of physiological shunt mechanism. On the other hand, the increased blood flow caused by inhibition of vasoconstrictor nervous tone seems to be directed through nutritional capillaries during the whole period of vasodilatation. This dilatation should therefore take place, both in the arterioles, and also more peripherally, in the precapillary sphincter region.

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Kallidin—a probable factor in the pathogenesis of malaria

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Tella & Maegraith (1966) suggested that bradykinin was involved in the pathological manifestations of malaria. It became of interest to see whether kallidin was also involved. Kallidin was prepared by incubating acid-treated plasma with urinary kallikrein. Plasma kallidinogen contents were then estimated by assaying the kallidin so produced against synthetic bradykinin.

Acid-treated plasma (Horton, 1958) was obtained from the blood of healthy rhesus monkeys (*Macaca mulatta*) and from those infected with *P. knowlesi* malaria (Tella & Maegraith, 1965). Kallikrein was prepared from pooled urine samples collected from fifteen healthy rhesus monkeys using a modification (Guth, 1959) of the method of Gaddum & Horton (1959). This monkey urinary kallikrein preparation gave a yield of 0.3 mg/ml. urine and an activity of 0.4 unit/mg when assayed against Glumorin. Recoveries of 80–90% were obtained by this method.

Maximum yield of kallidin was acquired by incubating 0.4 unit monkey urinary kallikrein with 5.0 ml. of acid-treated plasma at 37° C for 90 min. The yield from healthy monkeys was 8.9–10.3 mg/ml. plasma, and, when assayed on the isolated guinea-pig ileum, the biological activity was equivalent to 0.6–0.7 μg of synthetic bradykinin. The corresponding figures for the infected monkeys were 4.2–10.3 mg and 0.1–0.7 μg respectively, depending on the severity of the infection at the time blood samples were collected for kallidin estimation. Recoveries by this method were 70–80%.

The results showed that whereas the plasma levels of kallidinogen in healthy rhesus monkeys was approximately constant, in the infected animal they dropped sharply from the third day as the infection became intensified. There is thus a suggestion of an increase in the plasma level of circulating kallidin during the infection.

The behaviour of kallidinogen in these studies runs parallel with that of bradykininogen (Tella & Maegraith, 1966). Consequently, the same interpretations would apply.

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