in both acute and long-stay wards; and my thanks to Mr. Tromans, records officer, and Miss King, for their long-suffering but willing co-operation in the compilation of this report.

ADDENDUM.—Since this report was completed, figures for the year August 1, 1950, to July 31, 1951, have become available. Total admissions to the two "acute" geriatric wards were 590 (290 men, 300 women) compared with 479 in the previous year. The most significant change was that 46% of the women were discharged against 39% in the first year.

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SECOND INTERNATIONAL CONGRESS OF INTERNAL MEDICINE

SCIENTIFIC SESSIONS

The scientific meetings of the Second International Congress of Internal Medicine, held in London from September 15 to 18 under the presidency of Sir Russell Brain, took place at Friends House, Euston Road. The sessions took the form of symposia.

I.—THE SPRUE SYNDROME

On Monday, September 15, Dr. NANNA SVARTZ (Stockholm) took the chair for the first of the scientific sessions, which was on the sprue syndrome.

The opening speaker, Professor C. D. de LANGEN (Utrecht), reviewed the theories of production of steatorrhoea. He thought that too much attention had been paid to the disturbances of fat metabolism. His own observations had shown that there was also deficient absorption of other bowel contents—namely, vitamins, metabolites, water, and gas. It was wrong to assume that meteorism arose from bacterial fermentation and excess gas formation. It seemed likely that there was inadequate absorption of gas from the small bowel. Faulty absorption might result from circulatory disturbances, from damage to the intestinal mucosa, or from decreased bowel tone. Research workers had overlooked the importance of circulatory changes in producing steatorrhoea, but a study of the relationship between gas absorption and vascularity of the bowel would throw new light on the process of absorption.

Absorption of material through the gut wall was a biphasic process of filtration and reabsorption similar to that of renal excretion: it was governed by the relative forces of the hydrostatic pressure in the capillaries and the osmotic pressure of the bowel contents. This mechanical theory of intestinal absorption not only supported the findings of Frazer and other workers but laid emphasis on deficient absorption, as opposed to impaired fat metabolism, as the basic abnormality in the sprue syndrome.

Biochemical Changes and Vitamin Deficiencies

Dr. A. L. FROEHLICH (Antwerp) described some changes in the blood and bile and the vitamin deficiencies resulting from idiopathic steatorrhoea. The few observations on sodium and potassium metabolism suggested that there was a fall in blood sodium and chlorides, the potassium content remaining unchanged. A fall in blood potassium had been noted in one patient when oedema disappeared. The serum calcium fell significantly; the phosphorus, though not so constantly reduced, was between 1.35 and 2.5 mg.% in four patients. The alkaline phosphatase was sometimes raised. Reduction of the serum proteins was caused by a decrease in albumin; the globulin was normal or slightly raised. The blood fats, cholesterol, and phosphates were usually lowered, but neutral fat was normal. Similar changes had been found in patients with malnutrition and with Addisonian anaemia. There was little increase in the blood sugar after ingestion of glucose, but hypoglycaemia sometimes occurred later. This might be caused by delayed absorption, although the late hypoglycaemia suggested that the glucose was deposited in the liver and failed to reach the peripheral blood. Alterations of the blood fat levels after a fatty meal were less definite. Most workers had found that increase in the blood fat was absent or delayed, and Frazer had not found any increase in the chylomicron count. The serum cholesterol was either unaltered or diminished, and the increase in blood phosphates was less than normal. In Dr. Froehlich's experiments there had been no increase in the blood fat after a fatty meal, but hyperphosphatidaemia and hypercholesterolaemia had occurred as in normal subjects. In three patients otherwise showing the typical picture of steatorrhoea bile acids and choline were unaccountably absent from the duodenal fluid. This might have resulted from liver failure, and it was possible that such cases constituted a separate aetiological group. Dr. Froehlich had been unable to confirm the hypothesis that in steatorrhoea there was deficient synthesis of acetylcholine in the intestinal wall.

Vitamin-B deficiency was commoner in tropical sprue than in idiopathic steatorrhoea, but in both disorders hyperchromic anaemia was often found. The anaemia often responded to normal doses of vitamin B_{12} or to folic acid, but in some cases the response was poor. The commonest deficiency was that of the fat-soluble vitamins. The blood levels of vitamin A were usually low, and, although hypoprothrombinaemia was often encountered, it seldom gave rise to bleeding. In one patient a pigment similar to that found in experimental vitamin-E deficiency had been demonstrated. Commonest of all was deficiency of vitamin D, leading to osteoporosis and tetany, with associated low serum calcium and diminution of calcium excretion in the urine.

