Council was dissatisfied with these limited measures and in January 1965 it set up a small working party[5] to assemble all the facts about the medical examination of immigrants. This working party[6] under its chairman Dr. C. Metcalfe Brown, M.O.H. of the City of Manchester and M.O. to the Manchester Port Health Authority, made an interim report in July.[7] Its final report was adopted by the Council last week, and copies have now been sent to the Minister of Health and the Home Secretary.

At page 1423 of this issue of the *B.M.J.* we summarize the working party's main conclusions and recommendations. The working party stresses that *all* persons admitted to this country, other than short-stay visitors, should be medically examined before admission. There should be no differentiation in this respect, it thinks, between Commonwealth and alien immigrants. The alternatives of dispensing altogether with medical examination and the present system of examining only some immigrants are both rejected, the first because of "obvious health risks to the indigenous population." Examination should take place in the immigrant's country of origin, states the report. The practical difficulties of examining large and variable numbers of immigrants at port of entry greatly outweigh any theoretical advantages. Examination in his own country has advantages for the immigrant as well as for the receiving country. If disease is detected it can be treated, and apart from the immediate benefit to health this may result in a would-be immigrant becoming eligible for admission. If on the other hand the disease is such as to preclude admission despite treatment, he will have been spared from disrupting his life. The main argument against examination before embarkation is the possibility of widespread evasion or abuse. To minimize these hazards the working party recommends the use, on a part-time basis, of local doctors approved for the purpose by the British Government, backed up by a certain number of full-time Government medical officers from this country.

Finally there is the question of what the medical examination should cover. The working party has identified certain diseases whose presence in an immigrant it regards as a risk to the health of the inhabitants of this country. It recommends that immigrants with these conditions should not be permitted to enter "until they have a clean bill of health." The diseases in question are: tuberculosis, venereal disease, yaws, trachoma, keratoconjunctivitis, leprosy, smallpox, cholera, typhus, plague, yellow fever, typhoid and paratyphoid fevers, dysentery, and parasitic infections of the gastro-intestinal tract. For social and economic reasons, mental disorders, epilepsy, malignant diseases, drug addiction, and alcoholism are listed as additional grounds for exclusion. In order to establish an immigrant's freedom from these conditions the working party recommends a full general medical examination for all, "with special attention to the eyes and skin"; *x*-ray examination of the chest for those over the age of 12 years; and stool examination if the immigrant comes from a country where parasitic infections of the gastro-intestinal tract are common. But pre-entry examination is not considered enough. The working party emphasizes the importance of post-entry examination and follow-up of selected groups of immigrants. "We feel that the low natural immunity to disease of many immigrants, and the social conditions under which they live, make it imperative that a programme of post-entry investigation should be carried out in the interests of the immigrant himself." The working party therefore commends the steps which the Ministry has already initiated[8] to follow up those particularly at risk to tuberculosis, and hopes this policy will be pursued vigorously.

D. K. Stevenson's finding that the annual incidence of tuberculosis in Pakistanis in Bradford[9] was 30 times the rate for the indigenous population, and V. H. Springett's similar figure for Pakistanis and Sinhalese in Birmingham,[10] underline the seriousness of the situation. The crowded conditions in which many such immigrants live when first they reach this country provide ideal circumstances for further spread of tuberculosis. There is also a hazard from intestinal parasitic infections, such as amoebiasis and trichuriasis, a risk which is enhanced by the fact that many immigrants seem to gravitate to the catering trade. Hookworm infection may also prove a public health problem as S. N. Salem and S. C. Truelove have suggested.[11]

Few medical men would now challenge the need for medical screening of immigrants, whether Commonwealth or alien, before their entry to this country. Such examinations are not some elaborate form of colour bar, as has sometimes been implied, but a plain public health measure, long overdue. Apart from its first purpose in protecting the health of the citizens of these islands, proper medical examination is in the interests of the immigrant himself. From the economic aspect, too, examination before admission is highly desirable so as to prevent what could otherwise well prove a heavy burden on the hospital and other resources of this country. Now that Dr. Metcalfe Brown's working party has so clearly mapped the way, the Government should lose no time in following it.

