Born-type aggregometer by incubating aliquots (0·5 ml) of the platelet-rich plasma (PRP) with the prostaglandin for 1 min before the addition of sufficient adenosine diphosphate (ADP, 0·5–2 μM) to cause the second phase of platelet aggregation. In five experiments, the ID50 for the analogue, 6β-PGI$_1$ was 116 $\pm$ 20 ng ml^{-1} and thus was 250 times less active as an inhibitor in human PRP than prostacyclin (ID50, 0·5 $\pm$ 0·07 ng ml^{-1}). The finding that the epimer, 6α-PGI$_1$, was some 3–4 times less active than 6β-PGI$_1$ (ID50 350 $\pm$ 30 ng ml^{-1}) contrasts with the report by Tonga, Gandolfi & others (1977) suggesting that the 6α-epimer was the more active of the pair. Whether this discrepancy is the result of different stereochemical assignment, separation and purity of the compounds synthesized in the different laboratories, or some other experimental factor, remains to be resolved.

The present results indicate that although the stable analogue 6β-PGI$_1$ shares several of the properties of prostacyclin, its relative potency to prostacyclin depends on the system investigated. The analogue was less potent as a vasodilator and inhibitor of human platelet aggregation. Like prostacyclin the analogue inhibited the formation of mucosal erosions and also acid secretion in the rat stomach *in vivo* and *in vitro*. In the *in vivo* preparation where prostacyclin and the analogue were infused intravenously, prostacyclin was more potent as an antisecretory agents. However, following subcutaneous administration in the 3 h erosion study, the analogue was more active. Furthermore, in the *in vitro* isolated perfused stomach preparation, in which the prostaglandins require some 15–30 min to elicit a full response, the analogue was the more potent, presumably reflecting the rapid breakdown of prostacyclin under the incubation conditions. In the other *in vitro* systems where prolonged incubation was not required to achieve the maximal response, prostacyclin was the more potent, as an inhibitor of platelet aggregation and in contracting the rat stomach strip. Thus, although the stable analogue 6β-PGI$_1$ may be only a weak agonist for prostacyclin-sensitive sites, its relative potency to prostacyclin is greatly enhanced under conditions where stability is of importance.

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Action of chloroquine on pleurisy due to *Bordetella pertussis* hypersensitivity

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It is now generally agreed that chloroquine has a good effect on rheumatoid polyarthritis. On the contrary, its actions in the usual phamacological models of experimental inflammation (review by Swingle, 1974) are limited. We describe here its action on delayed *Bordetella pertussis* hypersensitivity in rats.

We have modified the technique previously described (Tarayre, Delhon & Lauressergues, 1977) in order to shorten duration of the sensitization period. Sprague Dawley male rats, 280–320 g, were used. *B. pertussis* suspension (Institut Pasteur—5 $\times$ 10^9 killed organisms ml^{-1}) was homogeneously mixed (v/v: 50/50) with Freund's complete adjuvant (Difco). 0·2 ml of this mixture was injected intramuscularly into both thighs of rats. 6 days later, 0·1 ml of *B. pertussis*-suspension was injected into the pleural cavity. 48 h later, the pleural exudate was taken and measured, and leucocytes counted with a Coulter Counter. After the cells had been spread on slides, the number of mononuclear and polynuclear cells was counted. In a first series of experiments, chloroquine diphosphate was given around the time of challenge (4 administrations of 10 mg kg^{-1}, orally). In a second series, the compound was given from the day of sensitization and during the duration of the experiment (9 administrations of 10 mg kg^{-1},

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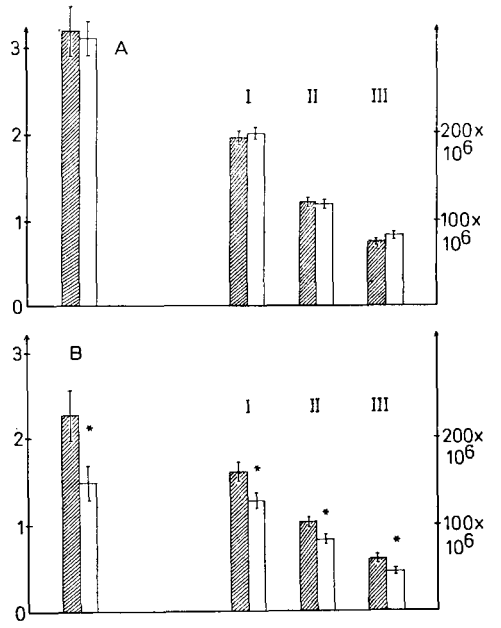

FIG. 1. A. Action of chloroquine on *B. pertussis*-pleurisy after treatment close to the time of challenge. Chloroquine diphosphate was given orally at a dose of 10 mg kg^{-1}, 24 and 2 h before; and 2 and 24 h after challenge. B. Action of chloroquine on *B. pertussis*—pleurisy after chronic treatment during sensitization period and near challenge. Chloroquine diphosphate was given orally at a dose of 10 mg kg^{-1}. The 1st dose of the compound was given 2 h before the 1st antigen injection. Rats were then treated every day. On the day of challenge injection, chloroquine was given 2 h before and 2 h after it. The last intubation occurred 24 h after challenge. For both types of treatment, the results given are the sum of 2 experiments with 18–20 rats per group each. Hatched columns controls; Open columns animals treated with chloroquine. I. Total leucocyte count per rat. II. Mononuclear leucocyte count per rat. III. Polynuclear leucocyte count per rat. Bars represent standard errors. * $P < 0.05$ as compared to controls. Ordinates, left-hand: Volume of pleural exudate (ml); right-hand: Cell count.

orally). Controls received the solvent alone under the same experimental conditions.

In spite of the changes in sensitization conditions compared with our previous work (Tarayre & others, 1977), there was a high pleural inflammation with predominance of mononuclear over polynuclear cells.

When the antimalarial agent was given at the time of challenge, no significant change in pleural inflammation parameters was observed (Fig. 1A).

On administration throughout the experiment, the drug produced a significant decrease of pleural exudate volume (35%) (Fig. 1B). A significant effect was also observed on cellular mechanisms. The total leucocyte count showed a 21%, the polynuclear count a 25% and the mononuclear count a 19% reduction. Moreover, in this experiment, the weight increase of treated and control rats was similar.

The useful actions attributed to chloroquine on some experimental inflammation models, whether immunological or not, were always seen at doses higher than those used clinically (review by Swingle, 1974). The drug's action on Freund's adjuvant polyarthritis is null or controversial (Newbould, 1963; Graeme, Fabry & Sigg, 1966; Ward & Cloud, 1966; Winter & Nuss, 1966; Perrine & Takesue, 1968). In addition, Swingle (1974) summed up the main properties of the antimalarial drug as they have been described *in vitro*. The effects on leucocyte chemotaxis and lymphocyte transformation are seen with low concentrations. The lack of inhibition of *B. pertussis* pleurisy when chloroquine was given around the time of challenge suggests that the effect on leucocyte chemotaxis described by Ward & Cloud (1966) did not play an important part at the doses used. On the contrary, lymphocyte transformation inhibition (Hurvitz & Hirschhorn, 1965) might be involved in the beneficial effect seen when the compound was given during the sensitization period.

According to the above results, this experimental model, described by Dieppe, Willoughby & others (1976), seems to be convenient for the evaluation in animals of the anti-inflammatory action of some anti-rheumatic substances.

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