatides, waxes, total and acetone-sol. unsaponifiable matter, free fatty acids (as oleic acid), sterols, and total acetone-sol. matter. As the free fatty acids were 24.6% of the total lipins, enzyme action probably took place while the samples were stored at -15°. Distillation of the extracts gave some separation and showed the presence of acids having a lower equiv. wt. than palmitic acid and of volatile unsaponifiable matter. A. W. M.

**Carotenoids. II. Isomerisation of  $\beta$ -carotene and its relation to carotene analysis.** B. W. Beadle and F. P. Zscheile (*J. Biol. Chem.*, 1942, 144, 21—33).—The carotene fraction from certain fresh plant materials, e.g., spinach leaves, peas, beans, asparagus, Lima beans, and broccoli, consists of  $\beta$ -carotene and an isomeride, neo- $\beta$ -carotene. Hence absorption spectra of pure  $\beta$ -carotene and plant carotene differ. Spectrophotometric analysis of a mixture of  $\beta$ - and neo- $\beta$ -carotene agrees with results obtained chromatographically (Al<sub>2</sub>O<sub>3</sub>, not MgO). Conversion of  $\beta$ - into neo- $\beta$ -carotene in hexane at 30° is examined; the reaction is reversible. On adding a conc. solution of  $\beta$ -carotene in hexane to the vegetables before extraction, approx. 5% of added  $\beta$ -carotene is isomerised to neo- $\beta$ -carotene under conditions of analysis; in absence of plant material  $\beta$ -carotene is unchanged under such conditions. A. T. P.

**Carotenoids. III. Distribution of pure pigments between immiscible solvents.** J. W. White, jun., and F. P. Zscheile (*J. Amer. Chem. Soc.*, 1942, 64, 1440—1443).—The distribution of  $\beta$ -carotene, cryptoxanthol, and zeaxanthol between hexane and methyl alcohol, diacetone alcohol, and  $\beta$ -methylpentane- $\beta\delta$ -diol has been measured.

Partition between hexane and diacetone alcohol (77—94.5 vol.-% satisfactorily separates  $\beta$ -carotene and dihydroxycarotene if monohydroxycarotene is absent.  $\beta$ -Carotene plus cryptoxanthol can be separated from dihydroxycarotene by 78.5% diacetone alcohol.  $\beta$ -Carotene and cryptoxanthol can be separated by 92% aq. methylpentanediol. W. R. A.

**Prolycopene.**—See A., 1942, II, 293.

**Spectrophotometric method for analysis of chloroplast pigments.** H. H. Haskin (*J. Biol. Chem.*, 1942, 144, 149—140).—Chlorophylls *a* and *b*, xanthophyll, and carotene are determined photometrically using selective filters. The pigments absorb differently at the wavelengths used, and are determined by calculation from the differences in total absorption. R. L. E.

**Alkaloids of *Lycopodium complanatum*, L., and *L. saururus*.**—See A., 1942, II, 336.

**Aconite alkaloids. Atisine. Isolation of two new alkaloids from *Aconitum heterophyllum*: heteratisine and hetisine.** Napelline.—See A., 1942, II, 335.

**Analytical classes of cannabinol compounds in marijuana resin.**—See A., 1942, III, 771.

## XXVIII.—APPARATUS AND ANALYTICAL METHODS.

**Improved inverted microscope.** C. Wolfson (*J. Lab. clin. Med.*, 1942, 27, 809—811). C. J. C. B.

**Simplified "lyophile" desiccator for small laboratories.** L. R. Christensen (*J. Lab. clin. Med.*, 1942, 27, 799—802). C. J. C. B.

**Adapter for micro-centrifuge shields.** J. C. Bock (*J. Lab. clin. Med.*, 1942, 27, 839—840).—An easily made adapter for micro-centrifuge shields is described. The changing of centrifuge heads is obviated. C. J. C. B.

**Simple modification of Hanike-Gibbs drop recorder.** W. H. Olson and H. Necheles (*J. Lab. clin. Med.*, 1942, 27, 802—804).—The recorder is modified so that the rates of a no. of different secretions can be measured at the same time. C. J. C. B.

**Antifogging effect of soap on eyeglasses and diagnostic mirrors.** L. Pelner (*J. Lab. clin. Med.*, 1942, 27, 798).—Soap exerts an antifogging action. C. J. C. B.

