slowly for one hour. Strain the stock off. Meanwhile mix the oil and flour till smooth. Add to the stock and bring to boiling point. Add parsley and serve.

The dosage of such a soup is naturally more vague than that of the former recipes, but half a pint will contain more than 375 I.U., and may contain 1,500 if a potent oil has been used. In fact, for ordinary purposes it would be well to substitute one ounce of an ordinary fat for one ounce of cod-liver oil, and so reduce the potency of the soup by one-half.

A batter to be used in deep-fat frying of fish, or for cod's roes, croquettes, etc., is the following:

Ingredients.—Two ounces of flour, one ounce of bread-crumbs, seasoning, one tablespoonful cod-liver oil, and milk to give coating consistency.

Method.—Mix flour, breadcrumbs, and seasoning in a bowl. Make a well in the centre and add the oil. Add enough milk to produce a coating consistency; beat for a few minutes, and set aside to cool for fifteen minutes.

The above recipes should give the average dietitian or the intelligent cook sufficient ideas of how to incorporate sources of vitamin D into the diet of people who are recalcitrant to fish-liver oils when served neat.

REFERENCE

Boas-Fixsen, M. A., and Roscoe, M. H. (1937-8). *Nutr. Abstr. Rev.*, 7, 823.

SYMMETRICAL PERIPHERAL GANGRENE IN CARDIAC FAILURE

BY

C. BRUCE PERRY, M.D., F.R.C.P.

AND

T. B. DAVIE, M.D., M.R.C.P.

(From the Departments of Medicine and Pathology,
University of Bristol)

Fishberg (1938) has recently called attention to the occasional development of symmetrical peripheral gangrene in cardiac failure. In view of the rare occurrence of this phenomenon and of the problems raised in an attempt to explain its pathogenesis, the following case would appear worthy of record.

Case Report

A man aged 64 first sought advice for rapidly increasing dyspnoea in September, 1937. He had suffered from a chronic cough for many years, but recently had been expectorating sputum which was pink in colour. With rest in bed he improved considerably, but in February, 1938, the dyspnoea became more severe and he suffered from "attacks" of dyspnoea and "faintness" associated with pain in the chest. He was admitted to hospital on February 28 with severe congestive failure, too ill to give a satisfactory history. He was cyanosed, extremely dyspnoeic, with congestion of the veins of the neck, enlargement of the liver, and oedema of the legs and lumbo-sacral region. The heart was considerably enlarged, the apex beat being in the sixth space in the anterior axillary line. There was an apical systolic murmur and a rather faint and indefinite aortic diastolic murmur. The blood pressure was 175/85. Rhonchi and rales were audible all over both lungs. He did not respond to treatment, and three days after admission petechial haemorrhages were noticed on the face, neck, and hands. At this stage both legs and feet became gangrenous, the whole area below the middle of the leg being cold and plum-coloured. The upper limit of the gangrenous area was sharply defined, though there was no definite line of demarcation. In view of the petechial haemor-

rhages and the sudden onset of the gangrene a tentative diagnosis of bacterial endocarditis with a saddle embolism of the common iliac arteries was made. The patient died that evening.

POST-MORTEM EXAMINATION

Chest.—There was some clear fluid in both pleural cavities, most marked on the right side, and both lungs were fixed to the pleura at the bases and posteriorly by old adhesions. The right lower lobe showed some bronchiectasis, and there was a diffuse fibrosis extending throughout both lower lobes. In places this resembled chronic fibroid phthisis, but no active tuberculous areas were found. Both lungs showed acute oedema. Histologically there was also a terminal haemorrhagic and purulent bronchopneumonia. The heart was greatly enlarged, weighing 23 oz., the enlargement being mainly due to left ventricular hypertrophy, though both ventricles were dilated. All valves were normal. The coronary arteries showed some atheroma, but this was not severe.

Abdomen.—There was some free fluid in the peritoneal cavity. The alimentary canal revealed patchy congestion, and the contents of the ileum were blood-stained. The spleen was small and hard. The liver showed atrophic cardiac cirrhosis and a very severe degree of "nutmeg" change. It weighed 35 oz. The kidneys were normal in size and had a smooth surface; there was some thickening of the arterioles and some fatty change in the cortex. On section the renal arterioles showed hypertensive hyalinization, but both intimal and medial changes were more marked in the arteries of the spleen. The aorta presented a moderate degree of atheroma, but the iliac, femoral, and popliteal arteries showed no obstruction on either side. The skull was not opened. The *post-mortem diagnosis* was congestive cardiac failure resulting from hypertensive heart disease, pulmonary fibrosis, and bronchiectasis.

Discussion

The occurrence of this symmetrical peripheral gangrene with no occlusion of the arteries supplying the part obviously calls for some explanation. The suggestion put forward by Fishberg is that with severe congestive cardiac failure a selective vasoconstriction occurs (? reflexly) as a result of the extreme diminution in cardiac output, so that a smaller amount of blood is distributed to the limbs and a larger to the brain and the other more important viscera. This view is supported by such cases as the one now recorded and by the observation that in the terminal stages of congestive cardiac failure the antecubital and external jugular venous pressures may be grossly different. Fishberg records an instance where the jugular pressure exceeded 20 cm. of water while the pressure in the antecubital vein was under 1 cm. of water. With improvement, which is rare, he has noted that the antecubital pressure rises to that in the jugular vein.

The murmurs heard during life would appear to have been caused by relative incompetence of the mitral and aortic valves due to the great enlargement and dilatation of the left ventricle.

Summary

A case of congestive cardiac failure, the result of hypertensive heart disease, is described in which symmetrical peripheral gangrene of the lower limbs developed shortly before death.

The mechanism of this is discussed, and it is suggested that this is due to a vasoconstriction which leads to a redistribution of the circulating blood in an attempt to protect the vital organs from the effects of the diminished cardiac output.

We are indebted to Dr. R. H. Parry, medical officer of health for the City of Bristol, for permission to publish this case.

REFERENCE

Fishberg, A. M. (1938). *J. clin. Invest.*, 17, 510.