P. F. M. Fellowes), concerts by Miss Jelly d'Aranyi and Mr. Norman Notley, and an exhibition of engineering models and scientific apparatus. To the exhibition contributions were sent by various official bodies, such as the Science Museum, the National Physical Laboratory, the Building Research Station, the Forest Products Research Laboratory and the Woolwich Research Department. Many of the exhibits related to structural engineering (unit construction of Warren-truss Bridges, a supporting structure in tubular steel scaffolding, the underpinning of Durham Cathedral and the protection of structural steelwork after erection by means of combined metallic coatings and paint), and to road engineering (an instrument for measuring the camber and gradient of roads, plant for automatically proportioning the ingredients of concrete and road material and models and diagrams illustrating the flow of traffic at complicated circus and bridged road junctions). Among scientific apparatus Sir Robert Hadfield showed a petrological microscope and a contact thermo-couple pyrometer, with specimens of the steel used for making the turbine blades of the *Normandie;* Mr. C. C. Paterson, an instrument for viewing the response curves of radio-frequency filters, and Dr. J. S. Owens, apparatus for measuring ultra-violet radiation in daylight, a water gauge reading to 0.1 mm of water and an evaporimeter for measuring the loss of water from different surfaces. There was an exhibit of the Harlandic-Synclock synchronous time system for ships, and Imperial Airways sent models of their Scipio flying boats and Heracles air liners.

DISCUSSION

DEEP-FOCUS EARTHQUAKES AND ISOSTASY

THE demonstration that the foci of numerous earthquakes lie from 100 to 700 kilometers below the surface has come as a matter of the greatest interest to students of the earth's structure and dynamics—so much so, indeed, that some seem inclined to conclude that this discovery proves that ordinary faulting can occur to depths of 400 to 700 kilometers,[1] and consequently that subcrustal readjustment and flowage does not take place in the manner postulated by the exponents of isostasy.

Before needless scientific confusion arises because of too ready acceptance of the belief that deep-seated faulting *is* thus proven, it may not be amiss to raise the question whether these deep-focus quakes necessarily must be regarded as due to ordinary faulting (*i.e.*, due to a sudden rupture or slipping induced by accumulated, elastically stored stress) or whether such deep-focus earthquakes may not be essentially similar in origin to "volcanic" earthquakes which result from surface or near-surface volcanic explosions.

The interesting and important experiments being conducted by Dr. Bridgman at Harvard are reported to demonstrate that at great confining pressures, and under varying conditions of rotational movement, various substances undergo explosive chemical transformations or physical changes of state.[2] Compilations by Turner[3] and Sharpe[4] also show that the bulk of the deep-focus earthquakes occur in the Japanese Archipelago, the East Indies, the West Coast of South America and the Himalayas—regions in which it has already been suggested that rocks such as normally occur near the earth's surface are now deeply invaginated in denser subcrustal material. These regions should, consequently, afford unusually numerous opportunities for the occurrence of rock rupture by sudden polymorphic transformations or explosive chemical reactions—such reactions, for example, as those which apparently caused the outbursts responsible for the diamond "pipes" of South Africa and for various "crypto-volcanic" features, such as have been described by Bailey and Bucher.

Pending proof that the deep-focus earthquakes *are* due to ordinary faulting, and are not due to instantaneous rupture produced by deep-seated "explosions," it would seem to be in order to consider that their bearing on the problems of tectonics and of isostasy remains indeterminate.

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[1] *Nature*, 136: 3446, 782–784, November 16, 1935.
[2] SCIENCE (Supplement), 82: 2135, 8, November 29, 1935.
[3] Int. Seismol. Summary, 1927, Jan.–Feb.–March issue.
[4] SCIENCE, 82: 2135, 523–524, November 29, 1935.

SELENIUM IN NUTRITION

EVER since the indication[1] that the so-called "alkali disease" of live stock of the north-central Great Plains might be due to traces of selenium in the indigenous grains and forage, the nutritional aspects of orally ingested selenium have received increasing amounts of attention. Some of the effects of the natural toxic grain have been revealed by the outstanding work of Franke and colleagues.[2]

The toxicological action of selenium salts was first studied by Gmelin,[3] who reported the production of a

[1] U.S.D.A., Circular 320, August, 1934.
[2] K. W. Franke, *Jour. of Nutr.*, 8: 597, 1934.
[3] Gmelin, "Versuche über die Wirkungen des Baryts. u. Stronians u.s.w. auf den thierischen Organismus," Tübingen, p. 43, 1824.

garlic odor in the breath and of metallic deposits in the intestinal walls. Selenium in the metallic state was described as innocuous, and the consensus of pharmacological and toxicological opinion at that time was that the toxic action of selenium salts was much the same as that of arsenic or other poisonous metals. Physiologically, reduction of selenium salts to the metallic state as, *e.g.*, by glucose, appeared to be the means of defense of the organism.