**Protection of thermostats in water-baths.** R. Finkelstein (*J. Lab. clin. Med.*, 1942, 27, 797).—On breakage Hg can ruin a water-bath by attacking the solder of joints in the bath and the metals in the thermostat. This can be avoided by placing a small test-tube around the bulb of the thermometer with the top of the tube below the water level. C. J. C. B.

**Use of economical electrodes in electrochemistry.**—See A., 1942, I, 342.

**Wax-resin compositions for moulage-making.** C. D. Clarke (*J. Lab. clin. Med.*, 1942, 27, 1098—1103). C. J. C. B.

**Simplified apparatus for catalase determination.** R. R. Thompson (*Ind. Eng. Chem. [Anal.]*, 1942, 14, 585).—A simple gas burette is connected to a reaction flask. The extracted catalase is treated with H<sub>2</sub>O<sub>2</sub>, the O<sub>2</sub> liberated giving a vol. for the catalase activity. J. D. R.

**Determination of barbituric acid derivatives.** F. H. Shaw (*Austral. J. Exp. Biol.*, 1942, 20, 47—48).—A micro-method, applicable to body fluids, is described. The solution containing the barbiturate is rendered alkaline and extracted with ether. After acidification, the free barbituric acid is extracted with ether-CHCl<sub>3</sub>, taken up in alkali, and then pptd. as the Ag salt which is determined by NH<sub>4</sub>CNS. The solubility of Ag barbiturate depends on  $p_{H}$ . It is insol. between  $p_{H}$  7.5 and 5.5. J. N. A.

**Photo-electric determination of oxalic acid and calcium, and its application to micro- and ultramicro-analysis of serum.** J. Sendroy, jun. (*J. Biol. Chem.*, 1942, 144, 243—259).—The method is described. The Ca is directly pptd. as Ca oxalate, and determined iodometrically, using a photo-electric method, by the extent of its reduction of a const. amount of Ce(SO<sub>4</sub>)<sub>2</sub>. 0.002 mg. of Ca or 0.02 c.c. of serum can be determined (error  $\pm 2\%$ ). J. N. A.

**Polarographic determination of lead in bones.** E. Weing (*Z. physiol. Chem.*, 1942, 273, 158—162).—The method described is claimed to be superior to others because smaller amounts of material are used, the preliminary chemical treatment of the bone ash is simple, and the polarograph is an automatic instrument and independent of the observer. J. N. A.

**Polarographic determination of antimony and bismuth.**—See A., 1942, I, 279.

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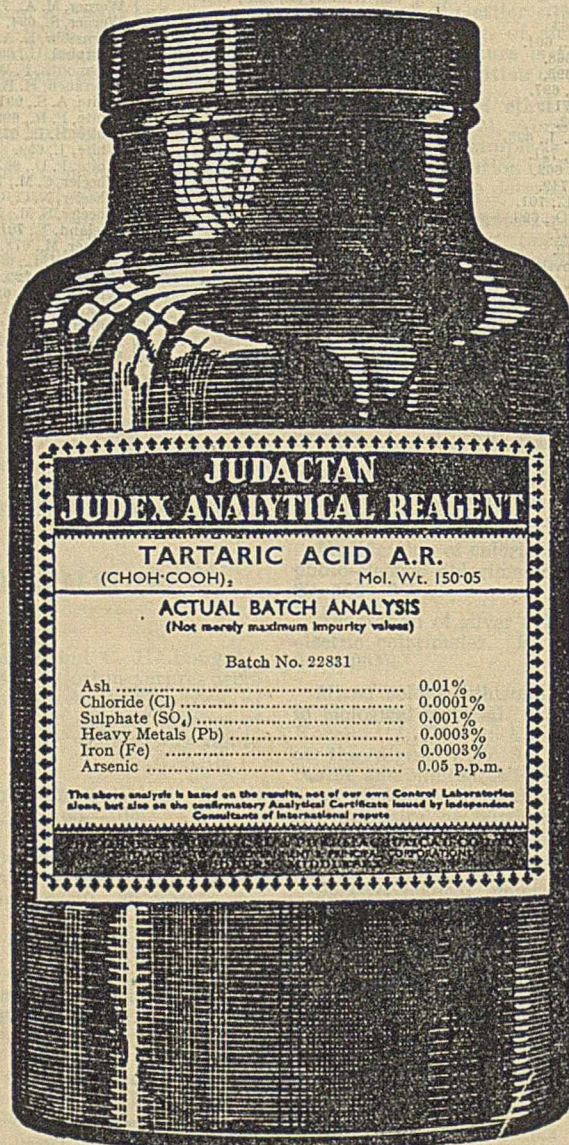
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