# BRITISH CHEMICAL AND PHYSIOLOGICAL ABSTRACTS

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**OCTOBER, 1943**





# A., III.—PHYSIOLOGY & BIOCHEMISTRY

## (INCLUDING ANATOMY)

### *CONTENTS*

I. General Anatomy and Morphology . . . 705
II. Descriptive and Experimental Embryology. Heredity 706
III. Physical Anthropology . . . . . 708
IV. Cytology, Histology, and Tissue Culture . . 708
V. Blood and Lymph . . . . . 710
VI. Vascular System . . . . . 717
VII. Respiration and Blood Gases . . . . 721
VIII. Muscle . . . . . 722
IX. Nervous System . . . . . 723
X. Sense Organs . . . . . 728
XI. Ductless Glands, excluding Gonads . . 734
XII. Reproduction . . . . . 738
XIII. Digestive System . . . . . 743
XIV. Liver and Bile . . . . . 744
XV. Kidney and Urine . . . . . 745
XVI. Other Organs, Tissues, and Body-Fluids . . 746
XVII. Tumours . . . . . 747
XVIII. Animal Nutrition . . . . . 750
XIX. Metabolism, General and Special . . . 756
XX. Pharmacology and Toxicology . . . . 758
XXI. Physiology of Work and Industrial Hygiene . 766
XXII. Radiations . . . . . 767
XXIII. Physical and Colloidal Chemistry . . . 768
XXIV. Enzymes . . . . . 768
XXV. Microbiological and Immunological Chemistry. Allergy . . . . . 770
XXVI. Plant Physiology . . . . . 778
XXVII. Plant Constituents . . . . . 779
XXVIII. Apparatus and Analytical Methods . . . 779
XXIX. New Books . . . . . —

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# BRITISH CHEMICAL AND PHYSIOLOGICAL ABSTRACTS

## A., III.—Physiology and Biochemistry (including Anatomy)

OCTOBER, 1943.

## I.—GENERAL ANATOMY AND MORPHOLOGY.

**Fate of skin homografts in man.** T. Gibson and T. B. Medawar (*J. Anat., London*, 1943, **77**, 299—310).—Controlled investigations of the reactions of autografts and homografts were made on a patient who had an extensive granulating area produced by thermal burns. Pinch autografts coalesced to form a continuous sheet. Homografts, at first indistinguishable from the autografts, showed degenerative changes after 15 days. Histological examination of the homografts showed no evidence that they were broken down by the reaction of lymphocytes or other mesenchyme cells. Foreign collagen was more resistant than foreign epithelium. W. J. H.

**Blood supply and innervation of choledocho-duodenal junction in cat.** J. W. Schulze and E. A. Boyden (*Anat. Rec.*, 1943, **86**, 15—39).—The extrinsic nerves to this junctional site are designated gastroduodenal nerve and gastroduodenal plexus. The former arises by confluence of branches from the hepatic plexus and the latter is composed of fibres from the hepatic plexus and recurrent fibres of the coronary nerve. The two gastroduodenal pathways give off small choledochal rami which may or may not anastomose before reaching the duct. They form a paracholedochal plexus which gives off branches to an intrinsic network in the adventitia of the duct. The intramural plexus enters the duodenal wall with the bile duct. The relationships of the nerves to the duodenal arteries are described. The finding of a sp. innervation to this junction has made possible an experimental analysis of the nervous pathways regulating the evacuation of the biliary tract. W. F. H.

**Hyoid region of placental mammals with especial reference to bats.** J. M. Sprague (*Amer. J. Anat.*, 1943, **72**, 385—472).—The characters of the hyoid complex indicate that the division of the Chiroptera into two suborders (Mega- and Micro-chiroptera) is a natural one. Certain characters are absolutely diagnostic of each group with no overlapping. This sharp definition is significant and agrees with data concerning the skull, teeth, and skeleton. A list of diagnostic hyoid characteristics of the two suborders is appended. W. F. H.

**Sequence of epiphyseal union in old world monkeys.** S. L. Washburn (*Amer. J. Anat.*, 1943, **72**, 339—360).—Sequence of union is described in four groups, viz., macaque, crested langur, maroon langur, and proboscis monkey. Species differences are few in no. Three maroon langurs exhibit early union of the epiphysis of the tip of the acromion process. Complete union of the epiphysis of the iliac crest is slightly earlier in macaques than in langurs. No sex differences in the order of epiphyseal union were found, except in the pubic symphysis. Maturation proceeds by regions, rather than by individual bones. Differences between man and monkey are cited. W. F. H.

**Phosphorylase in calcifying cartilage.** A. B. Gutman and E. B. Gutman (*Proc. Soc. Exp. Biol. Med.*, 1941, **48**, 687—691).—When ground epiphyses of young rabbits or rats are incubated with glycogen and inorg. P, the inorg. P content of the mixture rapidly falls. The fall is not affected in rate by NaF but is inhibited by phloridzin. If aq. extracts of epiphyses are used, the presence of adenylic acid is necessary to the reaction. V. J. W.

**Reaction of bone to multiple metal implants.** R. T. Bothie, L. E. Beaton, and H. A. Davenport (*Surg. Gynec. Obstet.*, 1940, **71**, 598—602).—Up to 4 pegs of different metals (12 tested) were implanted into the femora of cats and electrical potentials between them measured. The reaction of the bone was determined by *X*-ray photography and by post-mortem examination 203—259 days after the implantation. The electrical potentials follow the order of the electromotive force series of elements. The amount of reaction between bone and metal is not entirely parallel to the potential developed. Mn and Ti have not been previously tested; Mn was highly reactive, promoting much callus formation, and Ti was as well tolerated as V and stainless steel and bone tended to grow into contact with it. Theoretical reasons are given for regarding the reaction of bone to metal as based on chemical rather than electrical phenomena. P. C. W.

**Pathogenesis of spondylitis ankylopoietica.** E. Freund (*Edinb. Med. J.*, 1942, [iv], **49**, 91—109).—The condition is not rheumatic and is different from rheumatoid arthritis. Chronic inflammatory changes are present in intervertebral articulations. Articular processes, vertebral bodies and discs show infiltration and replacement by vascular fibrous tissue. Changes in intervertebral discs are secondary to immobilisation of joints by fibrous adhesions (unlike osteoarthritis). True osteoarthritic "lipping" is not present but osseous bridges between vertebral bodies are characteristic. Earliest changes occur in sacro-iliac joints. (13 photomicrographs.) H. S.

**Hypoplasia of mandible.** J. S. Llewellyn and A. D. Biggs (*Amer. J. dis. Child.*, 1943, **65**, 440—445).—Report of a case with a review of the literature. C. J. C. B.

**Hereditary ectodermal dysplasia of anhydrotic type.** H. Stadler and C. H. Blackstone (*J. Pediat.*, 1942, **21**, 229—237).—A report of 2 cases. C. J. C. B.

**Anomalies of aortic arch.** P. A. Herbut (*Arch. Path.*, 1943, **35**, 717—729).—A review with report of 12 cases. C. J. C. B.

**Technique for gross differential staining of peripheral nerves in cleared vertebrate tissue.** T. W. Williams (*Anat. Rec.*, 1943, **86**, 189—195).—Formalin fixation of fresh tissue is recommended and complete evisceration is suggested when convenient. The tissue is kept immersed in several changes of formalin for 1 week and is subsequently macerated in 3% KOH. Staining is effected by first immersing the specimen in a solution of glacial acetic acid 1 part, glycerin 1 part, and 1% aq. chloral hydrate 6 parts, for 2 days, and next transferring it to a solution of Ehrlich's hæmatoxylin 1 part, glycerin 1 part, and 1% aq. chloral hydrate 6 parts. The specimen is then dehydrated and cleared in methyl salicylate. By means of the method well-stained preps. of the peripheral and in many instances the central nervous system of forms representing nearly all vertebrate classes can be obtained. The stain is more sp. for myelinated than for non-myelinated nerves. W. F. H.

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

**Division of zygote producing trophoblast only.** V. Bonney (*J. Obstet. Gynaec.*, 1940, **50**, 217—218).—A case in which there was no evidence of development of the ovum but in which the trophoblast developed and eroded the Fallopian tube. P. C. W.

**Development of human knee joint.** L. J. McDermott (*Arch. Surg., Chicago*, 1943, **46**, 705—719).—The articular cavity of the knee joint develops by disappearance of cells from the substance of the dense blastemal interchondral disc between the tibia and the femur, with coalescence of several primary spaces. (13 photomicrographs.) F. S.

**Studies of normal development of New Zealand white strain of rabbit : oogenesis and external morphology of embryo.** A. J. Waterman (*Amer. J. Anat.*, 1943, **72**, 473—515).—Maturation is initiated by coitus in many ova within each ovary, beginning about the 4th hr. after mating. Ovulation time varies. Division of the first polar body in ovarian ova was found together with polyovular follicles. Stages of development from the 6th day after mating to the time of birth are described. Factors responsible for the variability among embryos of a single litter are discussed. W. F. H.

**Origin and differentiation of definitive germ cells in mice.** N. B. Everett (*J. exp. Zool.*, 1943, **92**, 49—91).—In *Peromysicus* and albino mouse embryos the primordial germ cells are first found in the gut endoderm of 2·6-mm. embryos. From here the cells migrate into the splanchnic mesenchyme and through the dorsal mesentery into the genital ridge, where some of them become located among the somatic epithelial cells. The ova which form in post-embryonic life are derived from these cells which enlarge *in situ* and then migrate into the ovarian cortex. The follicular cells arise from epithelial cells which are carried into the cortex with the enlarged oocytes. The theca cells arise from stroma cells represented by the septulæ and tunica albuginea. Transplants of genital ridge to host kidneys before sex cells are found in it will not form sex cells, although the associated genito-urinary ducts will develop. Transplants of genital ridge after sex cells have reached it form typical testicular or ovarian tissue. Ovaries of mice receiving 168 r. or more of *X*-rays were sterile. In these the epithelium continued to proliferate but the

ingrowths lacked sex cells and once all the oogonia are destroyed within an ovary there is no new formation of ova. J. D. B.

**Histogenesis of arteries of chick embryo.** A. F. W. Hughes (*J. Anat., London*, 1943, **77**, 266—287).—A description is given of the development of different arteries in the developing chick. Three types of vessels are recognised; type I is elastic; type III muscular; type II intermediate in type. Histogenesis of the different types of arteries is described. The mechanical tensions to which the vessels are subjected and the possible results that these may have on the developing vessels are discussed. W. J. H.

**Normal development of *Triturus pyrrhogaster*.** P. L. Anderson (*Anat. Rec.*, 1943, **86**, 59—73).—Tables and illustrations are given, correlating internal development with external appearance of the embryo up to the age of 6 weeks after fertilisation. W. F. H.

**Nervous system and regeneration of forelimb in adult *Triturus*. II. Rôle of sensory supply.** M. Singer (*J. exp. Zool.*, 1943, **92**, 297—315).—Regeneration of forelimb can occur when normal sensory supply alone is present. If in addition to destruction of the other nerve components the dorsal roots are cut there is also no loss of regenerative capacity in the limb. Hence the trophic influence of the sensory component on regeneration is initiated within the sensory neurons themselves. If, in the absence of other nerve components, the dorsal root ganglia are partially destroyed there is no regeneration of the limb. It is concluded that certain quant. requirements in respect to sensory fibre no. must be met before regeneration can occur and it is suggested that this no. is fairly close to the no. of sensory fibres normally present within the limb. J. D. B.

**Choline-esterase and behaviour in *Amblystoma*. I. Relationship between development of enzyme activity and early motility. II. Effects of inhibiting choline-esterase.** C. H. Sawyer (*J. exp. Zool.*, 1943, **92**, 1—11, 11—29; cf. A., 1942, III, 489).—I. A modified microchemical method, involving an alkalimetric titration of the acetic acid liberated on hydrolysis of acetylcholine and sensitive to $5 \times 10^{-9}$ mol. of ester (0·9 $\mu$g. of acetylcholine), was used for the determination of choline-esterase activity at different stages of development in *A. punctatum*. The results showed a close correlation between enzyme content and functional ability as expressed by behaviour manifestations.

II. The functional capacity of larvæ reared in solutions of choline-esterase inhibitors (eserine, prostigmine, and acetylcholine itself) is profoundly affected. On removal of a larva from the inhibitor solution the recovery of physiological and enzymic activities parallel each other. It is concluded that the choline-esterase content is a biochemical criterion of functional capacity in the neuromuscular apparatus and that the physiological development of this system can be quantitatively assayed in terms of its esterase activity. J. D. B.

**Effect of colchicine on limb regeneration in larval *Amblystoma*.** C. S. Thornton (*J. exp. Zool.*, 1943, **92**, 281—295).—Amputated limbs of larvæ maintained in colchicine solutions (1 : 1000 or 1 : 1500) fail completely to regenerate but undergo a progressive dedifferentiation which involves all the formed structures within the limbs. The results are interpreted as being due to the inhibition of blastema formation by the colchicine (cf. Schotté and Butler, A., 1942, III, 726). J. D. B.

**Origin of sensory neurons and sheath cells of IXth and Xth cranial nerves in *Amblystoma*.** C. L. Yntema (*J. exp. Zool.*, 1943, **92**, 93—119).—An experimental analysis of the development of the glosso-pharyngeal and vagus nerves in *A. punctatum* with the following conclusions. The lateral line ganglia of these nerves arises from dorso-lateral placodes. The visceral ganglion of IX arises from the second epibranchial placode. The anterior visceral ganglion of X arises from the third and fourth epibranchial placodes. The posterior visceral ganglion of X arises from the fifth epibranchial placode. The root ganglion of X arises from postbranchial neural crest which is situated medial to the 4th and 5th somites. The 3rd spinal ganglion arises from neural crest which lies medial to the anterior part of the 6th somite. Sheath cells of the IXth and Xth nerves arise from neural crest at the levels of origins of the various roots, and also from placodal ectoderm. J. D. B.

**Unilateral substitution of brachial region of spinal cord by corresponding half of medulla in *Amblystoma*.** S. R. Detweiler (*J. exp. Zool.*, 1943, **92**, 247—259).—The results of 22 experiments, 10 of which were successful, demonstrate that the morphological features which typify the future medulla are intrinsically determined at the time of operation (stages 24—26). In no case was there any nerve supply to the limbs from the grafted medulla. J. D. B

**Independent identical mutations to albinism in sex chromosome of fowl.** F. B. Hutt and C. D. Mueller (*Amer. Nat.*, 1943, **77**, 181—184).—Breeding tests on albinotic fowls (white Leghorns and Barred Plymouth Rocks) from New York State, Massachusetts, and Indiana indicated that all three were genetically identical, and were caused by a mutation in the sex chromosome. Reasons are also given for assuming that the three mutations arose independently. W. F. H.

**Mutation of genes by irradiation.** R. H. Fagan (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 239—242).—Brief review. P. C. W.

**High mutation frequency in *Drosophila pseudo-obscura*.** K. Mampell (*Proc. Nat. Acad. Sci.*, 1943, **29**, 137—144).—Certain strains of race *B* of this species show an abnormally high mutation rate. The mutations may occur at any time during the development of germ or somatic line. The phenomenon is due to a dominant "mutator" gene which increases the normal spontaneous rate about 34 times when heterozygous and about 70 times when homozygous. This represents a linear increase in the mutation rate with dosage of the gene. The latter is probably linked to the second chromosome. J. D. B.

**Inheritance of diabetes mellitus.** G. Berencsi (*Magyar Orv. Arch.*, 1941, **42**, 233—244).—There were very definite indications of inheritance in a no. of the families studied. In some cases the factor was dominant, in others recessive. Jews showed a greater disposition to diabetes than persons of other races. In diabetic families the non-diabetic members frequently showed gastro-intestinal tumours, possibly a different manifestation of a common abnormality of the cells from which the gastro-intestinal tract and the pancreas are derived. M. A. B.

## III.—PHYSICAL ANTHROPOLOGY.

**Anthropometry in pediatrician's office.** V. S. Vickers and H. C. Stuart (*J. Pediat.*, 1943, **22**, 155—171).—Norms are given for selected body measurements based on studies of children of North European stock. C. J. C. B.

**Metric study of undeformed Indian crania from Peru.** M. T. Newman (*Amer. J. phys. Anthrop.*, 1943, [ii], **1**, 21—45).—The 5 series described show a slightly greater variability than the mean of the North American Indian series described in the Hrdlička catalogues. The San Damian series shows particularly low variability, while the Machu Picchu series is highly variable. The variability of the latter indicates its composite nature, about half the series being coastal and about half highland in ancestry. The San Damian and Paucarcancha series represent two distinct highland strains. The identification of part of the Paucarcancha series with the Lagoa Santa type is doubted. W. F. H.

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

**Effects of advancing age on structure of anterior hypophyses and ovaries of female rats.** J. M. Wolfe (*Amer. J. Anat.*, 1943, **72**, 361—383).—A progressive decrease in the relative no. of eosinophils and an increase in the chromophobes takes place with age, and these changes were most marked early in life. Mitoses in eosinophils and chromophobes were most abundant in immature animals. Colloid degeneration appeared in 3-month-old rats. Adenomatous changes occurred in 27·8% of rats over 17 months old. The relative no. of granular basophils was not markedly altered by age. Age changes in the ovary included decrease in the no. of normal follicles and an increase in atretic follicles. Corpora lutea remained const. in no. from the age of 3 to 19 months. Cystic follicles first appeared in 12-month rats. W. F. H.

**Cytology of anterior pituitary of broody fowls.** F. Payne (*Anat. Rec.*, 1943, **86**, 1—13; cf. A., 1942, III, 867).—During the broody period there is a recession towards inactivity in basophils and acidophils and the development of "broody cells" through transformation of other cells. The transformations of the $A_1$ cells and the chromophobes is direct; basophils and $A_2$ acidophils probably first change into chromophobes and then into "broody cells." The possible significance of these changes is discussed. W. F. H.

**Experiments concerning mechanism of pituitary colloid secretion.** H. Selye (*Anat. Rec.*, 1943, **86**, 109—119).—Intravenous administration of hypertonic NaCl produces swelling and degeneration of anterior lobe basophils. Confluence of colloid material formed from such basophils produces cystic cavities throughout the gland tissue. The colloid accumulation, especially in the hypophyseal cleft, assumes such proportions that the posterior and middle lobes are separated from the anterior lobe by a wide cavity. It is concluded that there is a close correlation between anterior lobe cysts and the pituitary cleft and that anterior lobe basophils are concerned in the formation of colloid in both these situations. Isotonic salt solutions do not stimulate colloid formation and it thus appears likely that the pituitary changes described result from derangements in the osmotic equilibrium of the body. W. F. H.

**Method of repair in epithelial wounds of cornea.** L. B. Arey and W. M. Covode (*Anat. Rec.*, 1943, **86**, 75—86).—Repair of wounds in corneal epithelium of the white rat takes place by cell migration which quickly fills the defect. During the early stages the mitotic rate falls below normal in neighbouring epithelium and is almost lacking in the area being repaired. The new epithelial covering was

complete in 12 hr. but the reduced mitotic rate was not markedly increased until 96 hr. During the 6 days under observation the frequency of cell division, per unit epithelial area, never exceeded the normal rate. W. F. H.

**Microscopic anatomy of *Laterimeria* scale.** G. H. Roux (*S. Afr. J. med. Sci.*, 1942, **7**, *Biol. Suppl.*, 1—18). P. C. W.

**Factors influencing nucleoprotein content of fibroblasts growing *in vitro*.** J. N. Davidson and C. Waymouth (*Biochem. J.*, 1943, **37**, 271—277).—The effect of various substances on content of nucleoprotein-P of fresh explants from the 9-days-old embryo chick heart growing *in vitro* in roller tubes, and not on pure strains of fibroblasts, is determined. Cultures grown in Tyrode solution alone show a decrease in nucleoprotein-P which is probably due to presence of nucleases which hydrolyse the nucleic acid in cells which die. Addition of chick embryo extract causes a definite increase in nucleoprotein-P, and this increase is unaffected when the embryo extract is preheated at 100° for 5 min. Cryst. ribonuclease has no effect on growth-promoting properties of embryo extract, but the crude enzyme from pancreatin contains an unknown factor, which may be a P-containing protein degradation product, which causes marked stimulation of synthesis of nucleoprotein in presence of embryo extract. The effect is not only quant., but there are also qual. changes in the type of growth produced. This unknown factor is destroyed by heating for 30 min. at 100° in feebly alkaline solution. When cultures are grown in embryo extract containing a high concn. of mucinase there is a small increase in the amount of nucleoprotein-P. Extracts of mammalian embryos also cause an increase in nucleoprotein-P, and sheep embryo cartilage extract is very effective. Anterior pituitary extracts have no effect on nucleoprotein-P of the cultures. J. N. A.

**Identification of lead in bone tissue.** L. T. Fairhall (*U.S. Publ. Health Repts.*, 1943, **58**, 209—216).—A method of positively identifying Pb in bone tissue has been evolved which permits detailed investigation of the Pb deposit. The bone is decalcified in saturated aq. $SO_2$ with frequent changes. It is finally rinsed free from $SO_3''$. Sections can be cut satisfactorily from paraffin. Decalcification of rather large pieces of bone is fairly rapid. No Pb is removed from the bone in this process nor is the Pb deposit affected by the decalcifying medium. Bone sections are carried through xylene and alcohol to water and finally to a 1% $K_2CrO_4$–5% acetic acid solution in which they are left immersed for several days. Frequently a sufficient deposit of $PbCrO_4$ crystals is evident after 24 hr., but usually immersion for a few days is desirable. Sections are examined under a polarising or crystallographic microscope. The $PbCrO_4$ crystals are monoclinic prisms, usually well-defined, and of average length 2·5 μ. Identification of Pb in bone tissue by this procedure is definite since other metal chromates are either sol. in acetic acid or can easily be differentiated from cryst. $PbCrO_4$. While the epiphyseal portion of bone is rich in Pb, particularly in the early stage of Pb absorption, deposition occurs throughout the compact tissue on all surfaces over which blood passes. *X*-Ray studies of the bones of Pb-poisoned animals indicate that the amount of Pb, whether segregated or diffused, even in the absence of Ca, is insufficient to be revealed by ordinary *X*-ray photography. Deposition of Pb occurs in the fat cells of the red marrow. Pb storage in bone tissue occurs first as colloidally dispersed and finally as segregated cryst. masses. C. G. W.

**Giemsa stain from eosin and methylene-blue.** R. D. Lillie (*U.S. Publ. Health Repts.*, 1943, **58**, 449—452).—The variations in Giemsa stain due to variations in the composition of the constituent dyes are discussed. The relative constancy in character of the product of the oxidation of methylene-blue with a definite proportion of $K_2Cr_2O_7$ is recalled, and the substitution of eosinates of such crude azures for the usual constituents of Giemsa stain is proposed. The detailed method of prep. is described. C. G. W.

**Mallory-Heidenhain connective tissue stain. Employing domestic azocarmine B, aniline-blue, and orange G in balanced concentrations.** E. M. Schleicher (*Amer. J. clin. Path Tech. Sect.*, 1943, **7**, 35—39). C. J. C. B.

**Stain for *Treponema pallidum*.** T. G. Perrin (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 28).—Smears are prepared on slides from the exudate obtained by compressing the base of the chancre or by scraping the surface of the ulcer; they are permitted to dry spontaneously and stained for 2 min. while heating, or for 6 min. at room temp. The stain used is distilled water 10 c.c., commercial formalin 1 c.c., acetic acid 1 c.c., Ziehl's fuchsin 4 c.c.; it keeps well for a month. C. J. C. B.

**Use of filtra-pak bags in rapid method for paraffin sections.** E. S. Moss and G. V. Squires (*Amer. J. clin. Path. Tech. Sect.*, 1942, **6**, 93). C. J. C. B.

**Steel paraffin embedding boat.** A. Rhorer (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 40). C. J. C. B.

**Method for making sections of bones and teeth.** E. B. Ruth (*J. Lab. clin. Med.*, 1943, **28**, 751—755).—Fresh, fixed, or dried bone specimens are decalcified in 5% HCl. The decalcified bone is cleared in KOH glycerin solution, and finally in rising grades of glycerin. Glycerin is removed by washing in 70% alcohol, which is replaced by *n*-butyl alcohol. Paraffin is dissolved out of the sections in several changes of *n*-butyl alcohol, and the sections are placed in several changes of rising grades of glycerin. From 100% glycerin the sections are mounted in a thick glycerin jelly. C. J. C. B.

**Volumetric changes in fixed tooth specimens.** I. Jardeni (*Brit. Dent. J.*, 1943, **74**, 1—3).—Observations were made on pig embryos (length 17—19 cm.) following fixation in various solutions and on a fresh material of the same length. The effect of embedding was tested with paraffin and celloidin. The amounts of shrinkage and distortion following fixation are detailed. Shrinkage was uneven in each of the three dimensions. W. F. H.



## V.—BLOOD AND LYMPH.

**Blood.** F. H. Bethell, C. C. Sturgis, R. A. Hettig, and O. T. Mallery, jun. (*Arch. intern. Med.*, 1943, **71**, 854—903).—A review of literature for 1942. C. J. C. B.

**Culture of human marrow.** E. E. Osgood (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 540—549).—A summary of the author's work previously reported. P. C. W.

**Mammalian red cells as source of "small particles."** N. Sigurdsson (*J. Exp. Med.*, 1943, **77**, 315—322).—Small particles similar to those isolated from other tissues (cf. Kabat, A., 1940, III, 228) were isolated from hæmolysed and ultracentrifuged horse erythrocytes. One third of the dry wt. of the particles is lipins. The particles produce hæmolysins against homologous red cells when inoculated into a foreign species. Some at least of the particles isolated from whole organs represent disintegrated "stroma" as the particles can be isolated from red cells which do not contain visible granules. A. S.

**Formation of red blood corpuscles.** F. Duran-Jorda (*Lancet*, 1943, **244**, 513—514).—Embryological studies suggest that red blood corpuscles are formed all over the body from plasma cells. When the plasma cell disintegrates the nucleus with a small halo of protoplasm corresponds to the normoblast. The red blood corpuscle is a simple colloidal droplet. The accepted lymphocyte is a stage in the development of the normoblast. The platelets are remnants of broken-down plasma cells. C. A. K.

**Hæmopoiesis in chorionic villi of placenta of platyrrhine monkeys.** —See A., 1943, III, 618.

**Comparison of erythrocyte numbers in normal and [sex] hormone-treated fowl.** L. V. Domm, E. Taber, and D. E. Davis (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 49—50).—Red cell count is increased by androgens and decreased by œstrogens in fowls of both sexes, various ages, and castrated or normal. V. J. W.

**Blood pictures of active and hibernating ground squirrels.** J. Stuckey and R. M. Coco (*Amer. J. Physiol.*, 1942, **137**, 431—435).—The changes in the blood pictures of the ground squirrel due to hibernation (induced) were a decrease in total red cell count and hæmoglobin, a decrease in neutrophils and an increase in lymphocytes and monocytes, 0·75% decrease in sp. gr., 16% in creatinine, and 14% in glucose. M. W. G.

**Effect of change of altitude on blood in man.** R. C. Lewis, A. Iliff, A. M. Duval, and G. M. Kinsman (*J. Lab. clin. Med.*, 1943, **28**, 860—866).—Increases in red cell count, hæmoglobin %, and packed red cell vol. were usually found with increases in altitude (up to 8720 ft.). Sp. gr. changes were unimportant. C. J. C. B.

**Effect of change of altitude on corpuscular constants.** R. C. Lewis, G. M. Kinsman, A. Iliff, and A. M. Duval (*Amer. J. clin. Path.*, 1943, **13**, 208—214).—No changes were found in the mean corpuscular-hæmoglobin, -hæmoglobin concn. and -vol. (Wintrobe) in 7 subjects, at altitudes of 910, 5280, and 8720 ft. above sea level. C. J. C. B.

**Output of bile pigment by infants and children [relation to blood destruction].** R. J. Tat, T. J. Greenwalt, and W. Dameshek (*Amer. J. dis. Child.*, 1943, **65**, 558—570).—In 30 normal newborn infants, the daily fæcal bilirubin excretion (in mg. per day) for the 1st, 2nd, and 3rd 5-day periods of life was 8·6, 5·7, and 5·3; fæcal urobilinogen was a trace to 0·70. Below 2 years, fæcal urobilinogen was 2·5, and for children in 3—11-year group the vals. were 2—7. For 8 normal newborn infants the hæmolytic index, *i.e.*, (total daily urobilinogen or bilirubin in mg. × 100) ÷ (total circulating hæmoglobin in g.), was 0·5—29·7 (average 9·6). This range is higher than that found for the older age groups and corresponds to the increased destruction of blood which occurs in the early postnatal period. C. J. C. B.

**Rate of blood-regeneration after hæmorrhage.** S. Alstead (*Lancet*, 1943, **244**, 424—426).—350—1300 c.c. (average 710 c.c.) of blood were removed from 48 male subjects aged 27—92 (average 60) years. The min. hæmoglobin concn. occurred 7 days after venesection and 50% of subjects were still anæmic after 5 weeks; recovery was incomplete in 10% after 15 weeks. The low hæmoglobin concn. is attributed to over-dilution of the blood and was not related to the vol. of blood lost. C. A. K.

**Effect of vitamin-$B_1$ on survival time after hæmorrhage and application of cold.**—See A., 1943, III, 664.

**Photometric determination of iron in blood.** R. S. Pereira (*Rev. Brasil. Biol.*, 1943, **3**, 29—35).—A method for the determination of pseudohæmoglobin-Fe of whole blood is described. I. C.

**Iron treatment of anæmia with Ferro-Redoxon-Roche.** L. Feil (*Schweiz. med. Wschr.*, 1942, **72**, 1121—1125).—The prep. was given to 30 women during the puerperium and to 23 cases suffering from Fe-deficiency anæmia. A marked increase in reticulocyte and red cell count and in hæmoglobin concn. was observed in all cases. A. S.

**Refractory anæmia. III. Refractory anæmias with cellular marrow.** L. S. P. Davidson, L. J. Davis, and J. Innes (*Edinb. Med. J.*, 1943, **50**, 431—443; cf. A., 1943, III, 623).—6 cases of refractory idiopathic macrocytic anæmia with hypercellular megaloblastic marrows are described; treatment for 4—10 weeks was followed by complete recovery in all cases and prognosis is good. 2 cases of chronic granulocytopenia (cellular, normoblastic marrow with arrested myelocyte maturation), which have resisted treatment for over 2 years, are also described. H. S.

**Sternal marrow in Banti's syndrome and other splenomegalic states. Effect of splenectomy.** L. R. Limarzi, R. M. Jones, J. T. Paul, and H. G. Poncher (*Amer. J. clin. Path.*, 1943, **13**, 231—247).—In 33 cases of Banti's syndrome and allied conditions the marrow first shows myeloid hyperplasia, and there is moderate anæmia and leucopenia; splenectomy restores the blood to normal. Later in the disease, the marrow shows a "maturation arrest" of the myeloid and megakaryocytic tissue with peripheral leucopenia, neutropenia, thrombocytopenia, and myeloid immaturity; splenectomy may still restore the blood to normal. In the last stages of the condition, with cirrhosis of the liver, the marrow shows marked erythroid immaturity, as well as the above myeloid changes, and the results of splenectomy are poor except in children. In Felty's syndrome the hæmatological findings are similar to those in Banti's without cirrhosis and the effect of splenectomy is spectacular but transient. There is also a marked reticulum hyperplasia and erythrophagocytosis in the spleen. C. J. C. B.

**Anæmias of malnutrition.** H. C. Trowell (*Lancet*, 1943, **244**, 43—46).—A review. C. A. K.

**Effects of inanition and riboflavin deficiency on blood picture of the rat.**—See A., 1943, III, 665.

**Erythropoiesis in scurvy.** M. C. G. Israëls (*Lancet*, 1943, **244**, 170—172).—In 2 of 3 cases of scurvy bone marrow studies showed evidence of diminished erythropoiesis. There was no evidence of failure of maturation of erythroblasts. Ascorbic acid administration restored erythropoiesis to normal. C. A. K.

**Chronic hæmolytic anæmia with paroxysmal nocturnal hæmoglobinuria.** B. J. Hoffman and R. R. Kracke (*J. Lab. clin. Med.*, 1943, **28**, 817—827).—If the sleeping habits of the patient were reversed hæmoglobinuria still occurred during sleep, whether during day or night. No abnormal blood-pH variations were noted. The renal threshold for hæmoglobin was 120—180 mg.-%. The severity of nocturnal exacerbations of intravascular red blood cell destruction was lessened by aq. extracts of adrenal medulla or cortex. Synthetic adrenaline or deoxycorticosterone had no effect. Prostigmine, eserine, and pilocarpine were more efficacious than adrenal extracts in lessening nocturnal hæmoglobinuria; pilocarpine hydrochloride (3 mg. daily) was the most effective; on each occasion after its use hæmoglobin disappeared from the urine for days or even weeks. C. J. C. B.

**Morphology of erythrocytes in erythroblastosis fœtalis.** E. H. Reisner, jun. (*Arch. intern. Med.*, 1943, **71**, 230—255).—In 23 of 24 cases a biphasic Price–Jones curve consisting of a normocytic and a macrocytic peak, or 2 macrocytic peaks, was observed. In 1 case a monophasic microcytic curve was observed; the blood smear showed spherical microcytes, but 3 months later the smear was normal. The morphological changes in erythrocytes in erythroblastosis fœtalis resemble those observed in experimental hæmolytic anæmia due to antigen–antibody reactions. Blood smears in 6 cases showed varying nos. of megaloblasts and postmortem examination in 4 cases showed hepatic damage. The presence of megaloblasts and the macrocytosis may be the result of deficient erythrocyte maturation factor due to hepatic damage. C. J. C. B.

**Simplified procedure for typing and compatibility test for blood donors.** D. E. McBride and A. S. Giordano (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 43—45). C. J. C. B.

**Preparation of grouping serum.** R. A. Shooter (*Lancet*, 1943, **244**, 202—204).—The titre of blood-grouping serum is best tested against pooled red cell suspensions. Cold agglutinins active at 5—7° were found in 32% of 4710 sera, and 29 of 10,000 sera contained iso-agglutinins active at room temp. Methods of selecting high-titre sera and of preparing cold agglutinin-free sera are described. C. A. K.

**Blood group tests of Budapest university students.** V. Molnár (*Magyar Orv. Arch.*, 1941, **42**, 273—284).—Distribution of blood groups was: *O* 32·7, *A* 42·9, *B* 16·6, *AB* 7·8%. The vals. according to Bernstein were: *r* 57·2, *p* 29·8, *q* 13·0 and the Hirszfeld index 2·07. The distribution of groups was different in different races and in town and country folk. Distribution of *MN* factors was: *M* 29·4, *MN* 52·6, and *N* 18·0%. M. A. B.

**Origin of blood platelets.** G. Kiszely (*Magyar Orv. Arch.*, 1941, **42**, 251—263).—Wright's theory of the origin of blood platelets is supported by the disruption of the cells of *Triton* blood at the beginning of coagulation. The platelets were counted by diluting with vital stain. M. A. B.

**Simple method for separation of blood serum.** L. C. Steeves (*J. Lab. clin. Med.*, 1943, **28**, 994). C. J. C. B.

**Transfusion of liquid filtered serum.** S. Ackroyd, G. A. Harrison, and L. E. R. Picken (*Lancet*, 1943, **244**, 268—269).—The prep. of liquid filtered serum is described and the results of clinical use in 838 patients are discussed. A 4% reaction rate was noted. C. A. K.

**Method of re-infusion of abdominal cavity blood.** E. Anderes and M. Laszczower (*Schweiz. med. Wschr.*, 1942, **72**, 933—936).—Re-infusion of the patient's own blood from the abdominal cavity in cases of extrauterine pregnancy ruptures is recommended. A special filter pump is described. A. S.

**Red-cell suspension transfusions.** L. Watson (*Lancet*, 1943, **244**, 107—111).—46 transfusions of red-cell suspensions were given to 23 patients with various types of anæmia. The suspension contained about 18 g. of hæmoglobin per 100 c.c., and is of particular val. where an increase of the recipient's blood vol. is undesirable. A means of calculating the rise of hæmoglobin concn. is suggested. C. A. K.

**Use of carbon monoxide in determination of blood volume.** L. Szécsényi-Nagy and E. B. Hatz (*Biochem. Z.*, 1940, **306**, 108—112).—The procedure of Rusznyák and Hatz (A., 1935, 1517) is applied to determination of the CO content of blood (after inhalation of CO) and hence the blood vol. W. McC.

**Use of radioactive phosphorus for determining total circulating erythrocyte volumes.** R. S. Anderson (*Amer. J. Physiol.*, 1942, **137**, 539—543).—Net rates of transfer of radioactive $^{32}P$ into and out of rabbit erythrocytes *in vitro* were studied; the rate of loss of radioactivity from the circulating blood of rabbits into which such cells containing radioactive $^{32}P$ were introduced was low for 5—25 min. after injection. Vals. for circulating erythrocyte and total blood vol. can thus be calc. M. W. G.

**Synthesis and properties of radioactive dibromo-trypan-blue and radioactive dibromo-Evans-blue.** L. H. Tobin and F. D. Moore (*J. clin. Invest.*, 1943, **22**, 155—159).—The synthesis of radioactive brominated derivatives of trypan-blue and Evans-blue is described. The brominated dyes are colloidal in aq. solution, as shown by their relative non-diffusibility through Cellophane membranes, and their conduct on attempts at filtration. The charge is negative. By the Tiselius technique, the bromo-dyes, like the non-brominated ones, migrate preferentially with albumin if the concn. in plasma is about 0·05% or lower. The bromo-dyes are less sol. in water than the non-brominated dyes and stain Cellophane to a greater extent. The addition of 2 Br atoms produces a dye which is more red than the non-brominated dyes. C. J. C. B.

**Ferritin. IV. Occurrence and immunological properties.** S. Granick (*J. Biol. Chem.*, 1943, **149**, 157—167).—Ferritin that occurs in different organs of the same animal is immunologically identical, but it is species-sp. Methods for the isolation of ferritin from different organs and species are based on the solubility and heat-stability of horse-spleen ferritin. Horse, human, and dog *apo*ferritins have approx. the same sedimentation const. and mol. wt. of approx. $5 \times 10^5$. Human ferritin occurs in spleen, liver, and bone marrow. The lowest val. was obtained in lymphoid leukæmia following acute reaction to arsphenamine, and the highest vals. in monocytic and lymphocytic leukæmia. Ferritin from a case of cirrhosis of the liver was almost colourless and contained only 5% of Fe (instead of 23%). *apo*Ferritin is utilised in blood formation, as indicated by the low vals. obtained from frequently bled horses. It is present in decreasing amounts in the following organs: spleen, bone marrow, liver, testis, kidney, adrenal, pancreas, ovary, and lymph node. It is not present in blood, striped muscle, pituitary, and gastric mucosa. P. G. M.

**Antithrombin content of blood in anaphylaxis.** H. Dyckerhoff and G. Ruhl (*Biochem. Z.*, 1940, **303**, 316—323; cf. A., 1939, III, 892).—The antithrombin content of the blood of rabbits is increased by injection of protein (caseinogen, serum, papain), although blood coagulation time is unaffected. Since the antithrombin of rabbits' blood retards or prevents coagulation by thrombin of rabbit, sheep, horse, and ox, it is concluded that thrombin and antithrombin possess no species-specificity. W. McC.

**Action of proteolytic enzymes on fibrinogen solutions. Separation of fibrinogen from "pepsin-inhibitor."**—See A., 1943, III, 682.

**Highly active barium salt of heparin and its fractionation into two chemically and biologically different constituents.** M. H. Kuizenga

and L. B. Spaulding (*J. Biol. Chem.*, 1943, **148**, 641—647).—When ox lung is autolysed in presence of water at 35° for 0·5 hr. and then at 25—30° for 24 hr. the yield of heparin is about 2·5 times that previously obtained. The highest yield is $10^6$ units per 100 lb. of lung tissue. Purification of the crude heparin and the prep. of the Ba salt with activity of 85—100 units per mg. (85—90% yield) are described. The most active Ba salts when fractionated at pH 8·0 by pptn. with acetone yield a very active Na salt and another Na salt of lower activity which cannot be purified into a product of higher activity. J. N. A.

**Reaction of heparin with proteins and complex bases.** L. B. Jaques (*Biochem. J.*, 1943, **37**, 189—195).—The formation of stable compounds of fixed composition from heparin with casein, toluidine-blue, gelatin, and protamine (salmine) is described. Their dissociation consts. are markedly affected by pH, trichloroacetic acid, and inorg. salts. The formation of compounds with a sufficiently low dissociation const. is followed by loss of anticoagulant activity of the heparin. The results are discussed, particularly in relation to blood clotting. R. L. E.

**Coagulation of fibrin as polymerisation–crystallisation process.** U. Ebbecke (*Biochem. Z.*, 1940, **304**, 177—193).—The formation and development of fibrin as a system of acicular, fibrous, or granular crystallites are discussed. Theoretical aspects include the no. and type of nuclei, the non-sp. catalysing effect of surfaces (vessels and foreign particles), and the inhibitory effect of extraneous substances. The denaturation of fibrinogen is discussed in relation to induction period, formation of nuclei, development and breakdown of mol. chains, and stabilisation phenomena. F. O. H.

**Effect of vitamin-*K* on hypoprothrombinæmia induced by Dicumerol in man.** S. Shapiro, M. H. Redish, and H. A. Campbell (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 12—15).—Curative effect of 2-methyl-1 : 4-naphthaquinone observed by Overman *et al.* in rabbits (A., 1943, III, 6) also occurs in man. V. J. W.

**Prothrombin activity during pregnancy and lactation.** J. B. Field, R. S. Overman, and C. A. Baumann (*Amer. J. Physiol.*, 1942, **137**, 509—514).—Prothrombin activity of rat plasma was high during the last week of pregnancy; it decreased after parturition. Hypoprothrombinæmia induced by 3 : 3′-methylenebis-(4-hydroxycoumarin) was milder in pregnant rats than in the non-pregnant; in lactating rats the anticoagulant was only 18% as effective as in non-lactating controls. Post lactation, marked hypoprothrombinæmia was readily induced by the anticoagulant. M. W. G.

**Relation between prothrombin clotting time and plasma-fibrinogen concentration.** R. C. Page, E. J. de Beer, and Z. Bercovitz (*J. Lab. clin. Med.*, 1943, **28**, 910—912).—No relationship exists between prothrombin clotting time and plasma-fibrinogen concn. in normal subjects and in patients with a variety of gastrointestinal lesions. C. J. C. B.

**Prophylaxis of hypoprothrombinæmia of newborn infants with vitamin-*K*.** O. Ballon (*Schweiz. med. Wschr.*, 1942, **72**, 1119—1121).—Blood prothrombin concn. of newborn infants is lowest on the 1st day of life. Intravenous injection of 10 mg. of 2-methyl-1 : 4-naphthaquinol disuccinate into the mother 5—10 hr. before delivery increases the blood-prothrombin of the newborn. A. S.

**Coumarin and dicoumarin derivatives and prothrombin level.** J. Lehmann (*Lancet*, 1943, **244**, 458—459).—Coumarin and 2 of its derivatives, and 3 derivatives of dicoumarin, had no advantages over the original 3 : 3′-methylenebis-(4-hydroxycoumarin) when tested in rabbits for their effect on prothrombin level and toxicity. An active, stable, water-sol. substance for intravenous injection was not found. C. A. K.

**Glucose and citrate anticoagulant solution.** W. N. Hailstone (*Lancet*, 1943, **244**, 336—337).—Prep. of solutions and use of apparatus is described. The solution has a pH of 7·8 and caramelisation is avoided. C. A. K.

**Severe sublingual and paratracheal hæmorrhage in hæmophilia with recovery following tracheotomy.** K. H. Baird and M. Fox (*J. Pediat.*, 1943, **23**, 90—94).—A case report. C. J. C. B.

**Problem of thrombosis in obstetrics and gynæcology.** T. Koller (*Schweiz. med. Wschr.*, 1942, **72**, 1008—1009). A. S.

**Enumeration and differentiation of leucocytes in counting chamber with propylene glycol–aqueous stains.** T. G. Randolph (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 20—22).—0·1% of methylene-blue in propylene glycol is mixed with an equal vol. of 0·1% aq. eosin and the mixture used as diluting fluid for blood in the white-cell pipette. Red cells are hæmolysed and white cells are differentially stained and counted in the usual chamber. V. J. W.

**Tropical eosinophilia.** R. J. Weingarten (*Lancet*, 1943, **244**, 103—105).—A new disease entity includes bronchitis, leucocytosis, and eosinophilia (up to 88% of white cells). It occurs in India and responds rapidly to neoarsphenamine or quinquevalent org. arsenicals. C. A. K.

**Chronic pulmonary inflammation with blood eosinophilia.** M. Kartagener (*Schweiz. med. Wschr.*, 1942, **72**, 862—864).—Three types of pulmonary inflammation with up to 45% eosinophils in the white cell count are discussed. A. S.

**Spontaneous rupture of the spleen due to acute leukæmia or acute leukæmia due to trauma to spleen.** A. S. Rubnitz and E. B. Floersch (*J. Lab. clin. Med.*, 1943, **28**, 972—982).—Report of case and review. C. J. C. B.

**Leukæmia.** J. D. Kirshbaum and F. S. Preuss (*Arch. intern. Med.*, 1943, **71**, 777—792).—A clinical and pathological study of 123 fatal cases in a series of 14,400 necropsies. The types present were: stem cell leukæmia (28 cases), myelogenous leukæmia (53 cases), lymphatic leukæmia (37 cases), and monocytic leukæmia (5 cases). C. J. C. B.

**" Atypical " Gaucher's disease.** J. V. Petit and E. M. Schleicher (*Amer. J. clin. Path.*, 1943, **13**, 260—266).—An atypical case of Gaucher's disease is reported in a Jewish male, age 79 years. There were no clinical findings of Gaucher's disease. The bone marrow was primarily affected. A lymph node in the region of the porta hepatis was moderately and the spleen least involved. The latter organ showed only a few scattered Gaucher cells. (6 photomicrographs.) C. J. C. B.

**Biochemical changes in body fluids after death.** W. W. Jetter and R. McLean (*Amer. J. clin. Path.*, 1943, **13**, 178—185).—A lecture. C. J. C. B.

**Hypoproteinæmia in surgical patients.** D. Casten and M. Bodenheimer (*Surg. Gynec. Obstet.*, 1941, **72**, 178—191).—Discussion with reports of typical cases with the conclusion that hypoproteinæmia is due to disorder of the protein-producing organ (? liver) rather than to protein loss or lack. P. C. W.

**Constancy of chemical composition of serum-proteins regenerated on various dietary regimes.** W. A. Murrill and W. D. Block (*Arch. Biochem.*, 1943, **1**, 365—368).—There was no change of total N, total S, cystine, tyrosine, tryptophan, histidine, arginine, or lysine in the total serum-proteins of dogs whose diet was supplemented with casein, lactalbumin, beef serum, or yeast. E. R. S.

**Changes in serum-proteins of dogs during type I pneumococcal pneumonia.** B. Vassel, R. Partridge, and M. L. Crossley (*Arch. Biochem.*, 1943, **1**, 403—413).—Serum-protein level was const. for 50 hr. after inoculation, then the -albumin concn. fell to a min. of half the normal and the -globulin rose to a max. of twice the normal in 90—160 hr. The return to normal requires several days. The cystine content of the -globulins remained const., but that of the -albumins decreased with decreasing -albumin level. E. R. S.

**Barbour and Hamilton's drop method [to determine plasma-proteins].** F. A. de la Balze (*Medicina*, 1942, **2**, 449—473).—Barbour and Hamilton's method for the determination of the relative density of liquids is described with particular reference to proteins in plasma and serum. I. C.

**Fractionation of normal serum-proteins by electrophoretic and sodium sulphate methods.** H. L. Taylor and A. Keys (*J. Biol. Chem.*, 1943, **148**, 379—381).—Tiselius' electrophoretic method and fractionation with 22·5% $Na_2SO_4$ gave an average albumin/globulin ratio of 1·97 and 2·62 respectively in normal dialysed whole plasma, about 5% more N being assigned to the albumin fraction by the $Na_2SO_4$ technique. The correlation between the two methods is, however, better in normal than in pathological sera. E. C. W.

**Morphologic changes in normal rabbits induced by intravenous injection of crystallised bovine serum-albumin.** O. T. Bailey and C. v. Z. Hawn (*Amer. J. Path.*, 1943, **19**, 267—285).—Injection of a 25% solution of cryst. bovine albumin, 1 g. per kg. body wt., caused no change in the nutriton or behaviour of the rabbits. The spleen was enlarged and there was an increase in the amount of phagocytosis of leucocytes and of red blood cells in the pulp and sinusoids of the spleen. Phagocytosis of blood cells was also found in the Kupffer cells of the liver, in the lymph nodes, and in the bone marrow. Kidneys showed swelling and dispersion of the cytoplasm of the epithelium in the ascending loop of Henle and in the distal convoluted tubules. There was no albuminuria. These changes are supposed to indicate that albumin filtered out through the glomeruli and entered the tubular epithelial cells more rapidly than it was reabsorbed in the circulating blood. The histological changes were reversible, the organs returning to normal even after 12 injections. Cryst. bovine serum-albumin is not stored in the tissues of rabbits. (10 photomicrographs.) C. J. C. B.

**Simple chamber for estimating serum-pH with glass electrode.** A. H. Free (*J. Lab. clin. Med.*, 1943, **28**, 1033—1035).—A simple chamber made from a 4-way glass stopcock is described for use with a glass electrode and pH meter in the determination of serum-pH. The technique for measurement of serum-pH with the chamber is described. C. J. C. B.

**Blood-amylase activity in disease of carbohydrate metabolism and in non-diabetic pancreatic disease.** S. Z. Sorkin (*J. clin. Invest.*, 1943, **22**, 329—333).—The blood amylolytic activity of 25 normal patients was 0·5—4·4 mg. (average 2·6 mg.) of maltose per c.c. of serum per hr. Normal blood-amylase vals. were obtained in 56

diabetics, 11 patients with hepato-cellular liver disease, 8 with other types of liver disturbance, and 1 with glycogen storage disease. Markedly increased transitory blood-amylase activity was found in 3 patients with acute pancreatic œdema and a persistent increase was found in a patient with a carcinoma of the head of the pancreas. C. J. C. B.

**Clinical value of serum-amylase test.** E. F. Lewison (*Surg. Gynec. Obstet.*, 1941, **72**, 202—212).—Age, sex, diet, vitamin deficiency, and starvation had no effects on serum-amylase vals. 94% of 720 patients with diseases other than mumps or those of the biliary system had normal serum-amylase levels. The serum-amylase was raised in mumps and pancreatitis and depressed in liver diseases. P. C. W.

**[Blood] carbonic anhydrase in newborn infants.** S. S. Stevenson (*J. clin. Invest.*, 1943, **22**, 403—409).—The concn. of carbonic anhydrase in the blood of newborn infants is less than ½ and in premature infants ¼ of that found in adults. 13 infants who exhibited unexplained cyanosis showed low levels of carbonic anhydrase. Improvement in the cyanosis and general condition was accompanied by a rise in the blood concn. of carbonic anhydrase. C. J. C. B.

**Serum-choline-esterase and pathological conditions.** F. Huidobro and R. Croxatto (*Anal. Acad. Biol. Univ. Chile*, 1939, **3**, 91—103).—The choline-esterase activity of serum of patients does not depend on age, sex, diet, exercise, temp., pulse rate, and blood pressure, but mainly on the general condition. In 96% the serum-choline-esterase activity was decreased, and in 80% it varied with the general state. In 17% serum-choline-esterase in patients was within the normal range. I. C.

**Determination of serum-bilirubin and sulphanilamide.** F. Rappaport and F. Eichhorn (*Lancet*, 1943, **244**, 62—63).—Methods avoiding pptn. of protein are described in detail. C. A. K.

**Hypoglycæmia in a murderer.** D. Hill, W. Sargant, and M. E. Heppenstall (*Lancet*, 1943, **244**, 526—527).—A young man of 20 murdered his mother. Investigations showed that he developed an abnormal electroencephalogram whenever his blood-sugar fell to 80—90 mg.-% (in normal subjects such changes appear at 50 mg.-%). He was an abnormal individual with a psychopathic inheritance and he was considered not entirely responsible for his actions at the time of the murder. C. A. K.

**Micro-application of Fujita and Iwatake's method of determining blood-sugar.** W. Székessy (*Biochem. Z.*, 1940, **303**, 364—367; cf. A., 1932, 75).—Smaller quantities (5—50 mg.) of blood and reagents are taken and 0·001N-$Na_2S_2O_3$ is used for titration. W. McC.

