transfer of the nitrogen of one amino acid into other amino acids is also associated with a more fundamental synthesis of the carbon chain of the latter. We are now able to offer proof of the occurrence of extensive deamination and reamination of amino acids of the proteins in normal animals, by following the fate of the amino acid *l*-leucine, which contained two different and independent isotopes, namely, deuterium in the carbon chain and N^{15} in the amino group.

Four adult rats were kept on an ordinary stock diet (containing 15 per cent. casein) in nitrogen equilibrium without change of weight. To the diet was added an amount of *l-leucine* corresponding to 23 mg of nitrogen per rat per day for three days; the animals were then immediately killed by exsanguination. The leucine (possessing the natural configuration) contained 3.6 atom per cent. deuterium and 6.5 atom per cent. N^{15}; it was obtained by resolution of the synthetic racemic mixture described before.[4] While the total amount of nitrogen excreted corresponded to that in the diet, the excreta contained only 30 per cent. of the administered isotopic nitrogen, most of the remainder being incorporated in the body proteins. Only 8 per cent. was found in the "non-protein nitrogen" of the tissues.

The proteins of the liver, of the intestinal wall and of the remaining carcass were worked up separately, and the following analytically pure amino acids were isolated from the different proteins: three preparations each of arginine, tyrosine, glutamic acid, aspartic acid and leucine; two preparations of glycine and lysine, and one preparation of ornithine obtained by degradation of liver arginine. All the compounds, with the exception of the two lysine preparations, contained appreciable amounts of nitrogen isotope, a finding which corroborates our earlier observations after feeding tyrosine. The isotope concentration in the three leucine preparations was considerably higher than that of any other amino acid obtained from the same protein.

Indication of the mechanism responsible for the nitrogen transfer was obtained by the closer investigation of the leucine isolated from the proteins of the animals. The isotope (D and N^{15}) concentrations of the leucine isolated were considerably lower than those of the material fed. This was to be expected, as the dietary leucine was mixed with the ordinary leucine of the casein and with that of the tissue proteins into which the dietary leucine was introduced. If the leucine isolated from the tissues were only "diluted" by this "ordinary leucine," the ratio of the concentrations of the two isotopes in the compound should have been the same as in the material fed. The ratio (D : N^{15}), however, was altered considerably. It was 100 : 167 in the leucine administered and 100 : 103 in that isolated from the carcass. This result indicates that the carbon chain of leucine (characterized by the labeled hydrogen) had given up part of its nitrogen (in the present case labeled nitrogen) and in turn had "accepted" new nitrogen (which was normal). The other amino acids were obviously also involved in this process, in that they gave up normal nitrogen and accepted isotopic nitrogen from leucine.

The rapid introduction of a dietary amino acid into the tissue proteins, as well as the rapid and continuous deamination and amination of a large number of amino acids demonstrated in proteins of different organs has here taken place in normal adult animals on a stock diet. As all these reactions require the opening of peptide linkages in the protein, the finding is a new indication of the high chemical activity of the tissue proteins. The experimental details and their physiological and chemical implications will be presented elsewhere.

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A METHOD FOR PRODUCING PERSISTENT HYPERTENSION BY CELLOPHANE

IT has been found that arterial hypertension can be produced in dogs by wrapping one or both kidneys in Cellophane.

Dogs were anesthetized, a kidney freed from its bed, and after stripping off the fat on the surface, Cellophane sterilized in alcohol was gently wrapped around it and secured either with paper clips or a ligature. It is not necessary that very accurate approximation of the kidney and Cellophane occur. The kidney is gently replaced and the wound closed. This procedure may be repeated on the opposite kidney.

