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THE TOWN CHILD: A CRITICAL SURVEY

The lessons of the great social experiment of evacuation will take a long time to learn. They will be reflected, we hope, in future housing policy and especially in a new outlook on education. Evacuation threw a grim light on some aspects of town life never dreamed of by those whose acquaintance was only with the exterior aspect of the slums. No sooner had the children been bundled off to their country hosts at the beginning of the war than an outcry arose that many of them were dirty and verminous, deplorable in their habits, ill clad and ill shod, defiant and destructive. Allowance must be made for some exaggeration. Even had the children been little angels, they would not have been welcome guests to some of those on whom they were compulsorily billeted. But the complaints were by no means without substance, nor did they relate only to a very small proportion of the children. A group of professional women, acting upon a resolution from the National Federation of Women's Institutes, set themselves to find out the truth about these accusations, and *Our Towns: A Close Up*[1] is the horrifying result. The report is addressed to readers who have stout stomachs and an appetite for facts, however disagreeable the facts may be. It takes refuge in no polite euphemisms. It does not refer to scabies as "shelter rash" or to lousiness as "uncleanliness." Evacuation proved to be a window through which town life in its more squalid aspects was revealed. What it showed was poverty, but a poverty which was not so much lack of means as lack of personal discipline and social standard. One town clerk in a reception area states that as the children defiled on arrival "the heads of some of them could be seen crawling with vermin." In another area some of the children arrived in garments so verminous that these had to be immediately burned. In another, 23% of the evacuated children were found to have head lice, and, what is worse, the cleansers found many of them reinfested after every visit from relatives and friends. Dr. Kenneth Mellanby had previously given some idea of the extent of the infestation with head lice.[2] He had shown that in a sample of children from ten industrial cities over 50% of the girls between the ages of seven and eleven were infested. In the country infestation is regarded as a disgrace: in the towns it is treated as a lighter matter.

Other evidence which startled the people concerned out of their customary modesty related to insanitary habits among the children, testifying to a low standard of behaviour in the places from which they had come. Enuresis was found to be common at all ages up to fifteen years. Among the younger children this might be an infantile reversion due to a new sense of insecurity, but there were a substantial number of other cases of a more difficult type, without any detectable medical origin. A school medical officer in Surrey says that the problem of enuresis was much eased by the return to London of the worst cases of the slothful or untrained type; another in Essex attributes the condition to lack of early training. Faecal incontinence was also a great difficulty. "Some of the younger [children]," say the compilers of this volume, "were said to be wholly unaccustomed to use a closet and to have used the hearth or corners of the room for defaecation." This is a reflection not only upon the training of the children but upon the housing conditions. It is stated in *When We Build Again*, a publication of the Bournville Village Trust, that in 1935 Birmingham had 38,773 back-to-back houses, 51,794 houses without a separate water-closet, and 13,650 without a separate water supply. In Shoreditch there are said to be numerous three-story houses occupied as tenements by three families, sharing two closets, one at the top of the house and the other perhaps forty feet away in the yard outside.

All this and much more comes within the uncomfortable beams of this searchlight. The remedy lies partly in the provision of better and more sanitary housing, but that will not meet the case of children whose parents are neglectful and lazy and incompetent. The defects in behaviour reflect not only upon the parents but to some extent upon the teachers and upon the educational system, which instructs the children in remote geography but forgets entirely their own bedrooms and backyards. Schools could teach biology in such a way as to relate it to personal hygiene and behaviour. They could teach many other intimate and personal things which more fortunate children learn in their own homes. But, unhappily, the schools and their medical services do not get hold of the children until it is too late. It is not the Jesuit alone but the sanitary educationist who wants the child up to the age of seven, or even only up to the age of five. As things are, the five-year-old enters school suffering from complaints and faulty habits which the school doctors and nurses will spend the next nine years of his life in trying to eradicate. The best efforts of the school medical service are frustrated also by the reservoir of infestation and contagion in the pre-school child. The compilers of this report have been impressed by nothing so much as the need to multiply nursery schools in the poor quarters of towns. In such schools good eating and sleeping habits and cleanliness in body and behaviour could be inculcated, and the child learn discipline and self-respect. The wide extension of nursery schools will be costly, but less costly than dirt and disease and delinquency. It is the nursery school, if anything, which can break the insanitary entail and prevent the sins of the fathers—more often the sheer fecklessness of the mothers—from being visited upon the children. Prof. R. J. A. Berry, in a letter to the *Times* last month, pointed out that many of the unpleasant features revealed by the evacuation of children are precisely those characteristic of feeble-mindedness, and said: "If the parents themselves are feeble-minded then the sooner the child is removed, even if only for part of the time, to a nursery school, the better for the child and for those who sooner or later have to pay for its segregation. At the moment the educational system catches the defective a little too late."