**Micro-diffusion methods. Blood-glucose.** E. O'Malley, E. J. Conway, and O. Fitzgerald (*Biochem. J.*, 1943, **37**, 278—281).—A micro-diffusion method for determination of blood-glucose using a smaller micro-diffusion unit than the standard is described. 0·1 c.c. of oxalated blood is used and the results are very similar to those obtained with the Hagedorn–Jensen method modified by substitution of the Fujita–Iwatake method (A., 1932, 75) of deproteinisation with Cd instead of Zn. The range of the method for the blood sample which includes the internal control is 300 mg.-%, but for other blood samples done at the same time it is 500 mg.-%. Less manipulation and fewer solutions are required than for other methods. Anti-glycolytic precautions must not be taken in collecting the blood; the method is independent of glycolysis up to 5 hr. at room temp., or for 8—10 hr. if the blood is stored at 0°. J. N. A.

**Simplification of iodometric titration in blood-sugar determination by ferricyanide method.** L. Jendrassik and A. Polgár (*Biochem. Z.*, 1940, **304**, 271—274).—The Hagedorn–Jensen method of blood-sugar determination is simplified by the use of, first, a solution containing 1% of starch, 30% of KI, and 0·2% of $NaHCO_3$ and, secondly, 1 vol. of 45% aq. $ZnSO_4$ + 2 vols. of 40% (vol.) $H_2SO_4$. $Na_2S_2O_3$ solutions are best stabilised by means of 0·02—0·1N-NaOH. P. G. M.

**Significance of plasma-ascorbic acid levels in Nebraska children.** J. L. Gedgoud, V. M. Wilder, and J. A. Henske (*J. Pediat.*, 1943, **23**, 39—49).—On a daily intake of 60—80 mg. of ascorbic acid, a plasma level of 0·7 mg.-% or more was attained in 81% of children entering the hospital without infection, regardless of the entrance val. Of 12 children with infections, acute and chronic, 100—150 mg. daily was needed to raise the plasma level to 0·7 mg.-% or more in 11 over periods of 3—31 days. C. J. C. B.

**Effects of insulin on blood-lipins of man.** A. Kaplan, C. Entenman, and I. L. Chaikoff (*Endocrinol.*, 1943, **32**, 247—250).—Large doses of insulin (110—460 units), given to human subjects for treatment of schizophrenia, did not alter the concn. of the lipins in the blood. G. P.

**Blood constituents of carp and trout.** J. B. Field, C. A. Elvehjem, and C. Juday (*J. Biol. Chem.*, 1943, **148**, 261—269).—Blood samples were obtained by cardiac puncture of carp and trout anæsthetised by electric shock. They exhibit the following differences from mammalian blood: low erythrocyte count; high cell vol. and corpuscular hæmoglobin; low plasma-protein and hæmoglobin; non-protein-N is mainly amino-acid; total lipin and cholesterol are high, and above 50% of the lipins consist of cholesterol compared with 25% in human blood. The distribution of vitamin-*A* and -*C* varies widely in these two fish, whilst -$B_1$ is frequently absent and, when added to carp blood, is partly inactivated. The ratio carotenoids : -*A* is 25 : 1 in carp blood compared with 2·5 : 1 in trout blood, and the albumin : globulin ratios are 3·55 : 1 and 2·25 : 1, respectively. P. G. M.

**Hydrolysis of blood by acid and alkali in relation to study of alimentary ethyl acetate curve of blood. Effect of hormones, glucose, and poisons. Endogenous and exogenous alcohol of blood.** H. R. Kanitz and U. Sellschopp (*Biochem. Z.*, 1940, **306**, 77—90; cf. A., 1940, III, 630).—Esters in blood are hydrolysed by addition of 4N-NaOH or -$H_2SO_4$ and determined by oxidation. Application of this procedure and determination of volatile products in the blood of rabbits to which ethyl acetate alone or together with hormones (*e.g.*, thyroxine), glucose, or poison (nicotine, caffeine) is administered fail to indicate the origin of the products but show that complex metabolic processes occur. The alcohol vals. for untreated blood are much lower than those for blood treated with acid or alkali. W. McC.

**Colorimetric determination of amino-nitrogen in blood.** E. G. Frame, J. A. Russell, and A. E. Wilhelmi (*J. Biol. Chem.*, 1943, **149**, 255—270).—The method described is a modification of Folin's condensation with β-naphthaquinonesulphonate in alkaline solution (cf. Danielson, A., 1933, 965) for use with a photo-electric colorimeter. The method may be applied to samples containing 4—40 μg. of amino-N and duplicate determinations be made on 0·2 ml. of blood. The principal naturally-occurring substances that interfere are $NH_3$, uric acid, and allantoin. Unconjugated aminosulphonamides give a val. equiv. to 5—8% of their wt. as amino-N, whilst conjugated compounds do not react. The visible absorption spectra of the reaction products of 17 amino-acids are given. H. G. R.

**Plasma-amino-acid retention, as evidence of impaired liver function. Investigation in children with nephrosis and liver disease.**—See A., 1943, III, 654.

**Serum-acid phosphatase in prostatic carcinoma.**—See A., 1943, III, 659.

**Colorimetric determination of acetaldehyde in blood.** E. Stoty (*J. Biol. Chem.*, 1943, **148**, 585—591).—Acetaldehyde is distilled from a tungstic acid filtrate, collected in $NaHSO_3$, and determined colorimetrically with 4-hydroxydiphenyl. R. L. E.

**Determination of *p*-aminobenzoic acid, conjugated *p*-aminobenzoic acid, and *p*-nitrobenzoic acid in blood.** H. W. Eckert (*J. Biol. Chem.*, 1943, **148**, 197—204).—The oxalated or citrated blood is mixed with water and, after laking, with trichloroacetic acid. The mixture is filtered and treated with excess of $NaNO_2$, the unused material being subsequently destroyed by $NH_2{\cdot}SO_3NH_4$. *p*-Aminobenzoic acid is determined colorimetrically after addition of dimethyl-α-naphthylamine. Conjugated *p*-aminobenzoic acid is determined after acidic hydrolysis and *p*-nitrobenzoic acid after reduction by $TiCl_3$. H. W.

**Determination of dihydroxyacetone in blood.** W. J. Turner, B. H. Kress, and N. B. Harrison (*J. Biol. Chem.*, 1943, **148**, 581—584).—Ergothioneine is removed by $Ag_2SO_4$, and excess of Ag by NaCl. Dihydroxyacetone is determined by reduction of phosphomolybdic acid and back titration with $Ce_2(SO_4)_3$. A const. blank of 1·31 mg. per 100 ml. of blood, based on experiments, is deducted to allow for other reducing substances. The normal concn. of dihydroxyacetone in blood is below 0·7 mg. per 100 ml. R. L. E.

**Oxalate content of blood.** J. F. B. Barrett (*Biochem. J.*, 1943, **37**, 254—256).—There is no evidence that the amount of oxalate in blood is 2—4 mg.-% as claimed by Kamiya *et al.* (A., 1938, III, 93) and Merz and Maugeri (A., 1931, 1440). The ethyl oxalate method of Barber and Gallimore (A., 1940, III, 328) is confirmed, but the amount of oxalate involved is too small to permit very accurate determination. Moreover, it is not definite that all the oxalate found by this method pre-exists as such in blood. J. N. A.

**Determination of uric acid in whole blood and serum.** J. W. Mull (*J. Lab. clin. Med.*, 1943, **28**, 1038—1042).—The direct method of Benedict was most suitable for determining uric acid with the photoelectric colorimeter. Whole blood or serum may be used since there is a direct correlation between the findings. C. J. C. B.

**Blood-amines.** D. Richter and M. Lee (*J. Ment. Sci.*, 1942, **88**, 127—133).—Blood-alkylamines and -"amino-lipins" (lipins containing a basic N atom) were determined in 58 psychotic patients. The blood invariably contained less than 8 p.p.m. of alkylamines of the type of β-phenylethylamine or *iso*amylamine. The amino-lipin content was significantly higher in the blood of the schizophrenic than the other patients. G. D. G.

**Bromine content of human blood.** J. Straub (*Biochem. Z.*, 1940, **303**, 398—403).—Average Br content of human blood in Vienna, Budapest, and other places in Hungary is 0·300—0·543 mg.-%, but in Debrecen it is 1·066 mg.-%. The corresponding vals. for urine are 0·425—0·709 and 1·938, for drinking-water 0·020—0·065 and

0·120, and for cow's milk 4·932—6·444 and 15·492 mg.-%. The Br content of the blood of pigs and calves is much higher in Debrecen than in other places in Hungary. The results indicate that the Br content of blood and urine partly depends on dietary intake and that the Br of blood has some physiological rôle. W. McC.

## VI.—VASCULAR SYSTEM.

**Method for obtaining records of tortoise sinus with data on extrasystoles and length of cycles following stimulation.** F. E. Emery and T. A. Loomis (*J. Lab. clin. Med.*, 1943, **28**, 889—896).—Simultaneous graphic records of the contractions of the sinus venosus, auricles, and ventricles of turtles were obtained. An electrical stimulus applied during the refractory period of the sinus did not cause an extrasystole, but the rhythm may be disturbed. Electrical stimuli applied to the sinus during its quiescent period caused a contraction which may or may not be transmitted to the auricles and ventricles. The pause in the sinus (returning cycle) following an extra or induced contraction of the sinus may be shorter than, longer than, or equal to normal. The factors determining the length of this pause are governed mostly by the time in the cycle when the extrasystole is induced. The height of the next contraction of the sinus after an extrasystole of the sinus may be higher than normal even though the pause preceding it was shorter than normal. The strength of the stimulus may be changed from threshold to considerably above threshold without affecting the results. C. J. C. B.

**Potential changes in injured cardiac muscle.** M. Calabresi and A. J. Geiger (*Amer. J. Physiol.*, 1942, **137**, 440—446).—E.c.g. records of potential changes in injured hearts (exposed hearts of frogs) were compared with direct measurements of potential differences through a microvoltmeter (Burr, *et al.*, *Yale J. Biol. Med.*, 1936, **9**, 65). The surface of an area of injury in the beating heart is the site of important changes in electrical activity during systole and the monophasic electrogram obtained from the injured heart results predominantly from potential changes at the electrode over the injured area. M. W. G.

**Influence of inspiration on heart rate.** J. J. Lewis, A. E. Lewis, and V. E. Hall (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 52—54).—The cardiac slowing which accompanies inspiration is elicited best when the chest is held voluntarily in an inspiratory position against atm. pressure. If pulmonary pressure is reduced to −20 mm. Hg by suction against a manometer, slowing is less, and is less still if the chest is in an expiratory position. It is suggested that no recognised reflex is involved. V. J. W.

**One-ventricle pump and pulmonary arterial pressure of turtle.** R. A. Woodbury and G. G. Robinson (*Amer. J. Physiol.*, 1942, **137**, 628—636).—Pressure pulses from right and left aortas are synchronous and show equal pressures. Blood flow or systolic pressure rise occurs earlier in pulmonary artery than in aortas. During the last part of systole pulmonary pressure is 2 or more mm. Hg below that in aortas, while during diastole it descends more rapidly and to a lower val. than in aortas. Cardiac output is increased by an increased heart rate and/or increased diastolic filling of the ventricle. The presence of only one ventricle allows the turtle to regulate effectively distribution of blood flow between systemic and pulmonary areas. Adrenaline (intravascularly) increased peripheral resistance of the systemic vessels and muscle tone of the great pulmonary arteries, but had no effect on peripheral resistance of the pulmonary circulation. Cooling raises and warming lowers pulmonary systolic and diastolic pressure. At body temp. near 0°, diastole is excessively prolonged. M. W. G.

**Differential effects of respiration on right and left ventricles.** R. H. Shuler, C. Ensor, R. E. Gunning, W. G. Moss, and V. Johnson (*Amer. J. Physiol.*, 1942, **137**, 620—627).—Anæsthetised dogs (Na barbital) were used to measure direct cardiac vol. changes by oncometer, with ventricles exposed to intrathoracic pressure changes. Both ventricles increased in diastolic size and stroke vol. during inspiration; systemic arterial blood pressure fell during inspiration. Using a cinematographic method the ventricular areas could be mapped: the right ventricle showed increased diastolic size and stroke vol. during inspiration: the left ventricle decreased in these respects during inspiration; the reverse effects occurred during expiration. The main factor responsible for fluctuations in blood pressure during respiration is the changing output of the left ventricle. Sinus arrhythmia and intrathoracic pressure may modify these changes. M. W. G.

***U*-Wave patterns in abnormal electrocardiogram.** S. D. Solarz and S. R. Elek (*J. Lab. clin. Med.*, 1943, **28**, 936—947).—In 1000 cases with diagnostic e.c.g. patterns, 94 cases of abnormal *U* waves were found. The *U*-wave patterns were as follows. In left heart strain and in intraventricular block of the common type the *U* waves were in leads II and *CF*. In anterior wall infarction, abnormal (inverted or diphasic) *U* waves were found in leads $CF_2$ and $CF_4$ and occasionally in lead I. In posterior wall infarction inverted *U* waves are found most commonly in lead $CF_4$. No abnormal *U* waves were found in right heart strain or in intraventricular block of the indeterminate *S* type. A hitherto undescribed *U*-wave abnormality, a negative bowing of the *T*–*P* segment in lead I, is discussed. Instances of distortion of the *T* and *P* waves by large upright *U* waves and the confusion caused by the presence of prominent *U* waves in records of auricular fibrillation are pointed out. C. J. C. B.

**Myocardium in avitaminosis-*E* in guinea-pig.** S. A. Freire and B. F. Magalhães (*Rev. Brasil. Biol.*, 1943, **3**, 91—98).—Avitaminosis-*E* in the guinea-pig produces hyaline necrosis in the myocardium, especially in the sub-pericardial fibres. The e.c.g. shows damage of the ventricular myocardium. Necrosis in the myocardium and skeletal muscles is prevented by administration of vitamin-*E*. I. C.

**Cardiovascular action of bile salt with regard to inhibition of choline-esterase.** M. Schachter and S. Dworkin (*Amer. J. Physiol.*, 1942, **137**, 599—605).—Dogs under nembutal received 120 mg. (max. 300 mg.) and cats 30 mg. (max. 100 mg.) of bile salt intravenously. A short-lasting fall in blood pressure occurred, which persisted after atropine, vagotomy, and destruction of the spinal cord. In some experiments the fall in blood pressure was accompanied by cardiac slowing, which was abolished by atropine and vagotomy. The bile salt action is not due to inhibition of choline-esterase. M. W. G.

**Recovery from heart failure after cardiac massage.** H. K. Vernon (*Lancet*, 1943, **244**, 6).—Case report. Cardiac massage restarted the ventricles 4 min. after stoppage. After recovery from the operation (prostatectomy) the patient showed signs of parkinsonism. C. A. K.

**Paroxysmal tachycardia in 2-month-old infant with ventricular rate of 350.** J. P. Hubbard and G. W. Starbuck (*Amer. J. dis. Child.*, 1943, **65**, 582—584).—A case report. C. J. C. B.

**Scleroderma heart disease.** S. Weiss, E. A. Stead, J. V. Warren, and O. T. Bailey (*Arch. intern. Med.*, 1943, **71**, 749—776).—9 patients with generalised scleroderma and heart failure are described with 2 postmortem examinations. The sclerodermatous process was not confined to the skin but involved other organs and the cardiac failure was caused by myocardial scarring of an unusual type. (10 photomicrographs.) C. J. C. B.

**Fibrosis of endocardium and myocardium with mural thrombosis.** J. J. Smith and J. Furth (*Arch. intern. Med.*, 1943, **71**, 602—619).—(7 photomicrographs.) C. J. C. B.

**Diseases of heart.** A. Graybiel and P. D. White (*Arch. intern. Med.*, 1943, **71**, 713—729).—A review for 1942. C. J. C. B.

**Fraudulent use of digitalis to simulate heart disease.**—See A., 1943, III, 675.

**Treatment of angina pectoris.** M. Hochrein (*Schweiz. med. Wschr.*, 1942, **72**, 1171—1174).—A review. A. S.

**Blood pressure in dogs after destruction of central nervous system.** P. E. Galvão and J. Pereira, jun. (*Rev. Brasil. Biol.*, 1942, **2**, 177—183).—The arterial blood pressure of dogs whose central nervous system has been destroyed by forcing a 20% NaCl solution at 60 cm. Hg into the cisterna magna is 20—50 mm. Hg. I. C.

**Cardio-vascular centres in dogs after destruction of the central nervous system.** P. E. Galvão and J. Pereira, jun. (*Rev. Brasil. Biol.*, 1942, **2**, 185—192).—Pressor and depressor vasomotor reflexes are abolished. I. C.

**Blood pressure fluctuations in bronchial asthma.** H. Osgood (*J. Lab. clin. Med.*, 1943, **28**, 927—935).—Wide fluctuations of the systolic blood pressure, synchronous with respiration, are a const. finding in bronchial asthma, with max. in expiration, and min. in inspiration. The amplitude of these fluctuations parallels the severity of the asthma attack. C. J. C. B.

**Effect of drugs on circulation and on spleen volume.**—See A., 1943, III, 675.

**Vasomotor tone in hindlimb muscles of dog.** J. R. Pappenheimer and J. P. Maes (*Amer. J. Physiol.*, 1942, **137**, 187—199).—The dimensions of the vascular bed in the hindlimb muscles of the dog (anæsthetised) perfused with defibrinated blood at const. pressure from a pump–lung circulation are such that even a small degree of vasoconstriction increases the apparent viscosity of the blood. Extreme vals. for blood of normal corpuscular concn. are 2—8. At normal pressures the change of apparent viscosity accounts for ½ of the total change of resistance to blood flow caused by vasoconstriction. The ratio of the slopes of the pressure–flow curves of blood in unconstricted and constricted vessels of the hindlimb muscles are equiv. to those of Ringer's solution under the same conditions. This ratio is independent of the viscosity and depends only on the change in average dimensions of the blood vessels. In the innervated prep. the ratio of the slope is proposed as a measure of vasomotor tone. The val. varied from 1·0 (no vasomotor tone) to 3·5 (large tone produced by hæmorrhage). M. W. G.

**Hæmorrhagic lesions of coronary arteries.** J. P. English and F. A. Willius (*Arch. intern. Med.*, 1943, **71**, 594—601).—Hæmorrhagic lesions were present in the walls of the coronary arteries in 54 of

135 selected hearts over 40 years of age and were related to acute occlusion of the coronary artery in 20. The lesions showed large lipin-containing cells, proliferative intimal changes, and organisation. Smaller, less active lesions were usually found adjacent to calcified plaques. The intimal changes that coexisted with the hæmorrhage represent the primary factor; the hæmorrhage was secondary. (10 photomicrographs.) C. J. C. B.

**Experimental study of phlebitis following venoclysis with glucose and amino-acid solutions.** A. Horvitz, L. A. Sachar, and R. Elman (*J. Lab. clin. Med.*, 1943, **28**, 842—848).—Phlebitis is pronounced following a 3-hr. intravenous injection of a 2·5% amino-acid + 10% glucose solution in dog; the changes are not due to the amino-acids but to the hypertonicity of the solution, as the same changes are produced by isotonic 13% glucose solutions. Acid solutions produce more phlebitis than neutral solutions as shown by comparing the damage produced by a 7·5% solution of amigen at pH 4·6 with the same solution at pH 7·4. The size of the needle in relation to the calibre of the veins plays an important part in thrombus formation. C. J. C. B.

**Anatomic and physiological effects of arteriovenous fistula.** E. Holman (*Surgery*, 1940, **8**, 362—382).—There is a decrease in heart size 24—48 hr. after the establishment of large arterio-venous anastomoses in dogs; the cardiac size reverts to normal, followed by dilatation 4—5 days after the operation. If an undue proportion of blood is diverted through the fistula death may occur with marked decrease in cardiac size; cardiac size is also decreased in shock and hæmorrhage. The dilatation accompanying the fistula also affects the proximal artery and the proximal vein as well as the heart and fistula. In the growing animal hypertrophy may parallel dilatation and no decompensation occurs. 3 litter-mate dogs were studied: one with an aorta–vena cava fistula 12 mm. in circumference, one with one 18 mm. in circumference, and one left as a normal control. There was an increase in blood vol. and circulatory capacity proportional to the size of the fistula. The increase in blood pressure and decrease in pulse rate were greater when bilateral femoral fistulæ were closed than when only 1 fistula was closed. Transient raised systolic and diastolic pressures observed after closure of a fistula are due to the increased blood vol. which develops during the existence of the fistula; the permanent increased diastolic pressure is secondary to the removal of an area of decreased peripheral resistance. Venous pressures proximal to a fistula are determined by the vol. of blood flowing through the fistula and therefore by the size of the fistula. P. C. W.

**Improvements in calculation of renal resistance to blood flow. Charts for osmotic pressure and viscosity of blood.** H. Lamport (*J. clin. Invest.*, 1943, **22**, 461—470).—The similarity between blood flow and flow of plastic solids, as described by Bingham, is used to substitute a variable in place of a static val. for the yield pressure in this application of Poiseuille's law to the kidney. An expression for post-arteriolar resistance is added to those for arteriolar resistance so that total renal resistance to blood flow can be computed, if desired. A method is elaborated by which serum-protein of any albumin/globulin ratio can be converted by a chart (or formula) into an osmotically equiv. concn. of standard serum-protein and also the osmotic pressure of the blood concn. in the glomerulus by ultrafiltration to the degree indicated by the inulin/diodrast clearance ratio. These two vals. simplify and improve the calculation of renal resistance to blood flow. A chart for blood viscosity, as affected by the serum-protein concn. and the hæmatocrit, permits the calculation of renal resistance with allowance for the viscosity of the subject's blood. C. J. C. B.

**Effect of peptone on capillary permeability.** I. H. Shleser and S. C. Freed (*Amer. J. Physiol.*, 1942, **137**, 426—430).—Intracutaneous injection of 0·2 c.c. of a 2% solution of Difco Bacto-peptone increases the permeability of the capillaries of the rabbit skin; the effect is annulled by adrenal cortical extract, but not by corticosterone, compound *E*, or anhydrohydroxyprogesterone. As corticosterone prevents weak reactions to leukotaxine the capillary damaging factor in the peptone is not leukotaxine. M. W. G.

**Physiology and biochemistry of quantitative burns. [Changes in blood and lymph flow.]** W. W. L. Glenn, J. Muus, and C. K. Drinker (*J. clin. Invest.*, 1943, **22**, 451—459).—Methods of anæsthesia, cannulation of the principal lymphatic draining the foreleg of the calf, and for providing steady passive motion of the legs and so inducing const. conditions of lymph flow, are described. The technique of producing repeatable quant. burns of the leg of a calf weighing 125—200 lb. is given. There is no increase in capillary permeability in normal regions of the body distant from the burn. The increase in blood-urea and -creatinine is consistent with the usual findings attending oliguria. The increase in -creatine and -amino-N requires further study but it is suggested that the increase in creatine is due to tissue damage in the burned area. C. J. C. B.

**Reduction of fluid loss from damaged (burned) tissues by barbiturate.** H. K. Beecher and J. D. McCarrell (*J. Pharm. Exp. Ther.*, 1943, **78**, 39—48).—Pentobarbital Na given in sedative doses following burning of the skin or small intestine and mesentery in chickens or rabbits reduced the loss of fluid and protein from the burned surfaces. Pain was prevented by destruction of the cortex in chickens or local anæsthesia in the rabbits. The burns of the small intestine were made to circumvent the smaller degree of exudation from rabbit skin than from human skin owing to differences in structure. The fluid retained as a result of barbiturate therapy appears to remain within the vascular system. The effect is not due to differences in systemic arterial pressure. When barbiturates are used, there is a much drier skin surface and less capillary congestion of the duodenal mucosa following burns than is normal. Morphine does not have a similar effect. P. C. W.

**Experimental traumatic shock.** G. Ungar (*Lancet*, 1943, **244**, 421—424).—Trauma was applied to the adductor region of the thigh in mice, rats, and guinea-pigs by dropping a metal rod a no. of times. The mortality rate was proportional to the energy transmitted to the tissues but varied with the season. When a second trauma was applied 1—3 days after previous small trauma the mortality rate was significantly reduced. This protective action of previous trauma can be transferred by injection of serum from traumatised animals into animals of the same or other species. Injection of ascorbic acid reduces post-traumatic mortality. Nupercaine protects guinea-pigs against trauma whether injected at the site of injury or into some distant part of the body. C. A. K.

**Hypertensive effect of diets high in tyrosine.** G. J. Martin (*Arch. Biochem.*, 1943, **1**, 397—401).—When *l*-tyrosine formed 5 or 10% of the diet of rats, a syndrome was produced in which hypertension was the characteristic symptom. E. R. S.

**Prophylactic treatment of experimental renal hypertension with renin.** G. E. Wakerlin, C. A. Johnson, E. L. Smith, W. G. Moss, and J. R. Weir (*Amer. J. Physiol.*, 1942, **137**, 515—520).—Renin solutions were only partly purified and contained substances other than renin; they were equiv. to 1 g. of fresh kidney cortex per c.c. of solution and were administered in a dose of 1 c.c. per kg. body wt. Sera were examined for antirenin. Out of 4 dogs, 2 were completely protected, one partly, and the 4th not, against experimental renal hypertension (Goldblatt technique). Out of 4 the inactivated hog renin protected one dog. Out of 4 dogs, dog renin completely protected one, and partly one dog. Rabbit renin completely protected one. Inactive human renin was ineffective as was liver extract. 16 untreated controls all developed renal hypertension. The mechanism of this prophylaxis may be due to renin or to some substance in partially purified extracts, but antirenin is not involved. No toxic effects followed the injections, which were all given intramuscularly. M. W. G.

**Inability of purified or crude kidney extract (renin) to reduce blood pressures of hypertensive dogs.** M. Friedman, H. E. Kruger, and A. Kaplan (*Amer. J. Physiol.*, 1942, **137**, 570—572).—Hypertensive dogs were injected daily with kidney extract for 3—4 months. Some received purified renin (fraction *D*) intramuscularly, and others, subcutaneously and intramuscularly, a relative crude renin prepared by the Wakerlin and Johnson method (daily dose equiv. to 1 g. of fresh kidney cortex per kg. body wt.). No reduction in blood pressure occurred. In most of the dogs no increase in the ability of the plasma to neutralise the pressor effect of renin was observed. M. W. G.

**Renal hypertension and adrenalectomy in rats.** R. Dell'Oro (*Rev. Soc. argent. Biol.*, 1942, **18**, 13—23).—Permanent hypertension can be produced in rats by wrapping the kidney in gauze and collodion. Double adrenalectomy in these rats produces an abrupt and marked fall of blood pressure to normal vals. The injection of deoxicorticosterone to adrenalectomised rats increased the blood pressure, but the previous hypertensive level was not attained. In rats surviving after adrenalectomy, the pressure increased progressively and hypertrophy of the accessory adrenals was observed. I. C.

**Renin determination in blood of dogs during renal ischæmia.** R. Dell'Oro and E. Braun-Menendez (*Rev. Soc. argent. Biol.*, 1942, **18**, 65—70).—In dogs, after extirpating the right kidney, the left kidney was transplanted under the skin of the lumbar region. The renal artery was then constricted and the renin content determined in blood obtained by puncture of the renal vein through the skin and in blood of the femoral artery. No renin was found in plasma from the renal vein or femoral artery before constriction of the renal artery. After constriction and during the rise of blood pressure, 0·05—0·8 unit of renin was found in the femoral blood and 0·22—1 unit of renin in the renal venous blood. I. C.

**Experiments in study of immersion foot.** W. Blackwood and H. Russell (*Edinb. Med. J.*, 1943, **50**, 385—398).—Histological studies were made on the tails of rats kept at 3—4° in ½ in. of running artificial sea-water. Changes in muscle bundles appeared earlier than those in nerves; skin, other tissues, and blood vessels were relatively resistant to injury. Conspicuous damage resembling Zenker's necrosis was present at 48 hr.; exposure for longer than 96 hr. was fatal to majority of animals. Muscle lesions were polychromic staining of muscle fibres with loss of striations and characteristic "clods" of myoplasm; later fragmentation with sarcolemmal proliferation, capillary congestion, and polymorph and

monocyte infiltration. The destruction of muscle fibres was patchy and the reaction well advanced 3 days after exposure and max. by 14th day. Nerves showed Wallerian degeneration in 1—2 days; in 14 days myelin débris had been removed and there was greatly increased cellularity. The myoneural changes occur together and the tissues had not returned to normal within 2 months after exposure. Warming animals rapidly in warm water at 29° or heating them at 37° in an incubator for 3 hr. following exposure acccelerated the initial reaction but did not affect the reaction 1 month later. The periods and conditions of exposure were comparable with those under which the less severe forms of immersion foot develop in man. (18 photomicrographs.) H. S.

**Lymphatics of parietal tunica vaginalis propria of man.**—See A., 1943, III, 617.

## VII.—RESPIRATION AND BLOOD GASES.

**Lungs of Cetacea, with special reference to harbou : porpoise (*Phocæna phocæna*).**—See A., 1943, III, 617.

**Comparison of vagal respiratory regulation in cats and rabbits.** K. Bucher (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 17—20).—The trachea was obstructed in cats and rabbits under urethane or dial anæsthesia in various phases of the respiratory cycle. Cats reacted mainly with inspiratory, rabbits with expiratory, efforts, irrespective of the type of anæsthesia. A. S.

**Pneumometer for determination of maximal expiration in shortest time.** W. Hadorn (*Schweiz. med. Wschr.*, 1942, **72**, 946—950).—The apparatus is described. It is recommended in the diagnosis of pulmonary emphysema and bronchial asthma. A. S.

**Effect of section of one phrenic on contralateral phrenic nerve and respiration.** C. Petitpierre (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 32—33).—Section of one phrenic increases the amplitude of the action potentials recorded from the central cut end of the contralateral phrenic nerve in cats and rabbits and slows the respiratory rate, mainly by prolonging the expiratory phase; the increase in potentials was observed after double vagotomy but there is no further change in respiratory rate on phrenic section. Similar results were obtained when, in addition, the posterior nerve roots between $C_3$ and $C_7$ were cut on both sides. A. S.

**Respiration with Swiss Army gas mask.** M. Gukelberger (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 20—22). A. S.

**Influence of high temperature on central regulation of respiration.** A. Hámori (*Magyar Orv. Arch.*, 1941, **42**, 52—70).—The respiratory centre in the pons is the most, the pontobulbar inhibitory centre less, and the medullary centre least readily paralysed by heat. Respiratory movements are regulated by (i) the medullary centre which is practically unaffected by high blood temp., (ii) the centre in the pons which is more sensitive to heat, but cannot induce panting in the dog, (iii) the midbrain centre which is most sensitive, produces panting, and also controls the reflex respiratory movements elicited by heat or pain. Periodic breathing may occur when the higher centres are eliminated by heat paralysis, when competition between the pontobulbar inhibitory centre and the medullary centre occurs. M. A. B.

**Effect of lowered oxygen tension of inspired air on respiratory response of normal subjects to $CO_2$.** N. W. Shock and M. H. Solen (*Amer. J. Physiol.*, 1942, **137**, 256—258).—Men show individual differences in their respiratory response to 2% $CO_2$ with 21, 17, or 12% $O_2$. Generally the respiratory effects of increased $CO_2$ and lowered $O_2$ are additive, but not invariably. $O_2$ tension must be reduced below 91 mm. in inspired air before $CO_2$ begins to lose its efficacy as a respiratory stimulant. M. W. G.

**Comparison of acute and chronic toxicity of carbon dioxide with special reference to narcotic action.** J. H. Barbour and M. H. Seevers (*J. Pharm. Exp. Ther.*, 1943, **78**, 11—21).—In rats suddenly exposed to atm. with a high $CO_2$ content, 15% concn. is the max. tolerated. If the concn. is gradually increased during several days, the max. tolerated concn. may be 23%. Greater depression is produced by sudden than by prolonged exposure to a given concn., indicating acclimatisation. Following exposure to 11% $CO_2$, $O_2$ consumption is depressed by 15—25% and only returns to normal after 24 hr. exposure. During exposure to 11% $CO_2$ for 30 days, the plasma-pH falls to 7·09 within 0·5 hr., and remains below 7·15 throughout; the $CO_2$ content rises to 130—148 vols.-%. There is marked reticulocytosis but no increase in red or white blood cells. The sleeping time of rats anæsthetised with pentobarbital is increased in atm. of 10 or 20% $CO_2$ or in rats previously exposed to 10% $CO_2$ for 7 days. $CO_2$ delays the detoxication of pentobarbital by decreasing the blood flow through the liver as a result of splanchnic vasoconstriction. P. C. W.

**Carrot diet and susceptibility to acute anoxia.** D. Nelson, S. Goetzl, S. Robins, and A. C. Ivy (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 1—2).—Results of Campbell (A., 1939, III, 366) are confirmed. V. J. W.

**Effect of change of altitude on basal metabolism in man.** R. C. Lewis, A. Iliff, A. M. Duval, and G. M. Kinsman (*J. Lab. clin. Med.*, 1943, **28**, 851—858).—Change in altitude at least up to 8720 ft. did not affect the basal metabolism. C. J. C. B.

**Intravenous administration of oxygen.** F. S. Grodins, A. C. Ivy, and H. F. Adler (*J. Lab. clin. Med.*, 1943, **28**, 1009—1014).—Intravenous $O_2$ in dogs in small amounts (0·2—0·35 c.c. per kg.) causes marked respiratory changes, may lower blood pressure, and usually reduces the arterial $O_2$ content. C. J. C. B.

**Peripheral vascular response to acute anoxia.** D. I. Abramson, H. Landt, and J. E. Benjamin (*Arch. intern. Med.*, 1943, **71**, 583—593).—The inhalation of a 10% $O_2$–90% $N_2$ mixture for 10—27 min. slightly increased the blood flow in forearm and leg in most of 24 normal subjects, and decreased the flow in the hand. There was an increase in pulse rate and systolic blood pressure, and variable changes in rate of respiration. Responses to exercise are less satisfactory in anoxia. C. J. C. B.

**Oxygen therapy in shock.** G. Melton (*Lancet*, 1943, **244**, 481—484).—$O_2$ therapy was successfully used in 25 of 31 cases of hæmorrhagic or surgical shock. In severe cases the blood pressure was raised and the pulse and colour were improved; withdrawal of $O_2$ made the condition worse again. C. A. K.

**Use of oxygen in post-operative therapy of hyperthyroidism.** J. G. Schnedore, R. D. McClure, and A. B. McGraw (*Surg. Obstet. Gynec.*, 1941, **72**, 26—30).—6 of 12 hyperthyroid patients showed a depression of 2—7% in arterial $O_2$ saturation. There was a further fall in 10 of 11 patients 1 hr. after operation. $O_2$ therapy has a sedative effect, decreases the respiratory rate, and slows the heart and lowers the body temp. P. C. W.

**Treatment of carbon monoxide poisoning.** A. Thurnherr (*Schweiz. med. Wschr.*, 1942, **72**, 938—944).—Ultra-violet radiation or addition of thionin *in vitro* inhibits the formation, and accelerates the dissociation, of CO-hæmoglobin. Radiation and injection of thionin accelerate the decrease of the blood-CO concn. *in vivo* (rabbits); thionin was more effective than ultra-violet radiation. Radiation and re-infusion of blood in CO-poisoned animals was ineffective. A. S.

**Bronchoscopy in newborn infant.** F. D. Woodward and W. W. Waddell, jun. (*J. Pediat.*, 1943, **23**, 79—86).—A lecture. C. J. C. B.

**Pneumothorax in newborn infant.** G. W. Salmon and G. B. Forbes (*J. Pediat.*, 1943, **23**, 50—58).—Report of 3 cases. C. J. C. B.

**Spontaneous interstitial emphysema of lung, with mediastinal, retroperitoneal, and subcutaneous emphysema.** J. D. Adcock (*Arch. intern. Med.*, 1943, **71**, 650—657).—The literature is reviewed and a case reported. C. J. C. B.

## VIII.—MUSCLE.

**Electromyography.** G. Weddell, B. Feinstein, and R. E. Pattle (*Lancet*, 1943, **244**, 236—239).—Voluntary muscle action potentials were studied in animals and man after experimental denervation. Fibrillation can be distinguished from motor unit action potentials. Fibrillation indicates nerve interruption and is of val. in diagnosis and prognosis of nerve injuries. When there is no electrical activity more than a few weeks after injury severe changes such as fibrosis may be presumed to have occurred in the muscle. A case report is given. C. A. K.

**Muscle and nerve regeneration in rat.** H. M. Hines, J. D. Thompson, and B. Lazere (*Amer. J. Physiol.*, 1942, **137**, 527—532).—Regeneration in gastrocnemius, muscles and tibial nerves of albino rats at various periods after denervation was studied. After nerve crushing, regeneration was const. in animals of the same sex, wt., and age, and was slower in older than in younger rats. Functional reinnervation occurred at 12—15 days after denervation; fibrillary activity disappeared but increased sensitivity to acetylcholine was seen for as long as 35 days. Muscle strength increased rapidly after initial reinnervation until the 35th day; subsequent recovery was slower and largely accounted for by increase in muscle mass. At 84 days the muscles undergoing regeneration possessed 80—90% of the creatine content, wt., and strength of their contralateral controls. M. W. G.

**Histology of molluscan muscle.** M. Olson (*Biol. Bull.*, 1942, **82**, 190—194).—The fibres of the radula protractor of *Busycon canaliculatum* are plain, average 1 mm. in length, and consist of a cortex containing the coarse myofibrils and a medulla containing the nucleus. G. P. W.

**Properties of crustacean muscle.** F. P. Knowlton (*Biol. Bull.*, 1942, **82**, 207—214).—Experiments are described on the adductor of the claw in 3 species. The quick and slow types of contraction correspond to twitch and contracture in amphibian muscle. G. P. W.

**Innervation of crustacean muscles.** W. Holmes (*Nature*, 1943, **151**, 531—532).—Prawn muscle fixed in 80% alcohol, formalin, and glacial acetic acid and stained by Bodian's protargol method shows

a system of nerve fibrils, all traceable to main nerve trunks; no muscle fibre receives more than one or two fibril endings. When fixed in picric acid and similarly stained the fibrils appear more numerous and several end on each muscle fibre, but the extra fibrils seen are of connective tissue origin (cf. van Harraveld, A., 1939, III, 827). E. R. S.

**Acetylcholine and electrical stimulation of skeletal muscle in frogs.** N. Scheinfinkel (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 36—37).—Electrical stimulation produces contraction of the frog's sartorius muscle, loaded with 1—10 g., when acetylcholine has become ineffective. A. S.

**Effect of chloroform and ether on sensitivity of muscle to acetylcholine.** C. Torda (*J. Pharm. Exp. Ther.*, 1943, **77**, 350—356).—Up to 0·03 c.c.-% of $CHCl_3$ or 1·5 c.c.-% of ether increases the response of the frog's rectus to acetylcholine. Up to 0·16 c.c.-% of $CHCl_3$ or 3·7 c.c.-% of ether decreases the response. Higher concns. cause contracture. $CHCl_3$ and eserine potentiate each other, but $CHCl_3$ has no effect on atropine inhibition and atropine does not affect the $CHCl_3$ contracture. V. J. W.

**Autolysis of fish muscle.** B. Bailey, P. Koran, and H. C. Bradley (*Biol. Bull.*, 1942, **83**, 129—136).—In 10-day experiments at 38° the muscles of game fish (*Scomber scombrus, Germo alalunga, Sardia sardia, Xiphias gladius*) autolyse more rapidly and completely than do those of sluggish fish (*Cyprinus carpio, Lepidosteus osseus, Gadus callarias, Squalus acanthias*). This is apparently due to a difference in the properties of the proteins, and may indicate a means of maintaining activity by mobilisation of tissue-proteins when food is scarce. G. P. W.

**Loss of potassium from muscle after adrenalectomy and after iodoacetic acid poisoning in frogs.** J. C. Somogyi (*Verh. Ver. Schweiz. Physiol.*, 1941, **18**, 47—48).—The sartorius muscle of frogs, on direct electrical stimulation until fatigue, loses 0·192 mg. of K per g. Muscles of adrenalectomised frogs do not lose K on electrical stimulation. Muscles poisoned with iodoacetic acid (1 : 30,000) lose the same amount of K as normal muscle for the same degree of muscular work. A. S.

**Effects of pH and various concentrations of sodium, potassium, and calcium chloride on muscular activity of isolated crop of *Periplaneta americana* (Orthoptera).** J. T. Griffiths, jun., and O. E. Tauber (*J. Gen. Physiol.*, 1943, **26**, 541—558).—A satisfactory physiological salt solution for the isolated foregut of the American roach contains NaCl (14·0), $CaCl_2$ (0·4), KCl (0·2), and $NaHCO_3$ (0·2 g. per l.) and has a pH val. of 7·8—8·2. The effect of varying these amounts and the pH is determined. In solutions that contain Ca, but no K, approx. 50% of the crops exhibit an initial increase of tone and are arrested in rigor. In presence of K, but no Ca, all crops show an initial loss of tone and arrest in relaxation. With the above solution, durations of crop activity extending over 25 hr. are quite common, and several crops maintain contractions for more than 30 hr. There is a significant difference between the activities of crops from males and females. J. N. A.

**Effect of adrenal gland on phosphorylation of glycogen in muscle. III. Kinetics of phosphorolysis with muscle pulp in normal and adrenalectomised animals.** C. Montigel (*Helv. Chim. Acta*, 1943, **26**, 883—905; cf. A., 1942, III, 582).—In normal animals (rat, cat, dog) the phosphorolysis of glycogen by muscle is a reaction of the first order but in adrenalectomised animals it is of zero order. The course of the reaction in adrenalectomised animals *in vitro* and *in vivo* can be rendered largely normal by deoxycorticosterone and its acetate in certain doses. It is shown by increase of $[PO_4'''']$ in normal muscle that superconcn. of substrate in relationship to enzyme has the same effect on the type of reaction as adrenalectomy. This effect is counteracted by deoxycorticosterone. Deoxycorticosterone is thus an essential component of the enzyme system of glycogen phosphorolysis in muscle. H. W.

**Removal of malignant thymoma in case of myasthenia gravis.** F. Turnbull (*Arch. Neurol. Psychiat. Chicago*, 1942, **48**, 938—945).—Removal of a malignant thymoma in a woman 29 years of age, with myasthenia gravis, failed to cause improvement. W. M. H.

**Contractures of skeletal muscle in epidemic infantile poliomyelitis.** R. Scherb (*Schweiz. med. Wschr.*, 1942, **72**, 1009—1012). A. S.

## IX.—NERVOUS SYSTEM.

**Action of malonic acid on respiration of nerve.**—See A., 1943, III, 668.

**Convulsive attack by freezing the spinal cord in frogs of different countries.** M. Ozorio de Almeida, H. Moussatché, and M. Vianna Dias (*Rev. Brasil. Biol.*, 1943, **3**, 49—66).—The convulsive attack brought about by freezing the spinal cord of *Leptodactilus ocellatus* develops differently in animals of different regions. The degree of freezing required is smaller for frogs coming from hotter regions. Differences in the composition of the water may be responsible for these differences. I. C.

**Action of pentamethylentetrazole on choline-esterase activity of the spinal cord of *Bufo gay*.** J. Sepulveda and H. Croxatto (*Anal. Acad. Biol. Univ. Chile*, 1940, **3**, 31—40).—In *B. gay* the choline-esterase activity of the spinal cord between II and V vertebræ is higher than in segments above or below. Pentamethylentetrazole (0·010—0·050 g.) causes intense generalised convulsions and no change in cord choline-esterase activity. Eserine (0·0005—0·001 g.) decreases the choline-esterase activity of the spinal cord by 40%. I. C.

**Effects [on nervous function] of removal of skin in American frogs.** M. Ozorio de Almeida (*Rev. Brasil. Biol.*, 1942, **2**, 271—274).—Removal of the skin in American frogs brings about ataxia, incoordination, loss of the sense of position, and general depression. I. C.

**Induced resistance of central nervous system to experimental infection with equine encephalomyelitis virus.**—See A., 1943, III, 695.

**Psychology of pain.** E. Guttmann and W. Mayer-Gross (*Lancet*, 1943, **244**, 225—227). C. A. K.

**Vibration sensibility in face following retrogasserian neurectomy.** M. Brown and G. K. Yacorzynski (*Arch. Neurol. Psychiat., Chicago*, 1942, **47**, 813—820).—Vibration sensibility was studied in the face of 9 subjects after unilateral retrogasserian neurectomy. With the apparatus used the character of the oscillations at the skin surface was determined by a recording unit connected to the vibrating point. Amplitude thresholds at frequencies of 100 per sec. were raised and the upper limit of frequency of vibration perceptibly lowered in the lips and tongues of the subjects. On the forehead and cheek the vibrations delivered could not be perceived in most cases. Sensation of vibration is mediated by skin and deep pressure sense organs. Trigeminal neurectomy affects the sensibility dependent on the tactile end organs. W. M. H.

**Bulbar projection of trigemina nerve.** W. A. McKinley and H. W. Magoun (*Amer. J. Physiol.*, 1942, **137**, 217—224).—The character and distribution of action potentials recorded from points within the medulla of the cat (nembutal or decerebrated) on electrical stimulation of peripheral branches of the trigeminal are described. In their course in the sensory root and spinal fifth tract, fast conducting fibres of the 3 trigeminal divisions are laminated in a dorso-ventral order which is the inverse of their distribution in the face. Fibres of the mandibular division are detected as far caudally as the obex; those of the maxillary and ophthalmic division, as far caudally as the first cervical segment. The neurones of the spinal fifth nucleus fixed by primary fibres of the 3 trigeminal divisions are laminated in a dorso-ventral order similar to that of the spinal fifth tract, but with greater overlap. Secondary trigeminal pathways pass diffusely across the reticular formation to the ventro-medial part of the opposite side of the medulla and ascend in relation to the medial lemniscus. M. W. G.

**Effect of rotation on postural steadiness in normal and in schizophrenic subjects.** H. Freeman and E. H. Rodnick (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 53).—The amount of sway induced by rotation was studied in 30 normals and 30 male schizophrenics. The patients were less reactive to the stimulus than the normals. W. M. H.

**Thalamic stimulation and blood composition.** H. Wespi (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 41—42).—Stimulation of the nucleus centralis of the thalamus in cats produced marked leucocytosis with shift to the left in the differential count; negligible alterations of blood pressure and respiration were observed. A. S.

**Nigrohypothalamic fibres in cat.** D. L. Kimmel (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 51—52).—Lesions of the substantia nigra cause degeneration in fibres ascending to the hypothalamus by the homolateral mammillary peduncle. They end in homolateral nuclei of the mammillary bodies, the infundibular region, and the lateral hypothalamic area. None cross over. V. J. W.

**Intraneural conditioning. Cerebellar conditioned reflexes.** W. J. Brogden and W. H. Gantt (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 437—455).—Intraneural conditioned responses were established in dogs by stimulation of the neocerebellum with induced currents. The movements elicited were contractions of the ipsilateral ear, neck, or shoulder muscles and were made the basis of conditioned responses by preceding the electric shock with a bell or light. Both excitatory and inhibitory conditioned responses were produced with the same ease as by a painful shock applied to the skin of the leg. The cerebellum may integrate by itself or by its connexions with the thalamus and cerebral cortex. W. M. H.

**Cardiovascular changes in stimulation of motor cortex.** S. Hsu, K. Hwang, and H. Chu (*Amer. J. Physiol.*, 1942, **137**, 468—472).—A fall in blood pressure always occurs on electrical stimulation of the motor focal points of the sigmoid gyrus in chloralosed dogs. The extent and variation of the depressor effect is determined by the strength of current and differences in focal points. The drop in blood pressure from stimulation of any focal point is not influenced or abolished by vagal section. An increase of 20% occurs in heart rate. Amplitude of respiratory movements is decreased and rate

increased; in some cases there was apnœa throughout stimulation. An increase in renal vol. accompanied the fall in blood pressure. M. W. G.

**Fatigue of central nervous system.** E. Simonson (*Clin. Proc.*, 1943, **2**, 112—116).—Review of methods and results of measuring sensory and motor fatigue. P. C. W.

**Dyspraxia and apraxia following partial and complete section of corpus callosum.** A. J. Akelaitis, W. A. Risteen, R. Y. Herren, and W. P. Van Wagenen (*Arch Neurol. Psychiat., Chicago*, 1942, **47**, 971—1007).—In 18 cases of epilepsy the corpus callosum was sectioned in various degrees. In 10 cases motor function was normal after as before the operation; in 3 cases a kinetic dyspraxia ensued but was probably based on motor paresis with, in two cases, additional sensory changes; 4 out of 5 cases with diffuse or focal changes in the central nervous system had dyspraxia but in only one was exaggeration of kinetic dyspraxia attributed to partial section of the corpus callosum. Dyspraxia in either hand is found after the section of the corpus callosum only when damage to the appropriate hemisphere coexists. W. M. H.

**Hemiplegia and cerebrovascular disease.** L. J. Karnosh (*Cleveland Clin. Quart.*, 1942, **9**, 165—172).—185 records and post mortem examination of patients who had died of hemiplegia are analysed. There were 110 cases of cerebral hæmorrhage, with 87 (80%) cases with lesions in the middle cerebral artery branches. In 66 brains with thrombosis 77% showed softening in the same area. 5 patients with hemiplegia had primary hæmorrhage in the pons; there were 4 further cases with softening in the medulla. In 102 cases the apoplexy involved the external capsule, the upper and outer part of the putamen, and that part of the corona radiata which carries fibres from the facial and arm area. The most common symptom was a spastic paralysis of face and arm, rarely of the lower extremity; motor aphasia was common with right-sided paralysis; there was rarely hemianopia. In 22 cases the area of softening involved the internal capsule, the globus pallidus, and even the thalamus; in 5 cases the hæmorrhage extended into the lateral and third ventricles. The hemiplegia in these 27 cases was complete; there was flaccidity in 15 cases; tendon reflexes were generally absent or sluggish; the limbs usually remained flaccid; 10 patients had sensory disturbances, usually hypæsthesia to touch and pin-prick on the paralysed side, the facial area frequently not showing sensory defects. 2 patients had flaccid hemiplegia, hemianæsthesia, and hemianopia (there was a ruptured aneurysm in 1 case near the junction of the anterior choroidal with the internal carotid artery). In 5 cases of pontine hæmorrhage there were mainly flaccid weakness, respiratory difficulties, and, after the shock stage, external squint, difficulty in swallowing, sometimes compulsory laughing or crying. In 4 patients with thrombosis of the posterior inferior cerebellar artery there was unilateral hypotonia and ataxia with intact voluntary control, rotatory nystagmus, and loss of pain sense over the trigeminal distribution of the affected side (absence of corneal reflex). A. S.

**Effect of 180° rotation of the retinal field on visuomotor co-ordination.**—See A., 1943, III, 642.

**Effects of bilateral optic enucleation on voluntary muscular activity of albino rat.**—See A., 1943, III, 640.

**Fatigue and effort syndrome.** E. Guttmann and H. Rimoldi (*J. ment. Sci.*, 1941, **87**, 349—358).—With Mosso's ergograph, hysterical patients tend to produce slow, short, and abruptly-ending curves with marked differences in tempo between left and right hand; in anxiety, fast, long and irregular curves are obtained; whilst with true exhaustion, long, fast, regular curves are produced, frequently with a final effort. G. D. G.

**Prefrontal leucotomy.** G. W. T. H. Fleming and W. McKissock (*Lancet*, 1943, **244**, 361—362).—Good results in 12 cases of melancholia, 1 schizophrenic, 1 obsessional, and 1 manic depressive are reported. C. A. K.

**Results of prefrontal leucotomy.** E. L. Hutton (*Lancet*, 1943, **244**, 362—366).—Good results in 50 cases of schizophrenia, depressive, obsessional, and anxiety states are reported. Most of the patients gained wt. C. A. K.

**Parapyramidal fasciculotomy in brain stem.** J. H. Siris (*Arch. Neurol. Psychiat., Chicago*, 1942, **47**, 808—812).—In order to interrupt the parapyramidal pathways in a case of post-encephalitic Parkinsonism an incision was made in the reticular formation in the medulla. Relief of tremor was only transitory. Pain and temp. sensation was lost in the contralateral lower limb. Trigeminal sensation was not altered. W. M. H.

**Blood-sugar and consciousness.** W. Mayer-Gross and F. Berliner (*J. ment. Sci.*, 1941, **87**, 427—433).—During the pre-comatose stage, fluctuations of consciousness are associated with changes in the blood-sugar level. In coma, blood-sugar fluctuations have no influence on the degree of consciousness. There may be two levels of cerebral integration: cortical, disturbed in the pre-comatose stage, and diencephalic, impaired in coma. G. D. G.



**Cerebrospinal fluid-sugar and coma.** W. Mayer-Gross and F. Berliner (*J. ment. Sci.*, 1942, **88**, 82—88).—The clinical signs of hypoglycæmia are not more dependent on the c.s.f.-sugar than on the blood-sugar. The former runs roughly parallel to the latter during insulin hypoglycæmia. G. D. G.

**Pathologic anatomy of human nervous system in avitaminosis.** H. Ying-K'uei (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 271—319).—An endemic of so-called secondary *B* complex avitaminosis in 13 Chinese soldiers suffering from dysentery and tuberculosis was studied. The chief findings were neuropathy of the peripheral nerves, particularly the sensory fibres in beriberi, changes in the Betz cells in pellagra, and degeneration of the ascending fibres of the spinal cord, sometimes secondary to the neuropathy of the posterior roots and sometimes an independent process. W. M. H.