Nutritionally, selenium was regarded as of no importance. Only Gassmann[4] claimed it to be a normal constituent of human teeth and urine, but this was disclaimed by Fritsch.[5] However, in the light of the new relation to "alkali disease," consideration of the chemical similarity of selenium and sulfur and the importance of the latter in physiological oxidation-reduction systems made it seem probable that on sublethal levels of ingestion, selenium salts might play a rôle in the nutritive processes of animals.

To investigate the effects of the ingestion of inorganic selenium as distinct from the apparent organic combinations occurring in the natural toxic grain, sodium selenite was incorporated in a good stock diet for rats. At a level of 70 p.p.m. it arrested the growth of 3-weeks-old albino rats and caused emaciation and death within 2 weeks. A level of 35 p.p.m. caused slight and erratic growth, death occurring in about 6 weeks; 17.5 p.p.m. resulted in slightly subnormal growth and a variation in the lethal effect, some animals living as long as 267 days with no other symptoms than a slightly subnormal growth. Food consumption records indicated these growth effects to be due to inanition. This was verified by restricted intake experiments on control animals.

When adult rats were placed on a selenium diet at a level of 35 p.p.m. they showed the same variability in susceptibility, but in general withstood the poisoning more effectively than growing rats on the same level. After an initial inanition they gradually recovered weight and consumed normal quantities of ration. Occasionally such animals succumbed, entering a rapid cachexia and dying with the symptoms noted below. High levels of 420 p.p.m. fed to adult rats resulted in an inanition so severe that the animals virtually starved to death, although autopsy revealed subacute pathologic changes.

In acute selenium poisoning animals died after a short period (2 to 3 weeks). This category includes animals on high levels (70 p.p.m.) and those animals on lower levels which, due to individual susceptibility or constitutional weakness, died in about the same period of time. These rats lost weight rapidly or else gained weight slowly and erratically. They assumed a hunched position and were inclined to be restive. The urine became highly pigmented. Post-mortem examination revealed the liver as the seat of the most important pathological changes. When death was incident early the liver was inclined to be hemorrhagic and thin edged; when death occurred later it showed a tendency to fatty degeneration, friability and atrophy. Kidneys were also inclined to be hemorrhagic. Ascites was frequently observed.

In chronic selenium poisoning on diets low in selenium (17.5 p.p.m. and less) susceptibility was quite variable. Growth was slightly subnormal. Symptoms developed in these animals while they were still in a good nutritive state. Later the progress of cachexia was rapid, the urine became highly pigmented, and a rapid and progressive anemia developed with hemoglobin levels as low as 2.11 gm./100 cc. Liver cirrhosis, ascites, adhesions and regeneration of the liver were observed at autopsy. Heart and spleen were hypertrophied, testes and uterus atrophied. Pleural edema was frequent and an acute peritonitis was often terminal. These symptoms are identical with those reported by Franke for the natural toxic grain.

The importance of sulfhydryl in the oxidation-reduction relations of normal physiology made it seem probable that selenium might have a disturbing effect on such relations. Studies in the gross, by determination of the basic metabolic rates of rats on a 17.5 p.p.m. régime, gave no indication of significant variations from the normal. Using Rubner's formula for surface area and neglecting protein metabolism the mean basal metabolic rate of 16 experimental animals was found to be for males, 819 cal./24 hrs./sq. meter; for females, 731 cal.

The effects of metabolizable sulfur added as cystine at levels of 0.2, 0.4, 0.6 to the cystine low diet of Jackson and Block[6] did not mitigate the growth-arresting effects of selenium at a level of 35 p.p.m., nor did it have any postponing effect on the ultimate lethal action. For the more specific oxidation-reduction systems such as ascorbic acid and glutathione, determination in the livers of rats on a selenium diet showed no distinct trends in oxidizability from the range in the controls.

Early toxicologists postulated an action of selenium on the organism as a whole but did not demonstrate the toxicity for any specific cell function. By means of oxygen uptake studies in the Warburg apparatus we have demonstrated a reduction of the oxygen uptake rate in rat liver by sodium selenite when added in vitro at concentrations of M/300. Oxygen uptake was diminished approximately 50 per cent. during the first half hour and 70 per cent. during the second half hour. Diminished oxygen uptake could not be demonstrated

[4] T. Gassmann, *Zeit. Physiol. Chem.*, 97: 307, 1916; 98: 182, 1917.