Aetiology of the Syndrome

Professor A. C. FRAZER (Birmingham) sought to elucidate the many problems in the aetiology of idiopathic steatorrhoea and similar disorders. He considered that it was first necessary to determine the site of the fundamental defect. Although it had been postulated that liver failure and adrenal insufficiency were responsible for this group of diseases it seemed probable that changes in hepatic function were secondary, and that adrenal failure did not play a leading part. It had now been fully established that pancreatic enzymes were produced normally in steatorrhoea. There was, however, ample evidence to show that the small intestine was at fault. Fat-balance tests and the absence of an increase in blood fat after a fatty meal clearly pointed to deficient absorption; but defective absorption was by no means confined to fat, since it was readily demonstrable that glucose and urea, when given by duodenal intubation, were similarly affected. Insufficient absorption arose from mechanical rather than chemical causes. Radiographical studies shed considerable light on the causation of steatorrhoea. The "clumping" pattern so characteristic of steatorrhoea could be reproduced in normal subjects by giving substances such as fatty acids which stimulated mucus secretion in the small gut. The effects of these mucogenic stimuli had been reproduced in the laboratory and in isolated intestinal loops. If a non-flocculable barium sulphate suspension was used instead of a simple barium sulphate suspension in patients with steatorrhoea, "clumping" did not occur, but it was nevertheless evident that the small gut was both dilated and immobile. A third approach to the problem was through the knowledge that children with coeliac disease improved when wheat flour was withheld

from their diet. It had been shown that wheat gluten rather than wheat starch was the important factor. Wheat gluten did not exert its adverse influence through an allergic response, since other manifestations of allergy were absent. Professor Frazer concluded by postulating that the sprue syndrome was initiated by certain alimentary factors, such as infection, infestation, or the ingestion of rancid fats, which gave rise to increased mucus secretion and increased bacterial growth in the small bowel. The ensuing delay in absorption and decreased motility encouraged further bacterial changes and created a vicious circle that could best be broken by dietary restriction or control of intestinal flora.

Corticosteroid Therapy

Dr. CHESTER M. JONES (Boston) outlined current views on the sprue syndrome and described the response of this disease to corticosteroid therapy. He preferred to use the term "sprue syndrome" because of its wider implications. Although tropical sprue responded to replacement therapy with liver, folic acid, and vitamin B_{12}, response in non-tropical sprue, when there was an abnormality in the small intestine, was less constant. Nine patients with non-tropical sprue, having failed to improve under standard treatment, had been treated with cortisone or A.C.T.H. Treatment had not been given in the belief that their illness was due to failure of the adrenal cortex, but in the hope that corticosteroids might modify and improve intestinal absorption. All patients had shown striking clinical improvement. After three or four days' treatment abdominal distension had disappeared; within a week or ten days the stools had become less frequent and semi-formed, and the faecal fat had diminished. One patient had initially failed to improve because inadequate dosage had been given. Coincidental with clinical improvement there had been gain in weight, increase in the serum proteins, and correction of anaemia. Objective laboratory tests, such as the glucose and vitamin-A tolerance curves and chylomicron counts, had reverted to normal. Dr. Jones believed that corticosteroids actually stimulated the absorption processes which were deficient in the sprue syndrome.

From the enthusiastic discussion which followed the opening papers it was evident that world-wide interest was being aroused by the many problems of steatorrhoea. Among those who discussed the subject were Dr. F. Bulic (Yugoslavia), Dr. H. A. Salveren (Norway), Dr. A. Vaz Serra (Portugal), Dr. W. T. Cooke (Birmingham), Professor C. Jiménez Diaz (Madrid), Dr. R. H. Girdwood (Edinburgh), Dr. D. Adlersberg (U.S.A.), Professor E. Martin (Switzerland), Dr. C. Holten (Denmark), and Dr. J. L. Castro (Spain). Dr. BULIC described an epidemic form of sprue which had occurred in the Yugoslav Army and which had ceased when fresh supplies of fresh food had become available, Dr. SALVEREN commented on the low blood pressure in patients with steatorrhoea, and Dr. GIRDWOOD discussed the absorption of various anti-megaloblastic factors from the bowel.