**Pathology of senile brains. Silver-reducing structures in hippocampus.** L. S. King (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 241—256).—Changes in the hippocampal formation in senile brains are described. Substances occur within and outside the nerve cells which reduce ammoniacal Ag solutions without further chemical treatment. In the Alzheimer fibrillary change the process in which reducing substance occurs is not confined to the neurofibrils. Pyriform cells are found in the stage of transition to the fine-fibred type of senile plaque, but transitional stages in the formation of other types of plaque are not observed. W. M. H.

**Choline-esterase and mono-amine oxidase in human brain.** H. Birkhäuser (*Verh. Ver. Schweiz. Physiol.*, 1940, **17**, 8—9).—The acetylcholine quotient (c.c. of $O_2$ taken up by 100 mg. of tissue in 120 min.) in adult human brain was for thalamus 2·9 (±0·1; 23 cases), caudate nucleus 29·1 (±1·2; 22 cases), putamen 38·1 (±1·1; 18 cases), globus pallidum 10·0 (±0·5; 15 cases), cortex 1·6 (±0·08; 23 cases). The corresponding vals. for mono-amine oxidase were in persons above 60 years 104±8·7 (13), 93±5·6 (13), 78±9·2 (9), 63±6·2 (7), 50±3·1 (13), in subjects under 60 years, 90±5·1 (6), 88±4·3 (7), 73±3·3 (7), 81±5·1 (6), 47±1·9 (7), in small infants for thalamus 38±2·3 (4), caudate nucleus 24±0·4 (4), and cortex 19±2·9 (3). The choline-esterase vals. in 5 small infants were 4·8 (thalamus) and 16·8 (caudate nucleus). A. S.

**Acetylcholine-esterase content of brain tumours.**—See A., 1943, III, 659.

**Metabolic effects of potassium, temperature, methylene-blue, and *p*-phenylenediamine on infant and adult brain.** H. E. Himwich, A. O. Bernstein, J. F. Fazekas, H. C. Herrlich, and E. Rich (*Amer. J. Physiol.*, 1942, **137**, 327—330).—0·1M-K stimulates markedly aërobic metabolism of adult rat cerebral cortex but has only a small effect on infant brain. The acceleration of $O_2$ utilisation in the presence of 0·005N-*p*-phenylenediamine of the infant brain is 18% for 1st 10 min., rising to 73% from 50 to 60th min.; of the adult brain 124% in 1st 10 min. and diminishes rapidly. The decolorisation time of methylene-blue is 22 min. for adults and 35 min. for infants. There is also a smaller response to temp. increase in the infant brain. M. W. G.

**Distribution of alkaline phosphatase in normal and in neoplastic tissues of nervous system.** H. Landow, E. A. Kabat, and W. Newman (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 518—530).—With a histochemical technique based on the method used by Gomori and Takamatsu marked phosphatase activity was shown in the vascular endothelium of the central nervous system. In the nervous parenchyma it varies in different species and in different parts. Phosphatase activity of tumours of the central nervous system correlates with the activity of the corresponding normal tissue and the tendency to calcification, but is not so frequently found in the tumour blood vessels as in the normal. Phosphatase is found in areas of astrocytosis such as the chronic plaques in multiple sclerosis. Phosphatase in the vascular endothelium may be concerned with the passage of glucose through the capillary wall. W. M. H.

***In-vitro* oxidation of pyruvic and α-ketobutyric acids by ground preparations of pigeon brain. Effect of inorganic phosphate and adenine nucleotide.**—See A., 1943, III, 671.

**Histochemical vitamin-*C* determinations in choroid plexus epithelium and ependyma.**—See A., 1943, III, 667.

**Effect of diathermy on brain metabolism: changes produced on sugar, lactic acid, and pH of arterial and venous blood of the brain in paretic patients.** J. M. Looney and E. J. Borkovic (*J. Lab. clin. Med.*, 1943, **28**, 983—987).—There was no change in sugar utilisation and an increase in pH. The lactic acid arterio-venous difference is reversed so that during the period of continued temp. elevation, lactic acid is produced. There is no increase in brain metabolism during diathermy. C. J. C. B.

**Acid-soluble phosphorus compounds of cerebral tissue.** W. E. Stone (*J. Biol. Chem.*, 1943, **149**, 29—41).—Methods are described for the fractionation of cerebral P compounds and for the determination of ribose in nucleotides. The unidentified org. acid-sol. P (cf. A., 1940, III, 800) probably includes a hexose 6-monophosphate, aminoethyl phosphate, and an alcohol-sol. substance. Phospho-

glycerate, phosphopyruvate, and triose phosphate were not detected in cerebral tissue. The first post-mortem changes are hydrolysis of phosphocreatine and (partly) adenosine triphosphate, liberating adenosine diphosphate, adenylic acid, nucleoside or free pentose, and inorg. $PO_4'''$. R. L. E.

**Electroencephalography in cases of mental disorder.** W. G. Walter (*J. ment. Sci.*, 1942, **88**, 110—121).—The electroencephalograms of 72 mental patients were examined, both before and after hyperventilation. 25% gave definitely abnormal records; of these 12 were from epileptics, 2 from catatonics, 3 from cases of cerebral atrophy, and 1 from a case of cerebral hæmorrhage. No abnormalities were found in non-catatonic schizophrenia, manic-depressive psychosis, involutional depression, or psychoneurosis. The criterion of abnormality was the occurrence of waves with frequency less than 7 cycles per sec. and amplitude greater than 20 $\mu$v. The chief use of the electroencephalogram in mental disorder is in the diagnosis of epilepsy and the differentiation between organic and psychic disorders. Changes in resistance of the tissues may account for the peculiarities in certain schizophrenic records. G. D. G.

**Electroencephalograms of thiamin-deficient pigeons.** R. L. Swank and H. H. Jasper (*Arch. Neurol. Psychiat., Chicago*, 1942, **47**, 821—827).—Brain potentials were recorded during various stages of vitamin-*B* deficiency in pigeons. There is a progressive increase in amplitude of the waves; in opisthotonus lower frequencies become dominant and epileptiform waves may appear. In enopisthotonus the rapid frequencies largely disappear. Change towards the normal is observed within a few hr. of giving thiamin but is complete only after 10—17 days of normal diet if the deficiency has been extreme. The sequence of increase or synchrony of brain potentials and later depression is similar to the changes in $O_2$ and glucose deficiency. W. M. H.

**Value of electroencephalogram in cranial injuries.** M. A. Glaser and H. Sjaardema (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 689—696).—Electroencephalographic (e.e.g.) records were taken in 50 cases of head injury 4 days—28 years after the injury; the e.e.g. was normal in 19. E.e.g. records bore no relation to subjective symptoms. Abnormal e.e.g. were found in 18 of 27 patients with no neurological signs. High delta voltages often coincide with severe injury and medium or low delta voltages with less severe cases with clinical improvement. P. C. W.

**Effect of " yerba mate " on encephalogram.** J. B. Odoriz (*Rev. Soc. Argent. Biol.*, 1942, **18**, 190—202).—The ingestion of an infusion of " yerba mate " or that of caffeine produces transient modifications in the alpha rhythm, of the type seen when there is a diminution of the psychical and physical state of rest and increased attention. In a few cases of hypersensitivity to " mate," caffeine waves appeared which were also reproduced in dogs by large doses of the drug. I. C.

**Electroencephalogram accompanying hyperactive carotid sinus reflex and orthostatic syncope.** F. M. Forster, E. Roseman, and F. A. Gibbs (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 957—967).—Electroencephalograms were taken in 17 subjects with hyperactive carotid sinus reflexes, 3 subjects after $NaNO_2$, and in 35 controls. Slow waves are found in orthostatic syncope, but are not prominent in carotid sinus syncope of either the circulatory or the central type and usually no gross disorder of the cortical rhythm is found. In all types of carotid sinus syncope there is a sudden decrease in amplitude not easily distinguished from the ordinary attention response. During the period of unconsciousness slowing of cortical activity is shown with the Grass analyser in most cases of sinus circulatory syncope and increase in frequency in the cases of syncope of the central type. It is suggested that cortical activity is accelerated by stimulation of the carotid sinus. W. M. H.

**Effect on electrical activity of cortex of depressant and stimulant drugs.** F. A. Gibbs and G. L. Maltby (*J. Pharm. Exp. Ther.*, 1943, **78**, 1—10).—Electroencephalograms (e.e.g.) were recorded in six normal men following the intravenous administration of Na phenobarbital (2—3 doses of 320 mg. at 20 min. intervals), pentothal (63—90 mg.), morphine sulphate (2—3 doses of 16 mg., given also subcutaneously and intramuscularly), caffeine Na benzoate (1—2 doses of 49 mg.), benzedrine (2 doses of 10 mg.), and adrenaline (1 dose of 0·5 mg.). The e.e.g. was transformed into a spectrum by the Grass method of frequency analysis. The record was taken from the right occipital area with indifferent electrodes on the lobes of both ears. The first three compounds caused a shift in frequency to the slow side comparable to that in sleep and the last three compounds a shift to the fast side comparable to that during attention. Pentothal greatly increased the voltage level in the range of 14—30 per sec. during the initial stages; the other depressant drugs produced smaller changes. The direction of the change in frequency paralleled the clinical effects of depression or stimulation, although the direction in the change in voltage level varied with dosage and was inconst. within drugs of the same class. P. C. W.

**Use of testosterone propionate in treatment of involutional psychosis in male.** E. Davidoff and G. L. Goodstone (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 811—817).—20 male patients suffering from involutional psychoses were treated with testosterone propionate, administered intramuscularly for 6 weeks—3 months. 13 improved against 22 out of 48 of a control group treated with routine hospital measures. Severe psychosis failed to respond well to androgenic treatment. W. M. H.

**Hyperthyrotic catatonia.** R. E. Hemphill (*J. ment. Sci.*, 1942, **88**, 1—30).—Hyperthyroidism is not a significant factor in the production of mental disorder, except in toxic delirium and in a symptom-complex for which the name " hyperthyrotic catatonia " is suggested. In the latter, a phase with auditory and visual hallucinations and distortion of the body-image is succeeded by catatonic stupor. G. D. G.

**Alternating tremor and its relation to cortical pathways.** B. H. Balser (*Arch. Neurol. Psychiat., Chicago*, 1942, **47**, 962—970).—A case is reported of bilateral paralysis agitans which had been present for 12 years when thrombosis of the right middle cerebral artery developed, with resulting left spastic hemiplegia, from which the patient recovered during the next 3 years with 60% of voluntary motor power. The tremor (of paralysis agitans) never returned to the affected extremities, however. The rigidity, previously present, was not altered. Postmortem examination revealed complete destruction of the head of the caudate nucleus, the putamen, part of the globus pallidus, and portions of the internal capsule. $\frac{3}{5}$ of the pyramidal tract, the whole frontopontine system, and a part of the temporopontine system, the frontothalmic fibres to the anterior lateral thalamic nucleus, the corticorubral and the corticonigral fibres were affected. It is suggested that adjacent areas as well as area 6 are related to tremor. W. M. H.

**Cortical reorganisation of motor function. Studies on series of monkeys of various ages from infancy to maturity.** M. A. Kennard (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 227—240).—The recovery of function following ablation of areas 4 and 6 was studied in young macaques of varying age. The greatest capacity for reorganisation was found in the first 6 months of life and declined until the end of the first year, when spasticity begins to develop. Recovery is slow and is greatest when the ablations are in stages separated by long intervals. W. M. H.

**Tumours of brain in children and in adolescents.** J. H. Globus, J. M. Zucker, and J. M. Rubinstein (*Amer. J. dis. Child.*, 1943, **65**, 604, 661).—A clinical and anatomical review of 92 verified cases. C. J. C. B.

**Permeability of blood–brain barrier to neurotropic viruses.** U. Friedemann (*Arch. Path.*, 1943, **35**, 912—928).—A general review. C. J. C. B.

**Progressive degenerative encephalopathy. Occurrence in infancy, with antenatal onset simulating " swayback " of lambs : report of case.** N. W. Winkelman and M. T. Moore (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 54—71).—Resemblance to " swayback " in lambs is shown in the case of an infant, born partly asphyxiated and developing progressive spastic diplegia, convulsive seizures, marasmus, terminal pneumonia, and purulent meningitis. The brain showed maldevelopment, subcortical demyelination, liquefaction necrosis with cyst formation, underdevelopment, and degeneration of the cortex. On analogy antenatal origin is suggested. W. M. H.

**Homolateral dilatation of pupil, homolateral paresis, and bilateral muscular rigidity in diagnosis of extradural hæmorrhage.** B. Woodhall, J. W. Devine, and D. Hart (*Surg. Gynec. Obstet.*, 1941, **72**, 391—398).—3 cases are described. P. C. W.

**Spinal origin of vasoconstrictor fibres in arm of man.** J. R. Learmonth and R. L. Richards (*Quart. J. Exp. Physiol.*, 1943, **32**, 87—88).—In a patient with spastic right hemiplegia and athetosis of arm and shoulder girdle, reflex vasomotor response in an arm was normal when the most cranial source of its preganglionic vasoconstrictor fibres was the 3rd thoracic segment of the spinal cord. T. C. D.

**Central autonomic paralysis.** E. A. Stead, jun., R. V. Ebert, J. Romano, and J. V. Warren (*Arch. Neurol. Psychiat., Chicago*, 1942, **48**, 92—107).—Study of 6 cases of thrombotic medullary syndromes showed different degrees of involvement of the various autonomic functions. The efferent tracts concerned are anatomically distinct in the medulla. Horner's syndrome may not be associated with hyperhydrosis. In 2 cases sweating on heating the body and vasoconstriction on cooling were disturbed while vasodilation on heating and vasoconstriction from a full inspiration or from sensory stimuli were normal. W. M. H.

## X.—SENSE ORGANS.

**Aspects of Brazilian ophthalmology.** M. E. Alvaro (*Amer. J. Ophthal.*, 1943, **26**, 474—479).—Brief biographical notes are given on several early Brazilian ophthalmologists, followed by a discussion of certain diseases which are unusually prevalent in that country, and an indication of the standard Brazilian approach to certain universal ophthalmological problems. J. H. A.

**Soviet military ophthalmology.** A. A. Kolen (*Lancet*, 1943, **244**, 804—805).—Modern Soviet methods of treating war injuries of the eye are largely based on experience gained in the Russian civil war. In regard to plastic work on the face, in uncomplicated cases surgery can be undertaken much earlier after the injury than was formerly considered advisable, and methods of reconstructing the eyelids and conjunctival sac are described. Many eyes which would formerly have been excised have been saved by removing foreign particles from the ciliary body. Blood transfusion is widely used in treating traumatic vitreous opacities, traumatic iridocyclitis, and sympathetic ophthalmia. J. H. A.

**Designation of visual acuity.** A. Linksz (*Arch. Ophthal.*, 1943, **29**, 662).—Whereas civilian ophthalmologists record visual acuity by reference to a standard distance, in the U.S. Forces a standard size letter is used and the distance varied; the author describes how a reading by one method can be converted into the other. J. H. A.

**Relation of riboflavin to eye.** A. Pirie (*Brit. J. Ophthal.*, 1943, **27**, 291—301).—Following a review of the ocular and general signs of riboflavin deficiency, as described by Sydenstricker, Sebrell, Kruse, and others, this paper proceeds with a description of the chemical and physical properties of the vitamin and its biochemical functions are discussed. The distribution of riboflavin in the eye and the syndrome produced in animals fed on riboflavin-deficient diets are described. It is emphasised that some of the ocular signs may be due to concurrent deficiency of other vitamins of the *B* group. J. H. A.

**Riboflavin and riboflavin adenine dinucleotide in ox ocular tissue.** F. J. Philpot and A. Pirie (*Biochem. J.*, 1943, **37**, 250—254).—The amounts of total riboflavin and riboflavin adenine dinucleotide in ox corneal epithelium, substantia propria, conjunctiva, aqueous and vitreous humour, iris, lens, ciliary body, retina, choroid, Meibomian and lacrymal glands, and Meibomian gland secretions were determined by microbiological and the *d*-amino-oxidase methods. The vitreous and aqueous humour and lens contain only very small amounts of flavin. Some of the retinal and choroidal flavin is present as dinucleotide. The lacrymal and Meibomian glands contain more flavin than any other part of the eye, whilst the corneal epithelium contains much more than does the substantia propria and it is suggested that the corneal epithelium obtains flavin from the secretions of the extraocular glands as well as from blood in the limbal capillaries. J. N. A.

**Effects, other than anti-infectious, of sulphonamide compounds on eye.** M. E. Alvaro (*Arch. Ophthal.*, 1943, **29**, 615—632).—Sulphonamide compounds given orally reach the eye in 15 min., and attain their max. concn. in 6 hr. Changes in the eye which have been recorded as toxic effects are palpebral œdema, chemosis, conjunctival and scleral injection, iritis, mydriasis, cataract, narrowing of angioscotomas, retinal œdema and hæmorrhages, reduction of visual fields, optic neuritis, transient refractive changes, changes of accommodation, and heterophoria. Of these, much the most common is transient myopia, which is seen most commonly after administration of sulphanilamide, and may be due to increased refractive index of the lens or to spasm of accommodation. J. H. A.

**Congenital absence of lacrimal puncta in three members of a family.** A. E. Town (*Arch. Ophthal.*, 1943, **29**, 767—771).—This rare condition is reported in a father and two sons. The literature is reviewed, and the relevant embryology and treatment discussed. J. H. A.

**Orthoptics : education in binocular skill.** J. E. Lascaster (*Amer. J. Ophthal.*, 1943, **26**, 463—467).—Lack of binocular vision is usually manifested by a deviation of one eye or, rarely, both eyes from the normal position. The author contends that concentration on restoring the eyes to a straight position, as implied by the term "orthoptics," involves a faulty approach to the problem, which is really one of achieving by educational methods that muscular co-ordination which in most people develops unconsciously in childhood. The orthoptist's aim should be to teach binocular skill, and get the patient to use it when acquired. J. H. A.

**Objectives of orthoptic examination and treatment.** A. Linksz (*Amer. J. Ophthal.*, 1943, **26**, 552—558).—The objects of an orthoptic examination are to determine a patient's mode of fixation, to measure any deviation of the eyes from that position in which bifoveal fixation can be maintained, and to ascertain the binocular visual habits which he may have acquired as a result of such deviation. The object of orthoptic treatment is re-education so that a faulty habit or adaptive reaction is replaced by normal binocular function. Details of orthoptic procedure are discussed, and surgery is given its place in the picture. J. H. A.

**Convergence function in relation to basal metabolism.** S. V. Abraham (*Amer. J. Ophthal.*, 1943, **26**, 400—404).—In over 83% of a series of patients with subnormal convergence, the basal metabolic rate was below normal. It is not suggested that this finding indicates a pure hypothyroidism, but rather that care of the general health is of more importance in treatment than correction of minor refractive errors and periodic orthoptic exercises, which can only be of palliative val. J. H. A.

**Practical application of crossed prisms.** L. Bacon (*Arch. Ophthal.*, 1943, **29**, 772—774).—A method is described for estimating the strength of the prism required when both vertical and horizontal phorias (or tropias) are present. The resultant prism is always stronger than either of those required to correct each deviation separately, and its base lies between the bases of the other two, nearer to that of the stronger one. J. H. A.

**Objective strabismometry in young children.** M. C. Wheeler (*Arch. Ophthal.*, 1943, **29**, 720—736).—In a comparison of the different methods of angular measurements of the amount of deviation of a squinting eye in children, the author found that Priestley Smith's tape was more accurate than the perimeter, but that Hirschberg's method of observing the position in the squinting eye of the corneal reflexion of a candle flame is the best measure of the static angle of squint. Duane's screen test, in which the deviation is corr. by prisms, is accurate but inapplicable to very young children; like the synoptophore, it tends to give too high readings in convergent squint, since it reveals not only the static angle but also a phoria component. J. H. A.

**Clinical vascular physiology of eye.** W. F. Duggan (*Amer. J. Ophthal.*, 1943, **26**, 354—368).—In a discussion of a no. of retinal and other lesions generally attributed to allergy, toxins, viruses, or focal sepsis, the author stresses the concept of homeostasis, *i.e.*, those processes which are concerned with maintaining the *status quo* within the body, such as the relation between blood-sugar level, insulin, and adrenaline. These homeostatic reactions may become permanently stabilised outside the zone of normality; persistence of abnormal physiology leads in turn to pathological changes in structure, and clinical disease is present. Along such lines the author attempts to explain spasm of the central retinal artery, amblyopia following systemic hæmorrhage, exudative choroiditis, retinitis pigmentosa, and diabetic retinopathy, the immediate cause in all cases being local anoxæmia resulting from some degree of capillary or arteriolar spasm. J. H. A.

**Study of aniseikonia in case of increasing unilateral index myopia** H. M. Burian and K. N. Ogle (*Amer. J. Ophthal.*, 1943, **26**, 480—490).—The case of an elderly man who, over a period of 6 years, exhibited increasing unilateral myopia due to nuclear sclerosis of the lens is reported. Correction of this increasing myopia and the resultant increasing anisometropia was accompanied by a proportionate increase in aniseikonia, and the patient was not comfortable until this also was corr. This case is contrasted with one having a similar degree of anisometropia due to unilateral myopia presumably of axial type, in whom no aniseikonia was demonstrable. J. H. A.

**Devices to aid refraction.** G. F. Harding (*Amer. J. Ophthal.*, 1943, **26**, 407—408).—The author describes two devices, one consisting of a group of four spherical lenses differing slightly in dioptric val. and mounted in a row, the other of two cylindrical lenses of equal strength but opposite sign, which can be easily manipulated and eliminate much changing of lenses in the trial frame, with consequent lessening of fatigue for the patient. J. H. A.

**Correction of low astigmatism : subtraction test.** S. J. Beach (*Arch. Ophthal.*, 1943, **29**, 775—781).—The test described is as follows. The estimated correction is set up in the trial frame, astigmatism being corr. with a plus cylinder; two minus cylinders (−0·37 or −0·50 D.) are then taken, one in each hand, the axis of one being with and the other at right angles to the axis of the trial frame cylinder; these are held alternately in front of the correction in the frame and have the effect of respectively weakening and strengthening the trial frame cylinder, the exact power of which can thus be subjectively estimated. The axis of the cylinder may be determined by placing the spherical correction in the rear cell of the trial frame and a +0·25 D. cylinder in the front at each of the four cardinal axes in turn; in each position, the auxiliary minus cylinders are then applied as before; acceptance of the "with" cylinder means no astigmatism at that axis, acceptance of the "against" cylinder indicates that astigmatism is present but undercorr. J. H. A.

**Blue scleras, brittle bones, and deafness.** J. E. Farber and A. E. Margulis (*Arch. intern. Med.*, 1943, **71**, 658—665).—A family of 52 members is described, 12 of whom have the blue sclera syndrome. 7 of the 12 members have both blue scleras and brittle bones. 4 have these defects and deafness and 1 has so far only blue scleras. 8 of the affected members are males; 4 are females. C. J. C. B.

**Hereditary corneal dystrophy : history of condition and presentation of pedigree.** S. Schutz (*Arch. Ophthal.*, 1943, **29**, 523—534).—The history and the various ætiological theories of this disease are reviewed, and the pedigree is given of a family of whom 15 adults (11 affected) and 6 children (none affected) were examined. Reticular, nodular, and ring-shaped lesions were all found; the visual acuity was only slightly or not at all reduced and inheritance was dominant. This corresponds with type I in Bückler's classification. J. H. A.

**Strange case of Mr. Huxley's eyes.** H. Hartridge (*Lancet*, 1943, **244**, 657—658).—A review of Aldous Huxley's "The Art of Seeing." Huxley states that he had an attack of "keratitis punctata" at the age of 16, which left him with bilateral corneal opacities: for the next 25 years, in spite of increasingly strong glasses (for hypermetropic astigmatism), he found it more and more difficult to read, until he tried the Bates method of visual re-education, after practising which for 2 months he was able to read without glasses and the opacities were beginning to clear up. Hartridge attempts a physiological explanation of the disability and its cure which, in his view, owed more to nature than to Bates. He also defends the orthodox ophthalmologist against Huxley's method of treating eyes and ignoring the part which the mind plays in the visual process. J. H. A.

**Chemotherapy in experimental eye lesions.** J. M. Robson and G. I. Scott (*Lancet*, 1943, **244**, 100—103).—Corneal lesions were produced in rabbits by intracorneal injection of *Staph. aureus*. Ulceration, usually with hypopyon, was produced in control eyes treated with saline. In each animal one eye was used as control and then treated by local application of drugs; when applied 1 hr. after inoculation (and given repeatedly for 48 hr.) definite improvement ocurred with penicillin, 30% and 10% Na sulphacetamide, or 15% solution of sulphathiazole. Na formaldehydesulphoxylate was less effective, and tyrothricin and 2·5% Na sulphacetamide were of little or no val. None of the substances was effective when applied 24 hr. after infection. Penicillin removed *Staph. aureus* from the conjunctival sac. C. A. K.

**Clearing of œdematous corneas by glycerin.** D. G. Cogan (*Amer. J. Ophthal.*, 1943, **26**, 551).—Full-strength glycerin applied locally produces a temporary clearing of simple œdema of the corneal epithelium, an effect which may be useful when it is desired to examine the fundus in acute glaucoma. J. H. A.

**Development of corneal reflex in Amphibia. III. Influence of periphery on reflex centre.** J. J. Kollros (*J. exp. Zool.*, 1943, **92**, 121—142).—Grafting of a supernumerary eye in the otic area of *Amblystoma* and *Triturus* modifies the type of response which can be elicited from this area. Normally stimulation elicits head flexion but stimulation of the extra cornea results in retraction of the host eye of the same side. The results are interpreted in terms of Weiss' hypothesis of modulation of neurones by their terminal organs. J. D. B.

**Iontophoretic introduction of atropine and scopolamine into rabbit eye.** L. von Sallmann (*Arch. Ophthal.*, 1943, **29**, 711—719).—Atropine sulphate and scopolamine hydrobromide were introduced into the eye by means of a galvanic current, and their concn. in different parts of the eye was compared with that obtained when the drugs were applied by simple corneal lavage or subconjunctival injection. The amount of alkaloid introduced is much increased by iontophoresis, though the process gives rise to transient corneal opacities. Scopolamine penetrates into the posterior part of the eye rather more readily than atropine, but the concns. reached by means of transcleral application are no higher than when the corneal route is used. J. H. A.

**Penetration of sugars into aqueous humour.** C. B. Weld, W. H. Feindel, and H. Davson (*Amer. J. Physiol.*, 1942, **137**, 421—425).—Dogs were anæsthetised with Na amytal and injected intravenously with the sugar solution. Both the initial time of penetration and the subsequent increase in concn. of the 4 sugars (xylose, glucose, sucrose, raffinose) in the aqueous humour are inversely related to their mol. wt. Xylose, mol. wt. 150, has a rapid rate of penetration and a decrease in concn. gradient between the blood and the eye of 25% 3 min. after injection. With glucose (mol. wt. 180) the concn. gradient falls by 25% in 10 min. Sucrose showed slower diffusion, the concn. gradient being decreased by 25% only after 3 hr. Raffinose (mol. wt. 504) penetration at 3 hr. is measurable but so low as to indicate that it approaches the limiting size of a lipoid-insol. non-electrolyte mol. penetrating the blood–aqueous barrier. M. W. G.

**Rôle of ascorbic acid (vitamin-*C*) in secretion of intraocular fluid.** J. S. Friedenwald, W. Buschke, and H. O. Michel (*Arch. Ophthal.*, 1943, **29**, 535—570).—The authors have previously shown that ascorbic acid was present in the intraocular fluid in appreciably higher concn. than in the blood, a difference which involved the expenditure of energy and was, therefore, a secretory mechanism. In further experiments, mainly with guinea-pigs, it is now shown that ascorbic acid could be largely eliminated from the ocular tissues by dietary restrictions long before any constitutional features of scurvy appeared, and that this resulted in a decrease of the rate of secretion of the intraocular fluid. Ascorbic acid is stored in the ciliary stroma, and acts as a mediator in an oxidative–reductive reaction which takes place between epithelium and stroma. Oxidation of the acid by the ciliary epithelium is probably a necessary step in its secretion since, although ascorbic acid is present in the ciliary body of the cat, it is not oxidised there and, in this animal, is not secreted into the intraocular fluid. J. H. A.

**Principles of tonometer standardisation.** E. Sachs and F. L. MacCraken (*Arch. Ophthal.*, 1943, **29**, 782—792).—The onus of calibrating and standardising a tonometer should rest on the manufacturer. Failing this, two main methods have been used. In one, the quant. properties of the "unknown" instrument, *e.g.*, wt., radius of curvature, etc., are compared with the Schiotz standard; in the other, the performance of the "unknown" tonometer is tested on a cornea or cornea-like membrane and compared with the performance of a standard instrument. In the first method, the evaluation of results is complicated and any divergence from the standard cannot be corr., so that the authors recommend the second procedure, using a fresh pig's eye connected with a manometer, and taking alternate readings with the standard and "unknown" tonometers with the 5·5-g. wt. and with pressures of 15 to 40 mm. Hg in steps of about 5 mm. The results are plotted on a graph, to reveal any correction which must be added to or subtracted from readings subsequently taken by the "unknown" instrument. J. H. A.

**Anisocycloplegia.** S. J. Beach (*Amer. J. Ophthal.*, 1943, **26**, 522—524).—In 17 out of over 100 cases examined, the depth of cycloplegia in the two eyes differed by over half a dioptre. This phenomenon was independent of whether the cycloplegic was administered by the single or multiple drop method, and is apparently a property of the individual eye. It is not likely to cause trouble except to those who prescribe the unchecked cycloplegic acceptance or the objective atropine correction. J. H. A.

**Changes in iris pigmentation in metamorphosing amphibian larvæ.**—See A., 1943, III, 619.

**Extra-epidermal and supernumerary lenses in association with cyclopean eyes in *Amblystoma* embryos.**—See A., 1943, III, 619.

**Changes in mineral composition of rat lenses with galactose cataract.** P. W. Salit, K. C. Swan, and W. D. Paul (*Amer. J. Ophthal.*, 1943, **25**, 1482—1486).—461 rat lenses, 119 normal, the remainder with partial or complete galactose cataracts, were analysed for their content of water, ash, and certain anions and cations. The water content was 14—20% higher; total ash, $Na^{\cdot}$, $Ca^{\cdot\cdot}$, $SO_4^{\prime\prime}$, and $CO_3^{\prime\prime}$ were increased, $K^{\cdot}$, $PO_4^{\prime\prime\prime}$, and $Cl^{\prime}$ decreased in the cataractous group. On the whole, there is a greater increase in the negative than the positive ions with increasing opacity of the lens. J. H. A.

**Cataract in rats fed a low-protein diet.** C. Rezende and F. A. de M. Campos (*Arch. Ophthal.*, 1942, **28**, 1038—1041).—A no. of albino rats were fed on a low-protein diet (casein 10%). All the animals showed a poor growth curve, and cataractous changes were observed when they were about 2 months old. Since starch was the only carbohydrate in the diet, galactose cataract can be excluded, and it is thought that the opacities must be attributed solely to deficient protein intake. Proteins are known to have an anticataractogenic property both in galactose cataract and in normal lenses. J. H. A.

**Hereditary cataracta caerulea: six related cases.** O. Wolfe and R. M. Wolfe (*Amer. J. Ophthal.*, 1943, **26**, 404—406).—Six cases of blue-dot cataract in the same family are reported, four of whom were over 35 years of age and had in addition a well-marked posterior subcapsular opacity which necessitated intracapsular extraction. The two younger patients were operated on by the double-aspirating-needle technique, described by the authors in a previous paper. J. H. A.

**Effect of optical stimuli on [pituitary and] output of urine in albino rats.**—See A., 1943, III, 567.

**Spectral sensitivity of retinal receptors.** W. D. Wright (*Nature*, 1943, **151**, 726—727).—The visibility curve for the human eye was measured at low brightness by comparison with a field of 0·63 $\mu$. at $5·8 \times 10^{-6}$ erg per sq. degree per sec., using test fields 5′ and 20′ diameter, for foveal and 2° parafoveal vision. This reveals a hump on the parafoveal and a subsidiary peak on the foveal curve at 0·60 $\mu$., which is interpreted as the peak of the red receptor response and as showing the composite nature of the visibility curve. K. J. W. C.

**Physiology of colour vision.** (A) E. N. Willmer. (B) K. J. W. Craik. (C) A. H. S. Holbourn. (D) H. Hartridge. (E) E. N. Willmer (*Nature*, 1943, **151**, 632, 727—728; **152**, 190, 190—191, 191).—(A) The impossibility of matching greens or yellows with mixtures of the ends of the spectrum is attributed to the impossibility of achieving a high enough brightness by mixing red and blue.

(B) Willmer's theory of colour vision (see above) is criticised on the ground that mixtures of the extremes of the spectrum do not match green or yellow, nor does a wave-length of 560 $\mu$. look white, at brightness levels where spectral greens and yellows are seen as such, and where white light appears white.

(C) Willmer's theory of colour vision is criticised on the ground that three independent variables are required.

(D) Willmer's theory is criticised on the ground that a purple (a red plus blue) and a green do not match in colour at any brightness.

(E) The above objections are admitted to be serious, but modification of the hypothesis which will satisfy them is thought possible. K. J. W. C.

**Trichomatic theory of vision.** W. Peddie (*Phil. Mag.*, 1943, [vii], **34**, 426—430).—A discussion, with analogies, of the independence of colour receptors. K. J. W. C.

**Representation of intensity, hue, saturation, and magnitude of the resultant vector on the trichromatic theory.** W. Peddie (*Phil. Mag.*, 1943, [vii], **34**, 488—495).—Newton's and Abney's laws of colour-addition show the brightness of a resultant sensation to be the same as that resulting from the arithmetical sum of two white lights which individually matched the two colours in brightness. Yet the representation of colour and brightness in a three-dimensional colour-solid represents the sum as the resultant vector-length which would not ordinarily be the arithmetical sum of the individual vectors. It is claimed that if the individual vectors are drawn on a logarithmic scale to represent sensations, the magnitude of the resultant vector is correct. K. J. W. C.

**Standardised colour-vision testing lantern. II. Transport type.** L. C. Martin (*Brit. J. Ophthal.*, 1943, **27**, 255—259).—The author has modified a lantern which he designed in 1939 to make it specially suitable for use by transport undertakings. In addition to several minor alterations, the new model contains an orange-yellow filter as well as the original red, green, and white lights. The orange glass is combined with a suitable neutral filter to make the intensity of the transmitted light equal to that of the others. The aperture can be varied in size, but when the smallest " stops " are used even normal persons may sometimes make mistakes with colour combinations containing orange, so that this colour should be omitted when testing with such apertures. J. H. A.

**Improvement of colour vision by vitamin intake.** D. P. Le Galley and J. W. E. Harrisson (*Amer. J. Pharm.*, 1943, **115**, 95—99).—16 colour-blind subjects were divided into 4 groups of 4 each. One group received no additional vitamins (control), the other three groups received respectively a daily dose of -*A* (30,000 U.S.P. units), -$B_1$ (8 mg.), and -$B_2$ (16 mg.). Their colour vision was tested weekly for ten weeks by means of pseudo-isochromatic charts. The average improvement in the -*A* and -$B_1$ groups was in the region of 21%, as compared with 2% in the control and -$B_2$ groups. Subsequent administration of -$B_1$ to the -*A* group and -*A* to the -$B_1$ group produced further improvement of about 11%. J. H. A.

**Vitamin-*A* and dark adaptation.** J. Yudkin, G. W. Robertson, and S. Yudkin (*Lancet*, 1943, **245**, 10—13).—Using a modification of the Crookes adaptometer, the course of dark-adaptation in about 400 apparently normal subjects was measured. It was found that repeated measurements were remarkably const. for each subject, but that the course of adaptation varied considerably in different individuals. In particular, the cone–rod transition may occur early or late and bears no relation to the final rod-threshold, which in some of the cases was actually lower when the cone–rod transition was retarded; for this reason, any methods of testing dark-adaptation which rely on readings taken in its early stages are fallacious. Administration of vitamin-*A* was without effect in half the treated cases; in those whom it did affect, the final rod-threshold was always lowered, and some cases also showed alterations in the cone-threshold and cone–rod transition time. J. H. A.

**Hereditary macular degenerations.** R. I. Lloyd (*Amer. J. Ophthal.*, 1943, **26**, 499—508).—These degenerations are classified into four morphological types: (1) consisting of large white spots in the macular area (honeycomb or guttate choroiditis); (2) showing small and mainly peripheral white dots (retinitis punctata albescens, fundus albipunctatus cum hemeralopia congenita); (3) characterised by macular pigmentation with choroidal atrophy (juvenile maculo-cerebral degeneration, progressive macular degeneration); and (4) a macular choroidal degeneration with secondary retinal involvement, which, apart from the peripheral changes which often co-exist, may resemble a macular coloboma. J. H. A.

**Traumatic œdema of macula.** S. Philps (*Brit. J. Ophthal.*, 1943, **27**, 305—306).—Commotio retinæ may subside in a few days, or it may initiate a progressive lesion. Each of the four cases here reported illustrates one of the four typical stages in its course, viz., macular œdema, pigmenting changes replacing œdema, hole formation, and retinal detachment. J. H. A.

**Pigmentation of optic disc.** D. Kravitz (*Arch. Ophthal.*, 1943, **29**, 826—830).—Three cases are reported each of which exhibited a uniformly pigmented area on the optic disc of one eye. The literature is reviewed, the embryology discussed, and the presence of a cilioretinal vessel in all three cases is mentioned as of doubtful significance. J. H. A.

**Hypophysis and adrenals and retinal pigment of *Bufo arenarum*.** J. Sverdlick (*Rev. Soc. Argent. Biol.*, 1942, **18**, 207—214).—The retinal pigment of *B. arenarum* expands in light and contracts in darkness. The posterior hypophysis regulates the expansion caused by light; after hypophysectomy, moderate contraction of retinal pigment follows light adaptation and injection of extracts of posterior lobe in illuminated animals produces total expansion of the pigment in these but not in dark-adapted animals. The adrenals have no action on the expansion or contraction of the pigment in light and darkness. I. C.

**Choroidal detachment.** B. W. Rycroft (*Brit. J. Ophthal.*, 1943, **27**, 283—291).—Having reviewed the literature on choroidal detachment following glaucoma and cataract operations, the author discusses its ætiology and treatment in the light of his own investigations. He believes that the primary cause of the condition is sudden loss of the anterior chamber, which results in a forward movement of the lens, zonule, and ciliary body, with tearing of the choroid near the ova serrata. It is more prone to occur if the anterior chamber is deep, for then the forward movement is greater, and its occurrence is not influenced by variations in operative technique. Spontaneous reposition may occur; if it does not, there is often a fistula in the conjunctival flap preventing re-formation of the anterior chamber, and treatment should be directed to closing it. J. H. A.

**Historical survey of structure and function of cochlea.** T. H. Bast and J. Shover (*Ann. Otol. etc., St. Louis*, 1943, **52**, 281—329).—A detailed historical account of theories of hearing from Helmholtz' resonance theory to the resonance-volley theory of Wever and Bray. K. T.

**Ménière's disease. Surgical treatment.** W. E. Dandy. **Medical treatment.** H. G. Tobey (*Surg. Gynec. Obstet.*, 1941, **72**, 421—425, 425—430).—Reviews. P. C. W.

**Use of histamine in Ménière's disease.** B. T. Horton (*Surg. Gynec. Obstet.*, 1941, **72**, 417—420).—49 cases of Ménière's disease have been treated by the intravenous injection of 1 mg. of histamine over 1½ hr., repeated daily until the acute symptoms disappear, followed by maintenance doses of 0·1—0·2 mg. of histamine injected subcutaneously 2—4 times weekly. 15 of the cases are well and the remainder have had 1 or 2 recurrences which may be due to failure to persist with maintenance therapy. P. C. W.

## XI.—DUCTLESS GLANDS, EXCLUDING GONADS.

**Relation of certain endocrine glands to body weight in growing and mature guinea-pigs.** J. P. Mixner, A. J. Bergman, and C. W. Turner (*Endocrinol.*, 1943, **32**, 298—304).—The relative growth equation, $Y = aX^b$ ($Y$ = gland wt., $X$ = body wt., $a$ and $b$ are empirical consts.), was used to predict the wts. of endocrine glands of guinea-pig from their body wts. The vals. found for $a$ and $b$ were: pituitary ($X$ up to 1000 g.): male $a = 0{\cdot}183$, $b = 0{\cdot}708$; female $a = 0{\cdot}140$, $b = 0{\cdot}732$; thyroid ($X$ up to 1000 g.): male $a = 0{\cdot}598$, $b = 0{\cdot}730$; female $a = 0{\cdot}366$, $b = 0{\cdot}830$; adrenal ($X$ under 500 g.): male and female $a = 0{\cdot}657$, $b = 0{\cdot}949$; ($X$ over 500 g.): male and female $a = 0{\cdot}00381$, $b = 1{\cdot}789$; testis ($X$ up to 1000 g.): $a = 0{\cdot}00129$, $b = 2{\cdot}319$; ovary ($X$ up to 1000 g.): $a = 0{\cdot}637$, $b = 0{\cdot}762$. G. P.

**Interhormonal relations [on liver-glycogen].** I. Abelin (*Verh Ves. Schweiz. Physiol.*, 1942, **21**, 13).—Administration of adrenal cortex extracts or synthetic cortex hormones antagonises the glycogen-mobilising effect of thyroxine on the liver. Similar effects were observed following intraperitoneal injections of anterior pituitary hormones. A. S.

**Endocrine glands and pharmacology of colour in frogs.** A. O. M. Stoppani (*Rev. Soc. Argent. Biol.*, 1942, **18**, 215—224).—In *Bufo arenarum* and *Leptodactylus ocellatus* adrenaline, ephedrine, cocaine, and $CaCl_2$ produce pallor. Darkening is produced by Fourneau 933, veratrine, KCl, atropine, or eserine. Caffeine and nicotine darken *Leptodactylus* and have a complex action on *Bufo*. Removal of the hypophysis in *Bufo* suppresses the action of F. 933, veratrine, atropine, KCl, and eserine, and decreases the action of the other substances. Destruction of the adrenals in *Bufo* decreases the pallor produced by adrenaline, $CaCl_2$, cocaine, nicotine, or caffeine. Eserine or acetylcholine has no direct action on the melanophores of both frogs. Destruction of the autonomic nervous system does not greatly modify the action of drugs on the colour of *Bufo*. The melanophores of *Bufo* appear to be adrenergic effectors. I. C.

**Surgical pathology of thyroid gland.** A. C. Broders (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 620—632).—Macroscopic and microscopic descriptions are given of the various pathological changes in the thyroid gland. P. C. W.

**Liver failure following operation in hyperthyroidism.** G. Crile (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 438—444).—Impairment of liver function in hyperthyroid patients is related to the age of the patient more closely than to the basal metabolic rate. Liver failure characterised by confusion, delirium, and raised icteric index may occur in elderly patients on the 2nd or 3rd day after operation for hyperthyroidism. P. C. W.

**Graves' disease and myxœdema.** H. Zondek (*Lancet*, 1943, **244**, 75).—A woman of 34 had thyrotoxicosis; after treatment with I and irradiation of thyroid myxœdema developed and persisted for 4 years. Emotional disturbances later produced signs of thyrotoxicosis. C. A. K.

**Congenital myxœdema without mental retardation.** J. C. M. Fournier and J. M. Cervino (*J. Clin. Endocrinol.*, 1943, **3**, 265—267).—A 10-year-old patient is described. P. C. W.

**Anatomical classification of goitre.** W. C. MacCarty (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 550—555). P. C. W.

**Granulomas in struma fibrosa of thyroid.** W. M. German (*West. J. Surg. Obstet. Gynec.*, 1941, **49**, 120—131).—6 examples were found among 205 thyroid glands examined. Anatomical and histological descriptions are given and the lesions shown to be non-tubercular. P. C. W.

**Influence of age on effect of thyroidectomy in rhesus monkey.** W. Fleischmann, H. B. Shumacker, jun., and W. L. Straus, jun. (*Endocrinol.*, 1943, **32**, 238—246).—Thyroidectomy in adult rhesus monkeys has no effect on basal metabolic rate, on serum-cholesterol, or on creatine excretion in the urine. All the adult monkeys died of tuberculosis. In two 8-months-old monkeys thyroidectomy was followed by sharp rise in serum-cholesterol; in one of them, which survived the operation 151 days, there was a complete cessation of growth. G. P.

**Effect of sulphanilamide on conversion *in vitro* of inorganic iodine into thyroxine and di-iodotyrosine by thyroid slices.** A. L. Franklin and I. L. Chaikoff (*J. Biol. Chem.*, 1943, **148**, 719—720).—The degree of inhibition of the conversion of inorg. I′ into di-iodotyrosine and thyroxine is related to the concn. of sulphanilamide. P. G. M.

**Adrenal gland in hyperthyroidism.** E. C. Bartels, C. K. Stuart, and E. C. Johnson (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 424—437).—There was no change in blood-I or -cholesterol or clinical improvement in 1 hyperthyroid patient given daily injections of cortical extract for 10 days. Only 3 of 40 hyperthyroid patients maintained on a low-Na, low-Cl, and high-K diet showed low (less than 125 mg. per 100 ml.) concn. of Na in the urine on the 3rd day of the test indicating abnormal adrenal-cortical function. The increased urinary Cl excretion observed in some of the patients may indicate depressed adrenal-cortical function. P. C. W.

**Primary and secondary hyperparathyroidism.** L. J. Soffer and C. Cohn (*Arch. intern. Med.*, 1943, **71**, 630—649).—9 cases of hyperparathyroidism are reported. In 5 the condition was primary, due to adenoma of a parathyroid gland, and in 4 it was secondary, due to chronic renal diseases, multiple myeloma, or carcinomatous metastasis in bone. C. J. C. B.

**Ectodermal disorders in chronic hypoparathyroidism.** N. Learner and C. L. Brown (*J. Clin. Endocrinol.*, 1943, **3**, 261—264).—A case of chronic postoperative hypoparathyroidism showed pathological changes in skin, nails, hair, and ciliary epithelium (which recurred seasonally) and cheilitis and glossitis. All these symptoms disappeared under dihydrotachysterol therapy. P. C. W.

**Pinealoma.** W. O. Russe and E. Sachs (*Arch. Path.*, 1943, **35**, 869—888).—A clinicopathological study of 7 cases with a review. (8 photomicrographs.) C. J. C. B.

**Experimental necrosis of islets of Langerhans.** J. S. Dunn, H. L. Sheehan, and N. G. B. McLetchie (*Lancet*, 1943, **244**, 484—487).—Acute necrosis of almost all islet tissue of the pancreas was produced in rabbits by intraperitoneal injection of a synthetic styrylquinoline or by intravenous alloxan. The external secretory tissue of the pancreas and other endocrine glands were unaffected. The islet necrosis was accompanied by an initial rise of blood-sugar, followed by an intense hypoglycæmia with death in 12—48 hr. It is suggested that the islets are overstimulated and that the cells die from overstrain. C. A. K.

**Action of adrenaline on atropine–acetylcholine reversal phenomenon.** R. L. Stehle and K. I. Melville (*J. Pharm. Exp. Ther.*, 1943, **77**, 332—335).—During continuous infusion of adrenaline in the spinal atropinised cat, the usual pressor response to acetylcholine is replaced by a transient fall of blood pressure. During a similar infusion of pituitary extract the pressor response is not abolished but may be preceded by a transient depression if pressure is high. V. J. W.

**Adrenal cortex in systemic disease.** E. L. Sarason (*Arch. intern. Med.*, 1943, **71**, 702—712).—Cortical enlargement with depletion of lipin or reversal of lipoid pattern was found associated with imflammatory diseases, cachexia, pemphigus, and protracted vomiting. Cortical enlargement with increased lipin was encountered in cases of hypertension. No alterations were present in atherosclerosis. Extreme enlargement was found in 4 cases of erythroblastosis fœtalis. C. J. C. B.

**Influence of vitamin-$B_1$ on the adrenaline content of the adrenals and of blood.**—See A., 1943, III, 664.

**Reticuloendothelial system and vitamin-*C* storage after adrenalectomy.**—See A., 1943, III, 666.

**Symmetrical skin disease in adrenalectomised rats cured by cozymase and nicotinamide.**—See A., 1943, III, 666.

**Laryngeal and systemic histoplasmosis (Darling).** P. A. van Pernis, M. E. Benson, and P. H. Holinger (*Ann. int. Med.*, 1943, **18**, 384—393).—Report of a case. The destruction of the adrenal glands caused an Addison's syndrome. A. S.

**Use of adrenal cortical hormone in *Cl. welchii* infections in guinea-pigs.** M. Kepl, G. Caldwell, and A. Ochsner (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 25—26).—Subcutaneous injections of cortical extract prolonged survival time in guinea-pigs experimentally infected with *Cl. welchii*. V. J. W.

**Effect of deoxycorticosterone acetate on the hypertrophy of the adrenals caused by exercise.** M. Beznák and Z. Korényi (*Magyar Orv. Arch.*, 1941, **42**, 71—79).—Deoxycorticosterone acetate in a daily intraperitoneal dose of 100 μg. per kg. body wt. administered in 0·05 c.c. of arachis oil 1 hr. before work reduced the resultant adrenal hypertrophy to ⅓ in the cortex but to a smaller extent in the medulla. Mortality due to running was prevented. The arachis oil itself caused a 25% medullary hypertrophy in resting animals which was not inhibited by the sterone, and increased mortality in working animals. Deoxycorticosterone acetate prevented mortality in working animals receiving arachis oil. The toxicity of arachis oil may be due to the higher fatty acids. The mechanism of adrenal hypertrophy due to exercise is discussed. M. A. B.

**Life-maintaining and gluconeogenic properties of the cortin-like material excreted postoperatively.** E. H. Venning, M. M. Hoffmann, and J. S. L. Browne (*J. Biol. Chem.*, 1943, **148**, 455—456; cf. A., 1940, III, 409).—The cortin-like material in the urine of postoperative patients is less than half as effective as adrenal cortical extract (based on " cold unit " dosage) in maintaining life, growth, and liver-glycogen formation in adrenalectomised rats. The material may be derived from the cortex. R. L. E.

**Lactoflavin excretion in normal and adrenalectomised animals.** L. Laszt and L. D. Torre (*Vehr. Ver. Schweiz. Physiol.*, 1942, **21**, 23—24).—In normal rats the 24-hr. urinary lactoflavin excretion is 25 μg., in adrenelectomised rats 45 μg. After subcutaneous injection of 350 μg. of lactoflavin, the excretion in normals is half that of adrenalectomised animals; max. excretion occurs within 3½ hr. After previous administration of anterior pituitary or adrenal cortex the excretion in adrenalectomised rats becomes normal. After oral administration of 350 μg. of lactoflavin the 24-hr. excretion in normal rats is like that 3½ hr. after subcutaneous injection; after oral administration, adrenalectomised rats excrete half the normal amount. A. S.

**Anatomy and histology of swordfish pituitary.**—See A., 1943, III, 617.

**Developmental control of pars intermedia of hypophysis by brain.**—See A., 1943, III, 619.

**Carbohydrate tolerance of hypophysectomised dogs.**—See A., 1943, III, 671.

**Nitrogen retention, creatinuria, and other effects of treatment of Simmonds' disease with methyltestosterone.** S. C. Werner and R. West (*J. clin. Invest.*, 1943, **22**, 335—344).—The patients demonstrated renewed vigor, sense of strength, and libido, and redeveloped secondary sex characteristics after 100 mg. daily. There was no seborrhœa. There was N retention, associated with persistent wt. gain. Marked creatinuria developed after several weeks, and subsided some weeks after stopping treatment. C. J. C. B.

**Cushing's syndrome in children.** J. E. Farber, F. J. Gustina, and A. V. Postoloff (*Amer. J. dis. Child.*, 1943, **65**, 593—603).—Review and case report. C. J. C. B.

**Changes induced in Harderian gland of guinea-pig by injection of hypophyseal extracts.** G. K. Smelser (*Anat. Rec.*, 1943, **86**, 41—57).—Marked hypertrophy of the Harderian gland follows the injection of extracts of anterior pituitary. There is also an extensive discharge of lipin secretion from cells of the secretory epithelium. These effects are readily produced in thyroidectomised animals and it is thought that thyroidectomy actually enhances the response. W. F. H.

**Influence of Roentgen ray treatment of hypophysis and reproductive systems of ground squirrel and rat.** R. H. Denniston, II (*J. exp. Zool.*, 1942, **91**, 237—263).—Pituitaries or testes of ground squirrels and rats of various ages were *X*-rayed, at various times in the sex cycle, with dosages up to 6000 r. The results of this treatment on endocrine system and growth are detailed. The pituitary is extremely resistant to *X*-rays and no evidence of a " stimulating " dose was found. The cell types of the pituitary show a differential susceptibility to *X*-rays; α-cells lose their granules, the cytoplasm shrinks, and the nucleoli increase enormously in size; β-cells and chromophobes are apparently unaffected within the range of dosage used (7 to 4000 r). In young rats these cytological changes in the pituitary were accompanied by a 50% reduction in growth rate. J. D. B.