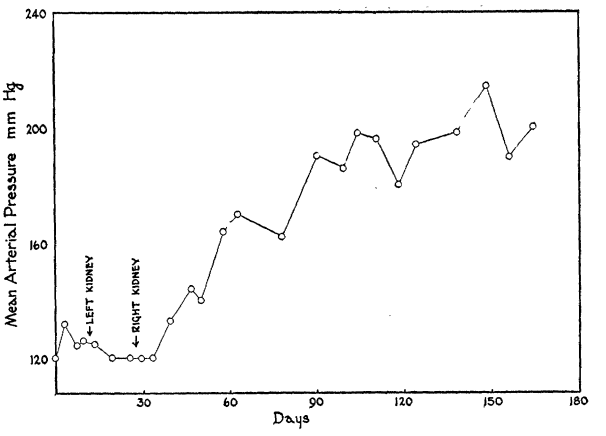

FIG. 1. Both kidneys were placed in Cellophane and blood pressure measured by intra-femoral puncture.

After about two to three weeks the arterial pressure, as measured by direct intra-arterial puncture of the femoral artery, begins to rise and may reach levels as high as 240 mm Hg mean pressure after a month or two. In some animals the pressure reaches a peak and tends to fall to lower levels, while in others the pressure has remained at high levels for seven months, *e.g.*, as long as the animals have been observed. Application of Cellophane to one kidney causes hypertension, but it is not so extreme as when both kidneys are treated.

At autopsy the kidney is found to be surrounded by a dense hull of tissue (fibroblastic and collagenous) as much as 4 to 5 mm thick. This is readily stripped from the surface of the kidney.

Hypertension produced by this method occurs whether the normal capsule is stripped or not before application of Cellophane. Denervation of the kidneys also does not interfere with its development. Removal of the offending kidney in animals in which hypertension has occurred after applying Cellophane to only one kidney causes the hypertension to disappear if it has not persisted for a long time.

We have not been able to produce hypertension by applying rubber (rubber surgical glove) to the kidneys. Other organs such as the heart, liver and adrenal glands have been enclosed in Cellophane, but it is too early to be certain of the results. In three experiments the pericardium showed signs of developing a constricting envelope, but the animals died before signs of constrictive pericarditis occurred.

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THE SUCCESSFUL TREATMENT OF MENINGO-ENCEPHALITIS, ASSOCIATED WITH CANINE DISTEMPER, WITH SULFANILAMIDE

MENINGO-ENCEPHALITIS associated with canine distemper has been considered for years an incurable disease. The mortality rate reported by veterinarians is from 90 to 100 per cent. Goldberg and Volgenau[1] reported on 73 cases treated at the New York State Veterinary College with one recovery. Our records indicate the rate to be essentially 100 per cent. when the disease is accurately diagnosed.

Canine distemper produced by the virus of Carré can be readily controlled by homologous serum, which has no effect on meningo-encephalitis. Sulfanilamide has little value as a therapeutic agent for distemper, in our own experience and in that of veterinarians generally.

The specific etiological agent for meningo-encephalitis associated with canine distemper is, however, unknown. Some believe that the virus of Carré is responsible, others that it is a bacterial invasion secondary to the virus of Carré and still others that it is due to certain specific bacteria and their toxins. In our work we have been unable to demonstrate any microorganisms in the brain or spinal fluid of dogs affected with meningo-encephalitis associated with canine distemper.

We have used sulfanilamide to treat fourteen dogs suffering from meningo-encephalitis associated with canine distemper. The sulfanilamide was administered in such dosage either by mouth or subcutaneously so that the sulfanilamide blood level was maintained at not less than 15 mgms per cent. Experience has shown that it is desirable to maintain this blood sulfanilamide level and that the administration of the drug should be started in the initial stages of the disease before extensive pathological changes are manifest.

Thirteen of the dogs have made complete recoveries. This is a recovery rate of about 93 per cent. compared to a mortality rate of from 90 to 100 per cent. in dogs not receiving sulfanilamide. There was a marked and rapid clinical improvement which usually took place in from 48 to 96 hours. Dogs with total anorexia started to eat. If the temperature was elevated it returned to normal. In the affected animals there was a lymphocytic leucopenia and a polymorphonuclear leucocytosis. After treatment with sulfanilamide, there was a favorable increase in the lymphocytes and a reduction in the polymorphonuclear leucocytes. The red cell count and the haemoglobin was not altered.

A detailed report of this work will appear later in another journal.

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