He had a patient with long-standing diabetes, complicated by retinitis, who developed post-partum necrosis of the anterior pituitary gland. Following this, not only did the insulin requirement fall but there was also remarkable regression of the retinitis. A Swedish team headed by Professor H. Olivecrona as neurosurgeon and Dr. R. Luft as endocrinologist have undertaken hypophysectomy in a group of patients who developed diabetes in childhood and after thirteen or more years had severe retinitis associated in some cases with renal damage. A progress report on the first twenty cases is published elsewhere in this *Journal* (page 752). There were five deaths attributable to the operation, a very high figure in such expert hands. It appears that hypophysectomy for the complications of diabetes is much more hazardous than in cases of malignant disease—partly because of the state of the arteries and partly because of the metabolic problems involved. Two of the remaining patients have died from their diabetic complications; the others failed to show any dramatic improvement, but the progress of the complications does seem to have been slowed or actually arrested. L. W. Kinsell and his group[2] in California have treated five patients in the same way and have reported similar results.

Removal of the pituitary in an attempt to arrest the progress of diabetic retinitis or renal damage is obviously a desperate measure. It is by no means certain that it will succeed, although the evidence so far justifies further investigation, especially since there is nothing else at the present time that offers any hope in these cases. The immediate mortality is high and the post-operative management extremely difficult. A less dangerous approach may well be found in some form of irradiation destruction of the pituitary gland or through chemical or hormonal inhibition of its activity.

INSULIN AND THE HEART

The technique of cardiac catheterization has been ingeniously adapted to the study of myocardial metabolism. By passing the catheter into the coronary sinus I. Ungar *et al.*[1] have been able to draw off venous blood as it leaves the heart, and by comparing its contents with that of samples drawn simultaneously from the femoral artery have obtained information of great interest. Their previous results have already been published,[2 3] and the present paper is on the special problems of the heart in diabetes mellitus.

Observations were made on 22 control subjects, 7 diabetic patients, all but one of whom needed insulin, and 9 dogs. The dogs were studied intact, when rendered diabetic with alloxan, and again after treatment with insulin. It was found that the heart in diabetic men and dogs uses less glucose and less lactate than normal. After insulin the dogs' hearts used more glucose, but lactate utilization was not improved during the observation period. The chief difference found was that the diabetic dog's heart was able to metabolize ketone bodies, whereas the heart in human diabetics utilized fatty acids. The authors concluded that the diabetic heart is deficient in the metabolism of carbohydrate, protein, and fat.

Many points of great interest emerge from their studies. Here is an example of how a technique adapted for a particular study may be usefully turned on to another. Here also is a striking example of species difference in tissue metabolism, and yet another warning against uncritically extending the conclusions of animal experiments to man. These studies confirm that the diabetic heart, instead of using chiefly lactate as a source of energy,[4] can metabolize fatty acids. Finally, the ubiquity of insulin is emphasized once again: not only does it largely control carbohydrate metabolism, but also, directly or indirectly, affects fat and protein too.

[1] Ungar, I., *et al.*, *Amer. J. Med.*, 1955, **18**, 385.
[2] Bing, R. J., *et al.*, ibid., 1953, **15**, 284.
[3] —— ibid., 1954, **16**, 504.
[4] McGinty, D. A., and Miller, A. T., *Amer. J. Physiol.*, 1932, **101**, 76.

CANCER OF THE LUNG IN INDUSTRY

The rapid rise in the mortality from cancer of the lung in recent years, and the increasing weight of evidence that the smoking of cigarettes is directly, and probably causally, associated with the disease, has drawn attention away from other less common circumstances in which lung cancer may develop. A large number of industrial processes have at different times been blamed for the disease,[1 2] and often with good cause. Prominent among these hazards are the mining of certain radioactive ores,[3] the refining of nickel,[4] the manufacture of chromates,[5 6] the manufacture of coal gas,[7] and industrial exposure to arsenical dust.[8 9] Evidence of a similar risk to workers in the asbestos industry was advanced by the Chief Inspector of Factories,[10] and by S. R. Gloyne,[11] who found in necropsy material that primary lung cancer occurred much more frequently in association with asbestosis than with silicosis.