**Rapid œdema during perfusion of hypophysectomised frogs.** S. Senderey (*Rev. Soc. argent. Biol.*, 1942, **18**, 244—256).—The œdema produced by perfusion of *Bufo arenarum* with Ringer's solution is greater after hypophysectomy, owing to the lack of the neurointermediary lobe. Grafts of the pars neuralis stop the disturbance,

but those of the pars distalis are ineffective. Hypophysectomy increases capillary permeability to water, trypan-blue, Congo-red, and gelatin. The fluid loss is compensated by increased reabsorption. I. C.

**Anatomical changes in rats after hypophysectomy.** V. G. Foglia and H. E. J. Houssay (*Rev. Soc. argent. Biol.*, 1942, **18**, 376—396).—Hypophysectomy in white rats diminished growth and the wt. of the body (15%) and of all organs, especially the testes (87%) and secondary sexual organs, adrenals (61%), and thyroid (59%). The % decrease of wt. in other organs was: kidney 56; heart 34; liver 30; thymus 20. The decrease of wt. is quicker in the testis, adrenal, and thyroid; it is not due to under-nutrition but to lack of stimulating hormones. I. C.

**Changes in thyroid and adrenals of rats after hypophysectomy.** H. E. J. Houssay (*Rev. Soc. argent. Biol.*, 1942, **18**, 410—415).—After hypophysectomy the height of the epithelium in the thyroid of male rats is decreased (37%); there is also great atrophy of the adrenal cortex and especially of its internal part; the medulla is not atrophic but does not grow. I. C.

**Thyroid-inhibiting action of hypophyses of rats fed with thyroid.** J. Reforzo-Membrives (*Endocrinol.*, 1943, **32**, 263—270).—Pituitaries or normal or thyroidectomised rats, fed desiccated thyroid, when injected into guinea-pigs decrease the height of the thyroid epithelium and basal metabolic rate. The power of the thyroid tissue of the injected guinea-pigs to oxidise *p*-phenylenediamine also decreases. G. P.

**Thyrotropic activity of anterior pituitary.** W. O. Thompson, P. K. Thompson, and S. G. Taylor (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 633—635).—Of 93 patients with basal metabolic rates below −10% 49 showed increases of over 10% in response to injections of pituitary thyrotropin; of those whose basal metabolic rate was below −30% only 1 responded; this patient was the only prepubertal case and her thyroid showed no atrophy. 12 of 24 patients with normal, and 14 of 28 patients with raised, basal metabolic rate (over 10%) showed increases. P. C. W.

**Isolation of thyrotropic hormone from anterior pituitary of whale.** C. Bomskov and L. Sladović (*Z. physiol. Chem.*, 1940, **264**, 274—286; cf. A., 1941, III, 873).—The prep. of thyrotropic hormone from whale anterior pituitary and from cattle glands is described. The hormone in the whale gland is not extracted by acetic acid, which extracts most of the hormone from cattle glands. A larger yield is obtained from the fresh whale gland than from the acetone-dried powder, and the highest yields are obtained by extraction with either 20% alcohol, 20% glycerol, 2% urea, or 1% bile followed by several extractions with dil. NaOH. Combination of the above solvents in a single extracting medium is of no use for extraction of the hormone. 1 kg. of dried powder from whale anterior pituitary contains 144,000 mouse units of the hormone, which is 63% of the amount present in cattle glands. J. N. A.

**Mol. wt. of the adrenocorticotropic hormone.** E. Burtner (*J. Amer. Chem. Soc.*, 1943, **65**, 1238).—Measurements of diffusion and sedimentation for this hormone (Li *et al.*, A., 1942, III, 599) give diffusion const. $10{\cdot}4 \times 10^{-7}$, mol. wt. 20,000±10%, frictional ratio 1·1, and axial ratio 3 : 1. R. S. C.

**Pituitary lactogenic hormone. IX. Content of sulphur-containing amino-acids.** C. H. Li (*J. Biol. Chem.*, 1943, **148**, 289—291; cf. A., 1943, III, 324).—Pituitary prolactin contains 4·31% of methionine and 3·11% of cystine. These acids together account for the total S. P. G. M.

**Non-specific results obtained with micro-method for assay of prolactin.** E. L. Lahr, R. W. Bates, and O. Riddle (*Endocrinol.*, 1943, **32**, 251—259).—Plucking feathers, incident to making local crop-sac tests for prolactin (micro-method of Lyons and Page), splitting the skin, or intradermal injection over the crop-sac of 0·5 ml. of air, water, or 0·9% NaCl produces a slight temporary increase in mitotic rate in the underlying epithelium of the crop. This is compatible with the successful use of the method. Org. and inorg. acids, aq. $NH_3$, ethyl or butyl alcohol, cresol, acetone, formaldehyde, bile, Na glycocholate, Na taurocholate, quinol, histamine, and androsterone when injected in 1—10-mg. doses over the crop-sac produced non-sp. positive macro- and micro-scopic reaction. Leucotaxine gave positive macroscopic reactions 48 hr. after injection, but increased mitoses were found in the crop-epithelium only up to 24 hr. G. P.

**Slowly-acting pituitary preparations in diabetes insipidus.** D. Court and S. Taylor (*Lancet*, 1943, **244**, 265—267).—Studies in 4 cases of diabetes insipidus showed that pitressin tannate suspended in peanut oil (5 i.u. per c.c.) is the safest and most effective slowly-acting prep. Its action lasts about 48 hr. Post-pituitary emulsion was clinically effective but paraffinomata developed at the sites of injection. C. A. K.

**Time relations of colour response.** H. Waring and F. W. Landgrebe (*J. Exp. Biol.*, 1941, **18**, 80—97).—In slowly-responding animals (*Xenopus*, *Anguilla*), the limiting factor is the rate of change of the concns. of pituitary hormones in the blood, and not the rate with which the melanophores react to changes in concn. The blood vol. of eels, and the distribution of melanotropic hormone (*B*) between pituitary and blood, are estimated. G. P. W.

## XII.—REPRODUCTION.

**Echinochromes as prosthetic groups of symplexes of high mol. wt. in eggs of *Arbacia pustulosa*.** R. Kuhn and K. Wallenfels (*Ber.*, 1940, **73**, [*B*], 458—464).—Ovaries of *Arbacia* collected in the Gulf of Naples in November, 1939—January, 1940, were dark violet-red and contained little echinochrome *A* but much echinochrome *B* and *C*; in February the brown-red ovaries no longer contained *B* or *C*, which had been replaced by *A*. *A*, *B*, and *C* can be removed from the ovaries by ether after addition of HCl. *A* and *B* but not *C* are removed from the ether by aq. $NaHCO_3$. The pigments are characterised by absorption spectrum and fluorescence. *B* has m.p. 173—175° (Kofler) and appears according to methylation experiments to be a hydroxylated derivative of naphthazarin. The echinochromes are present in the eggs as binary and ternary cymplexes built on the lines echinochrome–carrier and echinochrome–carrier–additional carrier. The ternary complexes of *A*, *B*, and *C* are sol. in 4% NaCl and salted out by $(NH_4)_2SO_4$, $CdSO_4$, $MgSO_4$, or $ZnSO_4$. Solutions in sea-water are unaffected by boiling or by addition of alcohol but give a ppt. with tannin or, in much greater dilution, with benzyldimethyldodecylammonium bromide. The colour and redox behaviour are described. The tenaciousness of the prosthetic group diminishes in the sequence $A > B > C$. Dialysis of the *A* or *B* + *C* symplex against distilled water until Cl is entirely removed is accompanied by almost complete pptn. of the pigment whilst the additional carrier remains in solution, from which it can be pptd. by $(NH_4)_2SO_4$, $MgSO_4$, $CdSO_4$, or $ZnSO_4$. It contains 8·0—8·5% N and is characterised by precipitability with 0·001N-HCl. The binary *A*-symplex is sol. in distilled and sea-water, from which it is completely pptd. by an equal vol. of alcohol; the ppt. is insol. in sea-water. Denaturation appears to have resulted from the removal of the additional carrier. The biological action of the symplexes is described. H. W.

**Light and sexual reproduction in Hydroids.** W. W. Ballard (*Biol. Bull.*, 1942, **82**, 329—339).—*Hydractinia* and *Pennaria* fail to spawn in continuous light or darkness, but spawn when light and darkness alternate. Light, following darkness, initiates maturation by a direct action on the germ cells themselves. Spawning occurs, apparently by muscular action, as soon as maturation is complete. Owing to a difference in the rate of the response to illumination, *Hydractinia* spawns shortly after sunrise and *Pennaria* several hr. later. G. P. W.

**Correlation between sexual reproduction and regeneration in Oligochaetes.** E. Liebmann (*J. exp. Zool.*, 1942, **91**, 373—387). J. D. B.

**Sex reversal in shipworm.** B. H. Grave (*Biol. Bull.*, 1942, **82**, 438—445).—In *Teredo navalis*, change from male to female, and vice versa, is normal and frequent. G. P. W.

**Reproduction in water-vole.** J. S. Perry (*Proc. Zool. Soc., London*, 1943, **112**, **A**, 118—130).—An account of reproduction in *Arvicola amphibius* based on field material comprising 128 males and 95 females. Details are given of reproductive organs, breeding season, œstrous cycle, and histological changes in genital tract and mammary glands. J. D. B.

**Reproduction in oysters. I.** V. L. Loosanoff. **II.** V. L. Loosanoff and J. B. Engle (*Biol. Bull.*, 1942, **82**, 195—206, 413—422).—Observations on adult *Ostrea virginica* in Long Island Sound. I. A systematic description of seasonal changes in the gonads. II. A description of spawning activities in oysters living at different depths. G. P. W.

**Sexual differentiation in molluscs. I. Pelecypods.** W. R. Coe (*Quart. Rev. Biol.*, 1943, **18**, 154—164).—A review. J. D. B.

**Primary and secondary sexual characteristics: their development in males from birth through maturity, with biometric study of penis and testes.** W. A. Schonfeld (*Amer. J. dis. Child.*, 1943, **65**, 535—549).—A reliable method of measuring the size of the penis and testes is described. During the first 2 years, there is only slight growth of the testes and increase in the circumference of the penis; the length of the penis continues to increase until 4—5 years of age. There is no further change until pubescence, when there is a rapid spurt reaching its max. during the post-pubescent period. The pubescent spurt may occur at 10—18 years of age. The secondary sexual characteristics were correlated with the size of the testes and penis. C. J. C. B.

**Physiology of endocrines in pregnancy, lactation, and puerperium.** E. Novak (*J. Clin. Endocrinol.*, 1943, **3**, 274—280).—A review. P. C. W.

**Growth of avian ovum.**—See A., 1943, III, 618.

**Incidence of ovulation determined by endometrial biopsy.** D. G. Morton and C. T. Hayden (*West. J. Surg. Obstet. Gynec.*, 1941, **49**,

15—28).—239 endometrial biopsy specimens were taken 20 days after the last day of menstruation and examined for the presence of the secretory phase of the cycle. Of the 116 normal patients 15% had anovulatory cycles (absence of secretory phase), of the 105 specimens from patients with menorrhagia and/or fibroids 39 showed anovulatory cycles (37%), and of the 18 specimens from women with irregular cycles 72% were anovulatory. There was little difference in the incidence of anovulatory cycles among the women below 40; those older than this showed a higher proportion of anovulation. A total of 142 specimens were taken from 33 normal women 5 days before the onset of the next period; only 7 anovulatory cycles were found. P. C. W.

**Human corpus luteum of pregnancy.** J. Gillman and H. B. Stein (*Surg. Gynec. Obstet.*, 1941, **72**, 129—149).—The gross anatomical and histological features of the corpora lutea are described in examples removed from 13 cases of intra-uterine pregnancy. There were structural alterations in the lutein cells and in the central cavity in corpora lutea from cases of ectopic pregnancy in which the embryo or fœtus had died. The corpus luteum grows rapidly between the 50th and 60th days of pregnancy, with a decrease in size later due to gradual obliteration of the central cavity and a decrease in thickness of the fibrous tissue surrounding the cavity. The theca lutein cells attain their greatest development at the 2nd—3rd month of pregnancy and disappear shortly after the obliteration of the central cavity; the granulosa-lutein cells persist throughout pregnancy. Both types of cell contain secretory granules, lipoid and chromidial substance while the granulosa-lutein cells contain vacuoles and colloid droplets in addition. Lipins and secretory granules are most numerous during the early months of pregnancy and gradually diminish while the colloid droplets are few in the early stages and increase during pregnancy. The secretion of the granulosa-lutein cells is non-stainable and appears as vacuoles chiefly in the cells nearest the central cavity; stagnation of the secretion leads to the formation of stainable colloid. Such colloid may also appear in 0·75% of the cells of a corpus luteum removed one day before menstruation. Ca is deposited in the colloid, which is regarded as a further sign of degeneration. Involution of the gravid corpus luteum may start as early as the second month. P. C. W.

**Differential growth in ovaries and genital tract near time of ovulation in rats treated with colchicine.**—See A., 1943, III, 620.

**Struma ovarii.** S. Sailer (*Amer. J. clin. Path.*, 1943, **13**, 271—277).—A report of 2 cases. (4 photomicrographs.) C. J. C. B.

**Relationship of progesterone to ovulation and luteinisation in persistent-œstrous rat.** J. W. Everett (*Endocrinol.*, 1943, **32**, 285—292).—A single injection of 0·5—1·0 mg. of progesterone to persistently œstrous rats caused dicœstrus lasting for 2—3 days. A second injection of progesterone, given on the 3rd day of dicœstrus or on the 1st day of œstrus following the artificial dicœstrus, produced corpora lutea. The second injection was less effective in this respect if given on the 1st or 2nd day of dicœstrus, and negligible in its effect when the new œstrus had persisted for longer than 8 days. G. P.

**Psychology of menstrual cycle.** S. Rosenzweig (*J. Clin. Endocrinol.*, 1943, **3**, 296—300). P. C. W.

**Emotional shock and endometrium.** A. A. Loeser (*Lancet*, 1943, **244**, 518—519).—4 women who had always menstruated regularly missed a period after an emotional shock. Endometrial biopsy a few weeks after the shock showed the stage of development that would normally have been reached at the time of shock, suggesting inhibition of release of the necessary hormones. Normal menstruation returned after 1 missed period. C. A. K.

**Metropathia hæmorrhagica.** B. Gilbert (*Clin. Proc.*, 1943, **2**, 97—111).—Discussion of cause and treatment. P. C. W.

**Cyclic vaginal response following administration of stilbœstrol in rats.** A. Coll and J. de L. Munoz (*Rev. Soc. argent. Biol.*, 1942, **18**, 233—237).—Injection of stilbœstrol dipropionate in castrated rats produces a cyclic vaginal response, œstrus alternating with dicœstrus. The optimal dosage is 0·3 μg. daily subcutaneously. With larger doses œstrus becomes longer and dicœstrus shorter. I. C.

**Supposed œstrogenic activity of cafesterol.** H. Hauptmann, J. França, and L. Bruck-Lacerda (*J. Amer. Chem. Soc.*, 1943, **65**, 993—994).—Coffee oil and various fractions therefrom, cafesterol, and oxcafestanediol-*A* have little, if any, œstrogenic activity. R. S. C.

**Implantation of solid pellets of œstrogen in treatment of menopausal symptoms.** G. H. Twombley and R. S. Millen (*Surg. Gynec. Obstet.*, 1941, **72**, 605—610).—Analysis of 46 cases. Œstrone pellets were ineffective while œstradiol pellets were effective. Dose was 1—3 25-mg. pellets. P. C. W.

**Experiences with stilbœstrol.** P. G. Fuerstner (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 742—745).—Analysis of the results of treatment in 64 cases of menopausal distress and for the suppression of lactation in 54 cases. P. C. W.

**Clinical investigation of stilbœstrol.** L. Felger and S. Gendel (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 746—752).—Favourable clinical report. P. C. W.

**Selective bismuth melanosis of female genital tract induced by treatment with sex hormones.** F. Sulman, S. Levy-Hochman, and H. G. Tietz (*Endocrinol.*, 1943, **32**, 293—297).—30—80% of female albino rats, treated simultaneously with injections of org. Bi compounds and œstrogens or chorionic gonadotropin, developed selective Bi melanosis of the vagina. Melanosis of the uterus occurred occasionally. Male rats treated with Bi and testosterone propionate rarely showed melanosis of preputial glands. G. P.

**Relative absorption rates of subcutaneous pellets of various crystalline compounds [sex steroids] in the rat.** T. R. Forbes (*Endocrinol.*, 1943, **32**, 282—284).—For the absorption of 90% of subcutaneously implanted pellets of cryst. substances the following periods were required: hexœstrol 54, diethylstilbœstrol dipropionate 140, diethylstilbœstrol methyl ether 145, diethylstilbœstrol methyl ether acetate 220, and testosterone dipropionate 272 days. Less than 30% of the pellets of anhydrohydroxyprogesterone were absorbed in 300 days. (Cf. A., 1941, III, 760; 1942, III, 749, 752.) G. P.

**Frozen plant juice as source of rabbit ovulating factor.** R. Bovasky and J. T. Bradbury (*Amer. J. Physiol.*, 1942, **137**, 637—639).—Oat juice retains its ovulating potency when stored in the frozen state for 21 months. There is a seasonal variation in the response of the rabbit to this plant substance. A simplified method is described for the extraction of the ovulation factor. M. W. G.

**Action of gonadotropic hormones on genital organs of dogs.** R. Sammartino and N. Arenas (*Rev. Soc. argent. Biol.*, 1942, **18**, 261—264).—The serum of a 1½-month pregnant mare produced in a normal bitch follicular and lutein phases, similar to the normal cycle. No reaction was obtained in a hypophysectomised hemicastrated bitch. Extracts of the serum were inactive in a castrated bitch but positive in the hypophysectomised. I. C.

**Preparation of chorionic gonadotropin by chromatographic adsorption.** P. A. Katzman, M. Godfrid, C. K. Cain, and E. A. Doisy (*J. Biol. Chem.*, 1943, **148**, 501—507).—Chorionic gonadotropin is prepared from pregnancy urine by the chromatographic adsorption of the active principle on permutit, eluting with an alcoholic solution of $NH_4$ acetate, and pptg. the hormone from the eluate with alcohol. $NH_4$ acetate increases the solubility of the hormone in alcohol. The purest preps. have a potency of 8500 i.u. per mg. J. E. P.

**Gonadotropic hormone in ovarian dysfunction.** V. L. Stevenson and L. A. Stevenson (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 723—726).—Favourable reports of the effect of pregnant mares' serum gonadotropin in the treatment of amenorrhœa, functional bleeding, and sterility. P. C. W.

**Reaction of chorionic gonadotropin with phenyl *iso*cyanate.** F. Bischoff (*Endocrinol.*, 1943, **32**, 260—262).—More than 90% of the activity of chorionic gonadotropin is destroyed by phenyl *iso*-cyanate (phenylcarbimide). G. P.

**Physical properties of allantoic and amniotic fluids of chick. I. Specific conductance. II. Hydrogen-ion concentration.** P. A. Walker (*J. Gen. Physiol.*, 1943, **26**, 495—502, 503—512).—I. By the 8th day of incubation, $\kappa$ of the allantoic fluid decreases, due to a decrease in concn. of $NH_3$ and increase in concn. of uric acid. The val. of $\kappa$ fluctuates from the 9th to the 13th day, but tends to increase slightly. Between the 13th and 16th days, $\kappa$ decreases and this is possibly connected with the change from mesonephritic to metanephritic excretion in the embryo; absorption of water from the contents of the allantoic sac begins at this period. From the 16th to the 19th day, $\kappa$ sharply decreases, due to absorption of water. The val. of $\kappa$ of the amniotic fluid is considerably higher than that of the allantoic fluid during the early period. From the 9th to the 14th day, there is practically no change in $\kappa$. There is a sudden and large decrease in $\kappa$ on the 14th and 15th days, possibly due to influx of protein from the albumin sac. At this period, there is max. total N in the fluid. After the 15th day $\kappa$ increases, and this coincides with the disappearance of this protein material.

II. The pH of the allantoic fluid from the 7th to the 13th day varies only slightly from 7·31 to 7·57. During the last week of incubation, the val. of pH suddenly decreases from 7·39 on the 13th to 5·87 on the 15th day. This is possibly due to an increase in concn. of $CO_2$, to a decrease in basic constituents, and to loss of Ca and $PO_4'''$. The average val. of pH of the amniotic fluid is slightly greater than neutrality, but on the 7th day it is slightly less than that of the allantoic fluid. From the 7th to the 15th day, pH gradually decreases and, by the 10th day, is slightly acid. This is possibly due to increased production of $CO_2$. After the 15th day, the pH rapidly becomes alkaline, attaining a val. of 7·9 on the 19th day. As the allantoic fluid is definitely acid at this period, the amnio-allantoic membrane cannot be permeable to $H^+$. This change in pH is possibly connected with absorption of certain materials from the amniotic fluid by the embryo (cf. Yamada, A., 1933, 734). J. N. A.

**Effect of pregneninolone on sexual development of *Rana pipiens*.** W. J. Eversole and S. A. D'Angelo (*J. exp. Zool.*, 1943, **92**, 215—227).—The administration of this steroid (ethinyltestosterone, anhydrohydroxyprogesterone) to tadpoles from early larval development, sexually undifferentiated stage, through metamorphosis is without effect on growth or differentiation of the larvæ, but markedly affects sexual development. All the animals so treated became males; genetic females cannot be identified. Pregneninolone given for 6 weeks to recently metamorphosed frogs does not alter sex ratio or gonad structure but markedly stimulates the oviduct and the caudal portion of the Wolffian duct. J. D. B.

**Modification of Bowman pregnancy test.** M. I. Mello (*Rev. Brasil. Biol.*, 1942, **2**, 343—348).—The following modifications of Bowman's pregnancy test are suggested: glacial acetic acid instead of sulphosalicylic acid; and I titration in Sörensen buffer solution (pH 5·9). Pregnancy urines gave min. vals. of 17·3 c.c. of 0·0005N-I and max. vals. of 72·6 c.c. Non-pregnancy urines reduce 7 c.c. of the same solution (average). The chemical test is positive in early cases of pregnancy (2 weeks after the last menstruation). I. C.

**New method for diagnosis of pregnancy.** M. I. Mello (*Rev. Brasil. Biol.*, 1943, **3**, 119—125).—The new test is based on the determination of gonadotropic hormone in urine: the hormone is adsorbed with kaolin in acid medium and then isolated by elution with 0·1N-NaOH. The solution is treated with Somogyi's Cu reagent and the excess not reduced is titrated with 0·005N-$Na_2S_2O_3$. Discrepancies between the classical biological tests and the new chemical test do not exceed 4·5%. I. C.

**Influence of race and sex on duration of gestation and weight at birth.** N. A. Anderson, E. W. Brown, and R. A. Lyon (*Amer. J. dis. Child.*, 1943, **65**, 523—534).—The mean birth wt. of 1731 white boys was 7 lb. 6 oz.; of 1658 white girls, 7 lb. 2 oz.; of 1144 Negro boys, 6 lb. 15 oz., and of 1153 Negro girls, 6 lb. 11 oz. The race and sex differences in birth wt. were of great statistical significance. The mean gestation period of white boys was 279, of white girls 279·9 days, of Negro boys 274·3 days. C. J. C. B.

**Therapy of seminal inadequacy. I. Use of pituitary, chorionic, and equine gonadotropins.** C. D. Davis, R. L. Pullen, J. H. M. Madden, and E. C. Hamblen (*J. Clin. Endocrinol.*, 1943, **3**, 268—273).—21 men with histories of 2—15 years of sterile mating were treated with one or more of the following courses of treatment which lasted 6 weeks with daily injections: (average total dose) chorionic gonadotropin 12,600 i.u., pituitary gonadotropin 10,800 r.u., equine gonadotropin 9700 i.u., or combinations of pituitary and chorionic gonadotropins, or of equine gonadotropin and testosterone. No significant improvement in seminal vals. took place though 4 wives became pregnant. P. C. W.

**Stimulation of mammary glands in hypophysectomised rats by œstrogen and testosterone.** S. L. Leonard (*Endocrinol.*, 1943, **32**, 229—237).—Œstradiol dipropionate stimulated growth of end buds of the mammary gland in hypophysectomised rats if the animals were younger than 70 days at time of hypophysectomy and if the treatment with the œstrogen was begun immediately after operation. The stimulation was of lesser degree than in normal animals and there was no development of acini. Testosterone propionate caused thickening of the ducts of the mammary gland in young hypophysectomised rats. G. P.

**Failing lactation.** M. Robinson (*Lancet*, 1943, **244**, 66—68).—A clinical study of causes of failure of lactation in 1100 cases is reported. The failure of milk secretion may concern water, solids, or both. Secretion is adversely affected by prematurity of the infant and by excessive muscular exercise. Most of the failures were related to the health of the mother. C. A. K.

**Reflex governing outflow of breast milk.** H. K. Waller (*Lancet*, 1943, **244**, 69—72).—Studies of the fat content of milk obtained from lactating mothers suggest that milk secretion is partly due to a reflex ("draught" reflex) initiated by the suckling infant. Reflex venous engorgement may compress the alveoli and force the milk into the larger ducts and sinuses. C. A. K.

**Influence of suckling stimulus on lactation.** H. L. Stewart and J. P. Pratt (*West. J. Surg. Obstet. Gynec.*, 1941, **49**, 98—103).—Records were taken of the milk output among 900 nursing mothers during the first 10 post-partum days. 87% showed an adequate milk supply for part-time or full breast nursing by the 5th day. Full breast nursing was present in 65% of those attempting to nurse with alternate breasts at each feed as compared with 74% of those nursing from both breasts at each feed. Primiparæ showed a higher incidence of full nursing from both breasts than multiparæ. There was deficient lactation at the 5th day in 14% of those attempting to nurse, and suckling from both breasts or other measures were unable to increase the milk supply by the 10th day. P. C. W.

**Gynæcomastia.** J. S. Richardson (*Lancet*, 1943, **244**, 304—305).—Case report. 125 mg. of testosterone propionate had no effect on the size of the breasts. C. A. K.

**Extracts of testis. I. Lipins from swine testis.** L. Ruzicka and V. Prelog (*Helv. Chim. Acta*, 1943, **26**, 975—994).—The fresh organs are extracted repeatedly with cold acetone, the extracts are evaporated to dryness in a vac.; the residue is treated with dry acetone which is subsequently removed. Alternatively the powdered and dried material is extracted with warm benzene, which is distilled under atm. pressure. The residue is dissolved in ether–acetone, frozen out at −75°, and filtered. The process is repeated on the filtrate. Solvent is removed from the filtrate finally in a vac., leaving the "benzene extract." The extracts are distributed between light petroleum and 70% ethyl alcohol, whereby the bulk of the fats and cholesterol remain in the petroleum. The material sol. in 70% ethyl alcohol is distributed between light petroleum and 50% methyl alcohol (the advantages of ethylene glycol are outweighed by the difficulty of its removal). The sol. material is treated in ether successively with aq. $NaHCO_3$, NaOH, and Girard reagent *T*. The product remaining in the light petroleum (after distribution between this solvent and 50% methyl alcohol) is hydrolysed and treated with $BaCl_2$, after which cholesterol is removed as far as possible as the acetate. The residue is separated into ketonic and non-ketonic portions which are subjected to chromatographic analysis. Thus are isolated $\Delta^5$-pregnen-3$\beta$-ol-20-one, *allo*pregnan-3$\beta$-ol-20-one, an unidentified *compound* C, $C_{21}H_{32}O_3$, m.p. 268°, softens at 257°, $[\alpha]_D$ −48° (±2°) to −39° (±2°) in pyridine [*oxime*, m.p. 238—239° (decomp.)], probably identical with testalolone, cholestan-3($\beta$) : 5 : 6-*trans*-triol, m.p. 236—241°, $[\alpha]_D$ +3·2°±2·1° in 96% of alcohol (diacetate, m.p. 166°), a *substance* E, $C_{13}H_{22}O_2N_2$, m.p. 270—280°, $[\alpha]_D$ +3·5°±3·5° in $CHCl_3$, and $\Delta^{3:5}$-cholestadien-7-one, m.p. 114·5°, $[\alpha]_D$ −305°±5° in $CHCl_3$. H. W.

**Action of androgens on male fish.** J. W. Burger (*Biol. Bull.*, 1942, **82**, 233—242).—Adult male *Fundulus*, normal and hypophysectomised, were injected with androgens. Testosterone propionate maintained or intensified the yellow coloration characteristic of the breeding male and stimulated the elaboration of the intra-testicular ducts, but had only a slight effect on spermatogenesis. Testosterone had a similar, but weaker, action on coloration and none on the testis. G. P. W.

**Partial masculinisation of ovary in rats treated with testosterone.** L. Marx (*J. exp. Zool.*, 1942, **91**, 365—371).—Ovaries of rats occasionally undergo partial masculinisation when given testosterone propionate (⅛ mg. for 22 days from the 8th post-natal day). The transformation of follicles into seminiferous tubules is described. Initial stages of this change were found to occur under various conditions but ovaries containing well differentiated testis-like areas were rare and occurred only among young females kept at elevated temp. J. D. B.

**Effect of testosterone on sexual pigmentation and sex characters of cricket frog (*Acris gryllus*).** B. Greenberg (*J. exp. Zool.*, 1942, **91**, 435—451).—Effects of implantation of pellets of testosterone propionate into males (before the breeding season) and females of this Anuran species resulted in development of male coloration in females and hypertrophy of vocal sacs and courtship behaviour in males. Gonoducts were hypertrophied, the Wolffian ducts of the female being least affected. J. D. B.

**Testosterone propionate in gynæcological and obstetric disorders.** T. E. Mandy and A. J. Mandy (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 604—608). P. C. W.

**Determination of concentration of spermatozoa in fowl and bull semen.** C. S. Shaffner and F. N. Andrews (*Anat. Rec.*, 1943, **86**, 99—107).—No significant variations in sperm cell vol. were observed. A 25% increase in vol. of solid material following centrifugation occurred when fowl semen was stored at 25°. No increase occurred when fowl semen was stored in ice water. Bull semen showed no change in vol. after centrifugation. The average coeff. of variability of the determination of sperm cell vol. and the coeff. of correlation of the hæmocytometer concn. count and centrifugation vol. determination for fowl and bull semen are given. W. F. H.

**Inhibition of sperm respiration and reversibility of effects of metabolic inhibitors.** H. A. Lardy and P. H. Phillips (*J. Biol. Chem.*, 1943, **148**, 333—341).—The reversibility of the effects of various inhibitors on ejaculated bull sperm was studied by removing the sperm from the inhibiting medium after a period of time and studying its metabolism in a fresh medium. Malonate, benzoate, F′, and quinol reversibly inhibited both respiration and motility. Hydroxyquinoline sulphate reversibly inhibited respiration but not motility. Indole, maleate, $SeO_3''$, $AsO_3'''$, *dl*-glyceraldehyde, and benzoquinone inhibited both respiration and motility irreversibly. CN′ and $N_3'$ inhibited respiration reversibly (only partially in the case of $N_3'$). Some of the substances inhibiting both respiration and motility were without effect on motility in presence of glucose, confirming the ability of oxidative and glycolytic processes separately to furnish energy for motility. The depressing effect of $N_3'$ on motility, however, was enhanced in presence of glucose. *p*-Phenylenediamine increased respiration to the normal rate for ejaculated spermatozoa in one specimen of sperm having a low respiration similar to that of epididymal spermatozoa. E. C. W.

**Inhibition of sperm glycolysis and reversibility of effects of metabolic inhibitors.** H. A. Lardy and P. H. Phillips (*J. Biol. Chem.*, 1943, **148**, 343—347; cf. preceding abstract).—The effect of various metabolic inhibitors on glycolysis of bull sperm was studied. Malonate and benzoate were almost without effect. CN′, maleate, and quinol stimulated glycolysis, reversibly only in the case of CN′. Iodoacetate, *dl*-glyceraldehyde, and $N_3'$ largely inhibited glycolysis, reversibly only in the case of $N_3'$. Benzoquinone completely inhibited glycolysis reversibly, as did F′. Pyruvate prevented inhibition by F′ of glycolysis, but not of motility. E. C. W.

**Behaviour of nurse cells and sperms in semen of *Littorina*.** T. M. Woodard (*Biol. Bull.*, 1942, **82**, 461—466). G. P. W.

**Factors controlling male activity in newt.** J. D. Ifft (*Biol. Bull.*, 1942, **83**, 111—128).—In *Triturus viridescens*, spermatogenesis and sperm discharge can be controlled at all seasons by varying the temp., which is apparently the factor responsible for the annual reproductive cycle. Variations in amount of illumination or of food had no effect. G. P. W.

**Familial eunuchoid gigantism.** K. Mansbacher (*J. Clin. Endocrinol.*, 1943, **3**, 257—260).—The cases of 2 brothers are reported. One aged 19 showed the normal symptoms of eunuchoid gigantism while his brother aged 10 showed the same physical characteristics of skeletal growth with normal sexual development. P. C. W.

**Cryptorchidism.** J. H. Lapin, W. Klein, and A. Goldman (*J. Pediat.*, 1943, **22**, 175—188).—Only 6 of 39 cases given endocrine treatment and followed for 2—9 years showed testicular descent. C. J. C. B.

**Orchidectomy for carcinoma of prostate. Orchidectomy for prostatic carcinoma.**—See A., 1943, III, 659.

**Hormonal therapy of prostatic carcinoma. Histological changes in carcinoma of prostate following resection and use of stilbœstrol.**—See A., 1943, III, 659.

## XIII.—DIGESTIVE SYSTEM.

**Gastric secretory depressant in gastric juice.** V. B. Scott, R. Moe, and A. Brunschwig (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 45—46).—Gray's method (A., 1943, III, 176) for extraction of urogastrone from urine does not extract the gastric secretory depressant from an alcoholic ppt. of gastric juice (cf. A., 1941, III, 591). V. J. W.

**Determination of peptic activity.** W. R. Hackett (*Lancet*, 1943, **244**, 430).—A range of dilutions of gastric juice in 0·4% HCl is incubated at 37° for 30 min. with a solution of serum-albumin of known concn. Residual albumin is determined turbidimetrically in the 1st dilution in which it appears and from this the quantity of albumin which 1 c.c. of gastric juice is theoretically capable of hydrolysing can be calc. C. A. K.

**Spontaneous motility of pyloric sphincter.** J. P. Quigley and M. R. Read (*Amer. J. Physiol.*, 1942, **137**, 234—237).—Studies of pyloric sphincter motility made with the pyloric diagraph (described fully) in unanæsthetised dogs show that normally the quiescent sphincter is continuously in the relaxed state. The active sphincter exhibits cyclic activity and is relaxed during more than half of each cycle. Sphincter opening is a characteristic phase of the cyclic activity and not the result of a passive stretching produced by material propelled from an adjacent portion of the gut. M. W. G.

**Motility of fasting stomach.** W. F. Anderson (*Lancet*, 1943, **244**, 40—42).—Gastric motility was recorded by a balloon method. Healthy subjects showed active contractions, tonus rhythm, or relative quiescence. There was no relation between acid-secreting power and motility of the fasting stomach. 10 patients with acute duodenal ulcer showed increased gastric tone with stronger contractions which were liable to become tetanic. In 2 patients with gastric carcinoma contractions were weaker and less frequent. In normal subjects ingestion of cold water inhibited gastric contractions but in 8 cases with peptic ulceration there was either no inhibition or actual stimulation of motility. C. A. K.

**Genesis of peptic ulcer in dogs after ligating common bile ducts.** J. L. Carr and F. S. Foote (*Surg. Gynec. Obstet.*, 1941, **72**, 198—201).—Ligation of the common bile duct caused the appearance of gastric or duodenal ulcers in 11 of 20 dogs after 3—8 weeks. The ulcers were started by physiological digestion of spontaneous hæmorrhages occurring in the mucosa due to the hæmorrhagic diathesis of obstructive jaundice. Once the continuity of the mucosa was interrupted the ulcers followed a normal course. P. C. W.

**Significance of hæmorrhagic pigment spots observed by gastroscopy.** J. M. Ruffin and I. W. Brown (*Amer. J. digest. Dis.*, 1943, **10**, 60—63).—These spots may occur in healthy individuals and can be produced by aspiration of the stomach. They probably have no clinical significance. N. F. M.

**Gastrogenic polyneuritis and tetany.** H. Roesli (*Schweiz. med. Wschr.*, 1942, **72**, 944—945).—Report of a case suffering from severe vomiting, alternating diarrhœa and constipation, polyneuritis, and tetany. The patient recovered after treatment with vitamins-$B_1$ and -$B_2$ and Ca. A. S.

**Generalised pruritis due to carcinoma of stomach and cured by gastrectomy.** L. Cardon (*Amer. J. digest. Dis.*, 1943, **10**, 63—65).—A case report. N. F. M.

**Changes in acinar cells of pancreas in response to presence of peptone in small intestine.** A. J. Ramsay, J. E. Thomas, and J. O. Crider (*Anat. Rec.*, 1943, **86**, 87—98).—Peptone stimulation depletes the acinar cells of zymogen granules and secretion accumulates in the ducts. In the vicinity of islets acinar cells retain their granules. The changes described resembled those observed during stimulation of the vagus nerves and did not occur during peptone stimulation in vagotomised animals. It is concluded that peptone stimulates the pancreas through a nervous mechanism dependent on the integrity of the vagus nerves. W. F. H.

**Stability of aminopolypeptidase in presence of pancreatic enzymes.**—See A., 1943, III, 682.

**Duodenal tube biliary tract drainage.** B. B. V. Lyon (*Amer. J. digest. Dis.*, 1943, **10**, 69—78).—A 25-year "follow-up" report on the first person to undergo treatment by this method. (A clinical lecture.) N. F. M.

**Rate of glucose absorption from intestine of diabetic rats.** F. Pauls and D. R. Drury (*Amer. J. Physiol.*, 1942, **137**, 242—245).—Diabetic rats store sugar with difficulty; on a high-carbohydrate diet they consume large amounts of sugar because of the loss in the urine. The rate of glucose absorption in diabetic rats is high after voluntary glucose feeding and not low as after forced glucose feeding. M. W. G.

**Relationship between roentgenographic abnormalities of gall bladder and constipation.** G. H. Laing, J. M. Beazell, and A. C. Ivy (*Amer. J. digest. Dis.*, 1943, **10**, 50—51).—In 372 patients undergoing cholecystography, there was a correlation between "constipation" and delayed emptying of the gall bladder, but none between constipation and the presence of gallstones. Colonic disturbance may cause delayed gall-bladder emptying. N. F. W.

**Phagocytosis in appendix of rabbit.** F. Baker and J. Enticknap (*Nature*, 1943, **151**, 532—533; cf. McEwen, *Brit. Med. J.*, 1904, II, 873).—Macrophages have been found ingesting iodophil bacteria, which form the greater part of the micro-organisms of cæcum. E. R. S.

**Chemotherapy of chronic ulcerative colitis.** M. A. Mills and T. T. Mackie (*Amer. J. digest. Dis.*, 1943, **10**, 55—57).—Improvement was noted in 78% of 109 cases treated with sulphathiazole, sulphaguanidine, and sulphadiazine, the last being preferred. N. F. M.

**[Intestinal] giardiasis with unusual clinical findings.** P. B. Welch (*Amer. J. digest. Dis.*, 1943, **10**, 52—53).—Of the 13 cases, all had radiologic evidence of duodenal or pyloric irritation. Eosinophilia occurred in 7 cases, and was abolished by atabrine therapy in 5 of these. N. F. M.

**Nutrient enema.** J. W. A. Mackenzie (*Arch. Dis. Childh.*, 1943, **18**, 22—27).—Glucose, NaCl, and predigested protein are absorbed from solutions introduced into the large intestine in considerable quantities. There is great variation in the amount of glucose absorbed. Absorption of NaCl by rectum is almost as great as by mouth. C. J. C. B.

## XIV.—LIVER AND BILE.

**Some aspects of liver.** S. Freeman and F. Grodins (*Int. Abst. Surg.*, 1941, March, 209—216).—A review. P. C. W.

**Liver function tests in aged.** H. A. Rafsky and B. Newman (*Amer. J. digest. Dis.*, 1943, **10**, 66—68).—Serum-cholesterol partition, bromsulphalein excretion, kephalin flocculation, and hippuric acid synthesis were studied in 50 normal subjects over 60 years of age. In 43 subjects at least one of the tests gave abnormal results. N. F. M.

**Mechanism of carbon dioxide fixation in cell-free extracts of pigeon liver.**—See A., 1943, III, 503.

**Components of vitamin-$B_2$ complex in Cohn liver extract in relation to sprue.**—See A., 1943, III, 500.

**Changes in liver of guinea-pigs deficient in vitamin-*C*.**—See A., 1943, III, 578.

**Enzymes of isolated cell nuclei of rat's liver.**—See A., 1943, III, 517.

**Liver-catalase.**—See A., 1943, III, 519.

**Liver-catalase activity of tumour-bearing animals.**—See A., 1943, III, 490.

**Fatty acid oxidation by liver enzymes.**—See A., 1943, III, 597.

**Hepatic cell mitochondria in fatty liver produced by high-sugar diet.**—See A., 1943, III, 544.

**Effects of excess dietary cysteic acid, *dl*-methionine, and taurine on rat liver.**—See A., 1943, III, 495.

**Effect of alcohol in experimental liver cirrhosis.**—See A., 1943, III, 589.

**Cirrhosis of liver : lipotropic action of parenterally administered amino-acids.** I. D. Fagin, M. Sahyun, and R. W. Pagel (*J. Lab. clin. Med.*, 1943, **28**, 987—993).—Liver specimens from patients with cirrhosis of the liver treated with amino-acids contained a greater % of protein and a smaller % of fat than specimens from patients who had not received amino-acids. These results indicate a lipotropic activity of the amino-acid mixture, due to their methionine content. The % of liver-fat and liver-protein varied inversely. C. J. C. B.

**Homologous serum jaundice.** Ministry of Health (*Lancet*, 1943, **244**, 83—88).—A review of the occurrence of jaundice following administration of measles serum, yellow fever vaccine, serum transfusions, etc. Case reports are given. C. A. K.

**Antitoxic principle in liver extracts.** G. G. Villela (*Rev. Brasil. Biol.*, 1942, **2**, 365—369).—An antitoxic principle has been obtained from aq. extracts of liver by adsorption with norit. The prep. protects mice against experimental intoxications with $CCl_4$ and arsphenamine. I. C.

**Antitoxic liver extracts and liver-glutathione in experimental toxicosis.** G. G. Villela (*Rev. Brasil. Biol.*, 1943, **3**, 99—103).—Experimental intoxications with $CCl_4$ and arsphenamine in mice decrease the total glutathione content of liver. A liver extract, which protects mice against experimental $CHCl_3$, $CCl_4$, and neoarsphenamine intoxications, maintains the glutathione content of liver at the normal level. I. C.

**Carcinogenic extract from human bile and gall-bladder.**—See A., 1943, III, 488.

**Distribution and excretion by the bile of iron, cobalt, and manganese.**—See A., 1943, III, 506.

## XV.—KIDNEY AND URINE.

**Changes which alter renal osmotic work.** J. D. Newburgh (*J. clin. Invest.*, 1943, **22**, 439—445).—A highly mathematical paper. The equation which gives the amount of work required for the idealised formation of urine has been examined to determine the amounts of excretory water and solids which will give the least work. The effect of changes in blood concns. on osmotic work is determined. The changes necessitated by limitation in osmotic work are predicted and examples given where these predictions agree with clinical findings. These considerations suggest certain therapeutic procedures, the validity of which can be determined only by actual trial. C. J. C. B.

**Simplified technique for measuring renal blood flow and tubular excretory mass.** T. Findley, J. C. Edwards, E. Clinton, and H. L. White (*J. Lab. clin. Med.*, 1943, **28**, 916—920).—A line chart is presented which permits prediction of diodrast-$T_m$ from a single urine specimen. When this is combined with the subcutaneous method for determining renal plasma flow and glomerular filtration rate, the entire procedure is greatly simplified. C. J. C. B.

**Renal displacement.**—See A., 1943, III, 449.

**The nephron as affected by mercury.** J. G. Edwards (*Amer. J. Path.*, 1942, **18**, 1011—1020).—59 rabbits and guinea-pigs and 24 frogs were injected intraperitoneally with $HgCl_2$. Only the proximal convolution was damaged, showing cell necrosis. Hypertrophy and/or hyperplasia of half or more of the outer layer of the glomerular capsule commonly occurred except in rabbits. (12 photomicrographs.) C. J. C. B.

**Diodrast and inulin clearances in nephrotic children with supernormal urea clearances.** K. Emerson, jun., and V. P. Dole (*J. clin. Invest.*, 1943, **22**, 447—450).—The elevated urea clearance in 4 nephrotic children was associated with increase of both inulin and diodrast clearances. C. J. C. B.

**Serum concentration and renal clearance of potassium in severe renal insufficiency in man.** N. M. Keith, H. E. King, and A. E. Osterberg (*Arch. intern. Med.*, 1943, **71**, 675—701).—When severe renal dysfunction develops in acute or subacute nephritis, in the terminal stage of chronic nephritis, or in marked congestion of the kidney, K is more slowly excreted into the urine and its blood concn. may be doubled. Several factors, both renal and prerenal, hinder further increase in serum-K concn. in man, less so in experimental animals. A sustained increase in serum-K in renal disease is of serious prognostic import and should forbid the therapeutic use of K salt. (10 photomicrographs.) C. J. C. B.

**Use of sulphonamides in renal insufficiency.**—See A., 1943, III, 582.

**Nature of renal lesion with sulphonamides and its prevention with urea.**—See A., 1943, III, 584.

**Changes in kidneys of guinea-pigs deficient in vitamin-*C*.**—See A., 1943, III, 578.

**Non-oxidase nature of kidney "laccase."**—See A., 1943, III, 597.

**Renin and production of cardiac and gastro-intestinal hæmorrhages and necroses [in dog]. Effect of angiotonin and renin on glomerular circulation in frog kidney.**—See A., 1943, III, 550.

**Uretero-intestinal implantation. IV. Ureterovesical reimplantation in dog.** H. M. Weyrauch, R. A. Burns, R. A. Peterfy, and F. Hinman (*Surg. Gynec. Obstet.*, 1941, **72**, 192—197).—There was little ascending infection and no obstruction among 6 dogs after a one-stage ureterovesical reimplantation. The infection and obstruction following uretero-intestinal implantation performed by the same technique must be due to the infective sources present in the large intestine. P. C. W.

**Urea clearance and diuresis in man.** R. Dominguez and E. Pomerene (*J. clin. Invest.*, 1943, **22**, 1—9).—The urea clearance in man rises continuously with diuresis at all levels of urine flow in health and renal disease (also after renal denervation). The relation between the clearance and diuresis is represented by $C = A(1 - e^{-kv}) + bv$ ($C$ = clearance, $v$ = urine flow in c.c. per min., and $A$, $k$, and $b$ are consts.). This equation is fitted to the data of 1 normal subject and of 3 patients with nephrosclerosis. C. J. C. B.

**Alkaptonuria with hyperuricæmia.**—See A., 1943, III, 506.

**Significance of glycosuria.** I. A. Mirsky and N. Nelson (*Arch. intern. Med.*, 1943, **71**, 827—835). C. J. C. B.

**Effect of glucose feeding on quantitative relationship between β-hydroxybutyric acid and acetoacetic acid in blood and urine.**—See A., 1943, III, 505.

**Quantitative study of urinary excretion of hypophyseal gonadotropin, œstrogen, and neutral 17-ketosteroids of normal men.** S. C. Werner (*J. clin. Invest.*, 1943, **22**, 395—401).—The urinary output of hypophyseal gonadotropin, œstrogen, and 17-ketosteroids of 4 normal men was determined over 3 months. Marked fluctuations were noted in the outputs of gonadotropin over long periods, and of œstrogen and 17-ketosteroid excretion over shorter periods. C. J. C. B.

**Urinary ketosteroids.** G. Pincus (*J. Clin. Endocrinol.*, 1943, **3**, 301—303).—Report of a conference. P. C. W.

**Excretion of pituitary hormones in urine.**—See A., 1943, III, 561.

**Determination of bile-pigment in urine.** K. Singer and R. Kubin (*J. Lab. clin. Med.*, 1943, **28**, 1042—1049).—A direct, rapid, photoelectric method, using Hammarsten's oxidation reaction, is described. By means of an appropriate filter and by employing the diluted, acidified but untreated urine as a standard, the otherwise necessary preliminary pptn. of the bile-pigments can be eliminated. C. J. C. B.

**Determination of acetone in urine.** A. Castiglioni (*Z. anal. Chem.*, 1940, **120**, 166—167).—Cotton-wool treated with 2 drops of a freshly prepared 1% solution of furfuraldehyde in 95% alcohol and 1 drop of 10% aq. NaOH forms the plug of a test-tube containing 2 c.c. of the urine to be tested. After boiling and keeping for 5 min. the plug is inserted in another test-tube in which 2 c.c. of conc. HCl are heated until HCl escapes through the wadding. If acetone (0·0001% or more) is present a bright red colour appears. F. N.

**Rapid simple technique for detection of acid-fast bacilli in urine.**—See A., 1943, III, 522.

**Routine analysis of urinary calculi.** J. H. Winer and M. R. Mattice (*J. Lab. clin. Med.*, 1943, **28**, 898—904).—A rapid, simple method using spot tests is described. C. J. C. B.

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

**Blast injury of lungs and brain.** P. B. Ascroft (*Lancet*, 1943, **244**, 234—235).—Case report with autopsy. C. A. K.

**Blast injuries.** J. V. Wilson and R. E. Tunbridge (*Lancet*, 1943, **244**, 257—261).—Pathological changes in 12 fatal cases are described in detail. C. A. K.

**Occurrence of crystals in skin of Amphipoda.** D. M. Reid (*Nature*, 1943, **151**, 504—505).—The $CaCO_3$ crystals in the skins of Amphipoda were examined in polarised light. It was possible to distinguish some families by the patterns produced, but not species. E. R. S.

**Reactivity of sheeps' wool keratin.** A. Schöberl and P. Rambacher (*Biochem. Z.*, 1940, **306**, 269—295).—The total S, cystine, and degree of injury of sheeps' wool from various parts of the animal and from various sources fall within the respective limits : 2·90—3·99, 7·75—11·96, and 0—32%. The back wool of Argentine sheep has a very low degree of injury (0—15%), as judged by the reactivity of the ·S·S· linking. Hydrolysis with 6N-$H_2SO_4$ at 125° liberates increasing amounts of amino-N with increased time of hydrolysis

(1—18 hr.), but the % of free cystine remains const. after 1 hr. This contrasts with the slight increase of free cystine with time of hydrolysis of edestin and albumin. Treatment with N-NaOH at 50° for ¼—10 hr. results in increasing amounts of $NH_3$ (82% of theoretical in 10 hr.) and free SH groups (12·1% in 10 hr.), with simultaneous reduction in ·S·S· linkings. Treatment with water alone at 120° for 24 hr. gives only 6·45% of the theoretical of $H_2S$ and 2·36% of SH groups. Elasticity of the fibre is markedly reduced by prolonged treatment with water. P. G. M.

**Factors influencing upstream immigration of pink salmon** (*Oncorhynchus gorbuscha*). F. A. Davidson, E. Vaughan, S. J. Hutchinson, and A. L. Pritchard (*Ecology*, 1943, **24**, 149—168).—At several localities upstream movements of pink salmon appear to be unrelated to water temp. and $CO_2$ concn. When sexually immature they may not attempt to enter the river if the water level is low whereas when sexually mature they attempt to migrate upstream whatever the water level. L. G. G. W.

**Pigment of beeswax.** R. Barré (*Rev. Canad. Biol.*, 1942, **1**, 485—490).—The presence of chrysin in beeswax is established by its conversion into a red pyranol by the action of Mg phenyl bromide. West American beeswaxes are richer than Eastern in chrysin, which is present only in traces in highly coloured, tropical beeswaxes. H. W.

## XVII.—TUMOURS.

**Skin carcinogenesis by a single application of 20-methylcholanthrene.** W. Cramer and R. E. Stowell (*Cancer Res.*, 1943, **3**, 36—42; cf. A, 1942, III, 396, 613).—Methylcholanthrene (0·6% solution in benzene) was applied to the unepilated skin of 14 mice of the Swiss strain by a single brush stroke which delivered a dose of about 0·3 mg. Six mice developed malignant tumours. The bearing of this result on the mode of action of carcinogens is discussed. F. L. W.