[1] *Our Towns: A Close Up.* By the Hygiene Committee of the Women's Group on Public Welfare (National Council of Social Service). Oxford University Press. (Paper 5s.; cloth 8s. 6d.)

[2] *Med. Off.*, Feb. 1, 1941.

ACID PHOSPHATASE AND PROSTATIC CANCER

The results obtained by Huggins and his colleagues[1] in the treatment of prostatic carcinoma have aroused much interest in the physiology of this gland, and more particularly in its ability to secrete a special type of phosphatase. The group of ferments to which it belongs, the phosphatases, have the power of liberating inorganic phosphate from hexose phosphate and other organic phosphorus compounds. They exist in many tissues, and their production is a manifestation of cellular activity. Many types

[1] *Arch. Surg.*, 1941, **43**, 209.

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have been described, but two are of outstanding clinical interest: the alkaline, which comes chiefly from bone cells, and the acid, which is formed in the prostatic glandular epithelium.[2] Curiously enough, only in man and monkey does the production of acid phosphatase occur in significant amounts, and then only after the onset of puberty.[3] Administration of male hormone to immature male rhesus monkeys leads to a very great increase in the concentration of acid phosphatase in prostate tissue. The suggestion has been put forward that the function of prostatic phosphatase is to split up hexose phosphate and other phosphoric esters in the course of glycogenolysis, upon which human spermatozoa largely depend for the energy requirements of prolonged motility after ejaculation. Although it is unlikely that the enzyme has any function in the glandular epithelium of the prostate, it is probably an important factor in the formation of prostatic calculi. Normally the ferment is excreted in the prostatic fluid, with the result that very little is found in the blood stream. The small amount of acid phosphatase that can be detected in the plasma of a healthy subject is derived from liver, spleen, bone, and kidney.

The information was of academic interest until the discovery of Gutman and his co-workers[4] that tumour cells derived from prostatic epithelium secrete large amounts of acid phosphatase brought the matter into the clinical field. With invasion of blood and lymph vessels by tumour cells a channel is opened to the ferment, so that it can appear in high concentration in the plasma. The Gutmans immediately applied this observation to the differential diagnosis of metastases derived from prostatic carcinoma.[5] They showed that by the method in use not more than 3 units of acid phosphatase per 100 c.cm. appear in the serum of a normal subject. Occasionally a slight increase is detected in the plasma of patients with tumours and other diseases of bone, liver, and other organs not associated with the prostate. Sullivan, Gutman, and Gutman[3] found in a series of 130 patients with bone metastases arising from cancer of the prostate that 110 had acid phosphatase values exceeding 3 units and 95 over 5 units per 100 c.cm. of serum. It would appear, therefore, that a value below 3 excludes bone involvement from prostatic cancer, and that one over 5 almost certainly indicates this. In 3 cases of Paget's disease the acid phosphatase value exceeded 5, but this did not lead to any trouble in diagnosis, because in this condition the alkaline phosphatase is raised, which is not the case with prostatic metastases. In only two instances did any confusion arise: one was in a case of carcinoma of the urinary bladder invading the prostate, and the other in a case of bronchogenic carcinoma with metastases in bone. It should be stressed that an indubitably high acid phosphatase is not usually found unless the skeletal system is involved. The data at present available do not permit one to state precisely how far extracapsular involvement of prostatic carcinoma must extend before a significant increase in plasma acid phosphatase can be detected. Huggins and Hodges,[6] however, showed that injection of testosterone is rapidly followed by an increase of acid phosphatase if there are any metastases in the bones. Accordingly this "provocative" injection of androgen may serve as an excellent diagnostic test of bone involvement when *x*-ray evidence is absent. The same workers went a stage further, and showed that removal of the male hormone by castration[1] or injection of oestrogen[7] led to remarkable clinical improvement, associated with a fall in the acid phosphatase value, which began within 24 hours.

