ments is further evidence of the dimnished renal blood flow. It is concluded that the decrease in urinary output during CPPB is a direct effect of the decrease in cardiac output.

Pa_{O_2} consistently increased when expiratory resistance was removed, rather than during CPPB, while Pa_{CO_2} increased during CPPB. Although an improvement in arterial oxygenation with continuous positive-pressure breathing has been reported,[1-3] Cheney *et al.* found no change in Pa_{O_2} in anesthetized patients subjected to a positive end-expiratory pressure.[5] While increased expiratory resistance prevented atelectasis, the elevated airway pressure caused an increase in pulmonary vascular resistance, resulting in shunting of blood away from ventilated alveoli, and Pa_{O_2} improved only when the expiratory resistance was released. Cheney believed that elevated expiratory resistance was responsible for the decrease in cardiac output, which when combined with constant oxygen consumption would contribute to a decraese in Pa_{O_2}. Philbin *et al.*[13] demonstrated that Pa_{O_2} was directly related to cardiac output, and thus the increase in cardiac output following the release of CPPB may account for the increase in Pa_{O_2} found after the re-establishment of IPPB.

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Drugs

PHENOBARBITAL AND DIPHENYLHYDANTOIN The half-life of phenobarbital in the sera of children treated for epilepsy with phenobarbital and diphenylhydantoin was determined. The serum half-life in children was shorter than that reported for adults. Phenobarbital may also depress the diphenylhydantoin levels, indicating microsomal enzyme induction. (*Garrettson, L. K., and Dayton, P. G.: Disappearance of Phenobarbital and Diphenylhydantoin from Serum of Children, Clin. Pharmacol. Ther. 11: 674 (Sept.) 1970.*)