notable difference in the nature of the pneumonia cases between those at this hospital and those reported from England. Complicated cases with septicæmia and abscess formation are a minority here. We wonder how Dr. Briggs and his colleagues ascertained their cases of lung infections; and whether these are reports only of the severe infections, or whether they represent the total of addicts seen with pulmonary infections at the three central London hospitals where the patients described were treated.

Addicts in New York City, in contrast to their London counterparts described in the article, are not emaciated. The conditions of life of an addict in New York City preclude any chronically ill person from being an active addict—ironically, one has to be physically fit to be an addict here. In contrast, the emaciation of the English addicts might be attributed to their having obtained, with relative ease, sufficient pure narcotic to suppress their desire for food. A further clinical difference is that the New York addict with pulmonary infection generally does not have on admission evidence of active cutaneous or venous sepsis. Even in the complicated cases, septic pulmonary embolism does not seem to be the ætiology in our experience, both because of the absence of thrombophlebitis and because of the usual gradual onset of the patient's illness. We suspect that most cases occur as an infection superimposed on the pulmonary œdema which follows intravenous injection of "narcotic" in this city.[2] This syndrome of pulmonary œdema is the cause of death in most of the about 360 identified addicts who die here each year.[3] Since in most of these cases narcotics are not toxologically identifiable in the organs,[2] the narcotic agent is perhaps not responsible. We should like to know what the situation is in England and Wales, where pure narcotic drugs are available to addicts.

We agree with the finding of Dr. Briggs and his colleagues that pulmonary sepsis and staphylococcal septicæmia often occur in patients without tricuspid endocarditis. In this regard, a third of the 28 addicts admitted to hospital with endocarditis, recently investigated, had a history of pneumonia, recent or remote. When those cases in which the pneumonia was presumed to be directly responsible for the development of endocarditis were excluded, a fourth of the remainder had a history of pneumonia. On the other hand, the frequency of a history of pneumonia in addicts who enter this and other hospitals with other diagnoses is less than 5%. This argues that there is a connection in susceptibility between the two diseases in addicts which cannot be explained at the present time.

Finally, we should like to know whether tetanus occurs in addicts in England as it does in this city.[4] The risk of this disease in addicts here is great, despite fairly high levels of group immunity. Presumably they are highly exposed. If tetanus is rare or non-existent in English addicts, who seem to be as careless in their injections as our addicts, the difference might be due to the peculiar but uniform inclusion of quinine in the "narcotic" that the New York City addict buys.

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MORTALITY TRENDS OF RENAL DISEASES

SIR,—I have read with much interest the review by Dr. Waters last week (p. 241), in which he discusses possible causes for the increase in the number of deaths from pyelonephritis and the concomitant decrease in deaths from nephritis. It is surprising that he makes no mention of the advances in diagnostic radiology. Credit must be given to the radiologists, rather than to the pathologists, for the demonstration of the high incidence of chronic pyelonephritis in children and young adults—a demonstration that led to the present appreciation of the importance of this condition as a major cause of death from hypertension and renal failure.

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TYPHOID AND CHOLERA VACCINES

SIR,—In the report by Dr. Ashcroft and his colleagues on the typhoid field trials in Guyana,[1] and the accompanying leading article,[2] there is considerable discussion on the observation that a single dose of vaccine was as effective as two doses. The question remaining unanswered was whether this was unique to the endemic area or whether these observations could be applied to non-endemic areas with a general and convenient recommendation for a single dose of typhoid vaccine to all populations.

This research laboratory has just completed the first year of a controlled cholera-vaccine field trial in 40,000 children, ages three months to fourteen years, in rural East Pakistan. A part of this study was to compare the effect of one dose of vaccine with two doses at an interval of one month. In children, ages five to fourteen years, protection provided by two doses of vaccine was not significantly different from that produced by a single injection, a finding comparable to the effect of typhoid vaccine in five to fifteen year old children in Guyana. In children under five, however, a single dose was virtually ineffective, while two doses provided a level of protection comparable to either one or two doses in older children. Evidence that the enhanced effect of a single dose in older children was indeed due to a "booster" effect on basal immunity in this endemic area was provided by two observations. First, a serological survey demonstrated that a higher proportion of the older population had circulating vibriocidal antibodies. Secondly, in the control group, the cholera case-rate in children ages five to fourteen was only half that of children under five, suggesting that this basal immunity was protective. (A detailed discussion of the effect of basal immunity on the epidemiological pattern of endemic cholera and its effect in vaccine field trials will appear shortly.[3]) Our conclusion is that the results of cholera-vaccine field trials in endemic areas must be interpreted with caution in making recommendations for vaccine schedules for non-endemic populations.

Since serological tests have not correlated with immunity to typhoid, the data from Guyana do not provide any means for estimating a level of basal immunity or any possible protection that may have been produced by such immunity in the control population. Thus, while it is true that "there is no evidence to support"[2] the hypothesis that basal immunity does exist in some of the children, neither is there substantial evidence against it. Certainly, the "numerous infections in the control group"[2] cannot be used as an argument that basal immunity has "no protective properties",[2] since there was not a comparable non-endemic control group of children for comparison.

The application of findings in cholera-vaccine field trials to typhoid-vaccine field trials may be debatable because of vast differences in pathogenesis and undoubtedly in mechanisms of immunity; however, until there is positive evidence against the role of basal immunity in the typhoid-vaccine studies, or until substantial evidence is presented indicating that one or two doses of vaccine provide the same measure of protection in non-endemic populations, there should be some caution in recommending single, instead of multiple, doses of typhoid vaccine to such groups.

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