**Experimental gastric tumours in mice.** V. J. Collins, W. U. Gardner, and L. C. Strong (*Cancer Res.*, 1943, **3**, 29—35).—Benzpyrene dissolved in sesame oil was given orally to 133 mice of the *C3H*, *NH*, *C57*, *A*, and *CHI* strains. All tumours involving the stomach arose in the forestomach and were squamous-celled. The highest incidence of gastric papillomas and carcinomas occurred in mice of the *C3H* strain. A high incidence of gastric tumours occurred in *C57* and *NH* mice and a low incidence in the *CHI* and *A* strains. No sexual difference was seen in *C3H* mice. The incidence of carcinomas was higher in ovariectomised *C3H* and *NH* mice than in intact mice. 22 carcinomas and 69 papillomas occurred. Gastric carcinoma from a *C57* mouse was transplanted to 3 normal mice of the same strain. F. L. W.

**Carcinogenesis after multiple irritation.** J. Lauridsen and H. E. Eggers (*Cancer Res.*, 1943, **3**, 43—46).—In mice of two strains treated with dibenzanthracene tumours appeared more rapidly in mice which were repeatedly lightly cauterised. The effect was more marked in the strain with low spontaneous incidence of cancer. On the other hand more sarcomas appeared in the strain of high spontaneous cancer incidence. F. L. W.

**Inheritance of susceptibility to tumours induced in mice. I. Tumours induced by methylcholanthrene in five inbred strains of mice.** W. J. Burdette and L. C. Strong (*Cancer Res.*, 1943, **3**, 13—20).—Differences in the time of appearance of tumours induced by a single injection of methylcholanthrene (1 mg. subcutaneously at 60 days of age) were found in five inbred strains of mice (*CBA*, *C3H*, *CHI*, *JK*, and *NH*). The predominant tumour type was spindle-cell sarcoma in some strains and rhabdomyosarcoma in others. The survival time of mice developing tumours did not parallel the susceptibility of the mice to induced tumours. F. L. W.

**Genetic analysis of induction of tumours by methylcholanthrene. IV. Probable remote induction of various types of gastric lesions.** L. C. Strong, V. J. Collins, and E. A. Durand (*Cancer Res.*, 1943, **3**, 21—28; cf. A., 1940, III, 853; 1942, III, 147, 396).—Gastric lesions including glandular hyperplasia, adenoma, adenocarcinoma, squamous-cell papilloma, and squamous-cell carcinoma were obtained in mice of the *NHO* strain after subcutaneous injection of 1 mg. of methylcholanthrene at 60 days of age. The carcinogen may have had some influence on the appearance of these lesions. These lesions occurred in mice that had also developed adenocarcinoma of the lung or spindle-cell sarcoma, or quite independently of any other form of neoplasia. F. L. W.

**Effects of a water-soluble carcinogen on early frog development.** J. B. Briggs and R. W. Briggs (*Cancer Res.*, 1943, **3**, 1—12).—Frog eggs were reared in solutions of the Na salts of the *endo*-succinates of 1 : 2 : 5 : 6-dibenzanthracene and of methylcholanthrene, the exposure beginning 1 hr. after fertilisation. Retardation of developmental rate, degeneration of embryos, and differential retardation of capacity for muscular movement were observed. There was no stimulative effect or any effect on the morphological organisation of the embryo. Control experiments with miscellaneous substances indicated that the effects were sp. to carcinogens. F. L. W.

**Quantitative analysis of dose–response data obtained with three carcinogenic hydrocarbons in strain *C3H* male mice.** W. R. Bryan and M. B. Shimkin (*J. Nat. Cancer Inst.*, 1943, **3**, 503—531).—1004 mice were injected with serial twofold doses of methylcholanthrene (0·00025—1·0 mg.), 3 : 4-benzpyrene (0·002—8 mg.), and 1 : 2 : 5 : 6-dibenzanthracene (0·002—8 mg.). The tumour incidence and latent periods of induction were analysed by the methods developed by Gaddum, Bliss, and Irwin and the results are shown in 27 graphs. The median tumour doses (T.D. 50) were: dibenzanthracene 0·016 mg., methylcholanthrene 0·021 mg., and benzpyrene 0·101 mg. The corresponding T.D. 95 doses were 0·084, 0·096, and 0·875 mg. The probit vals. corresponding to the tumour incidence were proportional to the logarithms of the doses injected. The standard deviations of the logarithms of the individual effective doses ($\lambda$) were $0·44 \pm 0·05$ for methylcholanthrene, $0·40 \pm 0·06$ for dibenzanthracene, and 0·55 for benzpyrene. The min. latent periods were 3·72 months (days ÷ 30) for dibenzanthracene, 2·48 for methylcholanthrene, and 3·02 for benzpyrene. The sp. induction times (the mean latent periods on the T.D. 50 dose) were 6·79, 5·14, and 5·23 months. Dibenzanthracene is thus most effective in that small doses induce tumours but treatment period is relatively long, when considered in carcinogenic units (*i.e.*, the T.D. 50 dose). E. B.

**Quantitative experiments on the production of subcutaneous tumours in strain *A* mice with marginal doses of 3 : 4-benzpyrene.** J. Leiter and M. J. Shear (*J. Nat. Cancer Inst.*, 1943, **3**, 455—477).—Doses of 0·05—0·20 mg. of benzpyrene in various fat solvents were injected into groups of 50 mice. When filtered lard was used there was considerable variation in the tumour incidence. The residue from filtration of lard contained some factors inhibiting tumour induction. The retarding factor appeared to be associated with saturated glycerides of high mol. wt. Tristearin, tripalmitin, and tricaprylin were also used and tricaprylin was found most suitable for experiments on tumour induction. E. B.

**Changes induced in a strain of fibroblasts from a strain *C3H* mouse by the action of methylcholanthrene.** W. R. Earle (*J. Nat. Cancer Inst.*, 1943, **3**, 555—558).—Fibroblasts from adipose and subcutaneous tissue were grown in the presence of 1 $\mu$g. of methylcholanthrene per c.c. of culture medium. The carcinogen reduced the rate of growth of the cultures. The first morphological change occurred after 40 days' cultivation, when the terminal processes of the cells became shorter and the cells became somewhat amœboid in appearance. The cell cytoplasm became granular. The induced cell changes remained when the carcinogen was removed. The control cultures showed similar changes after very prolonged cultivation. Injection of the changed cultures in the *C3H* mice induced tumours in 7·6—86% of such mice. The tumours could be transplanted into other mice (see following abstract). E. B.

**Morphology of sarcomas derived from fibroblasts previously treated with methylcholanthrene *in vitro*.** A. Nettleship (*J. Nat. Cancer Inst.*, 1943, **3**, 559—561).—The tumours were spindle-celled sarcomas and on the average were not less than 1 c.c. in vol. The tumours derived from control cultures, and cultures exposed to the carcinogen for short periods (6 days), showed least invasiveness and irregularity of cell pattern. These changes increased as the time of exposure of cultures increased (up to 406 days). The occurrence of metastases in the lungs and mitoses in the tumours was maximal in the tumours derived from a culture exposed for 111 days. E. B.

**Wave-length dependence of tumour induction by ultra-violet radiation.** H. F. Blum (*J. Nat. Cancer Inst.*, 1943, **3**, 533—537).—If mice are irradiated with ultra-violet light passing Pyrex and Corex D filters, the radiation penetrating is altered by absorption in the epidermis by protein or nucleic acids. E. B.

**Effect of intensity on tumour induction by ultra-violet radiation.** H. F. Blum (*J. Nat. Cancer Inst.*, 1943, **3**, 539—543).—Male mice of strain *A* were irradiated at different intensities. With intensities above $2 \times 10^{-5}$ erg per sq. cm. the tumour-inducing effect is independent of the intensity of irradiation. Below that intensity the carcinogenic action falls rapidly. E. B.

**Neoplasms and other lesions of the eye induced by ultra-violet radiation in strain *A* mice.** S. W. Lippincott and H. F. Blum (*J. Nat. Cancer Inst.*, 1943, **3**, 545—554).—Pathological changes were mainly in the cornea and consisted in vascularisation and hyperplasia of epithelial and connective tissue elements. Gross tumours of the eye occurred in 5% of exposed mice. Most of the tumours were fibrosarcomas, and hæmangio-endotheliomas of the substantia propria. No actual carcinomas were found. E. B.

**Some fundamental aspects of tumour development illustrated by studies with ultra-violet radiation.** H. F. Blum (*J. Nat. Cancer Inst.*, 1943, **3**, 569—581).—Groups of male strain *A* mice were exposed to ultra-violet light at regular intervals and the tumour development times ($t_d$) noted. In some of the groups irradiation was interrupted at 53, 68, and 117 days, *i.e.*, before tumours occurred. The logarithms of the individual $t_d$ for any particular exposure time were proportional to the probit of the tumour incidence. The incidence was less and the $t_d$ longer when less irradiation was given.

The changes leading to tumour formation must occur long before the tumour is observed. The development of tumours is discussed. E. B.

**Comparative effects of œstrogen, testosterone, and progesterone on benign mammary tumours of the rat.** J. Heiman (*Cancer Res.*, 1943, **3**, 65—68).—Progesterone inhibited the growth of the adenomatous portion of spontaneous rat mammary fibroadenoma. The % of takes was reduced. 18 mg. of progesterone inhibited the stimulating effect of 1 mg. of œstrone but not that of 2·5 mg. Progesterone alone did not inhibit the growth of fibromas in castrated females or in normal males; in castrated males the growth was inhibited. Progesterone in combination with testosterone reduced the % of takes from 66·6 to 8·3. Large doses of progesterone or testosterone neutralised the stimulating effect of œstrogen on growing fibroadenoma. Rat fibroma, myxoma, and sarcoma were not inhibited by progesterone. The growth of fibroadenomas in pregnant rats was not inhibited by progesterone. F. L. W.

**Reactions of hybrid and parabiotic pseudo-hybrid mice to inoculations of tumour *C*198.** A. M. Cloudman (*Cancer Res.*, 1943, **3**, 47—52).—Parabiosis between different stocks of mice is possible. The normal resistance of *C*57 black mice to implants of tumour *C*198 can be changed to susceptibility by parabiosis of these mice with mice of the (susceptible) leaden strain. F. L. W.

**Transplantable squamous-cell carcinoma in the rabbit.** H. S. N. Greene and W. H. Brown (*Cancer Res.*, 1943, **3**, 53—64).—The course of a squamous-cell carcinoma in the skin of a rabbit was followed from inception to death. The primary tumour conformed to a type known in man as spindle-cell epidermoid carcinoma. The tumour was successfully transplanted. F. L. W.

**Spontaneous mouse rhabdomyosarcoma.** A. Nettleship (*J. Nat. Cancer Inst.*, 1943, **3**, 563—568).—The tumour occurred in the thigh of a 3-month-old female mouse of strain *C*. The cells of the tumour were smaller than normal muscle cells, but some of them contained striations. No nerve cells were present. The tumour grew on transplanting into other mice but no metastases have been seen. E. B.

**Effect of a diet relatively low in cystine on the production of spontaneous mammary-gland tumours in strain *C3H* female mice.** J. White and H. B. Andervont (*J. Nat. Cancer Inst.*, 1943, **3**, 449—454).—None of a group of 45 virgin female *C3H* mice, fed on a low-cystine diet, developed tumours, while 97·5% of the controls developed tumours. The mice on low-cystine diet had irregular œstrous cycles and were very small. E. B.

**Comparison of dosage of methylcholanthrene on the production of leukæmia and sclerotic lesions in strain Dilute Brown mice on a restricted cystine diet.** J. White, G. B. Mider, and W. E. Heston (*J. Nat. Cancer Inst.*, 1943, **3**, 453—454).—Three groups of mice, (1) on dog chow, (2) on high-cystine diet, and (3) on low-cystine diet, were painted with methylcholanthrene. The mice of group (3) had a lower incidence of leukæmia but developed sclerosis of the aorta. E. B.

**Growth rate and number of spontaneous mammary carcinomas and riboflavin concentration of liver, muscle, and tumour of *C3H* mice as influenced by dietary riboflavin.** H. P. Morris and W. v. B. Robertson (*J. Nat. Cancer Inst.*, 1943, **3**, 479—489).—Mice were maintained on normal diet, and on diets rich and deficient in riboflavin. The absence of riboflavin did not reduce the average food intake but it caused reduction in body growth rate and in the growth rate of spontaneous mammary tumours. The addition of riboflavin to the diet increased the average no. of tumours occurring per mouse. The riboflavin content of the liver and tumour tissue fell to about half the normal val. while the content in the muscles was maintained at ⅔ of the normal val. when mice were fed on the deficient diet. E. B.

**Degradation of cystine by normal liver but not by transplanted hepatomas.** J. P. Greenstein (*J. Nat. Cancer Inst.*, 1943, **3**, 491—494).—Cystine and methionine were incubated with tissue extracts alone and in the presence of *dl*-alanine. $NH_3$ and $H_2S$ were liberated from cystine incubated with normal rat or mouse liver extract but not when incubated with extracts of hepatomas from rats and mice. The degradation occurred under aërobic or anaërobic conditions. E. B.

**Hydrogen-ion concentration of normal liver and hepatic tumours.** H. Kahler and W. v. B. Robertson (*J. Nat. Cancer Inst.*, 1943, **3**, 495—501).—The pH vals. of excised tissues were measured with a capillary glass electrode. The pH of the normal liver was 7·4 while that of hepatoma tissue from starved rats was 7·0. If excess of glucose were supplied to the animal the tumour-pH fell to 6·4 while the liver-pH was unchanged. E. B.

**Chemistry of tumours. VI. Isolation of antipodes of glutamic acid.** F. Kögl and H. Erxleben. **VII. Special place of *d*-glutamic acid in experiments with *d*-amino-acid oxidase.** F. Kögl, H. Herken, and H. Erxleben. **VIII. Stereochemical analysis of proteins from lymphogranuloma and other pathological tissues.** H. Erxleben and H. Herken (*Z. physiol. Chem.*, 1940, **264**, 198—219, 220—239, 240—250; cf. A., 1942, III, 615).—VI. The results of Chibnall *et al.* (A., 1940, III, 46, 507) on the isolation of *d*-glutamic acid from plant proteins, and of *l*-glutamic acid from hydrolysates of tumour proteins, are discussed, and it is pointed out that they do not invalidate those of Kögl. *d*-Glutamic acid is probably a normal physiological constituent, and this may also be the case with the small amounts (usually approx. 2%) found in normal tissues. When glutamic acid is isolated by the Foreman method *l*- tends to be conc. at the expense of *d*-glutamic acid. After hydrolysis of the proteins from pig kidney and calf lung and separation of glutamic acid, *dl*-glutamic acid is added to the filtrate, which is then worked up by the Foreman method. The recovered glutamic acid contains 93·3% of the *l*- and 35·7% of the *d*-acid which was added in the case of pig kidney, whilst with calf lung hydrolysate the corresponding amounts are 94·3% and 42·8%. Hydrolysis of Brown–Pearce tumour protein and determination of glutamic acid by the Foreman method gives 8·4% of *l*-glutamic acid, and when the filtrate is worked up afterwards by the HCl method a further small amount of *l*- and considerably more *d*-glutamic acid are obtained. Hydrolysis of Brown–Pearce tumour protein and isolation by the HCl method gives 6·7% of total which contains 1·8% of *d*-glutamic acid. The solubility of Ca *dl*-glutamate in ethyl alcohol (*d* 0·8338) at 19±1° is approx. 40 times that of Ca *l*-glutamate.

VII. Deamination of *d*- and *l*-aspartic acid, glutamic acid, leucine, phenylalanine, and valine by the amine oxidases present in rat kidney and liver occurs at different rates for each amino-acid and is very slow for *d*-glutamic acid. Deamination of *l*-amino-acids depends slightly less on concn. of the amino-acid than it does in the case of the *d*-acids. *d*-Leucine and *d*-valine are deaminated considerably more quickly than is *d*-glutamic acid by the enzymes from pig and sheep kidney. When mixed with other *d*-amino-acids, the rate of deamination of *d*-glutamic acid is considerably decreased, and this is not due to inactivation of the enzyme. The rate of oxidation of the other amino-acids is also affected. The results of Lipmann *et al.* (A., 1940, III, 424) are criticised on the ground of the very long time allowed for the enzymic reaction, and because the controls contained considerably more *d*-glutamic acid than could have been present in the hydrolysates of the tumour proteins.

VIII. Hydrolysis of the protein from a rabbit liver with diffuse hepatitis gave 5·7% of glutamic acid, of which 1·8% was the *d*-acid. A human miliary tuberculous lung yielded 5·7% of glutamic acid containing 1·9% of the *d*-isomeride. 6·4% of glutamic acid containing 1—6% of *d*-acid was isolated from human lymphogranulomatous tissue, whilst the mediastinal lymph gland of a pig with leukosis gave 5·8 and 2·4% respectively. The protein from a rabbit with Sanarelli virus myxoma gave 6·1% of glutamic acid containing 11% of the *d*-isomeride. It is concluded that the presence of more than normal amounts of *d*-glutamic acid in tissue-proteins is sp. for tumours and is not a general pathological condition. J. N. A.

**1 : 2 : 3 : 4-Dibenzphenanthrene and derivatives.**—See A., 1943, II, 296.

**Steric selection by peptidases in normal and carcinomatous sera. *d*-Peptidases in normal and carcinomatous tissue.**—See A., 1943, III, 682.

## XVIII.—ANIMAL NUTRITION.

**Food tables.** E. M. Widdowson and R. A. McCance (*Lancet*, 1943, **244**, 230—232).—Determinations of the chemical composition of individual mixed diets gave vals. sufficiently close to those derived from the use of food tables to warrant the use of the latter in dietary surveys. Up to 200 mg. of Ca daily may be obtained by drinking hard water. Fe intake may be enormously increased by contamination from kitchen utensils, especially chipped enamelled Fe saucepans. C. A. K.

**A.C.H. and Tuxford indices in nutritional assessment.** J. M. Latsky (*S. Afr. J. med. Sci.*, 1942, **7**, 217—225).—1000 children were graded as diseased, malnourished, or normal on the basis of extensive clinical and laboratory investigations. Of the 600 malnourished children the A.C.H. index [based on measurements of arm girth, chest depth, and hip width] selected only 17% and the Tuxford 66%. Of the 350 normal children the A.C.H. and Tuxford indices selected 12% and 40% respectively as malnourished. P. C. W.

**Diet and muscular fatigue.**—See A., 1943, III, 465.

**Nutrition of healthy and ill children in wartime.** G. Fanconi (*Schweiz. med. Wschr.*, 1942, **72**, 958—962).—A lecture. A. S.

**Physiology of nutrition and preserved food.** A. Fleisch (*Schweiz. med. Wschr.*, 1942, **72**, 957—958). A. S.

**Nutritional value of food preserves.** N. Bömmels (*Schweiz. med. Wschr.*, 1942, **72**, 967—968). A. S.

**Milk preserves and wartime nutrition.** M. Guigoz and A. Jaton (*Schweiz. med. Wschr.*, 1942, **72**, 970—972). A. S.

**Nutritional value of cocoa and chocolates.** C. del Boca and H. Zschokke (*Schweiz. med. Wschr.*, 1942, **72**, 976—977, 977—979). A. S.

**Nutritional value of soya bean.** Moser (*Schweiz. med. Wschr.*, 1942, **72**, 979—983). A. S.

**Protein intake in wartime.** A. Jung (*Schweiz. med. Wschr.*, 1942, **72**, 964—967).—The relative rôle of pigs, chicken, cattle, and cereals in the provision of nutritional proteins is discussed. A. S.

**Protein diet in pregnancy.** O. M. Holmes (*West. J. Surg. Obstet. Gynec.*, 1941, **49**, 56—60).—700 pregnant women (350 primiparæ and 350 multiparæ) were fed a high-protein diet and compared with an exactly similar series fed on a low-protein diet. The incidence of toxæmia was twice as high in both groups on the low-protein diet as in those on the high-protein diet; the incidence of prematurity was in the reverse direction. There was no difference in the wt. of the infants at birth. P. C. W.

**Intravenous injection of gelatin for nutritional purposes.** A. Brunschwig, V. B. Scott, N. Corbin, and R. Moe (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 46—48).—Positive N balance was maintained in protein-depleted dogs by intravenous injections of 12% solution of gelatin. Urinary N was greatly increased, almost all of it being non-protein. V. J. W.

**Nutritive value of milk-protein.**—See B., 1943, III, 215.

**Rôle of phosphorus, flavin, and hormones in utilisation of proteins.**—See A., 1943, III, 504.

**Oral use of amino-acids of hydrolysed casein (Amigen) in surgical patients.** R. Elman (*Amer. J. digest. Dis.*, 1943, **10**, 48—50).—Two patients with enterostomies who had intractable diarrhœa on ordinary liquid feeds responded well to 100—200 g. of Amigen daily + glucose and vitamins. Diarrhœa stopped, plasma-proteins rose, and the patients recovered. N. F. M.

**Nitrogen balance in experimental human deficiencies of methionine and cystine.** A. A. Albanese, L. E. Holt, jun., J. E. Brumback, jun., C. N. Kajdi, J. E. Frankston, and D. M. Wangerin (*Proc. Soc. Exp. Biol, Med.*, 1943, **52**, 18—20).—3 subjects on a methionine-deficient diet had a negative N balance which returned to normal when methionine was administered. Results with cystine were variable. (Cf. A., 1943, III, 662.) V. J. W.

**Importance of choline in nutrition.** V. G. Foglia (*Medicina*, 1942, **2**, 530—547).—Lack of choline in diet causes fatty degeneration of liver, hæmorrhagic lesions in the kidney, and retardation or cessation of growth. Its relations to vitamin-$B_1$ and metabolism of fats, proteins, and creatine are described. Choline is formed in the body through a transfer of the methyl groups from methionine to ethanolamine and transfers methyl groups to homocysteine to form methionine and creatine. I. C.

**Metabolism of arginine and leucine with special reference to respiratory exchange and heat production.**—See A., 1943, III, 504.

**Production of cirrhosis in liver of normal dog by prolonged feeding of a high-fat diet.**—See A., 1943, III, 485.

**Rôle of carbohydrates in infantile nutrition in wartime.** F. Stirnimann (*Schweiz. med. Wschr.*, 1942, **72**, 962—963). A. S.

**Artificial sweetening substances.** H. Staub (*Schweiz. med. Wschr.*, 1942, **72**, 983).—The chemical and pharmacological properties of saccharin and dulcin and their practical use are reviewed. A. S.

**Nutritional availability of iron in molasses.** R. S. Harris, L. M. Mosher, and J. W. M. Bunker (*Biol. Res. Lab., Mass. Inst. Tech., Publ.* 157; *Int. Sugar J.*, 1943, **45**, 215—216).—Cases affected with subclinical hypochromic anæmia are curable by ingesting foods (molasses) containing an abundance of readily available Fe. Chemical and biological tests were conducted with ordinary 1st, 2nd, and 3rd factory molasses, containing 3·2, 6·0, and 11·3 mg.-% of Fe, the "nutritionally available" Fe being 97, 85, and 54%. Fe "availability" varies considerably with various foods, being 100, 70, 50, and 24% with eggs, beef liver, beef kidney, and mutton, respectively. It does not depend on the total Fe content of the food. J. P. O.

**Iron in nutrition.**—See A., 1943, III, 455.

**Utilisation for hæmoglobin regeneration of the iron in salts used in the enrichment of flour and bread.**—See A., 1943, III, 455.

**Anæmia in women and children on war-time diets.**—See A., 1943, III, 456.

**Fluorine distribution.** C. N. Bromehead, M. M. Murray, and D. C. Wilson (*Lancet*, 1943, **244**, 490—491).—The occurrence of F in water in various parts of England and Wales and the distribution of dental fluorosis are described. C. A. K.

**Recent advances in treatment of hepatic diseases [and vitamins].**—See A., 1943, III, 566.

**Growth requirements of *Acetobacter suboxydaus*.**—See A., 1943, III, 602.

**Vitamin therapy in eye diseases. Vitamins in ophthalmology.**—See A., 1943, III, 468.

**Vitamin content of fruit and vegetable preserves.** Rorschach (*Schweiz. med. Wschr.*, 1942, **72**, 972—975). A. S.

**Experimental human vitamin-*A* deficiency and ability to perform muscular exercise.** G. Wald, L. Brouha, and R. E. Johnson (*Amer. J. Physiol.*, 1942, **137**, 551—556).—After 30 days of high vitamin-*A* nutrition, sedentary male subjects were maintained on an extremely low -*A* diet for 6 months; after 3—4½ months on the deficient diet and 6 weeks after return to a normal -*A*-supplemented diet, physical fitness for moderate and exhausting exercise was determined; no significant difference in performance was found. On the deficient diet, fæcal carotenoids fell to 1% of the previous val. to 0·5 μg. per c.c. Plasma-*A* maintained its initial max. level of 1·57—2·97 i.u. per c.c. The visual threshold of 3 subjects remained const. and min.; in 2 subjects it rose slowly to levels of 0·7—1·1 log unit above normal. M. W. G.

**Effect of various stages of vitamin-*A* deficiency in white rat on resistance to *Nippostrongylus muris*.** E. G. Riley (*J. infect. Dis.*, 1943, **72**, 133—141).—After 2 weeks' vitamin-*A* depletion there was increased resistance to the infection. After 4 weeks there was progressive decrease in resistance, manifested by a more intense and prolonged infection. F. S.

**Determination of vitamin-*A*.** E. M. Hume (*Nature*, 1943, **151**, 535—536).—The factor relating results of spectrophotometric tests with those of biological tests expressed in i.u. is 1740 (±7%). The potency of the U.S.P. reference cod-liver oil, reputed to be 3000 i.u., is 2619 i.u. The discrepancy is discussed. E. R. S.

**Critical study of Carr–Price reaction for determination of β-carotene and vitamin-*A* in biological materials.** M. Kaser and J. A. Stekol (*J. Lab. clin. Med.*, 1943, **28**, 904—909).—Excellent recoveries of cryst. β-carotene and vitamin-*A* were obtained with reproducible results. The macro-procedure described was satisfactory for routine analysis of plasma or serum. Preliminary saponification of plasma or serum has no effect on -*A* or carotene concns. in normal plasma. C. J. C. B.

**[No] carotene in annatto seed (*Bixa orellana*).** G. G. Villela (*Rev. Brasil. Biol.*, 1942, **2**, 159—164).—The pigment of the annatto seed is chiefly bixin. No carotene, lycopene, or cryptoxanthine could be detected. I. C.

**Carotene content of sweet potatoes.**—See B., 1943, III, 216.

**Determination of carotene oxidase in legume seeds.**—See A., 1943, III, 518.

**Vitamin requirements of thoroughbreds in training.** C. Way (*J. Amer. Vet. Med. Assoc.*, 1942, **100**, 335—339).—A prep. containing thiamin, riboflavin, and nicotinic acid improved the condition of thoroughbred horses in training and brood mares. Blood-sugar and serum-Ca and -P were determined in 116 horses, the mean vals. being 69·5, 8·8, and 3·5 mg.-% respectively. E. G. W.

**Gallstone formation and intake of *B* vitamins in cholesterol-fed guinea-pig.**—See A., 1943, III, 486.

**Uniformities in content of *B* vitamins in malignant neoplasms.**—See A., 1943, III, 491.

***B*-Vitamins in cancerous tissues. I. Riboflavin. II. Nicotinic acid. III. Biotin. IV. Pantothenic acid.**—See A., 1943, III, 491.

**Vitamin-*B* complex in ophthalmology.**—See A., 1943, III, 553.

**Thiamin excretion tests in children with paralytic poliomyelitis.** R. Ward, A. B. Sabin, V. A. Najjar, and L. E. Holt, jun. (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 5—7).—Excretion did not differ from that in normal controls. V. J. W.

**Vitamin-$B_1$ deficiency in pregnancy indicated by test for OBT principle.** O. Horwitz and D. L. Farley (*Surg. Gynec. Obstet.*, 1940 **71**, 313—316).—Level of vitamin-$B_1$ in the blood was assessed by determination of the OBT principle (a principle in human serum inhibiting saponin hæmolysis) (Farley, *Bull. Ayer Clin. Lab.*, 1939, **3**, 331). Subnormal levels were found in 13 of 100 pregnant women tested in the last three months of pregnancy. The OBT level was raised by the administration of yeast for 4 days. -$B_1$ deficiency caused loss of appetite. Neuritic symptoms developed among the patients with deficiency of OBT principle. One case of eclampsia had normal OBT level in the blood. P. C. W.

**Thiamin hydrochloride and energy value of glucose studied in rats by single food choice method.** C. P. Richter and K. K. Rice (*Amer. J. Physiol.*, 1942, **137**, 573—581).—Female rats on glucose and water survived for 36 days, rats on water alone for 4 days. Glucose rats at first very active were totally inactive during the last week; body wt., food and water intake decreased steadily, diœstrus occurred, but no sign of nutritional deficiency was observed though the animals were very emaciated at death. Glucose rats with access to 0·02% thiamin survived for 74 days and maintained activity for 50 days; body wt. decreased slowly. Intake of thiamin calc. in μg. per g. of glucose fell from 60 to 40, then increased to 100 in 70—80-day period. Signs of vitamin-*A* deficiency occurred and extreme emaciation before death. Rats given glucose and water with access

to 0·03% aq. cocarboxylase survived for 77 days, and also showed an increase in vitamin/glucose ratio during the latter half of the experimental period. M. W. G.

**Thiamin influence on laxative action.** S. Loewe, I. Loewe, and R. Knox, jun. (*Amer. J. digest. Dis.*, 1943, **10**, 65—66).—Experiments on rhesus monkeys showed that daily doses up to 100 mg. of thiamin per kg. did not increase the purgative effect of phenolphthalein. N. F. M.

**Hypersensitivity to thiamin.**—See A., 1943, III, 611.

**Treatment of nerve deafness with vitamin-$B_1$.**—See A., 1943, III, 555.

**Effect of hæmatin on stability of thiochrome in solutions of alkaline ferricyanide.** P. S. Owen, N. Weissman, and J. W. Ferrebee (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 59—60).—Traces ($1 : 10^7$) of hæmatin accelerate destruction of thiochrome in solutions containing alkaline ferricyanide. This property is not abolished by boiling, pepsin, CO, or strong acid or alkali, but the substance responsible is adsorbed on protein ppts. which thereupon become active, the fluid losing its activity. V. J. W.

**Vitamin-$B_1$-containing culture media and gonococci.**—See A., 1943, III, 524.

**Failure of seedlings of apple, peach, pear, and rose to respond favourably to vitamin-$B_1$.**—See A., 1943, III, 537.

**Vitamin-$B_1$ content of potatoes.** J. Meiklejohn (*Biochem. J.*, 1943, **37**, 349—354).—The *Phycomyces* growth method is suitable for the assay of vitamin-$B_1$ in potatoes, but the green sprouts contain a substance toxic to the fungus which is probably not either solanine or solanidine. The thiochrome method also fails with green sprouts. An adjuvant factor is present in the centres of the tubers between April and August and is always present in the skin layer. It operates only in presence of excess of -$B_1$, is not due to buffering or to N compounds, and is distinct from that of blood (Sinclair, A., 1940, III, 144). The -$B_1$ content of Majestic tubers increases during the growing season to 140 μg. per 100 g. at the time of lifting and falls during storage. The greater part of this loss occurs in spring and is chiefly due to sprouting, the -$B_1$ content of the white sprouts and young potato leaves being high. The -$B_1$ is conc. in the centre of the tuber rather than under the skin. H. G. R.

**Determination of vitamin-$B_1$ in navy beans.**—See B., 1943, III, 217.

**Lactoflavin metabolism in two patients with generalised eczema.** A. F. Kunz (*Schweiz. med. Wschr.*, 1942, **72**, 1154—1158).—Diminished urinary lactoflavin excretion was found in 2 patients suffering from generalised eczema, 1 patient having an oliguria. Administration of lactoflavin removed the lactoflavin deficit and produced diuresis. Nicotinic acid excretion diminished. The previous condition was restored on discontinuing the lactoflavin treatment. A. S.

**Adsorption of riboflavin by lactose ; influence of concentration.**—See B., 1943, III, 212.

**Clinical experiments with riboflavin, inositol, and calcium pantothenate.** M. G. Vorhaus, M. L. Gompertz, and A. Feder (*Amer. J. digest. Dis.*, 1943, **10**, 45—48).—Riboflavin (5 mg. daily by mouth) produced rapid healing in 5 out of 6 cases of decubital ulcer. Inositol was well tolerated in oral doses of 2 g. It did not affect bowel habits but benefited 2 cases of "dermatitis." Ca pantothenate (100 mg. intramuscularly bi-weekly) had no effect on alopecia or grey hair. N. F. M.

**Stimulating effect of adrenalectomy on hair growth and melanin deposition in black rats fed diets adequate and deficient in filtrate factors of vitamin-*B*. Effect of diet on postoperative survival.**—See A., 1943, III, 558.

**Nicotinic acid deficiency in the chick.** G. M. Briggs, jun., T. D. Luckey, L. J. Teply, C. A. Elvehjem, and E. B. Hart (*J. Biol. Chem.*, 1943, **148**, 517—522).—Nicotinic acid deficiency decreases growth, food intake, and muscle-nicotinic acid, and causes poor feathering and sometimes perosis and dermatitis. Chicks may synthesise about $\frac{1}{8}$ of their nicotinic acid requirement. Nicotinic acid esters have partial nicotinic acid activity, which varies with the length of the C side-chain. R. L. E.

**Nicotinic acid in angina pectoris.**—See A., 1943, III, 460.

**Treatment of asthma by nicotinic acid.**—See A., 1943, III, 551.

**Substitution of heated asparagine–glutamate mixture for nicotinamide as growth factor for *Bacterium dysenteriæ* and other microorganisms.**—See A., 1943, III, 604.

**Determination of nicotinic acid ; modifications of the microbiological method.** W. A. Krehl, F. M. Strong, and C. A. Elvehjem (*Ind. Eng. Chem.* [*Anal.*], 1943, **15**, 471—475).—Modifications in the basal medium proposed by Snell and Wright (A., 1941, III, 685) for the microbiological determination of nicotinic acid are described, and a different procedure is given for growing the necessary inoculum. These variations nearly double the response of the bacteria to quantities of nicotinic acid in the range 0·04—0·3 μg. and produce a linear standard curve. The modified method gives more consistent and uniform results than the original. J. D. R.

**Pyridoxine deficiency in turkeys.** F. H. Bird, F. H. Kratzer, V. S. Asmundson, and S. Lepkovsky (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 44—45).—Symptoms consisted of loss of appetite, poor growth, apathy, hyperexcitability, convulsions, and death, and were prevented by administration of 3 mg. of pyridoxine per kg. of diet. V. J. W.

**Biotin deficiency and other changes in rats given sulphanilylguanidine or succinylsulphathiazole in purified diets.**—See A., 1943, III, 510.

**Modification of yeast-growth assay method for biotin.** R. Hertz (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 15—17).—Addition to Snell's medium of 1 g.-equiv. per l. of a 10% biotin-free casein hydrolysate increases specificity and range of determinations. V. J. W.

**Effect of *p*-aminobenzoic acid when added to purified chick diets deficient in unknown vitamins.** G. M. Briggs, jun., T. D. Luckey, R. C. Mills, C. A. Elvehjem, and E. B. Hart (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 7—10).—Deficiency symptoms resulting from a purified diet (cf. A., 1942, III, 621) are partly removed by addition of 5 mg.-% of *p*-aminobenzoic acid and aggravated by administration of succinylsulphathiazole. It is suggested that an essential factor is produced by bacterial growth in the gut. V. J. W.

**Vitamin-*C* investigations in internment camp.** R. E. Flowerdew and O. B. Bode (*Lancet*, 1943, **244**, 428—430).—Vitamin-*C* nutrition was assessed in internees and other personnel in spring and autumn of 1942. The no. of subjects showing no ascorbic acid in urine after 3 daily test doses of 700 mg. of ascorbic acid per 10 stone body wt. was higher in spring than in autumn. Improvements in prep. and the eating of raw vegetables greatly improved -*C* nutrition in winter. C. A. K.

**Vitamin-*C* intakes at residential home.** L. J. Harris and M. Olliver (*Lancet*, 1943, **244**, 454—457).—Analyses of raw and cooked foods at a residential home for boys in 1941 and 1942 showed that daily intake of vitamin-*C* was highest in late summer (max. 55 mg. daily) and lowest in spring (min. 19 mg. daily). The change from old to new potatoes added 30 mg. of -*C* daily to the diet. Cabbages and sprouts were the next most important source of -*C*, and cooked vegetables were preferable to raw as much larger quantities could be taken. The water in which vegetables were cooked was used in gravies etc. C. A. K.

**Photodynamic effects in presence of ascorbic acid.** G. Boehm (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 3—4).—The frog's urinary bladder, in oxygenated salt solution, does not react when lactoflavin (0·3 mg.-%) or hæmatoporphyrin (2·5 mg.-%) is added in the presence of a strong source of light. Radiation in the presence of ascorbic acid (35 mg.-%) produces a strong contraction, repeatable after 5—10 min. interval. Acetylcholine is not involved, as shown by experiments with atropine and eserine. A. S.

**Occurrence of bound ascorbic acid.** K. Wachholder and A. Okrent (*Z. physiol. Chem.*, 1940, **264**, 254—266).—Cows' milk contains no bound ascorbic acid, and little, if any, is present in human milk. Similarly, plant tissues contain no bound ascorbic acid; rabbit and guinea-pig heart muscle contains fairly large amounts. The determination is carried out by comparison of extracts made with sulphosalicylic acid before and after hydrolysis with 0·5N-HCl at 100° in $CO_2$. The results with other animal tissues are inconclusive. The results of Holtz (A., 1941, III, 375) are criticised, and his findings are considered to be due to faulty technique. J. N. A.

**Vitamins and sore and bleeding gums.** C. C. Ungley and J. S. F. Horton (*Lancet*, 1943, **244**, 397—400).—In 51 naval patients with sore and bleeding gums the daily ascorbic acid intake was 16—80 mg. (average 37 mg.). They were no more "unsaturated" with ascorbic acid than healthy controls and there were no signs of scurvy. Local causes, *e.g.*, infection with Vincent's organisms, in 85% were adequate to explain the gum condition and both ascorbic acid and nicotinic acid were therapeutically ineffective. C. A. K.

**Trophic necrosis, skin-grafting, and vitamin-*C*.** G. Evans (*Brit. Med. J.*, 1943, I, 788).—Two cases of avitaminosis were treated with ascorbic acid; 150 mg. of ascorbic acid daily in one case for 8 weeks and in the second for 16 days were necessary before ascorbic acid was excreted regularly in the urine. I. C.

**Effect of hypophysectomy on concentration of ascorbic acid in rat adrenal.**—See A., 1943, III, 559.

**Influence of ascorbic acid on the gonadotropic hormone content of male rat pituitary gland.**—See A., 1943, III, 561.

**Ascorbic acid and chick explants.**—See A., 1943, III, 544.

**Ascorbic acid system as an agent in barley respiration.**—See A., 1943, III, 536.

**Ascorbic acid content of rhubarb.**—See A., 1943, III, 539.

**Ascorbic acid content of some tomato varieties and species.**—See B., 1943, III, 220.

**Apparent vitamin-*C* in certain foodstuffs.** F. Wokes, J. G. Organ, J. Duncan, and F. C. Jacoby (*Nature*, 1943, **152**, 14—15).—"Apparent vitamin-*C*" (determined as ascorbic acid with 2 : 6-dichlorophenol-indophenol), formed in germinating grains at pH 4·5—5·0, differs from reductones in being unstable in $HPO_3$ solution; only 5—10% condenses with formaldehyde at pH 4—5. The total apparent -*C* is therefore determined potentiometrically before and after removal of ascorbic acid at room temp. with 6% formaldehyde at pH 4—5. The formaldehyde method (visual or potentiometric) is preferred to the photometric method for the determination of -*C*. A. A. E.

**Raman spectra of *l*-ascorbic acid, tetronic acid, and related compounds.**—See A., 1943, I, 249.

**Hæmatologic and radiologic study of infants receiving massive doses of vitamin-*D* in rickets prophylaxis.** A. C. Rambar, L. M. Hardy, and W. I. Dishbein (*J. Pediat.*, 1943, **23**, 31—37).—The use of a single massive oral dose of an electrically activated vaporised ergosterol, containing 600,000 U.S.P. units of vitamin-*D*, was effective in preventing rickets in infants studied during the autumn and winter months. Repeated smaller doses of 100,000 U.S.P. units of the same agent given monthly during this period were also effective. No toxic findings occurred. C. J. C. B.

**Activation of provitamin-$D_3$ in rat.** E. Geiger and S. Lassen (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 11—12).—7-Dehydrocholesterol had no curative effect on rachitic rats kept in darkness. It had a slight effect if the rats were exposed to irradiation and sunlight, but a much larger effect was brought about by administration of irradiated 7-dehydrocholesterol, whether by mouth or subcutaneously. V. J. W.

**Chemical and biological stability of crystalline vitamin-$D_2$ and -$D_3$ and their derivatives.** W. Huber and O. W. Barlow (*J. Biol. Chem.*, 1943, **149**, 125—137).—The [α], ultra-violet absorptions, and m.p. are collected for vitamin-$D_2$ and -$D_3$, their 4-nitrobenzoates, 3 : 5-dinitrobenzoates, and 3 : 5-dinitro-4-methylbenzoates, and the -$D_3$–cholesterol additive compound (this is the most economical way of obtaining a cryst. -$D_3$ prep.). The various nitrobenzoic esters of -$D_2$ and -$D_3$ can be stored for at least 5 years at room temp. without decomp., and pure cryst. -$D_2$ and -$D_3$ can be obtained therefrom by direct saponification with methyl-alcoholic KOH. -$D_2$ is unchanged after 9 months, and -$D_3$ after 12 months, in amber evacuated ampoules at 4°, but if stored in presence of air, decomp. occurs after 1—2 months at 4° and after 2—3 days at room temp. Cryst. -$D_3$ would be a suitable reference prep. if suitably stored, and prepared afresh from purified esters every 9—12 months. Solutions of -$D_2$ or -$D_3$ in propylene glycol or in vegetable oils retain their biological potency in amber bottles, even in air at 38—40°, for at least 2—3 years, and milk (liquid or condensed) fortified with such solutions retains its potency for at least as long as it would be useful as a food. -$D_2$ and -$D_3$ have the same potency towards rats, but results of over 400 assays indicate that the usually accepted antirachitic value of 40,000 i.u. per mg. is 10—15% too low. E. C. W.

**Goitrogenic action of calcium and vitamin-*D*.**—See A., 1943, III, 476.

**Ergosterol content of certain yeasts and fungi.**—See A., 1943, III, 520.

**Modified antimony trichloride reagent for determination of certain sterols and vitamin-$D_2$ and -$D_3$.**—See A., 1943, III, 616.

**Factors influencing the A.O.A.C. chick method of vitamin-*D* assay.** I. Motzok, D. C. Hill, H. D. Branion, and J. S. Slinger (*J. Assoc. Off. Agric. Chem.*, 1943, **26**, 263—272).—A group size of 15 chicks did not give a very high degree of accuracy. Cross-bred chicks gave uniform response; White Leghorns showed the greatest variability. The strain of yeast may markedly influence the results. A. A. E.

**Vitamin-*E*, cod-liver oil, and muscular dystrophy.**—See A., 1943, III, 465.

**Muscular dystrophy in absence of testicular degeneration in vitamin-*E* deficiency.**—See A., 1943, III, 551.

**Factors influencing capillary permeability in vitamin-*E*-deficient chick.**—See A., 1943, III, 461.

**Tocopherol level in serum of normal subjects and patients with amyotrophic lateral sclerosis.**—See A., 1943, III, 552.

**Mechanism of biological action of vitamin-*K* and its synthetic analogues.** M. M. Shemiakin, L. A. Schukina, and J. B. Shvezov (*Nature*, 1943, **151**, 586—587).—Diethyl phthalate has a high antihæmorrhagic activity. The activity of vitamin-$K_1$ and -$K_2$ is due to their oxidative biodecomp. to phthalic acid. 2-Methyl-1 : 4-naphthaquinone and K 2-methyl-1 : 4-naphthaquinone-3-sulphonate (both highly active) are converted into phthalic acid by heating with water. E. R. S.

**Vitamin-*K* in hæmorrhagic disease of newborn infant. Clinical experience with water-soluble vitamin-*K*-like substance (tetrasodium 2-methyl-1 : 4-naphthaquinol diphosphate).**—See A., 1943, III, 547.

**Hæmorrhagic sweet clover disease. IX. Effect of diet and vitamin-*K* on the hypothrombinæmia induced by 3 : 3′-methylene-bis-(4-hydroxycoumarin) in the rat.**—See A., 1943, III, 457.

**Prevention of cincophen toxicity by use of vitamin-*K*.**—See A., 1943, III, 592.

**Determination of "citrin" in lemon preparations.** A. E. Goldfarb, E. Bueding, and P. Karp (*J. Lab. clin. Med.*, 1943, **28**, 1036—1037).—A modification of the method of Lorenz and Arnold (A., 1943, III, 191) for the determination of "citrin" in lemon preps. is described; the interference of glucose is eliminated by avoiding heating of the solutions after the addition of alkali. Lemon juice contains ¼ the amount of "citrin" found in lemon peel extract. C. J. C. B.

**Dietary factor, essential for guinea-pigs. I. Isolation from raw cream.** W. J. van Wagtendonk and R. Wulzen (*Arch. Biochem.*, 1943, **1**, 373—377).—Stiffness of the wrists of guinea-pigs was produced in 1 month by dietary means. This was cured by raw cream (1 g. per day), but not by cod-liver oil or α-tocopherol. 15 gallons of raw cream were churned to butter, this was saponified, and the fatty acids were steam-distilled; the distillate was extracted with ether and this washed with aq. KOH, and the ether solutes refluxed with acethydrazidotrimethylammonium chloride in ethanol–acetic acid (9 : 1). The product was neutralised and extracted with ether, then acidified and extracted with ether, giving 62 mg. with 500,000 units per g. (raw cream : 1·5 units per g.). The $HgI_2$ complex of this product had m.p. 60—61° (uncorr.), mol. wt. 820. The active ketone has mol. wt. approx. 200. The corresponding $HgI_2$ complex of methyl vinyl ketone had m.p. 120—121° (uncorr.). 3 mg. of yellow oil were recovered from the complex, having $10^6$ units per g. The $HgI_2$ complex isolated from pasteurised cream had m.p. 171—172° (uncorr.) and gave an inactive oil when decomposed with $H_2S$. E. R. S.

**Dermatitis on synthetic ration adequate for growth and reproduction in rat.** B. H. Ershoff (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 41—43).—Rats on a synthetic diet developed a dermatitis of the tail which was not due to lack of pantothenic acid, pyridoxine, or essential fatty acid. V. J. W.

## XIX.—METABOLISM, GENERAL AND SPECIAL.

**Basal heat production in relation to growth.** H. R. Benjamin and A. A. Weech (*Amer. J. Dis. Child.*, 1943, **65**, 1—35).—Observations of basal heat production are reported for 4 infants between the ages of 6 and 20 months. The daily fluctuations were large (9%). The association between basal calories and body wt. was closest. When heat production was referred to a unit of mass (cals. per kg.) there was no reduced change with age advance but when referred to unit of surface area or to unit of height, there was definite evidence of change with growth. Different infants of the same age vary significantly in the quantity of heat produced per kg. of body wt. Over the age span studied, prediction on the assumption of a const. heat output per kg. of wt. is not significantly less accurate than prediction on the basis of regression equations. The total error of prediction was between 11·3 and 13·7%. This error is composed of 2 components: variability among different infants, with vals. between 6·5 and 9·8% and variability in the individual infant, with vals. between 5·5 and 10·3%. The excess heat production per kg. of tissue of the infant over that of the adult was not due in any substantial degree to the normal changes in the rate of growth. C. J. C. B.

**Standards for basal metabolism of children 2 to 15 years of age.** R. C. Lewis, A. M. Duval, and A. Iliff (*J. Pediat.*, 1943, **23**, 1—18).—Standards for these children based on 1007 determinations on 70 healthy boys and 718 on 57 healthy girls are tabulated. C. J. C. B.

**Basal metabolism in rheumatic children.** E. E. Brown and V. P. Wasson (*J. Pediat.*, 1943, **23**, 19—23).—The basal metabolic rate in rheumatic children is slightly below normal (average −7% in 97 patients). The skin and hair of rheumatic children often become dry in the winter and spring and improve in the autumn. C. J. C. B.

**Evaluation of laboratory procedures in determination of basal metabolic rate.** L. E. Nolan (*Amer. J. clin. Path.*, 1943, **13**, 278—280).—Of 200 basal metabolism tests performed in 100 out-patients the test was satisfactory after 1 hr. rest in 88%. Of 400 basal metabolism tests performed in 200 patients hospitalised overnight, the result was satisfactory in 91%. The result of a single determination is as accurate as the average of 2 consecutive tests. C. J. C. B.

**Respiration rate of dairy bull spermatozoa.**—See A., 1943, III, 480.

**Effects of treatment of normal rats with saline anterior pituitary extract. II. Protein and energy metabolism.**—See A., 1943, III, 559.

**Origin of β-alanine in the animal body : microbiological assay of β-alanine.** J. R. Schenck (*J. Biol. Chem.*, 1943, **149**, 111—115).—β-Alanine is determined by measuring (in terms of total bacterial N) the growth of *Corynebacterium diphtheriæ* on a synthetic medium containing the substance under test. About 5 μg. of β-alanine is required for a test. Using this method, no increase in β-alanine was observed after incubation of tissue slices with aspartic acid or aspartylhistidine, and it could not be detected in the blood or urine of rabbits after injection of either of these substances, although even 1% conversion would have been detected. It is therefore improbable that β-alanine is derived from aspartic acid or carnosine from aspartylhistidine. E. C. W.

**Liver injury, liver protection, and sulphur metabolism.**—See A., 1943, III, 485.

**Hereditary aspects of cystinuria.**—See A., 1943, III, 543.

**Biological synthesis of urea.** F. Leuthardt and B. Glasson (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 25—27). A. S.

**Case of juvenile xanthomatosis.** F. K. Herbert (*Arch. Dis. Childh.*, 1943, **18**, 41—49).—A case of juvenile xanthomatosis is reported, with arrested growth, enlargement of the liver and spleen, renal disease with hypertension, and enormous increases in plasma-phospholipins (3970 mg.-%) and -cholesterol (2600 mg.-%). C. J. C. B.

**Rates of replacement of depot and liver fatty acids in mice.** DeW. Stetten, jun., and G. F. Grail (*J. Biol. Chem.*, 1943, **148**, 509—515).—The half-life of D in the depot and liver fats of rats on a high-carbohydrate diet after enrichment of the body-fat with D was 5—6 and 2·6—2·8 days respectively. Liver-fatty acids certainly have a shorter life than this suggests. Dietary choline has no effect on the rate of disappearance of D. R. L. E.

**Cerebral metabolism in fat-fed dog.** A. G. Mulder and L. A. Crandall, jun. (*Amer. J. Physiol.*, 1942, **137**, 436—439).—Ketosis was produced in dogs by starvation and fat-feeding. After 2—3 days' starvation 100 c.c. of olive oil were fed daily for 2—6 weeks. The brain of the fat-fed dog does not burn acetone bodies and has a R.Q. of approx. 1·0. The vals. for $O_2$ and glucose removed by brain in ketosis in mg. per 100 ml. of blood equal those for normally fed animals. M. W. G.

**Biochemical lesions [hyperglycæmia] produced by diets high in tyrosine.** G. J. Martin and W. C. Hueper (*Arch. Biochem.*, 1943, **1**, 435—437).—Inclusion of 5—10% of tyrosine in the diet of young rats produces a hyperglycæmia resembling diabetes. E. R. S.

**Pentosuria and diabetes mellitus in one patient.**—See A., 1943, III, 567.

**Carbohydrate metabolism of chick fibroblasts.**—See A., 1943, III, 452.

**Ketosis. XXI. Comparative metabolism of the hexitols.** C. Johnston and H. J. Deuel, jun. (*J. Biol. Chem.*, 1943, **149**, 117—124).—Sorbitol was rather more effective than glucose in increasing liver-glycogen when injected into rats, but muscle-glycogen is increased more effectively and rapidly by glucose. The effect of sorbitol in diminishing ketonuria was only half that of glucose in exogenous, and 25% in endogenous, ketosis. Sorbitol is probably converted into glucose in the liver, and its smaller effect in endogenous as compared with exogenous ketosis may be due to the failure of the damaged (fatty) liver to accomplish this conversion at the normal rate. Mannitol injections produced a slight increase in liver- but no increase in muscle-glycogen, while dulcitol had no effect on either. Experiments with ketosis, however, showed both mannitol and dulcitol to be ketolytic agents, though only half as effective as sorbitol, so that they must be potential sources of glucose. E. C. W.

**Metabolism of acetoin.** W. W. Westerfeld and R. L. Berg (*J. Biol. Chem.*, 1943, **148**, 523—528).—After oral or subcutaneous administration of acetoin to dogs up to 25% was excreted in the urine as βγ-butylene glycol. There were also traces of acetoin but no diacetyl. There was no increase in liver-glycogen. 12—14% of the glycol given by either route was excreted unchanged. 2 g. of acetoin given subcutaneously was fatal to 150-g. rats but they recovered from smaller doses. R. L. E.