Therapeutic deductions are obvious, but what is of interest here is the opportunity afforded for an objective test to indicate the response to operation. In an excellent review of the whole subject Sullivan and his colleagues[3] have summarized the clinical applications of acid phosphatase estimation. It provides independent evidence of the presence of metastases from carcinoma of the prostate, and it may be helpful in detecting the site of the primary tumour when the prostate is not unduly enlarged. It is of value also in determining when enucleation of the prostate is likely to be of no avail, because any increase in acid phosphatase means that the tumour cells have already reached the skeleton. After removal of a prostate which is afterwards found on histological examination to present signs of malignancy, repeated estimations of plasma acid phosphatase will detect any spread to the skeleton. Finally, if castration or other form of androgen control is carried out, the level of acid phosphatase in the blood serves as a useful means for early assessment of the treatment. The story is not yet finished, because there are hopes of further advances, direct and indirect. Even at this stage, however, there is a strong temptation for the academic investigator to point the moral that even from the most abstruse test-tube investigations matters of great clinical import and therapeutic interest may emerge.

THE PROTEIN RESERVOIR

It has now become so usual to estimate the serum proteins in the study of nutrition, shock, and metabolic disease that Peters[1] has done well to emphasize the implications of physiological work on this subject. Modern studies began when Starling showed that the exchange of fluid between the blood stream and the tissues depended on the balance between the capillary blood pressure and the colloid osmotic pressure of the proteins. The probability of a dual causation should always be remembered in the treatment of oedema. In heart failure oedema may prove intractable if, as the result of malnutrition, serum proteins are even slightly reduced. The ascites and oedema of cirrhosis are as much dependent on a fall in serum albumin as a rise in portal pressure. Because of its smaller molecular size and other properties, the serum albumin is the all-important factor in the maintenance of the colloidal osmotic pressure. Moreover, the globulin is but little reduced by albuminuria or malnutrition, and it may, indeed, be greatly increased in cirrhosis and other diseases in which albumin is diminished. Albumin and globulin are in fact independent variables in the serum, and no determination of the proteins that does not include separate measurement of these fractions is satisfactory. When the clinician finds the serum proteins reduced he is tempted to think they may be rapidly increased by the transfusion of plasma. This is a false analogy with the transfusion of red cells, which exist in a closed system within the blood vessels. Proteins, as Beattie and Collard have shown, flow freely in and out of the vascular system,[2] and the serum proteins are in equilibrium with the tissue proteins. The level of the proteins in the serum can be likened to the level of the fluid in a manometer which communicates with a much larger system. When a dog was starved of protein for 80 days the serum albumin was reduced by 70%, the haemoglobin by 50%, and the body weight by about 20%. But if the percentages are converted into total values, it is seen that of the total protein lost about 80% came from the tissues, only about 3% from the serum proteins, and the remainder

[2] Kutscher, W., and Wolbergs, *Z. physiol. Chem.*, 1935, **236**, 237.
[3] Sullivan, T. J., Gutman, E. B., and Gutman, A. B., *J. Urol.*, 1942, **48**, 426.
[4] *Amer. J. Cancer*, 1936, **28**, 485.
[5] Gutman, A. B., Tyson, T. L., and Gutman, E. B., *J. clin. Invest.*, 1938, **17**, 473.
[6] *Cancer Res.*, 1941, **1**, 293.
[7] Huggins, C., Scott, W. W., and Hodges, C. V., *J. Urol.*, 1941, **46**, 997.

[1] *J. Mt. Sinai Hosp.*, 1942, **9**, 127.
[2] *British Medical Journal*, 1942, **1**, 459; **2**, 301, 507.

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