[1] *Brit. med. J.*, 1961, **2**, 1624.
[2] *Brit. med. J. Suppl.*, 1961, **2**, 254.
[3] Commonwealth Immigrants Act, 1962, Clause 2, subsection (4).
[4] *Brit. med. J.*, 1964, **2**, 1547.
[5] *Brit. med. J. Suppl.*, 1965, **1**, 17.
[6] Dr. C. Metcalfe Brown, Dr. R. J. Dodds, Dr. H. R. C. Hay, and Dr. D. K. Stevenson.
[7] *Brit. med. J. Suppl.*, 1965, **2**, 21.
[8] Advisory letter to Medical Officers of Health on "Medical Arrangements for Long-term Immigrants," dated 22 April, 1965.
[9] Stevenson, D. K., *Brit. med. J.*, 1962, **1**, 1382.
[10] Springett, V. H., *Lancet*, 1964, **1**, 1091.
[11] Salem, S. N., and Truelove, S. C., *Brit. med. J.*, 1964, **1**, 1074.

New Rubella Syndrome

The most severe rubella epidemic for more than 20 years swept the United States during the early months of 1964. The outbreak affected some 1,800,000 people, and inevitably many women in early pregnancy were infected. The infants born to these mothers were extensively studied both clinically and with the aid of the recently developed virological techniques. It was soon apparent that many of the infants born with congenital defects were also suffering from additional disorders which had not previously been identified as part of the congenital rubella syndrome.[1]

This new syndrome has been named the "expanded rubella syndrome" or "acute congenital rubella," and appears to be due to widespread systemic infection with the virus. The most striking feature of the expanded rubella syndrome was thrombocytopenia, and many of the affected infants were born with a petechial or purpuric rash.[2–4] Though thrombocytopenia is known to be a complication of adult rubella,[5 6] it has only occasionally been noted in infants with the classical congenital rubella syndrome.[7] Infants with the expanded rubella syndrome—like those with classical congenital rubella—had a high incidence of cardiac lesions and defects of the

eye such as cataract, glaucoma, and microphthalmia. Most infants with the expanded syndrome, however, also had hepatosplenomegaly and a bulging anterior fontanelle and were of low birth weight.

Another feature of the syndrome was the presence of characteristic lesions in the metaphysis of the long bones.[8] These lesions appeared on *x*-ray films as abnormal areas of translucency. Similar bone changes were observed in half the infants born in the Philadelphia General Hospital during the epidemic period and are described by Dr. J. M. Lindquist and his colleagues at page 1401 of this week's *B.M.J.* Dr. Lindquist and his colleagues found osteoclasts in the tibial lesions of one child and postulate that the lesions are due to viral osteitis. A. J. Rudolph and his co-workers, on the other hand, think that the changes are due to a defect in the formation of bone which is secondary to metabolic or nutritional disturbances.[8] Similar radiological changes in the long bones are occasionally seen in newborn children suffering from other diseases, but they have not previously been reported in congenital rubella. Fortunately, the bone lesions —like the thrombocytopenia—generally resolve in the early months of life. The infants studied in Philadelphia showed a lower incidence of thrombocytopenia than those reported in other studies. Furthermore, none of them showed myocardial necrosis, jaundice, hepatitis, interstitial pneumonia, or anaemia—though these disorders have been noted in a few infants with the expanded rubella syndrome by other workers.[4 9]

Despite the severity of the congenital rubella which followed this outbreak the total incidence of congenital defect after maternal rubella in the first trimester was 10%,[10] which is no higher than that observed in previous surveys.[11 12] This suggests that although the particular strain of virus associated with the outbreak caused unusually severe disease it did not infect the foetus more often.

Epidemiological surveys have established that foetal infection may follow inapparent subclinical maternal rubella.[13 14] It may therefore be difficult to diagnose the congenital rubella syndrome if there is no history of maternal rubella or if—as sometimes happens—there is also no known exposure to infection during pregnancy. If a newborn infant is suspected to be suffering from congenital rubella, confirmation should be sought by taking throat swabs or other specimens for attempted isolation of the virus. Infants with the expanded rubella syndrome—like those with classical congenital rubella —suffer from a chronic infection with rubella virus, and the virus can readily be isolated from throat, urine, or rectum during the first months of life.[2-4] The affected infants are infectious, and cases of rubella have been observed among their attendants and family contacts.[3 15 16] Studies have also been reported that show that immunity to reinfection with rubella virus is associated with the presence of neutralizing antibody.[16 17] Volunteers who had been inoculated with the virus and persons exposed to infection from cases of the disease almost invariably failed to develop infection if they possessed detectable antibody. In contrast, those who lacked antibody suffered a high attack rate of clinical or subclinical rubella. Since the presence of neutralizing antibody is associated with almost certain immunity to rubella there is clearly hope of successful artificial immunization. The effects of a live virus vaccine have already been investigated in a small group of children.[18] The vaccine caused a mild illness with the production of antibody when it was inoculated subcutaneously, but there was no response if it was administered intranasally. Virus was present in the throat of the children inoculated subcutaneously and persisted up to three weeks after inoculation. Unfortunately, these children proved to be infectious and there were some cases of rubella among their contacts. This type of vaccine is probably, therefore, unsuitable for general use, but virological developments in the study of rubella are progressing rapidly and a better vaccine may yet be possible.