**Metabolism and toxicology of ethylene glycol and its diacetate.**—See A., 1943, III, 515.

**Absorption and excretion of fluorides. I. Normal fluoride balance.** W. Machle, E. W. Scott, and E. J. Largent (*J. Ind. Hyg.*, 1942, **24**, 199—204).—Intake and excretion of F were studied in a single healthy subject during 6 months. Average intake was 0·155 mg. per day in food and 0·299 mg. in drink; average daily excretion was 0·377 mg. in urine and 0·039 mg. in fæces. Urinary F content is a useful guide to intake. Over 16 weeks intake exceeded excretion by 5%, but during other periods excretion exceeded intake; it is concluded that under the conditions of this experiment there was no retention of F. E. M. K.

**Calcium metabolism and some present-day problems of nutrition.**—See A., 1943, III, 493.

**Vitamin-*D*, the parathyroid glands, and calcium metabolism.**—See A., 1943, III, 503.

**Metabolism of (1)*N*-acylsulphanilamides in dog.** E. J. Robinson and M. L. Crossley (*Arch. Biochem.*, 1943, **1**, 415—423).—Acetylsulphanilamide is excreted in the urine unchanged after oral administration. Other acylsulphanilamides are partly decomposed; 15% of butyrylsulphanilamide is excreted unchanged. Liver, kidney, and spleen contain an enzyme which can split off the acyl group, and the deacylation does not occur in the alimentary canal. E. R. S.

**Test of mitosis inhibiting substances on *Tubifex* eggs.** F. E. Lehmann (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 24—25).—The inhibitory effect of α-phenyl-β-*p*-anisylethylamine hydrochloride, phenylmescaline hydrochloride, quinol, benzoquinone, 1 : 4-naphthaquinone, 1 : 4-naphthaquinol, 2-methyl-naphthaquinol and -naphthaquinone, and 3-hydroxy-2-methylnaphthaquinone was tested on eggs of the worm *Tubifex*. A. S.

## XX.—PHARMACOLOGY AND TOXICOLOGY.

**Flavicin, an antibacterial substance produced by an *Aspergillus flavus*.** M. T. Bush and A. Goth (*J. Pharm. Exp. Ther.*, 1943, **78**, 164—169).—This substance is an org. acid, sol. in water and ether. 1·6 μg. per c.c. produced complete bacteriostasis for *Strep. hæmolyticus* and *B. anthracis*. Staphylococci required 8 μg. There was hardly any action on the coliform group. Median lethal intraperitoneal dose for mice was 40 mg. per kg. It had a powerful curative effect on pneumococcus I infections in mice. V. J. W.

**Absorption, excretion, and distribution of penicillin.** C. H. Rammelkamp and C. S. Keefer (*J. clin. Invest.*, 1943, **22**, 425—437).—Intravenous injection of 5000—40,000 Florey units of penicillin resulted in high initial plasma concn. followed by an abrupt fall associated with increased excretion in the urine. Traces of penicillin were found in the blood 30—210 min. after the injection. The average excretion after intravenous injection was 58% of the administered dose. Penicillin was rapidly absorbed after intramuscular and slowly after subcutaneous injections. Absorption from the body cavities was delayed, and this was reflected in the slow excretion of penicillin by the kidneys. The total amount found in the urine was somewhat lower than that obtained following intravenous injection. Fluid aspirated from the pleural and joint cavities, 22 and 13 hr. after the injection, showed persistence of penicillin. Absorption of penicillin from the duodenum was rapid, but oral and rectal doses were poorly absorbed. This may be due to the inactivating effect of acid and *E. coli*. In the presence of renal failure, penicillin was not excreted rapidly, and as a result, high concns. were maintained in the blood stream after intravenous injections. The concn. of penicillin in erythrocytes was less than 10% of that in plasma. No penicillin was found in the c.s.f., saliva, or tears, in subjects receiving it intravenously. C. J. C. B.

**Administration of penicillin.** M. E. Florey and H. W. Florey (*Lancet*, 1943, **244**, 387—397).—Penicillin was successfully used in 10 cases with staphylococcal infection, in 3 cases of actinomycosis, in 1 of sulphonamide-resistant streptococcal meningitis, and in 1 of subacute bacterial endocarditis due to *Strep. viridans*. Both generalised and local staphylococcal infections respond to the drug, especially bony lesions. The most practicable method of administration is intramuscular injection, though the intravenous route is effective. 3-hourly injections are necessary to maintain an adequate blood concn. owing to rapid excretion in the urine. Symptomatic improvement may precede fall of body temp. No toxic symptoms were seen. Local penicillin application was also successfully used in 172 infections of the eye, mastoid, wound sinuses, etc., due to staphylococci and streptococci. C. A. K.

**Penicillin *B* : preparation, purification, and mode of action.**—See A., 1943, III, 686.

**Nature of activity of sulphonamides for tubercle bacillus.** R. J. Fitzgerald and W. H. Feinstone (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 27—30).—*In-vitro* activity of sulphonamides against tubercle bacillus is correlated with their acid dissociation consts. and with their resistance to the inhibitory action of *p*-aminobenzoic acid. All the sulphonamides tested had the same activity against tubercle bacillus as against *E. coli*. V. J. W.

**Effects of sulphathiazole and sulphadiazine on man at rest and during exercise.** F. J. W. Roughton, R. C. Darling, H. W. Forbes, S. M. Horvath, S. Robinson, and J. H. Talbot (*Amer. J. Physiol.*, 1942, **137**, 593—598).—Normal males given sulphathiazole or sulphadiazine in daily doses of 2, 3, or 4 g. for 3 days (4—6 equally spaced doses on each day) showed no change at 24 and 72 hr. There were no disturbances in acid–base balance, in ability to perform moderate or exhausting work, or mental ability and co-ordination (Johnson Code test). Disturbances occur after sulphanilamide, especially in exhausting work; its effect may be due to the poisoning of carbonic anhydrase. For ambulant therapy (as far as immediate

effect is concerned) sulphathiazole and sulphadiazine (up to 4 g. per day) can be safely given. M. W. G.

**Concentrations of sulphadiazine in human saliva attained by chewing paraffin containing sulphadiazine.** J. H. Arnett, W. W. Spink, R. Boynton, and S. Agnew (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 54—56).—Concns. up to 8% were attained in saliva by chewing 1·5 g. of paraffin containing 0·325 g. of sulphadiazine. Blood concns. were very small. The method is recommended for pharyngeal infections. V. J. W.

**Distribution of sulphanilamide [after injection into animals].** F. Alexander (*Quart. J. Exp. Physiol.*, 1943, **32**, 21—28).—In sheep, rabbits, and mice, the concn. of sulphanilamide in the blood after intravenous injection corresponds with an even distribution of the substance throughout the body-water, but after 6 hr. the vol. of fluid through which the amount of sulphanilamide in the body would be evenly distributed, if the concn. in that fluid were the same as in the blood, becomes greater than the vol. of the body. In rabbits, 3—72 hr. after injection, the concn. in the kidneys and liver is greater than that in the blood. In the sheep 42—81%, and in the rabbit 50—79%, of the sulphanilamide is recovered in the urine. During the period when the concn. in the body is high, the proportion not recovered is destroyed or converted into a form not detectable by diazotisation. W. McC.

**Sources of error in sulphanilamide determinations.** A. H. S. Holbourn and R. E. Pattle (*J. Lab. clin. Med.*, 1943, **28**, 1028—1033).—When sulphanilamide is determined by forming a diazo-compound and coupling with dimethyl-α-naphthylamine or *N*-(1-naphthyl)ethylenediamine, the diazo-reaction should proceed at room temp. in moderate artificial light for 10 min. in the presence of 10% trichloroacetic acid. With 15% toluenesulphonic acid 20 min. is necessary. The coupling reaction should also be performed in moderate artificial light. The effect of sunlight might be important in tropical countries under camp conditions. C. J. C. B.

**Relative absorption and conjugation of sulphanilamides in man during a 3-hour period.** E. H. Loughlin, R. H. Bennett, M. E. Flanagan, and S. H. Spitz (*Amer. J. med. Sci.*, 1943, **205**, 223—229).—The acid and Na salts of sulphapyridine, sulphathiazole, and sulphadiazine appear in the blood within 5 min. of oral administration. The absorption of the acid salts is favoured by administration of $NaHCO_3$. The absorption of the Na salt, as demonstrated by blood levels obtained, was more rapid and greater than the absorption of the acid salt alone or the acid salt with $NaHCO_3$. Conjugated forms were present in the blood as early as 5 min. after oral administration of the acid and Na salts of these sulphonamide compounds, in some instances being the only form present. Conjugation was more marked when the acid salts of sulphapyridine and sulphadiazine were administered with $NaHCO_3$ than when given alone. C. J. C. B.

**Comparison of acetylation *in vivo* of phenylaminobutyric acid with *p*-aminobenzoic acid and sulphanilamide.**—See A., 1943, III, 671.

**Sulphonamide resistance in gonorrhœa.** J. Petro (*Lancet*, 1943, **244**, 35—38).—*In-vitro* studies in 44 cases of male gonorrhœa showed in 6 a sulphonamide-resistant gonococcus, which agreed with the lack of clinical response to sulphonamide therapy. Investigation of a female host of a resistant strain of gonococcus showed that such a strain can be transmitted and retain its resistance. C. A. K.

**Sulphathiazole in gonorrhœa.** F. J. G. Jefferiss and G. L. M. McElligot (*Lancet*, 1943, **244**, 65—66).—89·5% of 567 cases of acute gonorrhœa were successfully treated with sulphathiazole (6 g. a day for 2 days). The drug is more effective than sulphapyridine and much less toxic. C. A. K.

**Sulphapyridine in pneumococcal peritonitis.** I. Matheson (*Lancet*, 1943, **244**, 367).—2 cases of primary pneumococcal peritonitis were treated with sulphapyridine, one by mouth alone, the other by mouth and intraperitoneally. Both recovered. C. A. K.

**Sulphapyridine anuria successfully treated by ureteric catheterisation.** J. P. Cunniffe (*Brit. Med. J.*, 1943, II, 11).—Case report. I. C.

**Treatment of diffuse peritonitis by direct intraperitoneal introduction of sulphanilamide.** S. Rosenburg and N. M. Wall (*Surg. Gynec. Obstet.*, 1941, **72**, 568—578).—Rats injected intraperitoneally with cultures from cases of peritonitis or acute suppurative appendicitis showed survival in 11 of 21 cases when sulphanilamide (50—250 mg. as a suspension in saline) was given intraperitoneally at the same time (control mortality was 20 out of 21). When the infection was given 6—16 hr. before the sulphanilamide injection the protection was not so great but was still better than with subcutaneously injected sulphanilamide. P. C. W.

**Cultural observations on relative efficacy of sulphonamides in *Shigella dysenteriæ* infections.** A. V. Hardy, W. Burns, and T. DeCapito (*U.S. Publ. Health Repts.*, 1943, **58**, 689—693).—Bacteriostatic action of sulphonamides on *S. dysenteriæ* in culture differed. Sulphadiazine was most rapid in action and appears the most promising for the treatment of clinical infections due to the varieties of *Shigella* encountered in the U.S.A. Sulphasuxidine was more effective than sulphaguanidine, and was superior to sulphadiazine in convalescent and passive carriers of Sonne. C. G. W.

**Clinical response to sulphadiazine therapy.** A. V. Hardy and S. D. Cummins (*U.S. Publ. Health Repts.*, 1943, **58**, 693—696).—5 patients were in a crit. condition, 8 were severely, 6 moderately, and 2 mildly ill. All temp. due to uncomplicated *Shigella* infections were normal at 48 hr. and all but 3 at 24 hr. In cases of comparable severity treated with sulphaguanidine during the preceding year the clinical response was slower and less consistent. Sulphadiazine is a promising chemotherapeutic agent for these enteric diseases. C. G. W.

**Bacteriostatic action of sulphadiazine on *E. typhosa*.** A. V. Hardy (*U.S. Publ. Health Repts.*, 1943, **58**, 833—839).—Sulphadiazine was used in treatment of 19 chronic carriers, 4 convalescent carriers (including 1 treated as a case), 21 clinical cases, and 1 clinical relapse. Quant. cultural tests demonstrated that this sulphonamide has a marked bacteriostatic effect on *E. typhosa* in the enteric tract. The chronic-carrier state was not terminated by this treatment. C. G. W.

**Sulphaguanidine in paratyphoid-*B* infection.** T. F. M. Scott, P. B. Beeson, W. L. Hawley, W. Goode, E. Hathaway, and E. Jackson (*Lancet*, 1943, **244**, 487—490).—Sulphaguanidine was clinically and bacteriologically ineffective in cases of paratyphoid-*B* infection whether given in the acute stage, in convalescence, or in carriers. The drug was well tolerated; of 58 patients 2 had skin rashes and 1 abdominal discomfort. C. A. K

**Sulphathiazole treatment of acute septic endocarditis.** E. Macháček (*Schweiz. med. Wschr.*, 1942, **72**, 1015—1017).—A patient was free from symptoms for 3 months after 100 g. of sulphathiazole. A. S.

**Streptococcic bacteræmia cured with sulphadiazine.** W. M. M. Kirby and L. A. Rantz (*Arch. intern. Med.*, 1943, **71**, 620—629).—Report of a case of infection caused by hæmolytic streptococci of Lancefield group *C*, with a review of the literature. C. J. C. B.

**Sulphapyridine anuria.** R. H. L. Cohen and J. Spencer (*Lancet*, 1943, **244**, 74—75).—Case report. C. A. K.

**Clinical toxicity of sulphapyridine.** H. V. Williams (*Lancet*, 1943, **244**, 105—106).—Enlarged lymph nodes preceded skin rashes seen in 12 cases of gonorrhœa treated with sulphapyridine. 9 of the cases also had pyrexia, and enlargement of liver and spleen. C. A. K.

**Hæmolytic anæmia due to sulphapyridine.** E. I. Jones (*Lancet*, 1943, **244**, 201).—Hæmolytic anæmia (red cell count 1,430,000, hæmoglobin 42%) and jaundice occurred 11 days after starting an 8-day course of 24 g. of sulphapyridine in a man of 59. 1 month later administration of 1 g. of sulphapyridine reduced hæmoglobin concn. from 80 to 66% in 4 days. C. A. K.

**Dangers of repeated administration of sulphadiazine and sulphathiazole in children.** H. W. Fink and J. L. Wilson (*J. Pediat.*, 1943, **22**, 513—517).—177 children were given a sulphonamide drug during 2 or more courses and had no reaction during the first course. 86 were given sulphathiazole; 3 developed febrile reactions, none within the first 48 hr. 91 were given sulphadiazine; 4 developed febrile reactions, only 1 within the first 48 hr. Of the 7 cases reacting (4%), fever appeared first on the 2nd, 3rd, 4th, 5th, 7th, 14th, and 15th days respectively. 2 of 7 children with initial reactions were given a 2nd and a 3rd course of the drug without reaction. Sensitisation to sulphathiazole or sulphadiazine is not common in children, but the occurrence of 1 reaction greatly increases the probability of future reactions, as 5 of 7 children had repeated reactions. C. J. C. B.

**Treatment of leucopenia and granulopenia in rats receiving sulphaguanidine in purified diets.** A. E. Axelrod, P. Gross, M. D. Bosse, and K. F. Swingle (*J. Biol. Chem.*, 1943, **148**, 721—722).—The evidence, based on controlled addition of essential factors, suggests that the leucopenia and agranulocytosis which develop in rats receiving sulphaguanidine in purified diets are caused by inhibition of the synthesis of the essential factors by intestinal bacteria. P. G. M.

**Comparative effectiveness of arsenical compounds and sulphonamide drugs against bacterial infections.** E. E. Osgood, J. M. Armentrout, D. M. Ellis, I. E. Brownlee, and J. Joski (*J. Lab. clin. Med.*, 1943, **28**, 953—962).—From marrow-culture tests it is concluded that neoarsphenamine, in a concn. which can be maintained for half the time for 60 days clinically, if the directions for administration previously published (A., 1942, III, 770) are followed, deserves further trial in the therapy of subacute bacterial endocarditis and bacteræmias, due to *Staph. aureus*, *Strep. hæmolyticus*, *Hæmophilus influenzæ*, and *N. gonorrhœæ*. Sulpharsphenamine and arsphenamine are somewhat less and mapharsen and clorarsen are much less effective than neoarsphenamine in concns. clinically attainable. Sulphathiazole is the best sulphonamide; sulphadiazine should be used if sulphathiazole cannot be tolerated. C. J. C. B.

**Aminobenzoic acid esters of substituted monoalkylamino-alcohols.**—See A., 1943, II, 300.

**Chemotherapy by blocking bacterial nutrients.** H. McIlwain and F. Hawking (*Lancet*, 1943, **244**, 449—451).—*In vitro*, *Strep. hæmolyticus* requires pantothenate for growth. Pantoyltaurine, when present in concns. 200 times those of pantothenate, can prevent growth of this organism by displacing the pantothenate from the points at which it interacts with micro-organisms, like sulphanilamide and *p*-aminobenzoate. In rats the blood ratios of pantoyltaurine to pantothenate could be kept above 200 by frequent subcutaneous injections of pantoyltaurine to allow for rapid excretion. Such dosage for 4 days protected the rats completely from 10,000 lethal doses of a virulent strain of streptococcus (Richards). Smaller dosage was ineffective. Artificial raising of the blood-pantothenate level rendered previously effective doses of pantoyltaurine ineffective. In mice, which have a much higher blood-pantothenate than rats, pantoyltaurine did not protect against streptococcal infection. Sulphonamide-resistant streptococci and some strains of *C. diphtheriæ* are sensitive to pantoyltaurine. C. A. K.

**Accumulation and excretion of atebrin.** E. H. Dearborn, F. E. Kelsey, F. K. Oldham, and E. M. K. Geiling (*J. Pharm. Exp. Ther.*, 1943, **78**, 120—126).—Daily excretion was 4% of daily dose and max. concn. in tissues was attained in 2 weeks. Vomiting can be avoided by coating with cellulose acetate phthalate, when 80% of administered drug is absorbed. Concn. in tissues is lowered when sulphonamides are given at the same time. V. J. W.

**Comparative activity of quinine, quinidine, cinchonine, cinchonidine, and quinoidine against *Plasmodium lophuræ* infections of Pekin ducklings.** A. O. Seeler, E. Dusenbery, and C. Malanga (*J. Pharm. Exp. Ther.*, 1943, **78**, 159—163).—Quinoidine is less active and more toxic than any of the other 4, which are themselves about equal in both respects. V. J. W.

**"Anthiomaline" in lymphogranuloma inguinale.** W. E. Law (*Lancet*, 1943, **244**, 300—303).—Intramuscular or intravenous injections of "Anthiomaline" (Li Sb thiomalate) or Na Sb tartrate were preferable to sulphanilamide or surgical excision in 220 cases of lymphogranuloma inguinale in soldiers in West Africa. C. A. K.

**Propamidine in chronic wound sepsis.** W. R. Thrower and F. C. O. Valentine (*Lancet*, 1943, **244**, 133—136).—*In-vitro* studies showed that propamidine (4 : 4′-diamidinodiphenoxypropane dihydrochloride) has about the same bacteriostatic potency against *Staph. aureus* as sulphathiazole. It is also bactericidal. Effective concns. do not cause hæmolysis or inhibit phagocytosis, and the bactericidal action is not much reduced by the presence of pus. The successful local use of 0·1% propamidine in methylcellulose jelly in 10 cases with infected wounds is reported. C. A. K.

**Propamidine in chronic streptococcal infections.** A. H. McIndoe and A. R. Tilley (*Lancet*, 1943, **244**, 136—138).—11 cases of wounds, infected with β-hæmolytic streptococci and resistant to sulphonamides etc., responded in 4—10 days to 0·1% propamidine in a water-sol. jelly base. C. A. K.

**Propamidine in burns.** G. H. Morley and J. P. Bentley (*Lancet*, 1943, **244**, 138—139).—12 cases of burns were successfully treated with local application of 0·05—0·1% propamidine. C. A. K.

**Propamidine in wounds and burns.** F. Kohn, M. H. Hall, and C. D. Cross (*Lancet*, 1943, **244**, 140—141).—Propamidine was successfully used locally in the treatment of 5 cases of infected wounds (*Staph. aureus*, *B. proteus*, *Ps. pyocyanea*) and in 8 cases of burns. C. A. K.

**Metabolism of mononitroparaffins. II. Metabolic products of nitroethane.** E. W. Scott (*J. Ind. Hyg.*, 1942, **24**, 226—228).—Nitroethane is oxidised by $H_2O_2$ at pH 7, in presence of $FeSO_4$, to acetaldehyde and $HNO_2$. When mixed with rabbits' blood, with or without the addition of $H_2O_2$, 16—18% of nitroethane was converted into acetaldehyde; after intravenous injection acetaldehyde and increased $NO_2$ were demonstrated. (Cf. A., 1942, III, 625.) E. M. K.

**Treatment of tonsillitis, pharyngitis, and gingivostomatitis with bismuth salt of heptadienecarboxylic acid in cacao butter suppositories.** S. Silber (*J. Pediat.*, 1943, **23**, 59—68).—32 patients with tonsillitis, pharyngitis, and gingivostomatitis were treated with suppositories containing Bi heptadienecarboxylate. Subjective symptoms disappeared in 24—48 hr. Temp. dropped within 24 hr., and was normal in 36—48 hr. Local improvement appeared within 24 hr. C. J. C. B.

**Anti-streptococcal action of iodinin. Naphthaquinones and anthraquinones as its main natural antagonists.**—See A., 1943, III, 693.

**Evaluation of antiseptics and other anti-infectious agents.** W. J. MacNeal and N. C. Farnsworth (*J. Lab. clin. Med.*, 1943, **28**, 963—971).—A lecture. C. J. C. B.

**Control of sepsis.** J. M. Robson (*Edinb. Med. J.*, 1943, **50**, 399—414).—A lecture. H. S.

**[Treatment of] oxyuriasis [with gentian-violet].** E. B. Cram (*Amer. J. Dis. Child.*, 1943, **65**, 46—59).—Gentian-violet was the most effective drug. The daily dose for adults was 192 mg., and for children 10 mg. for each year of apparent (mental and physical) age, divided into 3 doses. Treatment was given for 10 days or for 8 days, a rest for 7 days, and then an additional 8 days. C. J. C. B.

**Fate of phenothiazine in domestic animals.**—See A., 1943, III, 671.

**Pectin in treatment of wounds.** C. A. Tomkins, G. W. Crook, E. Haynes, and M. Winters (*Surg. Gynec. Obstet.*, 1941, **72**, 222—227).—75 patients with various types of wounds were treated by the application of solutions of pectin (2—10%) and merthiolate with favourable results. P. C. W.

**Acridine powder in wound therapy.** G. Brownlee and I. M. Tonkin [with C. R. Kennedy] (*Quart. J. Pharm.*, 1943, **16**, 73—78).—The solubilities of the free bases and salts in water, sera, and acid and neutral urine, and the bactericidal activities against *Staph. aureus*, were determined for 5-amino- and 2 : 7- and 2 : 8-diaminoacridine. The data indicate that these substances, preferably as free bases, are suitable as chemotherapeutic wound dressings. Owing to its comparatively low toxicity to leucocytes and high bactericidal activity, 2 : 8-diaminoacridine merits special consideration. F. O. H.

**Action of wetting agents on micro-organisms. II. Synergistic effect of synthetic wetting agents on germicidal action of halogenated phenols.** E. J. Ordal and F. Deromedi (*J. Bact.*, 1943, **45**, 293—299; cf. A., 1943, III, 277).—Na laurylsulphonate and Na dioctyl sulphosuccinate enhanced the germicidal action of buffered solutions containing 2 : 4-di- or 2 : 4 : 6-tri-chlorophenol. The enhancing effect was primarily due to a synergistic action between the wetting agents and the undissociated phenols. F. S.

**Synthesis of physiologically active amines. Amines derived from phenyl styryl ketones.**—See A., 1943, II, 297.

**Pharmacology of methylamino*iso*octene (octin). I. Action on blood pressure.** R. P. Ahlquist (*J. Amer. Pharm. Assoc.*, 1943, **32**, 151—155).—Octin (6-methylamino-2-methylheptene) resembles the sympathomimetic amines in producing a sustained increase in blood pressure when intravenously injected into anæsthetised dogs or rabbits. The pressor action is not affected by cocaine. Repeated doses give decreasing pressor responses and increasing preliminary drops in pressure; the latter are due to direct myocardial depression. F. O. H.

**Pressor response to acetaldehyde and its potentiation by cocaine.** E. E. Nelson (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 23—24).—Pressor response in the dog to 2 mg. per kg. is roughly comparable with that to 1·25 μg. per kg. of adrenaline. Both are potentiated to the same degree by 10 mg. per kg. of cocaine. It occurs equally well after the adrenal vessels are ligatured. V. J. W.

**Potency of new extract of *Convallaria majalis* leaves. Assays by papillary muscle and cat methods.** J. R. Weeks and H. G. O. Holck (*J. Pharm. Exp. Ther.*, 1943, **78**, 180—186).—An extract from 3 g. of dried leaves contained 6·1 U.S.P. XI digitalis units determined by its action on cat papillary muscle, and 8·6 U.S.P. XI units and 11·8 U.S.P. XII units by the cat method. V. J. W.

**Use of pure digitalis glycosides (lanatoside *C*).** A. K. Hrenoff (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 757—761).—Lanatoside *C* had a rapid action, quick elimination, and was excellently tolerated in 32 patients with heart failure. P. C. W.

**Water exchanges due to anæsthetic drugs.** H. G. Barbour (*Anesthesiology*, 1940, **1**, 121—135).—The cells of the cerebral cortex become dehydrated during anæsthesia in mammals. With ether the blood becomes conc. chiefly due to contraction of the spleen. Blood also becomes conc. during morphine administration or withdrawal; the latter condition is associated with œdema of the brain. Amytal decreases blood concn. through taking up of red cells by the spleen and addition of water from intracellular sources. Third-stage anæsthesia is associated with loss of capacity to regulate temp. and this in turn is associated with breakdown of the reflexes governing water movement in response to body-temp. P. C. W.

**Anæsthetic action of methyl *iso*propyl ether.** J. C. Krantz, jun., C. J. Carr, W. E. Evans, jun., and S. E. Forman (*J. Pharm. Exp. Ther.*, 1943, **78**, 115—119).—Satisfactory anæsthesia was produced in the monkey and dog with no toxic results, but its rapid hydrolysis to methyl alcohol in the circulation makes it unsuitable for use in man. V. J. W.

**Production of ventricular tachycardia by adrenaline in *cyclo*propane anæsthesia.** C. R. Allen, J. W. Stutzman, and W. J. Meek (*Anesthesiology*, 1940, **1**, 158—166).—Doses of 3—12 μg. of adrenaline per kg. produced ventricular tachycardia for 30—140 sec. in dogs under *cyclo*propane anæsthesia, but not after decerebration. The decerebrated dogs responded when 5—10 times the doses of adrenaline were given. The tachycardia was annulled by a lesion of the pons, bilateral thoracic sympathectomy, or injections of ergotamine tartrate. Other evidence is adduced that *cyclo*propane stimulates a brain centre above the pons which sends impulses to the heart via the sympathetic nerves. Adrenaline acts directly on the heart so sensitised to produce tachycardia. P. C. W.

**Prophylaxis and treatment of ventricular fibrillation induced by adrenaline during *cyclo*propane anæsthesia.** C. L. Burstein, B. A. Marangoni, A. C. DeGraaf, and E. A. Rovenstine (*Anesthesiology*, 1940, **1**, 167—186).—Ventricular fibrillation was produced in dogs by injecting 10 μg. of adrenaline per kg. during *cyclo*propane anæsthesia (see preceding abstract). Procaine, *p*-aminobenzoic acid, the Ca double salt of benzylsuccinic and *p*-aminobenzoic acids (5—10 mg. per kg.), or Na *p*-aminobenzoate (10—40 mg. per kg.) when administered before the adrenaline, protected the dogs against the fibrillation. Intravenous injection of procaine during the tachycardia that precedes the fibrillation prevented the development of fibrillation and normal conditions were attained after a shifting of the pacemaker to the auricles and finally to the sinus node. Intracardiac injection of procaine was efficient in the treatment of 66% of dogs with ventricular fibrillation. P. C. W.

**Effect of various tissues on detoxication of evipal in dog.** S. J. Martin, H. C. Herrlich, and B. B. Clark (*Anesthesiology*, 1940, **1**, 153—157).—A concn. of 5% is optimal for inducing anæsthesia in dogs by intravenous evipan in doses of 30 mg. per kg. Liver damage by $CHCl_3$ anæsthesia, or removal of the liver and most of the gastrointestinal tract, increases the toxicity of evipan and prolongs the duration of anæsthesia produced by it. *In-vitro* incubation of evipan with various macerated tissues from the dog shows decreased anæsthetic action in the case of liver, skeletal muscle, or spleen (in decreasing order of activity), but no change with brain, kidney, or blood. P. C. W.

**Determination of sodium pentothal in blood.** L. M. Hellman, L. B. Shettles, and H. Stran (*J. Biol. Chem.*, 1943, **148**, 293—297).—Na pentothal shows an absorption max. at 288 mμ. No other barbiturate tested gave significant absorption in the concns. used. The facts are made the basis of a method of determining the drug. 90% recovery of added pentothal is obtained. P. G. M.

**Dangers of pentothal sodium anæsthesia.** A. R. Hunter (*Lancet*, 1943, **244**, 46—48).—A review, and report of a case of dermatitis following pentothal Na anæsthesia. C. A. K.

**Anæsthetic explosion due to static electricity.** E. M. Chivers (*Lancet*, 1943, **244**, 527). C. A. K.

**Analgesia and anæsthesia in labour.** D. G. Tollefson (*West. J. Surg. Obstet. Gynec.*, 1941, **49**, 44—55).—Analysis of 500 patients treated with pentobarbital Na and scopolamine. P. C. W.

**Stovaine analogues.**—See A., 1943, II, 293.

**Local anæsthetic action of esters of phthalic and aminophthalic acid.** L. W. Rowe (*J. Amer. Pharm. Assoc.*, 1943, **32**, 160—163).—Of the 41 esters (cf. Blicke *et al.*, A., 1941, II, 291; 1942, II, 14) tested for local anæsthetic activity (rabbit's cornea and frog's sensory nerve), relative toxicity, and irritant action, only *n*-propyl and *n*-amyl 3-aminophthalate are of promising activity. F. O. H.

**Duration of local anæsthesia in relation to concentrations of procaine and adrenaline.** A. J. Leser (*Anesthesiology*, 1940, **1**, 205—207).—Procaine and procaine + adrenaline were injected subconjunctivally in rabbits, and the duration of corneal anæsthesia was measured. Increasing the procaine concn. above 1% prolonged anæsthesia to a degree proportional to a simple multiple of log concn. Adrenaline 1 : 200,000 prolonged the anæsthesia produced by 1% procaine by 60%; lower concns. are less effective and higher are more effective though liable to damage the tissues. P. C. W.

**Value of oxygen and helium–oxygen mixtures before and after thyroidectomy.** W. M. Boothby and S. F. Haines (*West. J. Surg. Obstet. Gynec.*, 1940, **48**, 662—669). P. C. W.

**Analgesic action of pethidine by mouth.** R. V. Christie (*Lancet*, 1943, **244**, 294—295).—Pethidine was given as an analgesic in oral doses of 25—100 mg. to 335 patients suffering from pain. In 46% of 109 cases it was more effective than a mixture of acetylsalicylic acid, phenacetin, and codeine, in 21% equal, and in 33% inferior. In 13 patients it was less effective than $\frac{1}{6}$—$\frac{1}{4}$ grain of morphine. Toxic effects in 22 cases were slight and included nausea, vomiting, giddiness, and "hangover." There was no evidence of increased tolerance or addiction. C. A. K.

**Pethidine and pain.** G. Fitzgerald and B. McArdle (*Lancet*, 1943, **244**, 296—297).—Pethidine was given by oral, subcutaneous, or intravenous routes in doses of 50—100 mg. to patients with severe pain associated with neurological conditions. Its analgesic action lasts 6—8 hr. and in 7 cases it was more effective than ¼ grain of morphine; its efficacy was equal to but more prolonged than that of tab. codeinæ co. (N.W.F.). Side effects included transient giddiness, pallor, faintness, sweating, blurring of vision, nausea, tremulousness, and anxiety when the drug was given intravenously. C. A. K.

**Morphine-like properties of diphenylethylamine and related compounds.** E. C. Dodds, W. Lawson, and P. C. Williams (*Nature*, 1943, **151**, 614—615).—The capacities to depress the righting reflex in rats, raise the blood-sugar in rabbits, and produce dilatation of the pupil in cats are given for morphine, αβ-diphenylethylamine (*M*3), β-hydroxy-αβ-diphenylethylamine (*M*4), phenyl dimethylaminobenzyl ketone (*M*7), β-hydroxy-αβ-diphenyl-*n*-butyldimethylamine, α-phenyl-β-*cyclo*hexylethylamine, and β-phenyl-α-*cyclo*hexylethylamine. *M*3, 4, and 7, in doses of 50—400 mg. every 4 hr., relieved pain in patients with intractable pain due to pressure symptoms in inoperable cancer. E. R. S.

**Effects of age and sex on safety margin of vinbarbital sodium and of calcium 1-methyl-5-ethyl-5-*iso*butylbarbiturate in rats.** H. G. O. Holck, J. R. Weeks, D. R. Mathieson, and B. Duis (*J. Amer. Pharm. Assoc.*, 1943, **32**, 180—182).—The criterion of safety used is the ratio of 50%-lethal to 50%-effective (*i.e.*, in abolishing the righting reflex) dose (given intraperitoneally). This ratio for vinbarbital Na (Na 5-ethyl-5-β-methyl-Δβ-butenylbarbiturate) had vals. of 2·6, 4·3, 5·7, and 5·9 with rats aged 6—8·5, 3—5, 2, and 1 months, respectively, the change being due almost entirely to a decrease in the 50%-lethal dose with age. The safety ratio for the Ca barbiturate was 4·5 in rats aged 3—6 months. With both barbiturates, the safety margin for certain age groups of rats was greater in the males than in the females. F. O. H.

**Prolonged action of barbiturates.** V. V. Cole (*J. Pharm. Exp. Ther.*, 1943, **78**, 170—173).—Na pentobarbitone, given 22 hr. previously, protects rats against lethal doses of strychnine or picrotoxin. Against strychnine Na phenobarbitone is more effective after 22 hr. than when given 20 min. previously. Against picrotoxin it is equally effective after either interval. V. J. W.

**Effect of iminazole compounds on hæmatins etc.**—See A., 1943, II, 310.

**Pharmacology of gelsemine and dihydrogelsemine.** F. G. Henderson and K. K. Chen (*J. Amer. Pharm. Assoc.*, 1943, **32**, 178—179).—Both the alkaloids relax rabbit's intestinal muscle, contract isolated rabbit's uterus, lower arterial blood pressure in cats, and show the same toxicity (intravenous 50% lethal dose approx. 104 mg. per kg.) in mice. F. O. H.

**Effect of pervitin (deoxyephedrine) on fatigue of the central nervous system.** E. Simonson and N. Enzer (*J. Ind. Hyg.*, 1942, **24**, 205—209).—Pervitin and benzedrine both increased the fusion frequency of flicker when given in doses of 5—7·5 mg. and 10—15 mg. respectively; this is regarded as evidence of diminished fatigue of the central nervous system. Max. frequency of finger movements was used as a test of motor centres; pervitin had little effect on initial frequency but total score in 1 min. was increased after taking the drug. Benzedrine increased initial frequency but did not affect endurance. E. M. K.

**Action of benzole on certain central nervous regulating mechanisms.** F. Guerra (Perez-Carral) (*J. Pharm. Exp. Ther.*, 1943, **77**, 336—342).—In dogs and rabbits, inhalation of 0·5% benzole causes panting and shivering. Transection experiments indicate that the zone of integration for these effects is in the lower part of the pons and middle part of the medulla, possibly in the reticular formation. V. J. W.

**Excretion of metabolic product of salicylic acid.** C. Lutwak-Mann (*Biochem. J.*, 1943, **37**, 246—248; cf. A., 1943, III, 55).—The substance present in rat urine, after administration of salicylates, which causes it to darken in air after addition of alkalis has the properties of gentisic acid. The excretion of this substance is inhibited in rats poisoned with yellow P or $CCl_4$, and increased by phloridzin. Traces of gentisic acid in urine can be detected by the alkali test described. P. G. M.

**Amide-substituted phenylarsine oxides and their derivatives. A group of compounds of possible utility in treatment of syphilis.** H. Eagle, R. B. Hogan, G. O. Doak, and H. G. Steinman (*J. Amer. Chem. Soc.*, 1943, **65**, 1236—1237).—In 16 cases the ratio of treponemicidal activity (rabbit syphilis) to toxicity of phenylarsine oxide is favourably influenced by substitution by a carboxylamide or sulphonamide attached directly or indirectly to the aromatic nucleus. This effect is neutralised by substitution on the N in 14 out of 20 cases. Analyses (not methods of prep.) are recorded for *p*-$C_6H_4X$·AsO, in which X = C(:NH)·OEt, CO·NH·$CH_2$·CH(OH)·$CH_2$·OH, and CO·NH·$CH_2$·CN. R. S. C.

**Detoxication by *p*-aminobenzoic acid of certain quinquevalent arsenical drugs given in massive doses to rats.** J. H. Sandground and C. R. Hamilton (*J. Pharm. Exp. Ther.*, 1943, **78**, 109—114).—Administration by any route of 3—4 daily doses of 1—3 g. per kg. of *p*-aminobenzoic acid raised the % survival rate after lethal doses of carbarsone, tryparsamide, acetarsone, atoxyl, and phenylarsonic acid from 0 for certain of these compounds to 87—100. No inhibition of trypanocidal effect on *T. equiperdum* was produced. V. J. W.

**Quantity of *p*-aminobenzoic acid required to protect rats against high doses of carbarsone and arsanilic acid.** J. H. Sandground and C. R. Hamilton (*J. Pharm. Exp. Ther.*, 1943, **78**, 203—208).—All rats were protected against a 100% lethal dose of carbarsone or atoxyl by 3 previous daily injections of 750 mg. per kg. of *p*-aminobenzoic acid. 15 mg. per kg. protected 50% of rats against a 90% lethal dose. V. J. W.

**Time factor influencing *p*-aminobenzoate protection of rats receiving lethal doses of phenylarsonates.** J. H. Sandground (*J. Pharm. Exp. Ther.*, 1943, **78**, 209—214).—Injection of *p*-aminobenzoate 3 hr. before the arsenical prep. gives very much greater protection than when it is given 30 min. afterwards, and protection diminishes as this time is increased. This is due to the production in the body of tervalent arsenicals, against which *p*-aminobenzoate affords no protection. V. J. W.

**Trypanocidal activity and arsenic content of rat blood following intravenous administration of mapharsen.** H. N. Wright and L. Peters (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 3—4).—12·5 mg. per kg. of mapharsen was injected and As content and trypanocidal activity of blood determined at 15 min. to 10 days thereafter. Up to 1 hr. both are max. and decrease during the next 7 hr. After this trypanocidal activity decreases but As content increases, due presumably to a return of As from tissues to blood in a non-trypanocidal combination. V. J. W.

**Encephalopathy following mapharsen therapy.** A. M. Rabiner, I. S. Freiman, and N. Apter (*Arch. intern. Med.*, 1943, **71**, 836—843).—A case is described in which fatal cerebral symptoms developed after mapharsen therapy. The patient had received 45 injections of the drug, but the last was preceded by a treatment-free interval of 6 weeks. The brain changes consisted of petechial hæmorrhages and necrotic–degenerative lesions. (9 photomicrographs.) C. J. C. B.

**Agranulocytosis following neoarsphenamine.** C. McGibbon and F. Glyn-Hughes (*Lancet*, 1943, **244**, 173—175).—Case report. C. A. K.

**Study of excretion of organic antimonials using a polarographic procedure.**—See A., 1943, III, 657.

**Toxicity of lead azide.** L. T. Fairhall, W. V. Jenrette, S. W. Jones, and E. A. Pritchard (*U.S. Publ. Health Repts.*, 1943, **58**, 607—617).—Investigation of $Pb(N_3)_2$ as an industrial hazard indicated that the storage and distribution of Pb in the tissues following the ingestion of this compound are in general similar to that of other Pb salts. The acute toxic effect of this substance is associated with the $N_3'$ radical rather than with Pb. The effects of administering $NaN_3$ intraperitoneally, subcutaneously, and orally were compared with similar experiments with $Pb(N_3)_2$. The min. lethal dose of $NaN_3$ following injection is 35—38 mg. per kg.; up to 150 mg. per kg. of $Pb(N_3)_2$ could be injected intraperitoneally without causing death. This is equiv. to 66 mg. of $NaN_3$. The effect of exposure to $HN_3$ gas by inhalation was determined at various concns.; it was invariably fatal to rats in amounts beyond 1160 p.p.m. when breathed for 1 hr. The results of this investigation indicate clearly that $HN_3$ is a dangerous gas. C. G. W.

**Design of toxicity tests involving comparison with a standard preparation.** J. H. Gaddum (*Quart. J. Pharm.*, 1943, **16**, 78—86).—Statistical considerations indicate that in routine tests of the toxicity of successive batches of the same drug, a standard prep. should be simultaneously injected each time the test is done. It is convenient to use a smaller dose of the unknown prep. than of the standard and to reject the unknown if it kills more animals than does the standard. The calculation of the results of such tests and the selection of no. of animals and size of doses are discussed. F. O. H.

**Effect of massive doses of strychnine in animals whose central nervous system has been destroyed.** P. E. Galvão and J. Pereira, jun. (*Rev. Brasil. Biol.*, 1942, **2**, 173—175).—In animals whose central nervous system has been destroyed, strychnine in 100 times the lethal dose has no effect. I. C.

**Cardiovascular effect of trichloroethylene.** G. H. Marquardt, J. F. Mallach, and S. C. Werch (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 2—3).—Trichloroethylene causes constriction of the arterial side of capillary loops in man and the frog. It causes no irreversible change in the frog's heart unless complete stoppage occurs. V. J. W.

**Effect of introducing carboxyl group into phenol molecule on toxicity to goldfish.** W. A. Gersdorff (*Amer. J. Pharm.*, 1943, **115**, 159—167; cf. A., 1935, 1275).—Experiments at 27° with phenol, benzoic acid, and *o*-, *m*-, and *p*-hydroxybenzoic acid (concn. 0·09—1·46 g. per l.) show that *m*- and *p*-carboxyl, introduced into the phenol mol., decrease toxicity whilst *o*-carboxyl increases it. W. McC.

**Toxicity of several food-preserving agents.** K. E. Harshbarger (*J. Dairy Sci.*, 1942, **25**, 169—174).—The paired feeding method was used to determine the effects on the growth of rats of Ca or Na propionate, Na benzoate, zephiran, and Na benzoate + glycine. Diets containing 3% of Ca or Na propionate, 1% of Na benzoate, or 3% of Na benzoate + glycine were equal to the controls. 3% of Na benzoate alone diminished the growth rate and 3% of zephiran increased it. N. J. B.

**Mode of action of mustard gas in rats.** E. Rothlin, R. Jürgens, and T. Devrient (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 34—36).—The lethal dose $c \times t = 2000$ ($c$ = mg. of dichloroethyl sulphide per cu.m.; $t$ = 20 min.). 15% of the animals die 2—4 days following gassing with medium lethal doses (acute paralysis and inflammation) of the stomach, 33% after 10—14 days (paralytic ileus of the stomach, small intestines, and, sometimes, of colon with marked inflammation), 55% die later with similar changes, especially in lower small and large intestine, but little involvement of the lungs. Oral administration of 1—10 mg. of mustard gas produces similar gastro-intestinal changes. Prolonged subcutaneous administration of " Calcium-Sandoz " was beneficial. A. S.

**Poisoning by chlorinated naphthalene.** E. Collier (*Lancet*, 1943 **244**, 72—74).—12 cases of dermatitis and 1 case of acute yellow atrophy due to chlorinated naphthalene are reported. C. A. K.

**Phosphorus burns.** E. Obermer (*Lancet*, 1943, **244**, 202).—First aid treatment is described. C. A. K.

**Post mortem examination in cases of suspected poisoning.** H. J. Maldeis (*Amer. J. clin. Path.*, 1943, **13**, 165—168).—A lecture. C. J. C. B.

**Significance of toxicological procedures in medico-legal autopsy.** A. O. Gettler (*Amer. J. clin. Path.*, 1943, **13**, 169—177).—A lecture. C. J. C. B.

**Interbatch variations of Russell viper venom.** R. C. Page and E. J. de Beer (*J. Lab. clin. Med.*, 1943, **28**, 912—915).—Samples of Russell viper venom in $N_2$-filled ampoules were stable over at least 5½ years. C. J. C. B.

**Effect of venom of *Crotalus terrificus terrificus*, and its neurotoxic and hæmolytic fraction, on chicken embryo.** E. Biocca (*Rev. Brasil. Biol.*, 1942, **2**, 143—146).—Chicken embryos after 5, 12, 15, or 18 days of incubation show great resistance to the venom of *Crotalus*, and to its neurotoxic and hæmolytic fraction, when applied on the allantoic membrane. I. C.

**Preparation of scorpion toxin.** A. H. Mohammed (*Lancet*, 1943, **244**, 337).—A method of prep. of potent amorphous or cryst. scorpion toxin is described. 0·01 mg. kills a 100-g. rat. C. A. K.

**CTAB in surgery.** R. Williams, B. Clayton-Cooper, J. McK. Duncan, and E. M. Miles (*Lancet*, 1943, **244**, 522—525).—1% CTAB is very suitable for removing org. matter and is recommended for cleaning instruments, wounds, and skin. It is far more effective than soap in removing dirt and bacteria from the skin but some subjects may develop dermatitis. C. A. K.

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

**The " masculine " component and physical fitness.** C. C. Seltzer and L. Brouha (*Amer. J. phys. Anthrop.*, 1943, [ii], **1**, 95—108).—The " masculine " component is arbitrarily divided into, strong, medium, weak, and very weak categories. The degree of this component is related to physical fitness for hard muscular work. The higher is the physical fitness, the less frequent are the body types weak in masculinity. Weakness in " masculine " component is associated with lower physical fitness. The weaker is the component, the greater is the frequency of poor physical fitness. W. F. H.

**(A) Temperature of the skin of healthy individuals. (B) Influence of various room temperatures on the temperature of the skin.** A. Razgha and L. Zselyonka (*Magyar Orv. Arch.*, 1940, **41**, 504—511, 512—518).—(A) Skin temp. was correlated with room temp. in 49 human males. Skin temp. was highest and most const. on the forehead, and lowest and most variable on the toes. Variations of several degrees in these temp. did not necessarily indicate pathological cases.

(B) Decrease in skin temp. after exposure to room temp. for varying times was greatest on the fingers and toes; the temp. of the latter may take over 1 hr. to reach a steady state in cool room temp. At 20—21° the fall in temp. greatly exceeded that at 23—24°. A. A. M.

**Analysis of industrial morale.** E. D. Chapple (*J. Ind. Hyg.*, 1942, **24**, 163—172). E. M. K.

**Industrial exposure to tellurium : atmospheric studies and clinical evaluation.** H. H. Steinberg, S. C. Massari, A. C. Miner, and R. Rink (*J. Ind. Hyg.*, 1942, **24**, 183—192).—Te is added in pellets to the empty ladle after pouring molten Fe, and dense white fumes are evolved; 90% of the air samples taken near this source showed 0·1—1·0 mg. per 10 cu.m. while away from the source all were under 0·5 mg. Te per 10 cu.m. 62 workers exposed to these conditions were examined after 15 and 22 months, and presented symptoms of Te absorption, garlic odour of breath (71%) and of sweat (13%), dryness of mouth (33%), metallic taste (28%), and somnolence (16%). Te was present in 24-hr. urine specimens, the concn. varying with the intensity of exposure and with the severity of symptoms. E. M. K.

**Effects of daily exposures to arc welding fumes and gases on normal and tuberculous animals.** L. U. Gardner and D. S. McCrum (*J. Ind. Hyg.*, 1942, **24**, 173—182).—The concn. of fume in the animal

room averaged 32 mg. per cu.m. during an 8-hr. day, and of N oxides 2·5—3·2 p.p.m.; exposure was continued for one year. Guinea-pigs were most susceptible and all showed chronic tracheitis and bronchitis when killed; phagocytes packed with Fe particles were seen in the lung alveoli. Exposure to a higher concn. of fumes caused chemical pneumonia, often fatal, in half the animals exposed. Associated primary infection of the lungs with attenuated tubercle bacilli produced tubercles larger and more numerous than in control guinea-pigs; healing followed at the usual rate, although in exposed animals it was by calcification or scar formation instead of by resolution. Infection following exposure took exactly the same course as in controls. E. M. K.

**Effect of irritant gases on the rate of ciliary activity.** L. V. Cralley (*J. Ind. Hyg.*, 1942, **24**, 193—198).—Air containing the gas under test was passed through the excised rabbit trachea at a rate of flow adjusted to imitate conditions of natural breathing, and with temp. and humidity controlled. The tissue showed increasing sensitivity and was discarded 2½ hr. after the death of the animal. Formaldehyde, $Cl_2$, $SO_2$, HCl, $NO_2$, $NH_3$, and $H_2S$ caused cessation of ciliary movement not followed by recovery, formaldehyde being most active and $H_2S$ least. The response of ciliary activity to varying concn. of $SO_2$ agreed well with the effect of inhalation by human subjects. The rate of removal of dyes and bacteria placed on the mucosa of the naso-pharynx was depressed by inhalation of $SO_2$, the degree of depression varying with concn. of $SO_2$. E. M. K.

**Acute nephritis from exposure to carbon tetrachloride.** A. R. Smith (*Ind. Hyg. Bull., N.Y. State*, 1943, **22**, 171—172).—Serious damage may result from an unimpressive exposure to $CCl_4$. The concept of $CCl_4$ poisoning should be revised to include marked gastric irritation and severe kidney damage with little or no evidence of liver disease. C. G. W.

**Effects of exposure to toluene used as component of paints.** L. Greenburg, M. R. Mayers, H. Heiman, and S. Moskowitz (*Ind. Hyg. Bull., N.Y. State*, 1943, **22**, 122—125, 169—170).—Based on a study of 106 painters exposed to the inhalation of toluene of between 100 and 1100 p.p.m. for periods of 2 weeks—5 years, it is concluded that: (1) industrial exposure of man to toluene resulted in enlargement of the liver, macrocytosis, moderately decreased levels of erythrocyte counts, and abs. lymphocytosis; (2) such exposure did not result in leucopenia; (3) early chronic toluene intoxication in man is best evidenced by hepatomegaly and macrocytosis. C. G. W.

## XXII.—RADIATIONS.

**Peculiar growth lesions in frogs induced by irradiation of sperm cells with *X*-rays.**—See A., 1943, III, 491.

**Roentgen irradiation in acute peritonitis.**—See A., 1943, III, 484.

**Effect of centrifuging on production of *X*-ray-induced chromosomal aberrations.**—See A., 1943, III, 451.

**Effect of Roentgen radiation on tumour incidence in *Drosophila melanogaster*. Effects of *X*-rays and neutrons on mouse lymphoma chromosomes. Treatment of cancer with fast neutrons. Treatment of lymphosarcoma with radioactive phosphorus.**—See A., 1943, III, 571.

**Dependence of ultra-violet light hæmolysis on wave-length.** R. Stampfli and W. Wilbrandt (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 37—38). A. S.

**Types of tumour induced by ultra-violet radiation.**—See A., 1943, III, 488.

**Bactericidal effect of ultra-violet rays on non-spore-forming bacteria and mould spores.**—See A., 1943, III, 601.

**Skin sensitivity to light in prurigo æstivalis, eczema solare, and urticaria photogenica.** S. Epstein (*J. invest. Dermat.*, 1942, **5**, 225—241).—Subjects with prurigo æstivalis, eczema solare, or urticaria photogenica were exposed to Hg arc or C arc lamps (with or without filters) and α-, β-, or γ-rays, and the typical skin changes seen after exposure to sunlight were reproduced. The 3 forms of light-sensitivity are not due to sp. λλ. Prurigo lesions develop only when there has been marked radiation erythema and it may follow exposure to α-rays. C. A. K.

**Influence of different amounts of illumination on body weight of birds.**—See A., 1943, III, 487.

**Effect of temperature and light on oxygen consumption and rate of development of *Heliosoma*.** H. B. Roney (*Ecology*, 1943, **24**, 218—243).—Eggs of *H. trivolvis pseudotrivolvis* raised under various light filters over a range of temp. so that all eggs received $1·31 \times 10^{-3}$ g.-cal. per sq. cm. per hr. showed retarded hatching under all light conditions (except white light) when compared with darkness. Retardation was greatest with red light and progressively less with yellow, blue, and green but mortality was greatest under green light, and between 10° and 15°, $O_2$ consumption shows with all lights a series of metabolic peaks, less marked under green than under the other lights. L. G. G. W.

