Correspondence.

To the Editor of the "Post Graduate Medical Journal."

SIR,—My attention has been drawn to the case of liver abscess reported in your issue of October, pp. 374-376, and also to the reply in your November issue by Dr. V. S. Hodson, pp. 428-429. As I hold very distinct views on the subject of the treatment of liver abscess as the result of a somewhat extensive and varied experience of this disease during the last twenty years, I may be permitted to state that I consider both your correspondents are correct in their views. I appreciate fully the grounds upon which Dr. Hodson makes his reiterated statement on what he considers to be the inevitable and favourable result of emetine therapy in hepatic amœbiasis. I say hepatic amœbiasis advisedly because I consider with some show of reason that it is only when the *Entamœba histolytica* is active and multiplying within the abscess cavity that emetine can have its favourable therapeutic effect, since it is generally acknowledged now that emetine is a highly specific amœbicide. But two factors militate against this action from being invariably potent; firstly, the amount of pus produced by the cytotoxicity of liver tissue may be so large that the amount of dead tissue cannot be absorbed by the liver after the amœbæ have ceased to be an active factor; or, secondly, as happens very frequently, the abscess cavity becomes secondarily infected with micro-organisms. Should there be any communication through the diaphragm with the lung this infection is usually a *Streptococcus*, but should the abscess be situated in the lower part of the right lobe of the liver in immediate contact with the large intestine, it may be, as I have proved on two occasions, *Bacillus coli*.

I pointed out in the paper I read on this subject to the Tropical Section of the Royal Society of Medicine (*Proc. Roy. Soc. Med.*, November 5, 1931), the genesis of a liver abscess formation in its various stages and was able to illustrate it by

sections made from fatal cases. First, there is acute amœbic hepatitis—an engorged liver, greatly enlarged and studded with small pin-point abscesses, each containing active *E. histolytica*. I know of no test by which, on clinical grounds, these cases can be distinguished from small actually formed liver abscesses giving that gummatous appearance which Dr. Hodson describes. I imagine these specimens are very rare, because patients do not die at this stage, and I have been fortunate in being able to examine such a case microscopically. From the treatment point of view, this condition reacts miraculously to emetine injections, as one would expect. But there are other cases which at first cannot be distinguished by their clinical signs and symptoms by any test that I know of upon whose course neither emetine injection, or emetine-bismuth-iodide, have the slightest effect.

The treatment therefore divides itself into three headings: (1) Specific treatment with emetine which should always be undertaken; (2) aspiration of the abscess cavity, if it be single and uncontaminated on the same principle as aspiration of a cold abscess is undertaken. I have pointed out in previous papers how eminently successful this is; (3) in a secondarily infected abscess the only rational method, to my mind, is open operation and continuous irrigation of the abscess cavity by the Carrel-Dakin method. Emetine in these cases has not the slightest effect on the abscess itself, but should, of course, be given if a co-existing intestinal amœbiasis can be proved. I am strongly in favour of considering every case of hepatic amœbiasis as harbouring an intestinal infection as well. Should this not be got rid of by the generally approved methods, a further hepatic abscess formation may result. In conclusion, I am prepared to quote examples in support of each of these contentions.

Yours faithfully,

PHILIP MANSON-BAHR.

November 18, 1932.