[1] Rudolph, A. J., Yow, M. D., Phillips, C. A., Desmond, M. M., Blattner, R. J., and Melnick, J. L., *J. Amer. med. Ass.*, 1965, **191**, 843.
[2] Phillips, C. A., Melnick, J. L., Yow, M. D., Bayatpour, M., and Burkhardt, M., ibid., 1965, **193**, 1027.
[3] Horstmann, D. M., Banatvala, J. E., Riordan, J. T., Payne, M. C., Whittemore, R., Opton, E. M., and Du Ve Florey, C., *Amer. J. Dis. Child.*, 1965, **110**, 408.
[4] Cooper, L. Z., Green, R. H., Krugman, S., Giles, J. P., and Mirick, G. S., ibid., 1965, **110**, 416.
[5] Ackroyd, J. F., *Quart. J. Med.*, 1949, **18**, 299.
[6] Wallace, S. J., *Lancet*, 1963, **1**, 139.
[7] Brown, C. M., and Nathan, B. J., ibid., 1954, **1**, 975.
[8] Rudolph, A. J., Singleton, E. B., Rosenberg, H. S., Singer, D. B., and Phillips, C. A., *Amer. J. Dis. Child.*, 1965, **110**, 428.
[9] Korones, S. B., Ainger, L. E., Monif, G. R. G., Roane, J., Sever, J. L., and Fuste, F., ibid., 1965, **110**, 434.
[10] Sever, J. L., Nelson, K. B., and Gilkeson, M. R., ibid., 1965, **110**, 395.
[11] Manson, M. M., Logan, W. P. D., and Loy, R. M., *Rep. publ. Hlth med. Sub.*, No. 101, 1960. H.M.S.O., London.
[12] Lundström, R., *Acta Paediat. (Uppsala)*, 1962, **51**, Suppl. No. 133.
[13] Schiff, G. M., Sutherland, J. M., Light, I. J., and Bloom, J. E., *Amer. J. Dis. Child.*, 1965, **110**, 441.
[14] Avery, G. B., Monif, G. G. R., Sever, J. L., and Leikin, S. L., ibid., 1965, **110**, 444.
[15] Schiff, G. M., and Dine, M. S., ibid., 1965, **110**, 447.
[16] —— Smith, H. D., Dignan, P. St. J., and Sever, J. L., ibid., 1965, **110**, 366.
[17] Green, R. H., Balsamo, M. R., Giles, J. P., Krugman, S., and Mirick, G. S., ibid., 1965, **110**, 348.
[18] Plotkin, S. A., Cornfeld, D., and Ingalls, T. H., ibid., 1965, **110**, 381.

Potassium Chloride and Bowel Ulceration

Last year ulceration of the small intestine from treatment with thiazide diuretics combined with potassium chloride was discussed in these columns.[1] Since then the number of reported cases has grown to well over fifty, and this week at page 1409 Mr. W. B. Ashby, Mr. John Humphreys, and Mr. Sinclair J. Smith describe six more.

The clinical and pathological features are rather characteristic. A few patients taking the combined preparation complain of vague abdominal discomfort, sometimes accompanied by nausea, diarrhoea, or anorexia. This may be attributed to indigestion or to the drug itself, and if the tablets are stopped at this stage symptoms frequently disappear. If they are not, obstruction of the small bowel may develop after a period of weeks or months. In some cases gastro-intestinal haemorrhage[2] or perforation have been the first indications that something was wrong. At operation one or more annular ulcers are found to be narrowing the lumen of the jejunum or ileum, and resection is usually required. Microscopical examination shows superficial ulceration, with considerable submucosal inflammation and fibrosis, but the mural blood-vessels, at least in man, appear to be unaffected.

Although many of the patients described were suffering from chronic degenerative diseases for which therapy with

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[3] Lindholmer, B., and Räf, L., *Acta chir. scand.*, 1965, **129**, 434.
[4] Lister, R. E., *Lancet*, 1965, **2**, 794.
[5] Kiellbo, H., Stakeberg, H., and Mellgren, J., ibid., 1965, **1**, 1034.
[6] Björnberg, A., and Gisslén, H., ibid., 1965, **2**, 982.