**Physicochemical basis of mitogenetic radiation.**—See A., 1943, I, 260.

## XXIII.—PHYSICAL AND COLLOIDAL CHEMISTRY.

**Plasma model.** C. Wunderly (*Helv. Chim. Acta*, 1943, **26**, 755—768).—The rate of subsidence of red blood corpuscles is slightly diminished by maltose and inulin in somewhat increased concn., uninfluenced by dextrin, but increased by starch and, very appreciably, by glycogen. The action of these polysaccharides is therefore not simply related to the viscosity of their solutions but is probably controlled by the orientation of their macromols., their size, and the reactivity of their terminal groups. Gluconic and glycuronic acid restrict the aggregation of the red corpuscles in the same manner as maltose; they also restrict the action of glycogen. Synthetic mixtures of gelatin and glycogen are regarded as plasma models and on these the influence of pH variation between 3·4 and 7·3 and the effect of addition of $Na_2SO_4$, Na citrate and acetate, NaCl, and NaCNS is studied. Data are given for the dependence of the viscosity of these model mixtures on temp. H. W.

**Turbidity of suspensions of virus-proteins with varying acidity.** E. Pfankuch (*Biochem. Z.*, 1940, **303**, 342—348; cf. A., 1939, III, 729, 947).—Suspensions of tobacco- and aucuba-mosaic virus have low, approx. const. turbidity at pH greater than 4. Turbidity increases greatly from this level at pH 2·5, reaches a max. at approx. 3·2, and decreases rapidly to the const. level at 4. The turbidity of suspensions of potato *X* virus is also low and almost const. at pH greater than 5; it increases from this level at 2·5 to 3·5, remains unchanged between 3·5 and 3·9, and decreases to the low level at 5. This virus undergoes partial, irreversible aggregation and inactivation as acidity increases. The changes produced in the viscosity and turbidity of suspensions of tobacco-mosaic virus by acidity depend on polymerisation. W. McC.

**Polarographic researches on proteins. Fractionation of protein mixtures by electrophoresis.**—See A., 1943, II, 314.

## XXIV.—ENZYMES.

**Hydrogenase and symbiotic nitrogen fixation.**—See A., 1943, III, 602.

**Cytochrome oxidase.** E. Haas (*J. Biol. Chem.*, 1943, **148**, 481—493).—Cytochrome oxidase is extracted from pig heart muscle by aq. $NH_3$–$NH_4Cl$ at pH 10·4 after grinding and autolysis. The yield is 15 times and the activity per mg. 6 times those of previous preps. After ultrasonic treatment the enzyme can be obtained in sol. form; the solution is stable to high-speed centrifuging. Warburg's respiratory enzyme is present in the oxidase solution and takes part in its activity. R. L. E.

**Cytochrome oxidase and *d*-amino-acid oxidase in tumour tissue.**—See A., 1943, III, 490.

**Action of liver-enzymes on amino-acids.** P. Karrer and R. Appenzeller (*Helv. Chim. Acta*, 1943, **26**, 808—814).—The enzyme extract from dry trout liver powder readily degrades *dl*-alanine, -leucine, -aspartic acid, -methionine, and -phenylalanine but is less active towards *dl*-serine and scarcely attacks *dl*-histidine or -glutamic acid. The enzyme prep. from pigeon liver powerfully oxidises *dl*-alanine, -leucine, -methionine, and -phenylalanine but is inactive or nearly inactive towards *dl*-aspartic acid, -histidine, -glutamic acid, or -serine. Extracts from dry, powdered seagull or hen liver are very little active as judged by their behaviour towards *dl*-alanine, -leucine, -aspartic acid, -methionine, -histidine, and -phenylalanine. An extract from dry, powdered trout muscle is without action on *dl*-alanine, -methionine, -aspartic acid, -histidine, and -leucine. dl-*Ethylalanine* is obtained from *dl*-α-bromopropionic acid. H. W.

**Oxidative degradation of histidine in the animal body.** S. Edlbacher and H. Grauer (*Helv. Chim. Acta*, 1943, **26**, 864—882).—A dissociable enzyme, *dl*-histidine-oxidase, which oxidatively degrades *d*- and *l*-histidine is present in rat liver. It has a characteristic pH optimum at 7·0—7·5, is completely inactivated by HCN, but is not affected by $As_2O_3$. It consists very probably of a protein carrier and a co-enzyme which is also present in yeast juice which no longer contains codehydrogenases but in which according to Warburg and Griese various adenine-dinucleotides are present. The co-enzyme is certainly not identical with alloxazine-adenine-dinucleotide. The glyoxaline nucleus is not attacked during the oxidative degradation of histidine by the oxidase. Only small amounts of $NH_3$ can be detected and it remains uncertain whether its disappearance is due to re-aminations. The oxidase of guinea-pig kidney is dissociable, that from cat or pigeon liver is not. H. W.

**Action of ascorbic acid oxidase on reductone.** S. Filitti-Wurmser and J. B. Veiga-Salles (*Rev. Brasil. Biol.*, 1942, **2**, 203—208).—Aniline does not inhibit the oxidation of ascorbic acid, but decreases

by 60% the velocity of oxidation of reductone (hydroxypyruvaldehyde). The oxidation of reductone by ascorbic acid oxidase is one sixth as fast as the oxidation of ascorbic acid by the same enzyme. I. C.

**Liver-catalase activity of tumour-bearing animals.**—See A., 1943, III, 490.

**Change of *d*- into *meso*-tartaric acid by pancreas.** M. Betti and E. Lucchi (*Ber.*, 1940, **73**, [*B*], 777—778).—This change, disputed by Neuberg and Peiser (A., 1940, III, 140), is completely proved (cf. A., 1939, III, 721). R. S. C.

**Specific enzymic determination of nicotinic acid in blood.**—See A., 1943, III, 500.

**Theory of choline-esterase inhibition.** E. A. Zeller (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 43—44).—There is a linear relationship between speed of reaction and anticholine-esterase concn. in a system containing large amounts of acetylcholine, compared with the anticholine-esterase concn. The curve becomes parabolic when the concn. of the latter is increased and follows the equation $y = ax^b$ ($y$ = enzyme inhibition, $x$ = anticholine-esterase concn., $a$ and $b$ = consts.). This relationship held for the following anticholine-esterases: sulphanilamide, pyrazolone, morphine, novocain, percain, eserine. The inhibition with increasing choline concn. was greater than expected. Human brain- and guinea-pig's serum-choline-esterases were less inhibited by the same concn. of an anticholine-esterase than human serum-choline-esterase; the const. $b$ is smaller in the first 2 cases than in the latter. A. S.

**Specificity of the inhibitor in esterase inhibition by tri-*o*-tolyl phosphate.** H. Bloch (*Helv. Chim. Acta*, 1943, **26**, 733—739).—Of the three tritolyl phosphates only the *o*-compound inhibits choline-esterase and serum-lipase in the living rabbit. The *m*- and *p*-compounds are completely inactive whereas *in vitro* the *m*-derivative has a feeble activity. The inhibition is not due to liberated *o*-cresol since this is inactive. Tri-*o*-chlorophenyl phosphate has *in vitro* and *in vivo* an activity similar to that of tri-*o*-tolyl phosphate with which it is isosteric. H. W.

**Inhibition of urease activity by ascorbic acid.** L. A. Elson (*Nature*, 1943, **152**, 49).—With some preps. of urease, activity is completely inhibited by ascorbic acid ($1$ in $2 \times 10^5$); inhibition is prevented by addition of cysteine. A. A. E.

**Occurrence of sterically selective enzymes in the carcinomatous organism.** (A) H. Bayerle and F. H. Podloucky. (B) E. Waldschmidt-Leitz and R. Hatschek (*Z. physiol. Chem.*, 1940, **264**, 189—195, 196—197).—(A) *d*-Leucylglycine and *d*-leucylglycylglycine are not hydrolysed by human normal or carcinoma serum, nor by sera from rabbits with Brown–Pearce tumours, and *dl*-leucylglycine, *dl*-alanylglycine, *dl*-glutamylglycine and its ester are hydrolysed only to the extent of 50% by the above sera. After injection of *dl*-peptides into rabbits the sera are in a very few cases able to hydrolyse *dl*-peptides beyond 50%. Growth of Brown–Pearce tumours in rabbits is accelerated after injection of *d*-leucylglycine. The results of Waldschmidt-Leitz (A., 1942, III, 325) and the conclusions drawn from them are discussed, and their validity is doubted.

(B) The method used by Bayerle and Podloucky for determination of the extent of dipeptide hydrolysis is criticised. J. N. A.

**Detection of *d*-peptidases with *d*-amino-acid oxidase.** H. Herken and H. Erxleben (*Z. physiol. Chem.*, 1940, **264**, 251—253).—*d*-Leucylglycylglycine is hydrolysed by sera from rabbits with Brown–Pearce tumours and from human patients with sigmoid, œsophageal, rectal, and cæcal carcinoma, but it is unaffected by normal sera. The extent of hydrolysis is determined by titration if the *d*-substrate is used, and by use of *d*-amino-acid oxidase with the *dl*-substrate. J. N. A.

**Changes in properties of protyrosinase due to shaking.** T. H. Allen, A. B. Otis, and J. H. Bodine (*Arch. Biochem.*, 1943, **1**, 357—364).—35% of protyrosinase is converted into tyrosinase, 39% into inactive products, by shaking faster than 0·5 oscillation per sec. The changes did not proceed further after 50 min. shaking at 2 oscillations per sec. Variations of temp. and rate of shaking affected only the rate of change. A ppt. was also found containing amounts of protyrosinase and tyrosinase increasing with time of shaking. The decomp. of protyrosinase is a first-order reaction. When saponin, octanol, or dodecanol is present there is less inactive material produced, and a greater proportion of tyrosinase with increasing concn. of saponin etc. Na dodecyl sulphate converts protyrosinase entirely into tyrosinase, and when the product is shaken 40% is inactivated. No inactivation occurs when the concn. of dodecyl sulphate is above 0·0006M. E. R. S.

**Enzymic degradation of cell wall substances. III.** D. H. F. Clayson (*Chem. and Ind.*, 1943, 298—301; cf. A., 1943, III, 142, 273).—The saccharifying powers of crude enzyme extracts from moulds on oat straw subjected to various pretreatments are compared. Enzyme activity was greatest at about 45°, with pH approx. 4·0. A yield of about 15%, as glucose anhydride, is the highest so far recorded. R. H. H.

**Equilibria of isomerase and aldolase, and the phosphorylation of glyceraldehyde phosphate.** O. Meyerhof and R. Junowicz-Kocholaty (*J. Biol. Chem.*, 1943, **149**, 71—92).—A method is described for the determination of very small amounts of *d*-3-glyceraldehyde phosphate, involving oxidation by I to *d*-3-phosphoglyceric acid, pptn. as Ba salt, liberation of the free acid, and determination polarimetrically in presence of $MoO_4''$. The equilibrium const. for isomerase (catalysing the isomerisation of dihydroxyacetone phosphate to glyceraldehyde phosphate) is 20—25, using either the pure enzyme or preps. of zymohexase (isomerase + aldolase, which breaks down hexose diphosphate into aldotriose and ketotriose). The equilibrium const. for aldolase is about $1{\cdot}33 \times 10^{-4}$ at 38—40°. The equilibria are unaffected by the presence of inorg. $PO_4'''$, even if Warburg's "oxidising enzyme" and cozymase are also present. A diphosphoglyceraldehyde cannot therefore be formed, and in the oxidation of glyceraldehyde phosphate by cozymase, the rôle of inorg. $PO_4'''$ must be to form a loose additive compound, thereby making the carbonyl group more unstable towards oxidising agents. E. C. W.

**Effect of salt and substrate concentration on activity of adenosine triphosphatase.** J. W. Mehl and E. L. Sexton (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 38—40).—Rate of formation of inorg. P is increased by increasing KCl concn. or pH or by presence of glycine. It is independent of substrate concn. if enzyme concn. exceeds a known min. V. J. W.

**Occurrence of free and bound phosphatases in seeds.**—See A., 1943, III, 535.

**Essential steps in the enzymic breakdown of hexoses and pentoses. Interaction between dehydrogenation and fermentation.**—See A., 1943, III, 520.

**Co-enzymic reactions.** J. K. Parnas (*Nature*, 1943, **151**, 577—580).—A review. E. R. S.

**Mechanism of polysaccharide production from sucrose.**—See A., 1943, II, 294.

## XXV.—MICROBIOLOGICAL AND IMMUNOLOGICAL CHEMISTRY. ALLERGY.

**Synthesis of riboflavin by a yeast.** P. R. Burkholder (*Proc. Nat. Acad. Sci.*, 1943, **29**, 166—172).—In a medium consisting of $KH_2PO_4$ 0·5 g., $MgSO_4,7H_2O$ 0·5 g., $CaCl_2,2H_2O$ 0·3 g., $(NH_4)_2SO_4$ 2·0 g., KI 0·1 mg., glucose 20 g., asparagine 2·0 g., biotin methyl ester 1·0 μg. per l. with trace elements certain strains of yeast grown for 4—6 days produced 10—60 μg. of riboflavin per ml. of fermented liquor. Riboflavin production was favoured by appropriate concn. of salts, aëration, temp. of 30°, N sources such as asparagine, glycine, or methionine, and C sources such as sucrose, glucose, and fructose. Growth was favoured but riboflavin production was inhibited by hydrolysed casein, maltose, or galactose, and anaërobiosis. Small amounts of CN′ inhibited growth but stimulated riboflavin production. F. S.

**Enzymic reduction of furil and furoin.** C. Neuberg, H. Lustig, and R. N. Cagan (*Arch. Biochem.*, 1943, **1**, 391—395).—Yeast fermenting sucrose converts furil into furoin and hydrofuroin, which are optically active. E. R. S.

**Behaviour of $\Delta^1$-unsaturated steroid ketones on reduction by fermenting yeast.**—See A., 1943, II, 304.

**Pyruvic acid in yeast, blood, and spleen.**—See A., 1943, III, 505.

**Response of bacteria, yeast, and rats to [hydrogen] peroxide-treated biotin.**—See A., 1943, III, 501.

**Inhibition of bacteriostatic action of sulphanilamide by yeast extracts.**—See A., 1943, III, 581.

**Antagonistic fungi, *Aspergillus fumigatus* and *A. clavatus*, and their antibiotic substances.** S. A. Waksman, E. S. Horning, and E. L. Spencer (*J. Bact.*, 1943, **45**, 233—248).—*Fumigacin* is formed in Czapek–Dox medium during the 5th—9th day of the growth of *A. fumigatus*. It is conc. by adding norit to the culture filtrate, treating the norit with ether, and finally extracting with $CHCl_3$. It is insol. in water, crystallises from alcohol, contains C 62·7 and N 3·7%, and has m.p. 185—187°. Its chemical nature is unknown. It is active against Gram-positive bacteria and has little effect on Gram-negative bacteria or fungi. *A. clavatus* in the same medium produced clavacin, the greatest activity being at the 6th day. Clavacin is sol. in ether, $CHCl_3$, alcohol, and water and appears to be an acid. It is nearly as active against Gram-negative as against Gram-positive organisms. It has not yet been cryst. Both substances are highly toxic to animals. F. S.

**Phosphorus metabolism in moulds.** T. Mann (*Nature*, 1943, **151**, 619—620).—*Aspergillus niger* forms a polyphosphoric acid aërobically from orthophosphate, but not anaërobically. It is

extracted from the mycelium by trichloroacetic acid and is hydrolysed completely at 100° by N-HCl to orthophosphate. E. R. S.

**Isolation of fungus cerebrin from mycelium of *Aspergillus sydowi*.**—See A., 1943, II, 315.

**Biotin and growth of *Fusarium avenaceum*.** W. J. Robbins and R. Ma (*Bull. Torrey Bot. Club*, 1941, **68**, 446—462).—A strain of *F. avenaceum* which failed to develop in minerals–sugar media grew normally when the medium was solidified with agar. This effect is attributed primarily to the presence of biotin in agar although the growth produced exceeded that to be accounted for by the biotin content. Other unidentified growth-substances are probably concerned. Biotin is almost completely extracted from agar by aq. pyridine. A. G. P.

**Alcoholic fermentation with *Rhizopus suinus*. Inhibition of growth by aneurin.** W. H. Schopfer (*Arch. Sci. phys. nat.*, 1943, [v], **25**, *Suppl.*, 40—45; cf. A., 1943, III, 600).—Production of alcohol (later destroyed) by *R. suinus* at 22° in the dark on a buffered (initial pH 4) glucose medium containing asparagine or $NH_4$ tartrate, $MgSO_4$, and $K_2HPO_4$ is greatly increased by addition of aneurin, the increase being accompanied by a parallel retardation of growth. Added inositol slightly inhibits production of alcohol in presence and absence of added aneurin. The increase in production of alcohol caused by aneurin is accompanied by increased consumption of glucose. Added inositol counteracts the retardation of growth produced by aneurin. Added alcohol also retards growth and it is concluded that the action of alcohol on growth is primary and that of the acids (*e.g.*, fumaric) produced at the same time, secondary. W. McC.

**Pseudopyridoxine and certain fungi.** W. J. Robbins and R. Ma (*Proc. Nat. Acad. Sci.*, 1943, **29**, 172—176).—The addition of 1 " mμ mole " of pyridoxine per 25 ml. to a deficient basal mineral–glucose medium containing asparagine, biotin, and thiamin permitted considerable growth of the filamentous fungi *Ophiostoma catonianum*, *Ceratostomella ips* No. 255, *C. microspora*, *C. montium*, *C. multiannulata*, *C. piliferum*, *C. pluriannulata*, and *C. ulmi*. Pyridoxine could not be replaced by *dl*-alanine. There was no evidence for the existence of ψ-pyridoxine, a more active form of pyridoxine (Snell and Guiravol, A., 1943, III, 607) which is produced when pyridoxine is autoclaved with amino-acids. F. S.

**Lysins in blood of rats infected with *Trypanosoma cruzi*.** N. Denison (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 26—27).—Serum from such rats caused lysis of trypanosomes in culture in 2 hr., and, when 5 times diluted, in 3—5 hr. If the reticuloendothelial system was blocked with trypan-blue less lysin was produced. V. J. W.

**Slide agglutination and intradermal tests in experimental *Trypanosoma cruzi* infections.** H. A. Senekjie (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 56—59).—Flagellar (*H*) and somatic (*O*) agglutinins, skin-sensitising antibodies, and lysins are present in blood of rabbits infected or immunised with this organism. V. J. W.

**Stable trypanosome complement-fixing antigen.** D. J. Davis (*U.S. Publ. Health Repts.*, 1943, **58**, 775—777).—A stable antigen, easily prepared by freezing and thawing the cultural forms of *Trypanosoma cruzi* in saline with merthiolate (1 : 10,000), has fixed complement satisfactorily in the presence of sera from human beings and animals infected with this trypanosome. C. G. W.

**Kahn verification test in malaria.** A. de Groat (*J. Lab. clin. Med.*, 1943, **28**, 882—885).—The Kahn verification test was applied to a group of clinically non-syphilitic soldiers with malaria giving positive serological reactions for syphilis. These soldiers gave the general biological (non-syphilitic) type of reaction, indicating that the verification test can be employed as an aid in establishing the differential diagnosis of malaria and syphilis. C. J. C. B.

**Adaptation of Kahn shaker for homogenising specimens for bacteriological procedures.** L. Buxbaum (*J. Lab. clin. Med.*, 1943, **28**, 1000—1001). C. J. C. B.

**Rapid detection of production of acetylmethylcarbinol.** L. M. Coblentz (*Amer. J. Publ. Health*, 1943, **33**, 815—817).—Using a modified Voges–Proskauer test, a positive result could be obtained in 7 hr. at 30°. C. J. C. B.

**Effect of serum on hæmolysis by gramicidin and tyrocidine.** F. C. Mann, D. Heilman, and W. E. Herrell (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 31—33).—Presence of 5% of horse serum decreases the hæmolytic activity of gramicidin and abolishes that of tyrocidine on sheep red cells. V. J. W.

***In-vitro* and *in-vivo* studies on gramicidin, tyrothricin, and tyrocidine.**—See A., 1943, III, 510.

**Inhibition of bacterial respiration by sulphanilamide and by its inactive isomerides.**—See A., 1943, III, 506.

**Reactions of lower organisms to sulphonamides and *p*-aminobenzoic acid. Quantitative studies of sulphonamide resistance.**—See A., 1943, III, 507.

**Influence of trypsin and anti-protease on bacterial growth and sulphonamide action.**—See A., 1943, III, 519.

**Reactions and antibacterial properties of *p*-diazine di-*N*-oxide.**—See A., 1943, II, 309.

**Carbon monoxide inhibition of azotobacter in microrespiration experiments.** P. W. Wilson and C. J. Lind (*J. Bact.*, 1943, **45**, 219—232).—In micro-respiration experiments N fixation by *Azobacter vinelandii* was specifically inhibited by 0·1—0·6% of CO, a concn. 10 times greater than is required for a similar inhibition of the symbiotic system in inoculated red clover plants. There was a small inhibition of $NO_3'$ reduction and a slight non-sp. influence on growth by CO. *Azobacter* kept on N-free medium in air assimilated $NH_3$ and urea more rapidly than in $N_2$. The rate of uptake of $NO_3'$, $NO_2'$, asparagine, aspartate, and glutamate was low and variable. After adaptation, the rate of assimilation of $NO_3'$ and $NO_2'$ equalled $N_2$ fixation and asparagine uptake improved. F. S.

**Methane fermentation. VI. Influence of carbon dioxide concentration on the rate of carbon dioxide reduction by molecular hydrogen.** H. A. Barker (*Proc. Nat. Acad. Sci.*, 1942, **29**, 184—190).—In suspensions of *Methanobacterium omelianskii*, a methane-producing bacterium, the rate of $CO_2$ reduction, measured manometrically at 37° in an atm. of $H_2$, was increased by raising the $HCO_3'$–$CO_3''$ content of the medium within the pH range 6·0—7·8. Outside this pH range the rate was determined by $[H^+]$ or $[HCO_3']$. F. S.

**Iron requirements of heterotrophic bacteria.** W. S. Waring and C. H. Werkman (*Arch. Biochem.*, 1943, **1**, 425—433).—Max. growth on glucose–$(NH_4)_2SO_4$ of *Aerobacter aerogenes* and *indologenes*, *Escherichia coli*, and *Klebsiella pneumoniæ* occurred with 0·02—0·03 p.p.m. of Fe. *Pseudomonas aeruginosa* required 0·09 p.p.m. and *Serratia marcescens* 0·5 p.p.m. of Fe for max. growth and 0·3 p.p.m. for max. production of prodigiosin. The Fe requirement of an organism varies with the type of cytochrome system in the organism. The Fe content of an organism increases with increasing Fe content of the medium. Autoclaving of media containing less than 2 p.p.m. of Fe has no effect; more than 3—4 p.p.m. caused a decrease of growth. Positively charged $Fe(OH)_3$ sol reduces the $O_2$ uptake of *A. indologenes*, but not after treatment with K citrate (reversing the sign of the charge). Powdered Fe was as readily available as $FeCl_3$; $FeCO_3$, $Fe(OH)_3$, $FeAsO_4$, and $Fe_4[Fe(CN)_6]_3$ less readily; $Fe_4(P_2O_7)_3$, FeS, and $Fe_2O_3$ were much less readily available. The usual culture media contain adequate Fe for bacterial growth. E. R. S.

**Comparison of presumptive and confirmative media for bacteria of the coliform group and for fœcal streptococci.** A. A. Hajna and C. A. Perry (*Amer. J. Publ. Health*, 1943, **33**, 550—556).—Confirmation of coliform bacteria by inoculation of secondary tubes of brilliant-green–lactose–bile broth was as efficient as the more involved eosin–methylene-blue plate method. Confirmation of *Escherichia coli* by inoculation of secondary tubes of buffered lactose broth at 45·5° was also a simple practical method. The addition of 0·15% of Bacto bile salts No. 3 to this medium, with slight modification in the amounts of other ingredients, promises a method of efficiency comparable to the brilliant-green–lactose–bile broth method for the coliform group. Lauryl sulphate–tryptose broth gave many more positives for the coliform group than standard lactose broth with few false presumptive reactions. A new " EC " (*E. coli*) medium was as efficient for the isolation of coliform bacteria as lauryl sulphate–tryptose broth and gave no false presumptive on 147 samples of treated and untreated waters. This medium exhibits fæcal streptococci and other Gram-positive bacteria but not coliform bacteria. The " EC " medium can be used for the isolation of coliform bacteria at 37° or of *E. coli* at 45·5°. It can be used either as a primary medium for the growth of *E. coli* or as secondary medium for confirmation. A new " SF " medium is highly sp. at 45·5° for fæcal streptococci. The mere presence of growth and acid in this medium at 45·5° is confirmatory evidence of fæcal streptococci. The medium may be inoculated directly with water, milk, or sewage, or from other primary media, such as standard lactose broth. C. J. C. B.

**Application of decimal reduction time principle to study of resistance of coliform bacteria to pasteurisation.** L. I. Katzin, L. A. Sandholzer, and M. E. Strong (*J. Bact.*, 1943, **45**, 265—272).—The decimal reduction time is defined by $DRT = 2{\cdot}3/K = (t_2 - t_1)/\log_{10}(C_1/C_2)$, where $K$ is the unimol. reaction rate const., and $C_1$ and $C_2$ are the initial and final concns. of bacteria subjected to a const. lethal temp. for $(t_2 - t_1)$ min. The *DRT* of 66 strains of coliform bacteria in milk at 61° were all less than 2 min. F. S.

**Experimental blastomycosis in guinea-pig.** L. M. Nelson (*J. invest. Dermat.*, 1942, **5**, 257—267).—Guinea-pigs were experimentally infected by intraperitoneal injection of *Blastomyces dermatitidis*. 2—4 weeks later a tuberculin-like reaction occurred 48 hr. after intracutaneous injection of killed *Bl. dermatitidis*, the broth in which they grew, a saline extract of the organisms, or the polysaccharide of the organisms. C. A. K.

**Brucellosis : epidemiology, diagnosis, and control.** C. F. Jordan, I. H. Borts, D. M. Harris, and J. R. Jennings (*Amer. J. Publ. Health*, 1943, **33**, 773—779). C. J. C. B.

**Diagnosis of wound infection by bacterial enzymes.** D. McClean, H. J. Rogers, and B. W. Williams (*Lancet*, 1943, **244**, 355—360).—Guinea-pigs were infected intramuscularly with strains of *Cl. welchii*, *Cl. septicum*, and *Cl. œdematiens*, and killed 2—24 hr. after inoculation. Samples of œdema fluid, heart blood, injected muscle, and urine were immediately tested for the presence of hyaluronidase or lecithinase by methods which are described in detail. The enzymes produced by the bacteria can be detected in œdema fluid as soon as sufficient has formed for collection, and their presence is suggested as a test for early wound infection. C. A. K.

**Surface growth of *Clostridium welchii*.** E. Steinfield and M. Watson (*J. Lab. clin. Med.*, 1943, **28**, 998—999).—Test-tubes containing 12—15 c.c. of plain agar are heated to m.p. and allowed to cool slowly. Sterile defibrinated blood is added and then 50 mg. of Na ascorbate. The treated blood agar is poured into sterile Petri dishes and allowed to solidify. Within ½—1 hr. the plate darkens owing to the reduction of hæmoglobin. After streaking cultures or material presumably containing *Cl. welchii* over the surface of the treated blood agar, a Cellophane seal is placed over the agar surface; the $O_2$ tension is thus kept at a min. The plate can be examined by transmitted light and handled in any position. Subcultures can be made by lifting the edge of the Cellophane cover with forceps without undue distortion of the surface. C. J. C. B.

**Nutritional requirements of *C. diphtheriæ* and *L. casei*.** F. W. Chattaway, F. C. Happold, M. Sandford, B. Lythgoe, and A. R. Todd (*Nature*, 1943, **151**, 559; cf. A., 1941, III, 117).—A second growth-promoting factor for *C. diphtheriæ*, present in liver, is adsorbed by C, insol. in org. solvents, and unstable in alkaline though stable in acid solution. It differs from a growth factor for *L. casei* ϵ by giving a sol. Ag salt and by not being extracted by *p*-cresol at pH 3. E. R. S.

**Reactions from diphtheria toxoid.** J. H. Landes (*Amer. J. dis. Child.*, 1943, **65**, 519—522).—10 of 12 reactions in 7794 children occurred in previously immunised school children. There were no reactions following the first dose of 0·25 c.c. of fluid toxoid in 949 unimmunised white school children. There was no difference in the frequency of reactions between 5843 white and 1951 negro children or among the various age groups. C. J. C. B.

**Adaptation of gonococci in culture to sulphanilamides and other bactericides.** R. Spitzer (*Schweiz. Z. Path. Bakt.*, 1942, **5**, 275—292).—Different strains of *N. gonorrhœæ* showed uniform sensitivity towards additions of Hg oxycyanate and trypaflavine to the culture media but marked variations towards sulphanilamide, albucid, sulphapyridine, and especially sulphathiazole ranging from 1 : 4 to 1 : 128. The strains could be adapted to tolerate increasing concns. of sulphathiazole (up to a ratio of 1 : 1024 and averaging 1 : 364), sulphanilamide (up to 1 : 40, average 1 : 20), and trypaflavine (up to 1 : 50, average 1 : 40). This adaptation was sp. for the whole group of sulphanilamides, and persisted in ordinary ascites agar. Seroresistance was equally raised but phagocytosis and thermal resistance were not affected by these passages. E. M. J.

**Amino-acid requirements of *Lactobacillus casei*.** B. L. Hutchings and W. H. Peterson (*Proc. Soc. Exp. Biol. Med.*, 1943, **52**, 36—38).—Culture medium was made up to contain 20 mg.-% of each amino-acid except the one under test. No or slight growth occurred in absence of leucine, serine, phenylalanine, glutamic acid, valine, aspartic acid, cystine, arginine, tryptophan, or tyrosine, and was below normal in absence of alanine, *iso*leucine, threonine, lysine, methionine, and histidine. V. J. W.

**Use of *Lactobacillus casei* in microbiological assays.**—See A., 1943, III, 500.

**Influence of buffer and glucose in the *Lactobacillus casei* assay for pantothenic acid.**—See A., 1943, III, 577.

**Sulphapyridine bacteriostasis of *Lactobacillus arabinosus* and its counteraction.**—See A., 1943, III, 581.

**Abnormal agglutination reactions of a strain of *Leptospira icterohæmorrhagiæ*.** J. C. Broom and H. C. Brown (*Brit. Med. J.*, 1943, I, 783—784).—The sera of 34 cases of jaundice agglutinated a strain of *L. icterohæmorrhagiæ* (Jackson) which had been used for 8 years as a diagnostic antigen. These positive sera failed to produce adhesion with the same strain, failed to agglutinate other strains of *L. icterohæmorrhagiæ*, or protect guinea-pigs against infection with a virulent culture of *Leptospira*. No *Leptospira* were found in the urine, no infection could be produced in guinea-pigs, and the blood picture was not typical of Weil's disease. The non-sp. agglutinations are probably due to an acquired abnormal susceptibility of the strain used as antigen; this instability was lost on further subculture. I. C.

**Microbiological assay for *p*-aminobenzoic acid [using *Neurospora crassa*].**—See A., 1943, III, 577.

**Bacteriological aspects of meningococcal infection.** L. Thomas and J. H. Dingle (*J. clin. Invest.*, 1943, **22**, 353—359).—"Quellung" in sp. antiserum was a satisfactory method for the rapid identification of group I strains, and agreed in every instance with typing by agglutination. "Quellung" was enhanced by culturing the organisms in sp. antiserum for several hr. This reaction was not observed with group II strains. All except 2 of the 12 group I strains isolated had a high virulence for mice. There was no difference between the virulence of nasopharyngeal and c.s.f. strains of group I, between group I strains from cases and contact carriers, nor between group I strains from epidemic and sporadic cases. 3 of 5 group II strains from c.s.f. of cases were highly virulent for mice, while 2 were of low virulence. All of the carrier strains of group II of atypical meningococci were of low virulence. C. J. C. B.

**Immunological aspects of meningococcal infection.** L. Thomas, H. W. Smith, and J. H. Dingle (*J. clin. Invest.*, 1943, **22**, 361—373).—The agglutination test was positive in 33 of 34 convalescent sera from group I cases and in 2 convalescent sera from group II cases. Agglutinins were also present in 12 sera in the acute stage but a rise in titre between acute and convalescent sera was found in all except 2 patients. Agglutinins appeared on the 7th day. The sera of 4 carriers of group I organisms had agglutinin titres of 1 : 32—1 : 128. The sera of 5 of 6 carriers of group II organisms had no agglutinins for homologous or group I strains. The mouse-protection test was impracticable for sera from patients who had received sulphonamide, because of the protective action of small amounts of drug. Of 2 group II carrier sera, 1 protected against 100,000 and the other against 10 50% lethal doses. Of 14 group I contact sera, 1 protected against 100,000 and 1 against 10 50% lethal doses. "Quellung" was produced by 6 convalescent sera from group I cases when the organisms were incubated in the serum for 2—4 hr. The plate pptn. test was negative with 4 convalescent sera which possessed group I antibody by other methods. The complement-fixation test was positive in 16 of 26 convalescent sera from group I cases. Bactericidal tests with the fresh, undiluted sera of 3 group I patients against their own strains showed a diminution in bactericidal property during convalescence. C. J. C. B.

**Comparison of nasopharyngeal swab and cough plate in diagnosis of whooping cough and *H. pertussis* carriers.** J. J. Miller, C. W. Leach, T. M. Saito, and J. B. Humber (*Amer. J. Publ. Health*, 1943, **33**, 839—843).—With 342 comparative tests, in every week of the disease and in each age group the swabs yielded a higher % of cultures positive for *H. pertussis* than did the cough plates. The use of both methods was better than either alone. C. J. C. B.

**Relation between amounts of pertussis antigen injected and production of agglutinins.** J. A. Toomey, W. S. Takacs, R. Averill, and N. Lewis (*J. Lab. clin. Med.*, 1943, **28**, 835—842).—Rabbits given 10 doses of phase *B H. pertussis* organisms + toxin at 20 mg. per c.c. show a higher agglutinin titre against phase *B* organisms than the sera of those given the same no. of doses of 4 mg. per c.c. 20 and 45 mg. of phase *B* organisms alone for 10 injections produce comparable agglutinin titres in rabbits against phase *B* organisms and some demonstrable agglutinins against heterologous phase *A* bacilli. When rabbits are injected weekly over a long period with massive amounts of phase *B* organisms as antigen, there is no increase in titre against the phase *B* organisms, but in some instances there may be increased agglutinin titre against phase *A* bacilli as high as 1 : 160. C. J. C. B.

**Use of photronreflectometer for determination of optimal ratio between pneumococcus type I SSS and antipneumococcus type I rabbit serum.** D. S. Martin (*J. Lab. clin. Med.*, 1943, **28**, 870—881).—A photoelectric method for determining the relative pptn. rates obtained with various concns. of antigen and antibody is described. From a study of 11 const. antibody–varying antigen series, using pneumococcus type I polysaccharide and antipneumococcus type I rabbit serum, it was found that the fastest initial pptn. occurs in the mixture in which antigen and antibody are in optimal proportions, as shown by absence of both reagents in the supernatant, and the max. galvanometer deflexion is obtained in the region of antigen excess. The galvanometer deflexion 5 min. after mixing is directly proportional to the antigen concn. provided excess of antibody is present. C. J. C. B.

**Two artificial "*O*" forms of *Proteus vulgaris* and their enzymic properties.** O. Felsenfeld and G. Heilbrunn (*J. Bact.*, 1943, **45**, 273—275).—On gelatin the "*H*" form showed mainly a reaction of carboxylprotease. The "*O*" I form, originally formed by the activity of "reversible" narcotics and inhibitory agents (sulphonamides, ethanol, and ethylcarbamide), showed comparable activity with only quant. differences. The "*O*" II form, produced by "irreversible" narcotics and chemicals (chloral hydrate, phenol, malachite-green), showed an activity of aminoprotease, thus establishing a qual. difference. F. S.

**Metabolism of *Pseudomonas fluorescens* as interpreted by relationship between metabolic end products and electrical conductivity of culture medium.** L. A. Kazal (*J. Bact.*, 1943, **45**, 277—292).—In a synthetic glutamic acid medium, this amino-acid was utilised by *Ps. fluorescens* to produce $NH_3$, $CO_2$, and bacterial protoplasm as the principal metabolic products. Mathematical correlation of the chemical variables involved in this metabolic process indicated a const. mechanism of metabolism during 10 days' growth. There was also a linear relationship between sp. conductivity and $NH_3$-N is solution. The physical and chemical changes in the medium were therefore definitely correlated and the whole process of metabolism could be integrated by the measurement of one variable, sp. conductivity. F. S.

**Acute staphylococcal infection.** E. C. B. Butler and F. C. O. Valentine (*Lancet*, 1943, **244**, 194—197).—32 cases are described. The assessment of severity of acute staphylococcal infections involves repeated quant. blood-cultures (no. of colonies per c.c.), the antileucocidin titre, and clinical features including the primary focus. The val. of sulphonamide therapy is difficult to determine. C. A. K.

**Degradation of glucose by *Staphylococcus albus*.** L. S. Fosdick and G. W. Rapp (*Arch. Biochem.*, 1943, **1**, 379—389).—*S. albus*, isolated from human saliva or tooth scrapings, contains enzymes which promote all of the reactions of the Embden–Meyerhof scheme, but lactic acid is produced only under anaerobic conditions. It produces γ-hydroxy-α-ketovaleric acid and its aldehyde from Na pyruvate in $PO_4'''$ buffer. *S. albus* plays no part in dental caries but may have an inhibitory action. E. R. S.

**Group *A* hæmolytic streptococcus antibodies. Griffith type agglutinin and antistreptolysin titres in normal men and in acute infections.** L. A. Rantz, W. M. M. Kirby, and A. H. Jacobs (*J. clin. Invest.*, 1943, **22**, 411—417).—Agglutinins for group *A* hæmolytic streptococci of various Griffith types are present in the sera of healthy persons and are discovered more frequently and in higher titre in those with a past history of hæmolytic streptococcus infection. An agglutinin response to the infecting type of hæmolytic streptococcus, and often to other types as well, occurred in 50% of 24 cases of scarlet fever. Sulphonamide medication occasionally interfered with the development of agglutinins and antistreptolysin. C. J. C. B.

**Group *A* hæmolytic streptococcus antibodies. Griffith type agglutinin and antistreptolysin titres in carriers and non-carriers.** L. A. Rantz, A. H. Jacobs, and W. M. M. Kirby (*J. clin. Invest.*, 1943, **22**, 419—423).—33 of 64 children were group *A* carriers by culture of the excised tonsils. The sera of carriers contained larger amounts of agglutinins and antistreptolysin than non-carriers. When the carrier state was terminated by tonsillectomy, there was a const. decline in antistreptolysin titre within 60 days, and a less regular fall in agglutinin titre. C. J. C. B.

**Immunisation against rheumatic fever.** V. P. Wasson and E. E. Brown (*J. Pediat.*, 1943, **23**, 24—30).—In 1940—1941, 42 patients treated with hæmolytic streptococcal toxin suffered no relapses; in the control group of 33, 11 patients suffered attacks with 3 deaths. C. J. C. B.

***Streptococcus salivarius* and other non-hæmolytic streptococci of human throat.** J. M. Sherman, C. F. Niven, and K. L. Smiley (*J. Bact.*, 1943, **45**, 249—263). F. S.

**Counteracting sulphonamide inhibitors.** W. K. S. Wallersteiner (*Nature*, 1943, **151**, 586—587).—Sulphanilamidourea and sulphanilamidoallantoin inhibited growth of sulphanilamide-fast organisms, *Strep. viridans* and *Staph. aureus*, but sulphanilylurate did not. 7 : 7′-(Tetrazo-4 : 4′-diaminodiphenyl sulphone)-1-acetamido-8-naphthol-3 : 6-disulphonic acid and its Na salt are strong inhibitors of sulphanilamide-fast organisms. E. R. S.

**Action of tyrothricin on fæcal streptococci *in vitro* and *in vivo*.** E. C. Rodaniche and W. L. Palmer (*J. infect. Dis.*, 1943, **72**, 154—156).—Tyrothricin is highly bactericidal and bacteriostatic to *Strept. fæcalis* and related fæcal streptococci *in vitro*. Oral administration of tyrothricin may produce inhibition of the growth of streptococci in the intestine of mice. This inhibition is most readily demonstrated when sulphasuxidine is administered together with the tyrothricin. F. S.

**Transmissibility of syphilis.** H. Pariser (*J. invest. Dermat.*, 1942, **5**, 243—247).—Animal inoculation experiments showed that after introduction of *Spirochæta pallida* into the vagina of 3 women with secondary syphilis the organisms survived for 1½—4 hr. C. A. K.

**Treatment of resistant syphilis.** H. Beerman and M. Severac (*J. invest. Dermat.*, 1942, **5**, 269—282).—A strain of *Spirochæta pallida*, obtained from a patient who failed to respond to As and Bi treatment, was transferred to rabbits and from the 16th to 57th passage nearly every rabbit inoculated was infected. Arsphenamine, in dosage which cured rabbit syphilis due to other strains of spirochæte, failed to cure 13 of 51 infected rabbits. C. A. K.

**Rapid serodiagnosis test for syphilis.** F. Rappaport and F. Eichhorn (*Lancet*, 1943, **244**, 426—427).—A simple rapid flocculation test, suitable for serum and c.s.f., for diagnosis of syphilis is described. C. A. K.

**Normal reagin of Wassermann type in animal sera.** O. Bier and E. Trapp (*Rev. Brasil. Biol.*, 1942, **2**, 321—324).—A flocculating substance has been released from aggregates formed by the addition of Eagle flocculation antigen to ox and goat sera, using the salt dissociation method. The significance of this finding is discussed. I. C.

**SS agar for isolation of *Eberthella*, *Salmonella*, and *Shigella* groups from fæces, compared with MacConkey and bismuth sulphite agars and tetrathionate broth followed by MacConkey agar.** M. Mollov, J. E. Winter, and P. Steinberg (*J. Lab. clin. Med.*, 1943, **28**, 1021—1027).—SS agar is an excellent culture medium for isolating *Eberthella*, *Salmonella*, and *Shigella* from fæces. It gives fine definition of colony, provides for max. inhibition of coli, and facilitates isolation of intestinal pathogens when a large inoculum is used. In conjunction with SS agar, tetrathionate broth is of val. in the isolation of *Eberthella*, *Salmonella*, and a few of the *Shigella* group. In a small series of positive cases, SS agar was as efficient as or superior to Bi sulphite agar in the isolation of *E. typhosa* from fæces. C. J. C. B.

**Protective action of Vi bacteriophage in *Eberthella typhi* infections in mice.** W. E. Ward (*J. infect. Dis.*, 1943, **72**, 172—176).—More than 90% of mice infected with *Bact. typhosum* in the Vi phase and treated with one dose of sp. Vi bacteriophage were protected. Untreated mice and mice treated with a non-sp. phage had a mortality approaching 100%. F. S.

**Chronic granuloma following intradermal injection of typhoid vaccine.** I. L. Tilden and H. L. Arnold, jun. (*Arch. Path.*, 1943, **36**, 13—18).—A case report. (4 photomicrographs.) C. J. C. B.

**Stepwise liberation of poorly sorbed bacteriophages.** A. D. Hershey and J. Bronfenbrenner (*J. Bact.*, 1943, **45**, 211—218).—A poorly sorbed coliphage exhibited a stepwise increase after 60 min. incubation with a sensitive organism. Regeneration occurred in the same manner even in the absence of salt, where adsorption normally does not occur. The results indicated that the newly formed phage was held by structures not readily available for the sorption of external phage, *i.e.*, formed intracellularly. The stepwise rise implied liberation of phage by lysed bacterial cells. F. S.

**Influenza-like epidemic with numerous pulmonary infiltrations in a battalion.** E. Haemig and W. Heyden (*Schweiz. med. Wschr.*, 1942, **72**, 1113—1119).—105 men were taken ill, 59 showing marked pulmonary infiltration on *X*-ray examination with few or no auscultatory signs. Chemotherapy was unsuccessful. The infiltrations disappeared within 1—3 weeks. A virus pneumonia was diagnosed. A. S.

**Production of experimental influenza in mice by inhalations of atmospheres containing influenza virus dispersed as fine droplets.** C. G. Loosli, O. H. Robertson, and T. T. Puck (*J. infect. Dis.*, 1943, **72**, 142—153). F. S.

**Infectivity and immunising effect of St. Louis encephalitis virus propagated in mouse brain and chick egg.** M. G. Smith (*J. infect. Dis.*, 1943, **72**, 125—132).—Virus maintained on the chorioallantoic membrane of the developing chick for a long period was more infective, per intracerebral M.L.D., than mouse brain virus when inoculated subcutaneously in mice. The membrane virus was also more efficient as an immunising agent in mice. F. S.

**Post-infectious encephalomyelitis.** P. G. Stitt (*Amer. J. dis. Child.*, 1943, **65**, 585—590).—Mild symptoms of post-infectious encephalomyelitis following measles developed in a 9-year-old boy. When the symptoms began to subside he was vaccinated for smallpox. When the vaccinia reaction reached its height, there was increase in encephalomyelitic signs and symptoms, and Br poisoning as shown by a high blood-Br′. Improvement with a fall of blood-Br′ followed administration of NaCl. C. J. C. B.

**Ultracentrifuge as aid in detection of poliomyelitis virus.** J. L. Melnick (*J. Exp. Med.*, 1943, **77**, 195—204).—A purified and conc. macromol. fraction was regularly obtained from 16 infected human, monkey, and chimpanzee stools by means of differential ultracentrifugation, and inoculated intracerebrally into 16 monkeys of which 15 developed poliomyelitis. 11 stool specimen, tested in 11 monkeys by the intra-abdominal–intranasal method, produced poliomyelitis in 2 cases; the same specimens, tested separately by the ultracentrifugal-intracerebral method, produced poliomyelitis in 10 out of 11 animals. The latter method is at least 100 times as sensitive as the former. A. S.

**Development in epidemiology of poliomyelitis.** A. W. Fraser (*Arch. Pediat.*, 1943, **60**, 256—278).—A review. C. J. C. B.

**Attempts to propagate the virus of poliomyelitis in smaller laboratory animals.** J. L. Schwab, O. C. Woolpert, and N. P. Hudson (*J. infect. Dis.*, 1943, **72**, 97—107).—Attempts with 8 strains of poliomyelitis virus in rabbits, guinea-pigs, white rats, cotton rats, white mice, chickens, and pigeons all failed. F. S.

**Cell state as affecting susceptibility to a virus. Enhanced effectiveness of rabbit papilloma virus on hyperplastic epidermis. Oral papillomatosis on rabbits : virus disease.**—See A., 1943, III, 490.

**Latent pneumotropic virus of mice.** H. V. Karr (*J. infect. Dis.*, 1943, **72**, 108—116).—A virus isolated from lungs of apparently healthy mice by serial lung passages produced fatal pneumonia in mice. It was immunologically unrelated to influenza *A* virus or to the latent pneumotropic virus of Horsfall and Hahn (*J. Exp. Med.*, 1940, **71**, 391). F. S.

**Psittacosis (ornithosis) virus in English pigeons.** C. H. Andrewes and K. C. Mills (*Lancet*, 1943, **244**, 292—294).—Psittacosis virus was identified by impression preps., histological sections and filtration of infected organs, and by serological tests in apparently normal pigeons obtained from southern England and in pigeons recently arrived from America. C. A. K.

**[Vector of] Rocky Mountain spotted fever.** R. R. Parker, G. M. Kohls, and E. A. Steinhaus (*U.S. Publ. Health Repts.*, 1943, **58**, 721—729).—The rickettsia of Rocky Mountain spotted fever has been recovered from *Amblyomma americanum* nymphs collected from vegetation. Old and recent case data suggestive of the transmission of spotted fever by this tick are discussed. The evidence of spontaneous infection in *A. americanum*, together with the suggestive case data, is considered sufficient to establish this tick as the third species transmitting spotted fever to man in the United States. C. G. W.

**Typhus rickettsial agglutination tests in Middle East Forces and Egypt.** C. E. van Rooyen and W. G. C. Bearcroft (*Edinb. Med. J.*, 1943, **50**, 257—271).—In 73 cases of typhus in the Middle East the rickettsial agglutination (R.A.R.) was superior to Weil–Felix (W.F.) test and also differentiated between murine and epidemic infections. In 174 controls the R.A.R. gave fewer false positive results than the W.F. test. Both become positive from the 7th, and reach max. about the 14th, day of illness but R.A.R. persists longer than W.F. Cross-agglutinations occur between murine and epidemic strains in R.A.R. H. S.

**Endemic murine typhus.** F. Smith and R. W. Evans (*Lancet*, 1943, **244**, 142—143).—4 cases are described, in which the titre with proteus *OX* 19 suspension rose to 1 in 1260. C. A. K.

**Laboratory infection with murine typhus.** M. Van den Ende, E. H. R. Harries, C. H. Stuart-Harris, A. J. Steigman, and R. Cruickshank (*Lancet*, 1943, **244**, 328—332).—12 laboratory workers were accidentally infected with murine typhus, probably owing to inhalation of infective droplets during intranasal inoculations of mice. All had been previously immunised with typhus vaccine, and the disease was moderately severe in 3 cases and mild in the others. Neutropenia with relative or abs. monocytosis occurred during the 1st week of infection. C. A. K.

**Inactivation of vaccinia virus by mild antiseptics.** W. B. Dunham and W. J. McNeal (*J. Lab. clin. Med.*, 1943, **28**, 947—953).—The following agents were effective in inactivating vaccine virus in 3 min.: phenol, 3%; tincture of I, 1%; $HgCl_2$, 0·1%; propylene glycol, 70%; liquor antisepticus, N.F. VI, undiluted; listerine, undiluted; tincture of metaphen, diluted to quarter strength 0·05%; lysol, 2%, and amphyl, 1%. C. J. C. B.

**Traumatic causation of Pfeiffer's glandular fever.** H. Pinösch (*Schweiz. med. Wschr.*, 1942, **72**, 1012—1013).—A patient developed glandular fever following injury to a finger. A. S.

**Ætiology of rheumatism.** W. M. Levinthal (*Edinb. Med. J.*, 1943, **50**, 415—430).—Evidence is produced to support the conception of rheumatism, acute and chronic, as a continual antigen–antibody reaction in or on the cells of mesodermal tissues. The antigen is a dissolved bacterial substance and the antibody is present in the cells rather than in the blood stream. Debility of the antibody-producing system is the basic cause of rheumatism. H. S.

**Fixation of complement in tropical dermatosis.** F. W. Eichbaum (*Rev. Brasil. Biol.*, 1942, **2**, 285—300).—Complement fixation tests in cases of leishmaniasis brasiliensis, pemphigus foliaceus, leprosy, and blastomycosis with two preps. of the Witebsky–Kuhn–Klingenstein antigen (W.K.K.) gave different results only in cases of blastomycosis, in which a positive reaction was obtained with the commercial prep. of the antigen, and a negative with the antigen prepared in the laboratory. The reactivity of the sera of leishmaniasis, pemphigus, and blastomycosis is not influenced by sp. absorption with the W.K.K. antigen; in leprosy sera have been obtained which are absorbed by tubercule bacilli and by the W.K.K. antigen and sera the reactivity of which is not affected by absorption by these two antigens. The W.K.K. reaction therefore in the tropical dermatosis studied, except in some cases of leprosy, is non-sp. I. C.

**Preparation and purification of serum anti-*Latrodectus mactans*.** I. Pirosky, R. Sampayo, and C. Franceschi (*Rev. Soc. argent. Biol.*, 1942, **18**, 170—175).—Immunisation was produced by subcutaneous injection of the cephalothorax of the spider *L. mactans* in the horse. The sp. antitoxin of the anti-*Latrodectus* serum was purified by digestion with pepsin. I. C.

**Removal of bivalent cations from solution by beef heart antigens.** L. F. Pierce and E. L. Breazeale (*J. invest. Dermat.*, 1942, **5**, 249—255).—Ca and Mg were removed from solution by Kahn and Hinton antigens and could be recovered from the floc by washing with saline. A new theory of the mechanism of serological reactions in syphilis is suggested. C. A. K.

**Artificial antigen with blood-group *A* specificity.**—See A., 1943, III, 545.

**Ascorbic acid and histamine content of rabbit's blood during anaphylactic shock.** F. Eichbaum and M. Rocha e Silva (*Rev. Brasil. Biol.*, 1943, **3**, 39—44).—Ascorbic acid (200—500 mg.) has no effect on the intensity of anaphylactic shock or on the histamine content of blood after injection of the antigen in rabbits. Ascorbic acid does not release histamine from cells into plasma and does not prevent diffusion of histamine from the blood cells in *in vitro* anaphylaxis. I. C.