II.—FLUID AND ELECTROLYTE BALANCE

On Tuesday, September 16, the day was devoted to a symposium on the clinical importance of disturbances of fluid and electrolyte balance. In the morning the chair was taken by Dr. A. GIGON (Basle) and in the afternoon by Sir HAROLD HIMSWORTH (London).

Professor J. G. G. BORST (Amsterdam) opened the symposium by describing the part played by the kidney in governing fluid balance. He emphasized the importance of recognizing three types of diuresis. The first, water diuresis, took place after drinking water. Absorption of water led to a fall in the electrolyte concentration in the blood and so inhibited the production of antidiuretic hormone by the pituitary. As a result, the kidneys excreted large quantities of urine with a low electrolyte content. The second, saline diuresis, occurred when salt and water were ingested together, producing an increase in plasma volume, a rise in venous pressure, and (in accordance with Starling's law) increased cardiac output. The kidneys responded by passing excess urine rich in salt and with a slight increase in potassium concentration. This type of diuresis was found in many pathological states, including heart failure, where it had been found that, if venous pressure increased to such an extent that cardiac output, instead of continuing to rise, began to fall, there was a corresponding diminution in salt excretion. Such patients were "over the top of the curve" of both cardiac output and salt excretion. The third type of diuresis was the rhythmic diurnal variation found in normal individuals, who passed more urine by day than by night. It was important to allow for this when measuring diuresis in pathological states. The third type of diuresis normally dominated the other two: it was abolished by cortisone therapy and in Cushing's syndrome.

Hormonal Influences

Dr. F. T. G. PRUNTY (London) discussed the effect of the adrenal hormones on fluid and electrolyte metabolism. He thought it better to study water and electrolytes separately. Patients with Addison's disease maintained in normal water balance with desoxycorticosterone (D.C.A.) did not show normal diuresis after drinking water, and excreted excessive urine at night. This was the basis of the Kepler test. The use of D.C.A. in Addison's disease corrected the loss of sodium and chloride in the urine, and ultimately water loss as well. There was probably an increased reabsorption of sodium and chloride by the distal urinary tubules; it was uncertain whether there was a direct effect on tubular absorption of water. The pituitary's tendency to correct water loss was prevented by the serum hypotonicity in Addison's disease. Cortisone could restore normal water diuresis in these patients by directly altering tubular reabsorption.

The effects of A.C.T.H. on sodium and chloride excretion were well known, but the reasons for the extreme variability in sodium retention were not. Excess sodium excretion sometimes occurred during A.C.T.H. treatment, but more often after withdrawal of the hormone. This effect was not due to adrenal cortical insufficiency nor to contamination of A.C.T.H. with posterior pituitary. The conflicting results from giving D.C.A. and cortisone to patients with Addison's disease suggested that there might exist an adrenal hormone concerned directly with electrolyte metabolism. In A.C.T.H. therapy oedema could arise without obvious fluid retention, suggesting there was transference of fluid from the cells to the extracellular fluid. Further studies on these problems were in progress.

Renal Failure

Professor ROBERT PLATT (Manchester) spoke of the control of water and electrolytes in renal failure. The primary cause of renal failure was a diminution in the number of functioning nephrons. Animals surgically deprived of most of their kidney substance had excretory disturbances like patients with renal failure. Even in advanced renal failure the daily excretion of urea, creatinine, and electrolytes remained normal. Water excretion and fluid intake were increased. Both in patients and experimental animals there was glomerular and tubular enlargement: it had been estimated that the glomeruli could filter at one and a half times their normal rate. It was clear that, if water and electrolyte excretion was maintained after the glomerular filtration rate had been reduced to one-fifth, each tubule must excrete five times as much fluid as before. This was largely due to decreased reabsorption. The high rate of excretion could be maintained only by a state of permanent osmotic diuresis where the solute load determined the urinary volume and concentration. Many of the features of water diuresis in the failing kidney were more easily explained if it was assumed that the tubules could excrete a hypotonic fluid.

Other evidence for tubular excretion was the knowledge that certain substances were secreted by the tubules in health and disease, and that hypotonic urine was secreted by the tubules of certain aglomerular fish. From the clinical viewpoint it was important to remember that patients with renal failure needed plenty of water and salt: protein restriction reduced the blood urea and so lessened osmotic diuresis.