R. Doll[12] has now published an important analysis of the records from a large asbestos works. Records of coroners' necropsies were available for 105 employees who died between 1933 and 1952 and for whom asbestosis had been suspected as a contributory cause of death. Lung cancer was found in 18 cases, in 15 of them associated with asbestosis. All those with both conditions had been employed in the industry for at least nine years before 1933, by which time national dust-reducing regulations had become effective. Following up this suggestive evidence, Doll compared the mortality of 113 employees, who had all worked for at least twenty years in places where they were liable to be exposed to asbestos dust, with that to be expected if

[1] Smith, W. E., *Acta Un. int. Cancr.*, 1953, **9**, 443.
[2] Doll, R., *British Medical Journal*, 1953, **2**, 521.
[3] Lorenz, E., *J. Nat. Cancer Inst.*, 1944, **5**, 1.
[4] Chief Inspector of Factories, *A.R. Chief Inspector of Factories 1948*, 1949, H.M.S.O., London.
[5] Machle, W., and Gregorius, F., *Publ. Hlth Rep. (Wash.)*, 1948, **63**, 1114.
[6] Brinton, H. P., *et al.*, ibid., 1952, **67**, 835.
[7] Doll, R., *Brit. J. Industr. Med.*, 1952, **9**, 180.
[8] Hill, A. B., and Faning, E. L., ibid., 1948, **5**, 1.
[9] Perry, K., *et al.*, ibid., 1948, **5**, 6.
[10] Chief Inspector of Factories, *A.R. Chief Inspector of Factories 1947*, 1949, H.M.S.O., London.
[11] Gloyne, S. R., *Lancet*, 1951, **1**, 810.
[12] Doll, R., *Brit. J. Industr. Med.*, 1955, **12**, 81.
[13] Kennaway, E. L., and Kennaway, N. M., *Brit. J. Cancer*, 1947, **1**, 260.
[14] —— —— ibid., 1953, **7**, 10.
[15] James, W. R. L., *Brit. J. Industr. Med.*, 1955, **12**, 87.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25318/1

their mortality had been the same as that of the whole male population of England and Wales. Up to June, 1953, 39 of the men had died, whereas the expected number was 15.4. The excess was wholly accounted for by deaths from lung cancer (11 deaths, only 0.8 expected) and from other respiratory and cardiovascular diseases (22 deaths, only 7.6 expected). This high risk of contracting lung cancer became progressively less as the duration of employment under the old dusty conditions decreased.

The vulnerability of the lung to siliceous dusts, on the other hand, does not seem to make that organ vulnerable at the same time to cancer.[13] In coal-miners Sir Ernest and Lady Kennaway[14] found that the mortality from lung cancer varied between different coalfields and was low in South Wales, where pneumoconiosis is most prevalent. W. R. L. James[15] has recently made a detailed comparison of necropsy data in South Wales relating to 1,827 coal workers and 1,531 other males aged over 21 years. Primary lung cancer was found in 3.3% of the miners and in 5.4% of the non-miners. The histological types of cancer, and the distribution of metastases, were similar in the two groups. James suggests the possibility that a relatively early age of death for those affected by pneumoconiosis may explain the lower incidence of lung cancer among them at necropsy. There is certainly no evidence that coal dust has any carcinogenic influence.