**Cross-desensitisation in allergic diseases.** K. Maunsell (*Lancet*, 1943, **244**, 3—6).—Patients with asthma, eczema, or hay fever were studied. Local skin desensitisation was produced by injecting increasing dosage of allergic extracts intracutaneously. Desensitisation to one allergen was often associated with desensitisation to other allergens; there was no decrease in histamine response in desensitised areas. C. A. K.

**Significance of inhalant allergens in treatment of bronchial asthma in children.**—See A., 1943, III, 464.

**Intramuscular histaminase in hay fever.**—See A., 1943, III, 587.

**Respiratory allergy from arsphenamines.**—See A., 1943, III, 514.

**Survey of skin testing [for protein-sensitivity] : suggested method in epileptics.** D. C. Dewar (*J. ment. Sci.*, 1941, **87**, 397—408).—Epileptics were scratch-tested, and a week later tested intradermally, in the forearm. G. D. G.

## XXVI.—PLANT PHYSIOLOGY.

**Assimilation *in vitro*.** E. Baur and F. Niggli (*Helv. Chim. Acta*, 1943, **26**, 994—994).—Assimilation is observed under the influence of light with a paste of lecithin, phytol, and glycerol containing chlorophyll and methylene-blue. In a paste composed of glycerol, geraniol, lecithin, and methylene-blue, the chlorophyll has been successfully replaced by cibacetorange 3*R*. (Cf. A., 1943, III, 536.) H. W.

**Lipoxidase in legume seeds.** H. Süllmann (*Verh. Ver. Schweiz. Physiol.*, 1942, **21**, 39—40). A. S.

**Bud formation, abscission, and flower production of *Gardenia* as affected by light and temperature.** C. G. Keyes (*Proc. Amer. Soc. Hort. Sci.* [1939], 1940, **37**, 1034—1036).—Bud formation in gardenias when the night temp. was 21·1° was reduced and extra illumination failed to increase bud formation at all temp. 12·8—21·1°. Bud abscission was greatest at the high night temp. and was not reduced by extra illumination at any temp. Extra light increased flower production only when temp. was high during periods of light and low during periods of darkness. L. G. G. W.

**Effect of temperature and photo-period on the growth and flowering of miscellaneous annuals.** K. Post and C. L. Weddle (*Proc. Amer. Soc. Hort. Sci.* [1939], 1940, **37**, 1037—1043).—13 annual flowering plants are classified according to the conditions of temp. and photo-periods under which they flowered most abundantly, flowered earliest, and made most growth. L. G. G. W.

**Response of plants to intermittent supplementary light.** E. P. Hume (*Proc. Amer. Soc. Hort. Sci.* [1939], 1940, **37**, 1059—1065). Horticultural varieties of *Matthiola incana*, *Delphinium ajacis*, *Chrysanthemum segetum*, *and Callisthepus chinensis* were given intermittent illumination of periods varying from 0 to 100 sec. alternating with periods of darkness from 0 to 100 sec. *C. chinensis* flowered as early with intermittent light as with continuous light and more quickly than the normally illuminated controls. *Chrysanthemum* also responded to intermittent illumination but only if the time of illumination was not less than ⅓ of the possible total. Above this they flowered at the same time as plants receiving continuous illumination. *Delphinium* responded by earlier flowering to illumination as little as 13·6% of the total time, whilst with *Matthiola* earliest flowering occurred with continuous illumination and intermittent light of not less than 50% of the possible time. Smaller % of light showed less stimulation and 13% of light failed to accelerate flowering. L. G. G. W.

**Tetraploid lily.** S. E. Emsweller and P. Brierley (*Proc. Amer. Soc. Hort. Sci.* [1939], 1940, **37**, 1006).—Tetraploid plants of *L. formosanum*, Stapf, resulted when apical growing points of diploid plants were treated for 2 hr. with 0·1—1·0% aq. colchicine and the bulblets that formed in the axils of the terminal leaves of the treated plants removed and planted. Some of the tetraploids flowered and set seeds and the tetraploidy was inherited. Flowers, leaves, and stomata were all larger in the tetraploids than in the diploids. L. G. G. W.

**Rate of respiration in potato tubers at high temperatures in relation to treatment with ethylene chlorohydrin.** N. W. Ward (*Proc. Amer. Soc. Hort. Sci.* [1939], 1940, **37**, 871—873).—Treatment with ethylene chlorohydrin increases the respiration rate of potato tubers but at 21·1° the respiration rate after rising rapidly for a day begins to fall so that after 10 days treated and control tubers respire at the same rate. At 32·2° and 35° treated tubers still have a higher respiration rate than the controls after 12 days. Tubers (control and treated) after 19 days at 21·1° when moved to 32·2° show an increase in respiration rate but this is not sufficient to cause breakdown and indicates that treated tubers kept at 21·1° for long enough to allow the respiration rate after its initial increase to fall back to normal may be planted in soil at 32·2° with little danger of breakdown due to high respiration rate. L. G. G. W.

**Effect of colchicine pretreatment on frequency of chromosomal aberrations induced by *X*-radiation.** R. T. Bramfield (*Proc. Nat. Acad. Sci.*, 1943, **29**, 190—193).—Treatment of onion roots for 45 min. with a 0·05% solution of colchicine before *X*-irradiation reduced chromatid aberrations to 1·03% as compared with 3·51% of chromosomes in non-colchicine-treated roots. The frequency of chromosome aberrations was 1·75% and 1·83% in the pretreated roots and controls. The colchicine pretreatment reduces chromosome movement in prophase when chromatid effects are induced, thus giving less opportunity for fusion of broken ends into new combinations. F. S.

**Growth factors controlling tomato stem growth in darkness.** F. W. Went and D. M. Bonner (*Arch. Biochem.*, 1943, **1**, 439—452).—Stem growth almost ceases after an initial spurt in darkness, but revives when the leaves are immersed in 10% sucrose and the growth rate is proportional to the no. of leaves immersed. Auxin limits growth only after decapitation, when growth is restored by indolylacetic acid. Caulocaline limits growth within 20 hr. of cutting off the roots. Roots form caulocaline, and a technique is described for testing substances for caulocaline activity. Coconut milk and pea diffusate contain caulocaline. E. R. S.

**Possibility of non-bacterial fixation of atmospheric nitrogen in association with energy-releasing reactions.**—See B., 1943, III, 199.

## XXVII.—PLANT CONSTITUENTS.

**Winter sources of vitamin-*C*. Vitamin-*C* content of oranges and peaches.**—See A., 1943, III, 578.

**Determination of flavones or quercetin-like substances in natural products.** L. S. Weatherby and A. L. S. Cheng (*J. Biol. Chem.*, 1943, **148**, 707—709).—The flavone content of certain plant products was determined by the method described earlier (A., 1942, II, 292). Lemon peel has the highest quercetin equiv. of the substances tested (6·3 mg. per g. on dry basis). Red rose petals have a higher val. (4·4) than white (2·7) or yellow rose petals (2·8). P. G. M.

**Lichen substances. XCV. Zeorin group. II.**—See A., 1943, II, 314.

**Gums from carob bean, *Ceratonia siliqua*, L.** B. W. Lew and R. A. Gortner (*Arch. Biochem.*, 1943, **1**, 325—337).—Commercial carob bean gum consists of *d*-mannose and *d*-galactose (approx. 3·5 : 1), and contains no pentose or uronic acid. The gum had $[\alpha]_D^{30}$ +43°, falling to 0° and rising to an equilibrium val. of +28° on hydrolysis (6—7 hr.). The consumption of and products of oxidation by $HIO_4$ indicate 1 : 2 linkages, probably α-galactoside and β-mannoside. 4 methods using $HIO_4$ gave 18, 17, 16, and 13 hexose units per mol., which consists of 6—8 mannose units followed by 2 galactose units repeated, with at least one galactose end unit. E. R. S.

**Lignin. XL. Enzymic degradation of polymeric carbohydrates. Fractionation of linden wood and enzymic degradation of the fractions.**—See A., II, 315.

**Proteins of *Vicia faba*.**—See A., 1943, III, 596.

**Sapogenins from *Trigonella foenum-graecum*, *Beth* root, and *Balanites ægyptica*, Wall. Isolation and structures of thirteen new steroidal sapogenins. New sources for known sapogenins.**—See A., 1943, II, 304.

**Alkaloids of *Bocconia arborea*, Wats.**—See A., 1943, II, 312.

**Alkaloids of *Thermopsis rhombifolia* (Nutt), Richards.**—See A., 1943, II, 311.

## XXVIII.—APPARATUS AND ANALYTICAL METHODS.

**Cotton as suture material.** S. A. Localio and J. W. Hinton (*Surg. Gynec. Obstet.*, 1941, **72**, 615—618).—Tensile strength was determined in samples of silk, linen, and cotton threads as bought and after varying sterilising procedures. Cotton should be boiled for 10—20 min. and used while wet. The tensile strength is decreased by autoclaving, or by drying after boiling, but is increased after boiling if tested while still wet. Cotton was successfully used in 50 major surgical operations. P. C. W.

**Improved animal board.** H. M. Kaplan, I. J. Tetwin, and E. H. Davis (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 48—49). C. J. C. B.

**Stand for holding donor bottle in inverted position.** C. H. Binford (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 50). C. J. C. B.

**Profile printing in photomicrography of blood cells.** W. P. Murphy (*Arch. intern. Med.*, 1943, **71**, 814—827).—11 photomicrographs are given of various blood conditions in which this method of printing has been used. C. J. C. B.

**Colour photographs simplified.** V. J. Connolly (*J. Lab. clin. Med.*, 1943, **28**, 921—923). C. J. C. B.

**Permanent preservation of stained and unstained blood films and bacterial smears by paraffin coating.** F. B. Queen (*Amer. J. clin. Path. Tech. Sect.*, 1943, **7**, 50). C. J. C. B.

**Electric lantern slide pointer.** N. A. Murray (*J. Lab. clin. Med.*, 1943, **28**, 1019—1029).—The method of construction of a simple type is detailed. C. J. C. B.

**Electronic device for location of metallic fragments *in vivo*.** C. Fenning (*J. Lab. clin. Med.*, 1943, **28**, 867—869). C. J. C. B.

**Modified electroscope especially suited for measuring substances with low energy radiation.** A. M. Seligman (*J. clin. Invest.*, 1943, **22**, 281—284).—A modification of the Lauritsen electroscope, which increases its sensitivity, is described. This makes it possible to measure the soft radiation from radioactive S with an accuracy comparable to that of Geiger–Müller counters. C. J. C. B.

**Roller rim-drive kymographs.** R. P. Walton, F. M. Cook, and A. B. Cullen (*J. Lab. clin. Med.*, 1943, **28**, 1002—1008).—The mechanism is fully illustrated. C. J. C. B.

**Apparatus for preparation of parenteral solutions.** A. G. Keller (*J. Lab. clin. Med.*, 1943, **28**, 1015—1019). C. J. C. B.

**Volumetric respirometer for aquatic animals.** P. F. Scholander, N. Haugaard, and L. Irving (*Rev. Sci. Instr.*, 1943, **14**, 48—51).—A closed-circuit respirometer in which continuous direct measurements can be made at any intervals and for many hr. is described. It is capable of showing accurately the use of 0·1 ml. of $O_2$ per hr. J. L. E.

**Separation of biological fats from mixtures by adsorption. II. Separation of phosphorus- and nitrogen-free lipin fractions.** W. Trappe (*Biochem. Z.*, 1940, **306**, 316—336).—The adsorbability of natural substances increases in the order : hydrocarbons, cholesteryl esters, triglycerides, cholesterol, fatty acids, phosphatides. Adsorption on $Al_2O_3$ (Brockmann) results in saponification of triglycerides and autoxidation of unsaturated fatty acids. Some catalytic change occurs in cholesterol and its esters by adsorption on activated $SiO_2$ from HCl-containing solvents. By the use of light petroleum, $CCl_4$, or benzene, different fractions can be adsorbed on activated $SiO_2$. No adsorption takes place from ether solution. A steroid is present in human serum-, liver-, and adrenal-lipins, which differs from cholesterol and its esters and accompanies the hydrocarbon fraction. P. G. M.

**Determination of sodium pentothal in blood.**—See A., 1943, III, 763.

**Determination of atebrin in blood and urine.** J. M. Masen (*J. Biol. Chem.*, 1943, **148**, 529—539).—Atebrin is extracted after addition of NaOH by *iso*propyl–*iso*butyl alcohol–light petroleum, washed, transferred to aq.-alcoholic HCl, and measured by fluorescence; 0·1 mg. per l. can be accurately determined. Atebrin is excreted unchanged at first, but later decomp. products appear in the urine. R. L. E.

**Determination of sodium dehydro*iso*androsterone sulphate in water or urine.** N. B. Talbot, J. Ryan, and J. K. Wolfe (*J. Biol. Chem.*, 1943, **148**, 593—602).—Na dehydro*iso*androsterone sulphate in an *n*-butanol extract from water or urine is hydrolysed by $BaCl_2$ and determined colorimetrically (cf. A., 1942, III, 504). R. L. E.

**Determination of quinine in human plasma ; determination of quinidine.** B. B. Brodie and S. Udenfriend (*J. Pharm. Exp. Ther.*, 1943, **78**, 154—158).—1 part of plasma, 39 of water, and 10 of 20% $HPO_3$ are mixed and centrifuged. The fluorescence of the protein-free supernatant fluid is compared with that of a standard containing a known amount of quinine. An identical method can be used for quinidine. V. J. W.

**Colorimetric determination of phosphorus.**—See B., 1943, III, 199.

**Determination of phosphorus by molybdenum-blue method.**—See A., 1943, I, 263.

# INDEX OF AUTHORS' NAMES, A., III.

OCTOBER, 1943.


Abelin, I., 734.
Abraham, S. V., 729.
Abramson, D. I., 722.
Ackroyd, S., 712.
Adcock, J. D., 722.
Adler, H. F., 722.
Agnew, S., 759.
Ahlquist, R. P., 762.
Akelaitis, A. J., 725.
Albanese, A. A., 751.
Alexander, F., 759.
Allen, C. R., 762.
Allen, T. H., 769.
Alstead, S., 710.
Alvaro, M. E., 728, 730.
Anderes, E., 712.
Anderson, N. A., 741.
Anderson, P. L., 707.
Anderson, R. S., 712.
Anderson, W. F., 743.
Andervont, H. B., 749.
Andrewes, C., 777.
Andrews, F. N., 742.
Appenzeller, R., 768.
Apter, N., 765.
Arenas, N., 740.
Arey, L. B., 708.
Armentrout, J. M., 760.
Arnett, J. H., 759.
Arnold, H. L., jun., 776.
Ascroft, P. B., 746.
Asmundson, V. S., 754.
Averill, R., 774.
Axelrod, A. E., 760.

Bacon, L., 739.
Bailey, B., 723.
Bailey, O. T., 714.
Baird, K. H., 713.
Baker, F., 744.
Ballard, W. W., 738.
Ballon, O., 713.
Balser, B. H., 728.
Barbour, H. G., 762.
Barbour, J. H., 721.
Barker, H. A., 772.
Barlow, O. W., 755.
Barré, R., 747.
Barrett, J. F. B., 716.
Bartels, E. C., 735.
Bast, T. H., 734.
Bates, R. W., 737.
Baumann, C. A., 713.
Baur, E., 778.
Bayerle, H., 769.
Beach, S. J., 730, 732.
Bearcroft, W. G. C., 777.
Beaton, L. E., 705.
Beazell, J. M., 744.
Beecher, H. K., 719.
Beerman, H., 775.
Beeson, P. B., 760.
Benjamin, H. R., 756.
Benjamin, J. E., 722.
Bennett, R. H., 759.
Benson, M. E., 735.
Bentley, J. P., 761.
Bercovitz, Z., 713.
Berencsi, G., 708.
Berg, R. L., 757.
Bergman, A. J., 734.
Berliner, F., 725, 726.
Bernstein, A. O., 726.
Bethell, F. H., 710.
Betti, M., 769.
Beznák, M., 736.
Bier, O., 776.
Biggs, A. D., 706.
Binford, C. H., 780.
Biocca, E., 766.
Bird, F. H., 754.
Birkhauser, H., 726.
Bischoff, F., 740.
Blackstone, C. H., 706.
Blackwood, W., 720.
Bloch, H., 769.
Block, W. D., 714.
Blum, H. F., 748.
Bode, O. B., 754.
Bodenheimer, M., 714.
Bodine, J. H., 769.
Boehm, G., 754.
Bommels, N., 750.
Bomskov, C., 737.
Bonner, D. M., 779.
Bonney, V., 706.
Boothby, W. M., 763.
Borkovic, E. J., 726.
Borts, I. H., 773.
Bosse, M. D., 760.
Bothie, R. T., 705.
Bovasky, R., 740.
Boyden, E. A., 705.
Boynton, R., 759.
Bradbury, J. T., 740.
Bradley, H. C., 723.
Bramfield, R. T., 779.
Branion, H. D., 755.
Braun-Menendez, E., 720.
Breazeale, E. L., 778.
Brierley, P., 728.
Briggs, G. M., jun., 753, 754.
Briggs, J. B., 747.
Briggs, R. W., 747.
Broders, A. C., 734.
Brodie, B. B., 780.
Brogden, W. J., 724.
Bromehead, C. N., 751.
Bronfenbrenner, J., 776.
Broom, J. C., 773.
Brouha, L., 752, 766.
Brown, C. L., 735.
Brown, E. E., 756, 775.
Brown, E. W., 741.
Brown, H. C., 773.
Brown, I. W., 743.
Brown, M., 724.
Brown, W. H., 749.
Browne, J. S. L., 736.
Brownlee, G., 762.
Brownlee, I. E., 760.
Bruck-Lacerda, L., 739.
Brumback, J. E., jun., 751.
Brunschwig, A., 743, 751.
Bryan, W. R., 748.
Bucher, K., 721.
Bueding, E., 756.
Bunker, J. W. M., 751.
Burdette, W. J., 747.
Burger, J. W., 742.
Burian, H. M., 730.
Burkholder, P. R., 770.
Burns, R. A., 746.
Burns, W., 759.
Burstein, C. L., 763.
Burtner, E., 737.
Buschke, W., 731.
Bush, M. T., 758.
Butler, E. C. B., 775.
Buxbaum, L., 771.

Cagan, R. N., 770.
Cain, C. K., 740.
Calabresi, M., 717.
Caldwell, G., 736.
Campbell, H. A., 713.
Campos, F. A. de M., 732.
Cardon, L., 744.
Carr, C. J., 762.
Carr, J. L., 743.
Casten, D., 714.
Castiglioni, A., 746.
Cervino, J. M., 735.
Chaikoff, I. L., 715, 735.
Chapple, E. D., 766.
Chattaway, F. W., 773.
Chen, K. K., 764.
Cheng, A. L. S., 779.
Chivers, E. M., 763.
Christie, R. V., 763.
Chu, H., 724.
Clark, B. B., 763.
Clayson, D. H. F., 769.
Clayton-Cooper, B., 766.
Clinton, E., 745.
Cloudman, A. M., 749.
Coblentz, L. M., 771.
Coco, R. M., 710.
Coe, W. R., 738.
Cogan, D. G., 731.
Cohen, R. H. L., 760.
Cohn, C., 735.
Cole, V. V., 764.
Coll, A., 739.
Collier, E., 766.
Collins, V. J., 747.
Connolly, V. J., 780.
Conway, E. J., 715.
Cook, F. M., 780.
Corbin, N., 751.
Court, D., 737.
Covode, W. M., 708.
Craik, K. J. W., 732.
Cralley, L. V., 767.
Cram, E. B., 761.
Cramer, W., 747.
Crandall, L. A., jun., 757.
Crider, J. O., 744.
Crile, G., 734.
Crook, G. W., 762.
Cross, C. D., 761.
Crossley, M. L., 714, 758.
Croxatto, H., 724.
Croxatto, R., 715.
Cruickshank, R., 777.
Cullen, A. B., 780.
Cummins, S. D., 760.
Cunniffe, J. P., 759.

Dameshek, W., 710.
D'Angelo, S. A., 741.
Darling, R. C., 758.
Davenport, H. A., 705.
Davidoff, E., 727.
Davidson, F. A., 747.
Davidson, J. N., 709.
Davidson, L. S. P., 711.
Davis, D. C., 741.
Davis, D. E., 710.
Davis, D. J., 771.
Davis, E. H., 780.
Davis, L. J., 711.
Davson, H., 731.
Dearborn, E. H., 761.
De Beer, E. J., 713, 766.
De Capito, T., 759.
De Graaf, A. C., 763.
De Groat, A., 771.
De La Balze, F. A., 714.
Del Boca, C., 751.
Dell'Oro, R., 720.
Denison, N., 771.
Denniston, R. H., 2nd, 736.
Deromedi, F., 762.
Detweiler, S. R., 707.
Deuel, H. J., 757.
Devine, J. W., 728.
Devrient, T., 765.
Dewar, D. C., 778.
Dingle, J. H., 774.
Dishbein, W. I., 755.
Doak, G. O., 764.
Dodds, E. C., 763.
Doisy, E. A., 740.
Dole, V. P., 745.
Dominguez, R., 746.
Domm, L. V., 710.
Drinker, C. K., 719.
Drury, D. R., 744.
Duggan, W. F., 730.
Duis, B., 764.
Duncan, J., 755.
Duncan, J. McK., 766.
Dunham, W. B., 777.
Dunn, J. S., 735.
Durand, E. A., 747.
Duran-Jorda, F., 710.
Dusenbery, E., 761.
Duval, A. M., 710, 722, 736.
Dworkin, S., 718.
Dyckerhoff, H., 712.

Eagle, H., 764.
Earle, W. R., 748.
Ebbecke, U., 713.
Ebert, R. V., 728.
Eckert, H. W., 716.
Edlbacher, S., 768.
Edwards, J. C., 745.
Eggers, H. E., 747.
Eichbaum, F., 778.
Eichbaum, F. W., 777.
Eichhorn, F., 715, 776.
Elek, S. R., 717.
Ellis, D. M., 760.
Elman, R., 719, 751.
Elsor, L. A., 769.
Elvehjem, C. A., 715, 753, 754.
Emerson, K., jun., 745.
Emery, F. E., 717.
Emsweller, S. E., 778.
Engle, J. B., 738.
English, J. P., 718.
Ensov, C., 717.
Entenman, C., 715.
Enticknap, J., 744.
Enzer, N., 764.
Epstein, S., 767.
Ershoff, B. H., 756.
Erxleben, H., 749, 769.
Evans, G., 754.
Evans, R. W., 777.
Evans, W. E., jun., 762.
Everett, J. W., 739.
Everett N. B., 706.
Eversole, W. J., 741.

Fagan, R. H., 708.
Fagin, I. D., 745.
Fairhall, L. T., 709, 765.
Fanconi, G., 760.
Farber, J. E., 730, 736.
Farley, D. L., 752.
Farnsworth, N. C., 761.
Fazekas, J. F., 726.
Feder, A., 753.
Feil, L., 711.
Feindel, W. H., 731.
Feinstein, B., 722.
Feinstone, W. H., 758.
Felger, L., 740.
Felsenfeld, O., 774.
Fenning, C., 780.
Ferrebee, J. W., 753.
Field, J. B., 713, 715.
Filitti-Wurmser, S., 768.
Findley, T., 745.
Fink, H. W., 760.
Fitzgerald, G., 763.
Fitzgerald, O., 715.
Fitzgerald, R. J., 758.
Flanagan, M. E., 759.
Fleisch, A., 750.
Fleischmann, W., 735.
Fleming, G. W. T. H., 725.
Floersch, E. B., 714.
Florey, H. W., 758.
Florey, M. E., 758.
Flowerdew, R. E., 754.
Foglia, V. G., 737, 751.
Foote, F. S., 743.
Forbes, G. B., 722.
Forbes, H. W., 758.
Forbes, T. R., 740.
Forman, S. E., 762.
Forster, F. M., 727.
Fosdick, L. S., 775.
Fournier, J. C. M., 735.
Fox, M., 713.
Frame, E. G., 716.
França, J., 739.
Franceschi, C., 777.
Franklin, A. L., 735.
Frankston, J. E., 751.
Fraser, A. W., 776.
Free, A. H., 714.
Freed, S. C., 719.
Freeman, H., 724.
Freeman, S., 744.
Freiman, I. S., 765.
Freire, S. A., 718.
Freund, E., 705.
Friedemann, U., 728.
Friedenwald, J. S., 731.
Friedman, M., 720.
Fuerstner, P. G., 739.
Furth, J., 718.

Gaddum, J. H., 765.
Galvão, P. E., 718, 765.
Gantt, W. H., 724.
Gardner, L. U., 766.
Gardner, W. U., 747.
Gedgoud, J. L., 715.
Geiger, A. J., 717.
Geiger, E., 755.
Geiling, E. M. K., 761.
Gendel, S., 740.
German, W. M., 735.
Gersdorff, W. A., 765.
Gettler, A. O., 766.
Gibbs, F. A., 727.
Gibson, T., 705.
Gilbert, B., 739.
Gillman, J., 739.
Giordano, A. S., 711.
Glaser, M. A., 727.
Glasson, B., 757.
Glenn, W. W. L., 719.
Globus, J. H., 728.
Glyn-Hughes, F., 765.
Godfrid, M., 740.
Goetzl, S., 721.
Goldfarb, A. E., 756.
Goldman, A., 743.
Gompertz, M. L., 753.
Goode, W., 760.
Goodstone, G. L., 727.
Gortner, R. A., 779.
Goth, A., 758.
Grail, G. F., 757.
Granick, S., 712.
Grauer, H., 768.
Grave, B. H., 738.
Graybiel, A., 718.
Greenberg, B., 742.
Greenburg, L., 767.
Greene, H. S. N., 749.
Greenstein, J. P., 749.
Greenwalt, T. J., 710.
Griffiths, J. T., jun., 723.
Grodins, F., 744.
Grodins, F. S., 722.
Gross, P., 760.
Guerra, F. (Perez-Carral), 764.
Guigoz, M., 750.
Gukelberger, M., 721.
Gunning, R. E., 717.
Gustina, F. J., 736.
Gutman, A. B., 705.
Gutman, E. B., 705.
Guttmann, E., 724, 725.

Haas, E., 768.
Hackett, W. R., 743.
Hadorn, W., 721.
Hailstone, W. N., 713.
Haines, S. F., 763.
Hajna, A. A., 772.
Hall, M. H., 761.
Hall, V. E., 717.
Halmig, E., 776.
Hamblen, E. C., 741.
Hamilton, C. R., 764.
Hámori, A., 721.
Happold, F. C., 773.
Harding, G. F., 730.
Hardy, A. V., 759, 760.
Hardy, L. M., 755.
Harries, E. H. R., 777.
Harris, D. M., 773.
Harris, L. J., 754.
Harris, R. S., 751.
Harrison, G. A., 712.
Harrison, N. B., 716.
Harrisson, J. W. E., 733.
Harshbarger, K. E., 765.
Hart, D., 728.
Hart, E. B., 753, 754.
Hartridge, H., 731, 732.
Hathaway, E., 760.
Hatschek, R., 769.
Hatz, E. B., 712.
Haugaard, N., 780.
Hauptmann, H., 739.
Hawking, F., 761.
Hawley, W. L., 760.
Hawn, C. v. Z., 714.
Hayden, C. T., 738.
Haynes, E., 762.
Heilbrunn, G., 774.
Heilman, D., 771.
Heiman, H., 767.
Heiman, J., 749.
Hellman, L. M., 763.
Hemphill, R. E., 728.
Henderson, F. G., 764.
Henske, J. A., 715.
Heppenstall, M. E., 715.
Herbert, F. X., 757.
Herbut, P. A., 706.
Herken, H., 749, 769.
Herrell, W. E., 771.
Herren, R. Y., 725.
Herrlich, H. C., 726, 763.
Hershey, A. D., 776.
Hertz, R., 754.
Heston, W. E., 749.
Hettig, R. A., 710.
Heyden, W., 776.
Hill, D., 715.
Hill, D. C., 755.
Himwich, H. E., 726.
Hines, H. M., 722.
Hinman, F., 746.
Hinton, J. W., 779.
Hochrein, M., 718.
Hoffman, B. J., 711.
Hoffmann, M. M., 736.
Holbourn, A. H. S., 732, 759.
Holck, H. G. O., 762, 764.
Holinger, P. H., 735.
Holman, E., 719.
Holmes, O. M., 751.
Holmes, W., 722.
Holt, L. E., jun., 751, 752.
Horning, E. S., 770.
Horton, B. T., 734.
Horton, J. S. F., 754.
Horvath, S. M., 753.
Horvitz, A., 719.
Horwitz, O., 752.
Houssay, H. E. J., 737.
Hrenoff, A. K., 762.
Hsu, S., 724.
Hubbard, J. P., 718.
Huber, W., 755.
Hudson, N. P., 776.
Hueper, W. C., 757.
Hughes, A. F. W., 707.
Huidobro, F., 715.

Humber, J. B., 774.
Hume, E. M., 752.
Hume, E. P., 778.
Hunter, A. R., 763.
Hutchings, B. L., 773.
Hutchinson, S. J., 747.
Hutt, F. B., 707.
Hutton, E. L., 725.
Hwang, K., 724.

Ifft, J. D., 743.
Iliff, A., 710, 722, 756.
Innes, J., 711.
Irving, L., 780.
Israels, M. C. G., 711.
Ivy, A. C., 721, 722, 744.

Jackson, E., 760.
Jacobs, A. H., 775.
Jacoby, F. C., 755.
Jaques, L. B., 713.
Jardeni, I., 710.
Jasper, H. H., 727.
Jaton, A., 750.
Jefferiss, F. J. G., 759.
Jendrassik, L., 715
Jennings, J. R., 773.
Jenrette, W. V., 765.
Jetter, W. W., 714.
Johnson, C. A., 720.
Johnson, E. C., 735.
Johnson, R. E., 752.
Johnson, V., 717.
Johnston, C., 757.
Jones, E. I., 760.
Jones, R. M., 711.
Jones, S. W., 765.
Jordan, C. F., 773.
Joski, J., 760.
Juday, C., 715.
Jürgens, R., 765.
Jung, A., 751.
Junowicz-Kocholaty, R., 770.

Kabat, E. A., 726.
Kahler, H., 749.
Kajdi, C. N., 751.
Kanitz, H. R., 716.
Kaplan, A., 715, 720.
Kaplan, H. M., 780.
Karnosh, L. J., 725.
Karp, P., 756.
Karr, H. V., 777.
Karrer, P., 768.
Kartagener, M., 713.
Kaser, M., 752.
Katzin, L. I., 772.
Katzman, P. A., 740.
Kazal, L. A., 775.
Keefer, C. S., 758.
Keith, N. M., 745.
Keller, A. G., 780.
Kelsey, F. E., 761.
Kennard, M. A., 728.
Kennedy, C. R., 762.
Kepl, M., 736.
Keyes, C. G., 778.
Keys, A., 714.
Kimmel, D. L., 724.
King, H. E., 745.
King, L. S., 726.
Kinsman, G. M., 710, 722.
Kirby, W. M. M., 760, 775.
Kirshbaum, J. D., 714.
Kiszely, G., 712.
Klein, W., 743.
Knowlton, F. P., 722.
Knox, R., jun., 753.
Kögl, F., 749.
Kohls, G. M., 777.
Kohn, F., 761.
Kolbros, J. J., 731.
Kolen, A. A., 729.
Koller, T., 713.
Koran, P., 723.
Korényi, Z., 736.
Kracke, R. R., 711.
Krantz, J. C., jun., 762.
Kratzer, F. H., 754.
Kravitz, D., 733.
Krehl, W. A., 753.
Kress, B. H., 716.
Kruger, H. E., 720.
Kubin, R., 746.
Kuizenga, M. H., 712.
Kunz, A. F., 753.

Lahr, E. L., 737.
Laing, G. H., 744.
Lamport, H., 719.
Landes, J. H., 773.
Landgrebe, F. W., 737.
Landow, H., 726.
Landt, H., 722.
Lapin, J. H., 743.
Lardy, H. A., 742, 743.
Largent, E. J., 757.
Lascaster, J. E., 729.
Lassen, S., 755.
Laszczower, M., 712.
Laszt, L., 736.
Latsky, J. M., 750.
Lauridsen, J., 747.
Law, W. E., 761.
Lawson, W., 763.
Lazere, B., 722.
Leach, C. W., 774.
Learmonth, J. R., 728.
Learner, N., 735.
Lee, M., 716.
Le Galley, D. P., 733.
Lehmann, F. E., 758.
Lehmann, J., 713.
Leiter, J., 748.
Leonard, S. L., 741.
Lepkovsky, S., 754.
Leser, A. J., 763.
Leuthardt, F., 757.
Levinthal, W. M., 777.
Levy-Hochman, S., 740.
Lew, B. W., 779.
Lewis, A. E., 717.
Lewis, J. J., 717.
Lewis, N., 774.
Lewis, R. C., 710, 722, 756.
Lewison, E. F., 715.
Li, C. H., 737.
Liebmann, E., 738.
Lillie, R. D., 709.
Limarzi, L. R., 711.
Lind, C. J., 772.
Linksz, A., 729.
Lippincott, S. W., 748.
Llewellyn, J. S., 706.
Lloyd, R. I., 733.
Localio, S. A., 779.
Loewe, I., 753.
Loewe, S., 753.
Loomis, T. A., 717.
Looney, J. M., 726.
Loosanoff, V. L., 738.
Loosli, C. G., 776.
Loughlin, E. H., 759.
Lucchi, E., 769.
Luckey, T. D., 753, 754.
Lustig, H., 770.
Lutwak-Mann, C., 764.
Lyon, B. B. V., 744.
Lyon, R. A., 741.
Lythgoe, B., 773.

Ma, R., 771.
McArdle, B., 763.
McBride, D. E., 711.
McCance, R. A., 750.
McCarrell, J. D., 719.
MacCarty, W. C., 735.
McClean, D., 773,
McClure, R. D., 722.
MacCraken, F. L., 731.
McCrum, D. S., 766.
McDermott, L. J., 706.
McElligot, G. L. M., 759.
McGibbon, C., 765.
McGraw, A. B., 722.
Macháček, E., 760.
Machle, W., 757.
McIlwain, H., 761.
McIndoe, A. H., 761.
MacKenzie, J. W. A., 744.
Mackie, T. T., 744.
McKinley, W. A., 724.
McKissock, W., 725.
McLetchie, N. G. B., 735.
MacNeal, W. J., 761, 777.
Madden, J. H. M., 741.
Maes, J. P., 718.
Magalhães, B. F., 718.
Magoun, H. W., 724.
Malanga, C., 761.
Maldeis, H. J., 766.
Mallach, J. F., 765.
Mallery, O. T., jun., 710.
Maltby, G. L., 727.
Mampell, K., 708.
Mandy, A. J., 742.
Mandy, T. E., 742.
Mann, F. C., 771.
Mann, T., 770.
Mansbacher, K., 643.
Marangoni, B. A., 763.
Margulis, A. E., 730.
Marquardt, G. H., 765.
Martin, D. S., 774.
Martin, G. J., 720, 757.
Martin, L. C., 733.
Martin, S. J., 763.
Marx, L., 742.
Masen, J. M., 780.
Massari, S. C., 766.
Matheson, I., 759.
Mathieson, D. R., 764.
Mattice, M. R., 746.
Maunsell, K., 778.
Mayer-Gross, W., 724, 725, 726.
Mayers, M. R., 767.
Medawar, T. B., 705.
Meek, W. J., 762.
Mehl, J. W., 770.
Meiklejohn, J., 753.
Mells, M. I., 741.
Melnick, J. L., 770.
Melton, G., 722.
Melville, K. I., 735.
Meyerhof, O., 770.
Michel, H. O., 731.
Mider, G. B., 749.
Miles, E. M., 766.
Millen, R. S., 739.
Miller, J. J., 774.
Mills, K. C., 777.
Mills, M. A., 744.
Mills, R. C., 754.
Miner, A. C., 766.
Ministry of Health, 745.
Mirsky, I. A., 746.
Mixner, J. P., 734.
Moe, R., 743, 751.
Mohammed, A. H., 766.
Mollov, M., 776.
Molnár, V., 711.
Montigel, C., 723.
Moore, F. D., 712.
Moore, M. T., 728.
Morley, G. H., 761.
Morris, H. P., 749.
Morton, D. G., 738.
Moser, 751.
Mosher, L. M., 751.
Moskowitz, S., 767.
Moss, E. S., 709.
Moss, W. G., 720.
Motzok, I., 755.
Moussatché, H., 723.
Mueller, C. D., 707.
Mulder, A. G., 757.
Mull, J. W., 716.
Munoz, J. de L., 739.
Murphy, W. P., 780.
Murray, M. M., 751.
Murray, N. A., 780.
Murrill, W. A., 714.
Muus, J., 719.

Najjar, V. A., 752.
Nelson, D., 721.
Nelson, E. E., 762.
Nelson, L. M., 772.
Nelson, N., 746.
Nettleship, A., 748, 749.
Neuberg, C., 770.
Newburgh, J. D., 745.
Newman, B., 744.
Newman, M. T., 708.
Newman, W., 726.
Niggli, F., 778.
Niven, C. F., 775.
Nolan, L. E., 756.
Novak, E., 738.

Obermer, E., 766.
Ochsner, A., 736.
Odoriz, J. B., 727.
Ogle, K. N., 730.
Okrent, A., 754.
Oldham, F. K., 761.
Olliver, M., 754.
Olson, M., 722.
O'Malley, E., 715.
Ordal, E. J., 762.
Organ, J. G., 755.
Osgood, E. E., 710, 760.
Osgood, H., 718.
Osterberg, A. E., 745.
Otis, A. B., 769.
Overman, R. S., 713.
Owen, P. S., 753.
Ozorio de Almeida, M., 723, 724.

Page, R. C., 713, 766.
Pagel, R. W., 745.
Palmer, W. L., 775.
Pappenheimer, J. R., 718.
Pariser, H., 775.
Parker, R. R., 777.
Parnas, J. K., 770.
Partridge, R., 714.
Pattle, R. E., 722, 759.
Paul, J. T., 711.
Paul, W. D., 732.
Pauls, F., 744.
Payne, F., 708.
Peddie, W., 733.
Pereira, J., jun., 718, 765.
Pereira, R. S., 711.
Perrin, T. G., 709.
Perry, C. A., 772.
Perry, J. S., 738.
Peterfy, R. A., 746.
Peters, L., 765.
Peterson, W. H., 773.
Petit, J. V., 714.
Petitpierre, C., 721.
Petro, J., 759.
Pfankuch, E., 768.
Phillips, P. H., 742, 743.
Philpot, F. J., 729.
Philps, S., 733.
Picken, L. E. R., 712.
Pierce, L. F., 778.
Pincus, G., 746.
Pinösch, H., 777.
Pirie, A., 729.
Pirosky, I., 777.
Podloucky, F. H., 769.
Polgár, A., 715.
Pomerene, E., 746.
Poncher, H. G., 711.
Post, K., 778.
Postoloff, A. V., 736.
Pratt, J. P., 741.
Prelog, V., 741.
Preuss, F. S., 714.
Pritchard, A. L., 747.
Pritchard, E. A., 765.
Puck, T. T., 776.
Pullen, R. L., 741.

Queen, F. B., 780.
Quigley, J. P., 743.

Rabiner, A. M., 765.
Rafsky, H. A., 744.
Rambacher, P., 746.
Rambar, A. C., 755.
Rammelkamp, C. H., 758.
Ramsay, A. J., 744.
Randolph, T. G., 713.
Rantz, L. A., 760, 775.
Rapp, G. W., 775.
Rappaport, F., 715, 776.
Razgha, A., 766.
Read, M. R., 743.
Redish, M. H., 713.
Reforzo-Membrives, J., 737.
Reid, D. M., 746.
Reisner, E. H., jun., 711.
Rezende, C., 732.
Rhorer, A., 709.
Rice, K. K., 752.
Rich, E., 726.
Richards, R. L., 728.
Richardson, J. S., 741.
Richter, C. P., 752.
Richter, D., 716.
Riddle, O., 737.
Riley, E. G., 752.
Rimoldi, H., 725.
Rink, R., 766.
Risteen, W. A., 725.
Robbins, W. J., 771.
Robertson, G. W., 733.
Robertson, O. H., 776.
Robertson, W. v. B., 749.
Robins, S., 721.
Robinson, E. J., 758.
Robinson, G. G., 717.
Robinson, M., 741.
Robinson, S., 758.
Robson, J. M., 731, 761.
Rocha e Silva, M., 778.
Rodaniche, E. C., 775.
Rodnick, E. H., 724.
Roesli, H., 744.
Rogers, H. J., 773.
Romano, J., 728.
Roney, H. B., 767.
Roschach, 752.
Roseman, E., 727.
Rosenburg, S., 759.
Rosenzweig, S., 739.
Rothlin, E., 765.
Roughton, F. J. W., 758.
Roux, G. H., 709.
Rovenstine, E. A., 763.
Rowe, L. W., 763.
Rubinstein, J. M., 728.
Rubnitz, A. S., 714.
Ruffin, J. M., 743.
Ruhl, G., 712.
Russe, W. O., 735.
Russell, H., 720.
Russell, J. A., 716.
Ruth, E. B., 709.
Ruzicka, L., 741.
Ryan, J., 780.
Rycroft, B. W., 734.

Sabin, A. B., 752.
Sachar, L. A., 719.
Sachs, E., 731, 735.
Sahyun, M., 745.
Sailer, S., 739.
Saito, T. M., 774.
Salit, P. W., 732.
Sallmann, L., 731.
Salmon, G. W., 722.
Sammartino, R., 740.
Sampayo, R., 777.
Sandford, M., 773.
Sandground, J. H., 764, 765.
Sandholzer, L. A., 772.
Sarason, E. L., 735.
Sargant, W., 715.
Sawyer, C. H., 707.
Schachter, M., 718.
Scheinfinkel, N., 723.
Schenck, J. R., 757.
Schleicher, E. M., 709, 714.
Schnedore, J. G., 722.
Schöberl, A., 746.
Scholander, P. F., 780.
Schonfeld, W. A., 738.
Schopfer, W. H., 771.
Schukina, L. A., 755.
Schulze, J. W., 705.
Schutz, S., 730.
Schwab, J. L., 776.
Scott, E. W., 757, 761.
Scott, G. I., 731.
Scott, T. F. M., 760.
Scott, V. B., 743, 751.
Seeler, A. O., 761.
Seevers, M. H., 721.
Seligman, A. M., 780.
Sellschopp, U., 716.
Seltzer, C. C., 766.
Selye, H., 708.
Senderey, S., 736.
Senekjie, H. A., 771.
Sepulveda, J., 724.
Severac, M., 775.
Sexton, E. L., 770.
Shaffner, C. S., 742.
Shapiro, S., 713.
Shear, M. J., 748.
Sheehan, H. L., 735.
Shemiakin, M. M., 755.
Sherman, J. M., 775.
Shettles, L. B., 763.
Shimkin, M. B., 748.
Shleser, I. H., 719.
Shock, N. W., 721.
Shooter, R. A., 711.
Shover, J., 734.
Shuler, R. H., 717.
Shumacker, H. B., jun., 735.
Shvezov, J. B., 755.
Sigurdsson, N., 710.
Silber, S., 761.
Simonson, E., 725, 764.
Singer, K., 746.
Singer, M., 707.
Siris, J. H., 725.
Sjaardema, H., 727.
Sladovic, L., 737.
Slinger, J. S., 755.
Smelser, G. K., 736.
Smiley, K. L., 775.
Smith, A. R., 767.
Smith, E. L., 720.
Smith, F., 777.
Smith, H. W., 774.
Smith, J. J., 718.
Smith, M. G., 776.
Soffer, L. J., 735.
Solarz, S. D., 717.
Solen, M. H., 721.
Somogyi, J. C., 723.
Sorkin, S. Z., 714.
Spaulding, L. B., 712.
Spencer, E. L., 770.
Spencer, J., 760.
Spink, W. W., 759.
Spitz, S. H., 759.
Spitzer, R., 773.
Sprague, J. M., 705.
Squires, G. V., 709.
Stadler, H., 706.
Stämpfli, R., 767.
Starbuck, G. W., 718.
Staub, H., 751.
Stead, E. A., jun., 728.
Steeves, L. C., 712.
Stehle, R. L., 735.
Steigman, A. J., 777.
Stein, H. B., 739.
Steinberg, H. H., 766.
Steinberg, P., 776.
Steinfeld, E., 773.
Steinhaus, E. A., 777.
Steinman, H. G., 764.
Stekol, J. A., 752.
Stettin, De W., jun., 757.
Stevenson, L. A., 740.
Stevenson, S. S., 715.
Stevenson, V. L., 740.
Stewart, H. L., 741.
Stirnimann, F., 751.
Stitt, P. G., 776.
Stone, W. E., 726.
Stoppani, A. O. M., 734.
Stoty, E., 716.
Stowell, R. E., 747.
Stran, H., 763.
Straub, J., 716.
Straus, W. L., jun., 735.
Strong, F. M., 753.
Strong, L. C., 747.
Strong, M. E., 772.
Stuart, C. K., 735.
Stuart, H. C., 708.
Stuart-Harris, C. H., 777.
Stuckey, J., 710.
Sturgis, C. C., 710.
Stutzman, J. W., 762.
Süllmann, H., 778.
Sulman, F., 740.
Sverdlick, J., 733.
Swan, K. C., 731.
Swank, R. L., 727.
Swingle, K. F., 760.
Szécsényi-Nagy, L., 712.
Székessy, W., 715.

Taber, E., 710.
Takacs, W. S., 774.
Talbot, J. H., 758.
Talbot, N. B., 780.
Tat, R. J., 710.
Tauber, O. E., 723.
Taylor, H. L., 714.
Taylor, S., 737.
Taylor, S. G., 737
Teply, L. J., 753.
Tetwin, I. J., 780.
Thomas, J. E., 744.
Thomas, L., 774.
Thompson, J. D., 722.
Thompson, P. K., 737.
Thompson, W. O., 737.
Thornton, C. S., 707.
Thrower, W. R., 761.
Thurnherr, A., 722.
Tietz, H. G., 740.


[Continued on page iii of ...

Tilden, I. L., 776.
Tilley, A. R., 761.
Tobey, H. G., 734.
Tobin, L. H., 712.
Todd, A. R., 773.
Tollefson, D. G., 763.
Tomkins, C. A., 762.
Tonkin, I. M., 762.
Toomey, J. A., 774.
Torre, L. D., 736.
Torda, C., 723.
Town, A. E., 729.
Trapp, E., 776.
Trappe, W., 780.
Trowell, H. C., 711.
Tunbridge, R. E., 746.
Turnbull, F., 723.
Turner, C. W., 734.
Turner, W. J., 716.
Twombley, G. H., 739.

Udenfriend, S., 870.
Ungar, G., 720.
Ungley, C. C., 754.

Valentine, F. C. O., 761, 775.
Van den Ende, M., 777.
Van Pernis, P. A., 735.
Van Rooyen, C. E., 777.
Van Wagenen, W. P., 725.
Van Wagtendonk, W. J., 756.
Vassel, B., 714.
Vaughan, E., 747.
Veiga-Salles, J. B., 768.
Venning, E. H., 736.
Vernon, H. K., 718.
Vianna Dias, M., 723.
Vickers, V. S., 708.
Villela, G. G., 745, 752.
Vorhaus, M. G., 753.

Wachholder, K., 754.
Waddell, W. W., jun., 722.
Wakerlin, G. E., 720.
Waksman, S. A., 770.
Wald, G., 752.
Waldschmidt-Leitz, E., 769.
Walker, P. A., 740.
Wall, N. M., 759.
Wallenfels, K., 738.
Waller, H. K., 741.
Wallersteiner, W. K. S., 775.
Walter, W. G., 727.
Walton, R. P., 780.
Wangerin, D. M., 751.
Ward, N. W., 779.
Ward, R., 752.
Ward, W. E., 776.
Waring, H., 737.
Waring, W. S., 772.
Warren, J. V., 728.
Washburn, S. L., 705.
Wasson, V. P., 756, 775.
Waterman, A. J., 706.
Watson, L., 712.
Watson, M., 773.
Way, C., 752.
Waymouth, C., 709.
Weatherby, L. S., 779.
Weddell, G., 722.
Weddle, C. L., 778.
Weech, A. A., 756.
Weeks, J. R., 762, 764.
Weingarten, R. J., 713.
Weir, J. R., 720.
Weissman, N., 753.
Welch, P. B., 744.
Weld, C. B., 731.
Went, F. W., 779.
Werch, S. C., 765.
Werkman, C. H., 772.
Werner, S. C., 736, 746.
Wespi, H., 724.
West, R., 736.
Westerfeld, W. W., 757.
Weyrauch, H. M., 746.
Wheeler, M. C., 730.
White, H. L., 745.
White, J., 749.
White, P. D., 718.
Widdowson, E. M., 750.
Wilder, V. M., 715.
Wilhelmi, A. E., 716.
Willbrandt, W., 767.
Williams, B. W., 773.
Williams, H. V., 760.
Williams, R., 766.
Williams, P. C., 763.
Williams, T. W., 706.
Willius, F. A., 718.
Willmer, E. N., 732.
Wilson, D. C., 751.
Wilson, J. L., 760.
Wilson, J. V., 746.
Wilson, P. W., 772.
Winer, J. H., 746.
Winkelman, N. W., 728.
Winter, J. E., 776.
Winters, M., 762.
Wokes, F., 755.
Wolfe, J. K., 780.
Wolfe, J. M., 708.
Wolfe, O., 732.
Wolfe, R. M., 732.
Woodard, T. M., 743.
Woodbury, R. A., 717.
Woodhall, B., 728.
Woodward, F. D., 722.
Woolpert, O. C., 776.
Wright, H. N., 765.
Wright, W. D., 732.
Wulzen, R., 756.
Wunderly, C., 768.

Yacorzynski, G. K., 724.
Ying-K'uei, H., 726.
Yntema, C. L., 707.
Yudkin, J., 733.
Yudkin, S., 733.

Zeller, E. A., 769.
Zondek, H., 734.
Zschokke, H., 751.
Zselyonka, L., 766.
Zucker J. M. 728.



## ERRATUM.

**Abstracts A., III, 1943.**

| Page | Line | |
|---|---|---|
| 512 | 11* | *For* "Borque" *read* "Bourque." |

* From bottom.

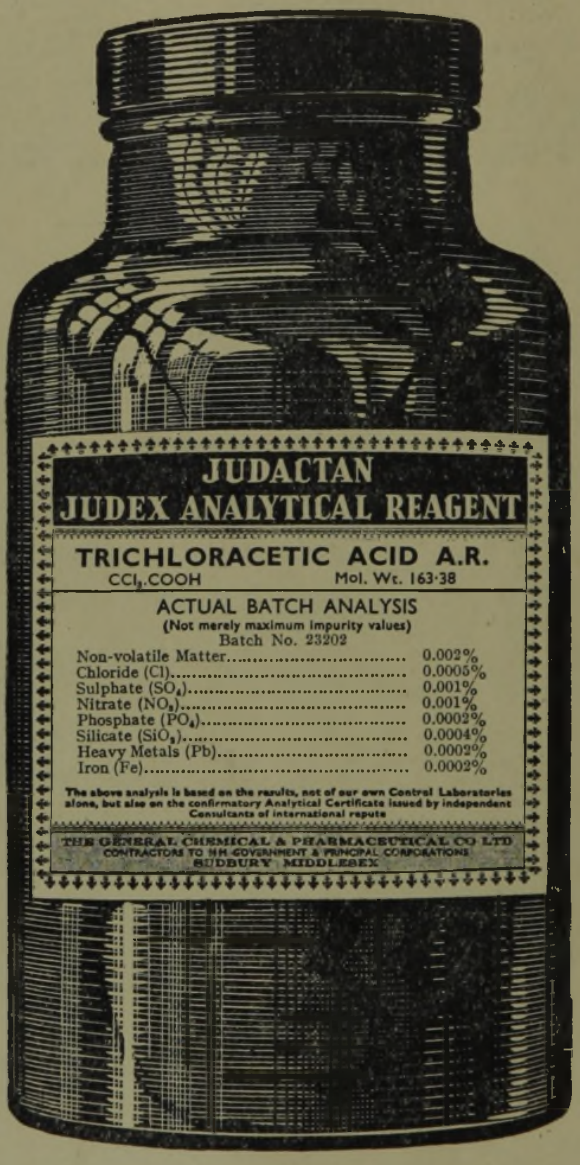
# JUDACTAN

## ANALYTICAL REAGENTS WITH ACTUAL BATCH ANALYSIS

ACTUAL

BATCH

ANALYSIS




Each Batch subjected to INDEPENDENT ANALYSIS before label is printed

You are invited to compare the above actual batch analysis with the purities guaranteed by the specifications of any competing maker in this Country or abroad

**THE GENERAL CHEMICAL & PHARMACEUTICAL CO. LTD.**

**Chemical Manufacturers, Judex Works, Sudbury, Middlesex**

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