Diabetic Coma

Dr. RENÉ S. MACH (Geneva) outlined the changes in fluid and electrolyte balance in diabetic coma. During diabetic acidosis patients became severely ill from excessive loss of electrolyte and fluid from the kidney. Estimates of the magnitude of these losses had been made in diabetic patients who had ceased to take insulin and during the treatment of diabetic coma. Sodium was lost partly as sodium chloride and partly in combination with ketones. The sodium was derived mainly from extracellular fluid. It was not known if any diminution of intracellular sodium took place. Diabetic patients lost potassium derived entirely from the cells, and retained potassium when insulin was given. Water was lost not only in the urine but also from hyperventilation and vomiting. If the patients were given water but insufficient salt, hypochloraemia resulted; the tonicity of extracellular fluid depended on the amounts of salt and water lost by the kidneys compared with the amount ingested. Loss of cellular potassium began immediately acidosis occurred and progressed slowly. The plasma potassium level depended on many factors, of which the acid-base balance, insulin and glucose therapy, and saline administration were of practical importance. The losses of salt and water were produced in several ways. Hyperglycaemia produced osmotic diuresis, and water loss was accentuated by anorexia, vomiting, and hyperventilation. Acidosis caused sodium and potassium loss because inorganic base was removed with ketones, and because acidosis favoured cation excretion.

Malnutrition

Professor R. A. MCCANCE (Cambridge) described the conclusions which he and Dr. E. M. WIDDOWSON (Cambridge) had drawn from their investigations on the body changes in malnutrition. Recent technical advances had made it possible to study in detail the changes in body fluids and in the various organs. The hallmark of malnutrition was not a fall in body weight or even diminution of body fat. The really important change was a big increase in the extracellular fluid together with a fall in the total cell mass. The increase in extracellular fluid was brought about largely because fluid replaced the body space which had previously been occupied by fat and cells; additional factors were a fall in the serum proteins and a high salt intake. There was no proof that the changes were produced by renal or endocrine disorders or by vitamin deficiency. Visible oedema was variable and bore no relation to the vast increase in extracellular fluid. The liver wasted in starvation, and it had been possible to show that there was a decrease in individual cell mass due to loss of water and protein. In conclusion, starvation produced no gross changes in electrolyte balance or in cellular structure—a happy coincidence which probably explained the survival of the human race through periods of famine.

Cellular Hyperhydration and Other Topics

Professor J. HAMBURGER (Paris) offered some stimulating thoughts on cellular hyperhydration. Although the clinical signs arising from disturbances of the extracellular fluid were recognized, disorders of cellular fluid were not so well defined. Cellular hyperhydration could be produced either by reducing the osmotic pressure of the extracellular fluid (by infusion of water or removal of salt by dialysis) or by chemical agents such as potassium cyanide or histamine.

Cellular hyperhydration produced gastro-intestinal symptoms, asthenia, headaches, cramps, and nervous symptoms. There were changes in the electroencephalogram, oliguria, and nitrogen retention. The clinical diagnosis could be confirmed by laboratory tests. Cellular hyperhydration could be caused by loss of salt from the body, Addison's disease, shock, or renal diseases with oliguria. Cellular hyperhydration could occur at the same time as extracellular dehydration or generalized hyperhydration with oedema. It was possible that some of the symptoms previously attributed to extracellular dehydration might, in fact, arise from cellular hyperhydration. Care was needed in selecting correct treatment, since the imprudent infusion of hypotonic solutions would aggravate the condition.

Dr. O. J. BROCH (Oslo) spoke on the significance of sodium loss and the role of the serum proteins in regulating osmotic pressure. Some patients suffered loss of total bases from the serum whilst the chloride and bicarbonate remained normal, an apparent acid surplus resulting. Possibly there was a corresponding reduction of the base equivalent of the serum proteins, which might sometimes behave as bases. Serum proteins could therefore vary their base binding capacity without quantitative alteration. Large fluctuations of sodium occurred in the extracellular fluid, but unless there was also an upset of water equilibrium they gave rise to no ill effects.

Effect of Surgery

Mr. A. W. WILKINSON (Edinburgh) described the disturbances in body fluids in surgical patients. The metabolic changes which followed surgical operations reflected local disturbances at the site of injury. They were part of the body's response to injury and could not be modified easily. Unless there was rapid loss of body fluids, infusions of fluid were not required. Starvation and loss of gastric juice in pyloric stenosis presented difficult problems. Duodenal, biliary, and pancreatic fistulae led to loss of intestinal secretions and were a constant drain on the body fluid and electrolytes. All efforts should aim at minimizing the discharges, and if necessary they should be replaced through a jejunostomy. Drowsiness and apathy were the salient features of potassium deficiency; the cardiographic changes did not correspond closely to either the clinical state or the level of serum potassium. Alteration of the acid-base balance of blood was of only secondary importance to changes in sodium and potassium metabolism.