SALMONELLA IN EGG PRODUCTS

During the past few years many outbreaks of paratyphoid fever have been traced, epidemiologically and bacteriologically, to bakeries, with confectionery containing imitation cream as the most frequently suspected source of infection. In an investigation into a widespread outbreak of paratyphoid fever in South Wales, S. Thomson[1] and A. R. Culley[2] suspected that a contaminated ingredient used in the bakeries was the means by which the imitation cream was affected, and in a paper published last week K. W. Newell[3] reports an investigation of six small outbreaks of paratyphoid fever due to *Salm. paratyphi* B, phage type 3a var. 2. These outbreaks all occurred during the last half of 1954: there were 38 clinical cases of the infection, and 14 symptomless excreters were found. In all these outbreaks imitation cream used to embellish cakes came under suspicion, and the source of contamination was thought to be a particular batch of Chinese frozen whole egg in use at all the bakeries concerned. Unfortunately very little of this batch was available for examination, and the organism was not found, though samples taken from what was left contained *Salm. thompson* and *Salm. aberdeen*. In another paper Miss Muriel E. Smith and Dr. Betty C. Hobbs[4] describe the bacteriological investigations which were complementary to the epidemiological inquiry. In later investigations—reported by Newell and his colleagues[5] at a symposium on "Food Microbiology and Public Health" held last July in Cambridge—*Salm. paratyphi* B, phage type Taunton, was found in a sample of Chinese whole egg.

Meanwhile interest had been aroused in the bacteriology of egg albumen, which is used in bakeries in its frozen or crystalline forms for the preparation not only of cooked foods but also of uncooked and lightly cooked products. An examination of Chinese crystalline albumen showed that salmonellae were present in from 15–75% of samples and the serotypes included a preponderance of *Salm. thompson*. *Salm. paratyphi* B was found in 11 samples and included the phage types Taunton, 3b, and a type designated 1 var. 5. *Salm. paratyphi* B was also found in frozen albumen. Methods for the isolation of these organisms from dried albumen are in common use in most laboratories. A 10 to 20% suspension of dried material in broth and also in selenite F[6] is incubated at 37° C. Subcultures are made on to desoxycholate citrate agar[7 8] and Wilson and Blair agar[9 10] after 24 hours and also after three days' incubation.

The presence in frozen whole egg or in crystalline egg albumen of *Salm. paratyphi* B and of the serotypes of salmonella responsible for some outbreaks of food poisoning in this country makes it desirable that some control should be exercised over these products, and their bacteriological quality should be improved either by altering methods of production or by treating the material on its arrival in this country. The *Medical Officer*, in a recent annotation[11] on dried egg albumen, describes a method of control which could be applied at the ports—that is, the examination of 5% samples from all batches received, and the holding of all consignments showing any positive samples. It is recommended that such positive consignments should be used for commercial purposes only, or held pending successful heat treatment to render the substances safe. Those batches found negative on two samplings could be released on the condition that they were used in heated products only. There would certainly be difficulties in putting this policy into practice, and it does not take into account the considerable quantities of these egg products already held in this country both by private importers and by the Ministry of Agriculture, Fisheries, and Food.

At present there are several outbreaks of paratyphoid fever in Southern England which appear to be connected with bakeries, and perhaps related to egg products. It is hoped that despite financial and international difficulties it will be possible for some co-ordinated policy to be formulated which will make safe these previously unsuspected sources of infection. In any event, if these outbreaks of paratyphoid fever are to be prevented, stricter bacteriological control will be necessary of foodstuffs susceptible to infection, both imported and home-produced.

[1] Thomson, S., *Monthly Bull. Minist. Hlth (Lond.)*, 1953, **12**, 187.
[2] Culley, A. R., *Med. Offr*, 1953, **89**, 243, 257.
[3] Newell, K. W., *Monthly Bull. Minist. Hlth (Lond.)*, 1955, **14**, 146.
[4] Smith, M. E., and Hobbs, B. C., *ibid.*, 1955, **14**, 154.
[5] Newell, K. W., Hobbs, B. C., and Smith, M. E., *J. appl. Bact.*, 1955 (in press).
[6] Leifson, E., *Amer. J. Hyg.*, 1936, **24**, 423.
[7] —— *J. Path. Bact.*, 1935, **40**, 581.
[8] Hynes, M., *ibid.*, 1942, **54**, 193.
[9] Wilson, W. J., and Blair, E. M. McV., *J. Hyg. (Camb.)*, 1931, **31**, 138.
[10] —— *ibid.*, 1938, **38**, 507.
[11] *Med. Offr*, 1955, **94**, 153.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25318/2