[5] R. Fritsch, *Zeit. Physiol. Chem.*, 104: 59, 1918; 109: 186, 1920.

[6] R. W. Jackson and R. J. Block, *Jour. Biol. Chem.*, 98: 465, 1932.

in the livers of rats which had previously received a selenium ration at levels of 70, 35, and 177.5 p.p.m.

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NORTH AMERICAN FISH-HOOKS

BIREN BONNERJEA in his recent article in SCIENCE,[1] "North American Fish-Hooks," states that he has come to the conclusion that the barbs on fish-hooks (in America) might have been an original invention, concluding the article by saying, "it seems to me that ethnographers and archeologists in the field would do well to bear this [looking for proof to that end] in mind."

While in charge of the Quileute Indians at LaPush, Washington, from 1905 to 1909, the writer did much excavating about the hill on which the Indian village is situated. The first two feet of debris there included numerous Hudson Bay beads, as well as other white man's things. Below this level only aboriginal things were found, among which were many Indian fish-hooks, of the barbed-outside type, these being found often in the lowest debris exposed there, often six feet below the stratum that contained the blue trade beads. This proves conclusively that the barbed hooks were in use before the coming of the white man to that region.

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TERMITES IN CENTRAL NEW YORK STATE

DURING a recent discussion on the distribution of Termites, mention was made of the scarcity of these interesting insects north of the Mason and Dixon line.

In the late spring of 1933, a nest of Termites (*Reticulitermes flavipes*) was discovered on the north bank of Cascadilla Creek, several hundred yards southeast of the Cornell University stadium, by Mr. Wade H. Hadley, Jr., and the writer. Note of this northern station for this species of Termite might be of interest to the entomologist.

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SCIENTIFIC BOOKS

THE AUTONOMIC NERVOUS SYSTEM

The Autonomic Nervous System. Anatomy, Physiology, and Surgical Treatment. By JAMES C. WHITE, assistant professor and tutor in surgery, Harvard Medical School, assistant visiting surgeon, Massachusetts General Hospital, Boston. xviii + 386 pp. The Macmillan Company, 1935. Price $7.00.

THIS important monograph written by a surgeon is a highly significant contribution both to the anatomy and the physiology of the autonomic nervous system. In addition it represents the most authoritative clinical discussion of surgery of the autonomic nervous system that has yet appeared. After a brief historical account of the autonomic system, in which the date and title of Eustachius' celebrated plates are erroneously stated and the name of Gaskell is not to be found, White gives an excellent brief discussion of the anatomy of the autonomic nervous system which can be safely recommended to students. Indeed it is much more complete and concise than that to be found in any current text-book of anatomy or physiology. It is to be regretted, however, that the unimaginative publishers have virtually ruined White's excellent tabular summary of the innervation of the principal organs on pages 35–46 by lack of suitable indentation and complete disregard for the niceties of typography. The chapter on general physiology is sound, but dull, suggesting that too many physiologists tried to help the conscientious author.

The most significant disclosure in the book is described on pages 92–98 in which it is pointed out that the conventional operation for removing the sympathetic supply of the upper extremities destroys the cells of origin of the post-ganglionic fibers, whereas the conventional operation for the removal of the sympathetics of the lower extremities (ablation of lumbar ganglia 1, 2, 3 leaving intact the lower lumbar and sacral ganglia) leaves the cells of origin of the post-ganglionic fibers virtually intact. When the post-ganglionic cells are removed the tissues formerly innervated by them become sensitized to adrenaline. Consequently when such an animal or a human being secretes adrenin or is given adrenaline, extreme vasoconstriction occurs in the sensitized part. This, White argues, gives an intelligent physiological explanation for the virtual failure of the conventional operation for sympathectomy of the upper extremity. Having pointed this out, White then describes a procedure for pre-ganglionic denervation of the upper extremity, and in the few cases thus far tried this gives more satisfactory immediate results than the older procedure. It is based upon the fact, originally disclosed by Langley and long overlooked, that the pre-ganglionic vasoconstrictors of the upper extremities emerge from the spinal cord at levels below the third or fourth thoracic, often extending as far down as the tenth thoracic. Consequently when the thoracic sympathetic trunk is merely cut through at Th. 3 or 4, the pre-ganglionic vasoconstrictors of the upper extremity are entirely removed and the post-ganglionic cells remain intact. Regeneration, however, may occur.

White, like Livingston, whose recent book, "The

[1] 82: 2134, p. 492.