In the discussion which followed the opening papers Dr. D. A. K. BLACK (Manchester) described the changes in pure potassium deficiency induced by taking exchange resins together with extra sodium. Potassium deficiency was associated with sodium retention and increase in the volume of extracellular fluid. Potassium deficiency could be partly compensated by alkalosis, but when it was accompanied by acidosis, as in diabetic coma, symptoms were severe. Here, treatment of the acidosis by lactate might spare the necessity of giving intravenous potassium. The following members also spoke: Dr. G. Sala (Italy), Dr. E. Coelho (Lisbon). Dr. E. Azerad (France), Dr. L. Wislicki (Israel), Dr. A. Svanborg (Stockholm), Dr. M. Soriano Jimenez (Madrid), Dr. A. Cajdos (France), and Professor Ask-Upmark (Sweden).

III.—NEUROTROPIC VIRUS DISEASE

On Wednesday morning, September 17, the subject of the symposium was "Some aspects of neurotropic virus disease." Sir RUSSELL BRAIN was in the chair.

Dr. J. R. PAUL (Yale) opened the discussion with a review of poliomyelitis and the virus encephalitides. He emphasized that countries with a high standard of sanitation had experienced high rates of poliomyelitis during the last generation, while in countries with more primitive sanitation poliomyelitis had remained a largely endemic disease occurring particularly in infants, as it was in the nineteenth century. He thought it probable that in these more primitive countries the children gained immunity which protected them against the disease when they became older, while this was not happening in the countries with a higher standard of sanitation; here the disease was tending to occur in epidemic

form and often in adults. Although the virus had been found in sewage, flies, and food, the exact importance of these extrahuman agents in the spread of the disease was still uncertain: they were certainly not essential links in the chain. The most important source of transmission seemed to be man, during the early or late stages of clinical or subclinical infection.

Attempts to eliminate the virus in a community by quarantine measures were unlikely to succeed. And the fact that there were at least three strains of poliomyelitis virus made active immunization methods very difficult. However, attempts were being made to produce a concentrated immune serum for inducing passive immunity during an epidemic.

In discussing other forms of virus encephalitis, Dr. Paul emphasized the importance and frequency of mumps as a cause of meningo-encephalitis, even in the absence of parotitis. Antibody titres could be helpful in the diagnosis of this condition. In St. Louis encephalitis, Japanese B encephalitis, and eastern and western equine encephalomyelitides the virus was transmitted by arthropods, and these diseases tended to occur, as does poliomyelitis, in the summer. In many cases there appeared to be a viraemia without evidence of neurological involvement, and he thought that the occasionally severe and spectacular infections in man were only accidents in the general epidemiological picture.

Coxsackie Virus and Poliomyelitis

Dr. HERDIS VON MAGNUS (Copenhagen) discussed the Coxsackie group of viruses. This virus was originally isolated in 1947 in the faeces of two cases of paralytic poliomyelitis and since then had been recovered from patients suffering from various clinical pictures. It had been clearly established that strains of the virus had been the cause of cases of Bornholm disease and herpangina, but its significance in other diseases was still uncertain. In particular the relation to poliomyelitis was difficult to be sure about, as both Coxsackie virus and poliomyelitis virus had at times been isolated from the same case. At the moment a virus laboratory could help the clinician only in selected cases, as isolation of the virus and serological examination of paired sera were the only reliable methods, and about 15 different serological types of this virus were known to exist.

Dr. W. RITCHIE RUSSELL (Oxford) discussed some outstanding clinical problems of poliomyelitis. There was still much to learn about the clinical aspects of the disease, and segregation of cases in poliomyelitis centres would assist scientific study. Dr. Ritchie Russell went on to discuss treatment. In patients with pharyngeal palsy, postural drainage was particularly important. He also spoke of the difficulty of deciding what was the best type of respirator to use. There was also division of opinion on the correct treatment of paretic muscles in the early stages of the disease.

Following the major papers, short contributions were made by other speakers, including Dr. O. THORDARSON (Reykjavik), who described an epidemic of myalgia in Iceland thought to be due to a strain of Coxsackie virus. Professor C. H. STUART-HARRIS (Sheffield) described a small epidemic of herpangina in which a Coxsackie virus had been obtained from the faeces of one of the patients.

IV.—ANTIBIOTICS

In the afternoon of Wednesday, September 17, with Sir LIONEL WHITBY (Cambridge) in the chair, Sir ALEXANDER FLEMING (London) reviewed the development of antibiotics in medicine. It was striking how penicillin had converted many serious diseases into trivial complaints. Only a few of the antibiotics found by research had come into use, since many were toxic and others ineffective. In clinical use it was necessary to select that antibiotic which was effective against the particular infecting microbe and also to ensure that it reached the microbe (and not only the blood stream) in therapeutic concentration. Combinations of antibiotics had to be chosen with care. Jawetz had divided antibiotics into two groups: group I including penicillin, streptomycin, and bacitracin, and group II chloramphenicol, aureomycin, and terramycin. It was safe and often helpful to combine drugs in group I, but there was no advantage in mixing drugs in group II. The greatest care was needed in combining a drug from group I with one from group II, since if the organism was sensitive to the group I drug there would be antagonism, although if it was insensitive there would be synergism.

Professor F. MAGRASSI (Naples) described the dosage and complications of antibiotic treatment. There had been some change in the behaviour of antibiotics since the early days when their clinical effect had paralleled the laboratory findings; nowadays this was not always true. A number of biological factors were probably concerned with the action of antibiotics in the body. The direct toxic effects of antibiotics varied enormously; penicillin was quite safe, whereas streptomycin, and to a less extent aureomycin and chloramphenicol, could be dangerous. Complications could also arise from allergy and from rapid destruction of bacteria. The extent to which antibiotics interfered with the development of natural immunity was uncertain. Secondary effects of antibiotic therapy arose from the development of resistant organisms and from suppression of normal intestinal bacteria which were useful to the body.

Behaviour in the Body

Professor A. KEKWICK (London) outlined the behaviour in the human body of the five commonly used antibiotics. After absorption streptomycin, aureomycin, and terramycin were rapidly distributed throughout the extracellular fluid, but penicillin and chloramphenicol were more widely distributed and were poorly concentrated in certain tissues. Behaviour of antibiotics at the blood–brain barrier varied; penicillin and terramycin concentrated poorly in the cerebrospinal fluid, but chloramphenicol diffused rapidly. The concentration of antibiotics in tissues and body fluids was described in detail. All antibiotics were excreted by the kidneys, but, whereas penicillin and terramycin were predominantly disposed of in this way, chloramphenicol and aureomycin were to a large extent broken down by the body. Absorption of penicillin from the intestine was very irregular and there was practically no absorption from the skin. It was possible to forecast the behaviour of the common antibiotics after administration in their usual doses.

Professor C. JIMÉNEZ DIAZ (Madrid) deprecated the indiscriminate use of antibiotics because of the harmful results which might accrue and described the uses of antibiotics in certain diseases. He emphasized the increasing number of strains of resistant organisms and the occurrence of "crossed resistance" between aureomycin and terramycin or chloramphenicol. Antibiotics might have useful therapeutic effects apart from their use in infections. Aureomycin could prevent the development of some experimental anaemias, and had some effect on hepatic cells, since it had benefited patients with hepatitis and cirrhosis. Aureomycin was also of value in post-irradiation sickness.

Much stimulating discussion followed the opening papers, but space does not permit it being described in detail. Dr. NANNA SVARTZ (Stockholm) described electron microscopic studies on the effects of antibiotics on bacteria. Professor C. D. DE LANGEN (Utrecht) considered possible means of reducing the barrier between the blood stream and the infection processes. Dr. B. OLHAGEN (Sweden) drew attention to the appearance of acid-fast organisms other than tubercle bacilli in the sputum of patients treated with antibiotics. Other speakers were Dr. K. G. Bengtsson (Sweden), Dr. E. Nyman (Sweden), Dr. J. Andreu-Urra (Spain), Dr. F. van Goidsenhoven (Belgium), Dr. V. Gorlitzer von Mundy (Austria), Dr. Najib Farah (Egypt), Dr. F. di Raimondo (Spain), Dr. L. V. Schneider (U.S.A.), Dr. M. Janbon (France), and Dr. F. Musotto (Italy).