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Drugs and Their Actions

INTRAOCULAR PRESSURE Changes in intraocular pressure (IOP) in man were compared following topical application of propranolol, lidocaine, and two beta-adrenergic blocking agents devoid of local anesthetic activity. Propranolol and lidocaine had equal local anesthetic effects, and both decreased IOP in normal patients and in patients with simple chronic glaucoma. Neither of the two non-anesthetic beta blockers affected IOP. The authors concluded that the ability of topical propranolol to decrease IOP is related to its anesthetic activity, not to its beta-blocking effect. None of the compounds lowered IOP in acute glaucoma. (*Musini, A., and others: Comparison of the Effect of Propanolol, Lidocaine, and Other Drugs on Normal and Raised Intraocular Pressure in Man, Am. J. Ophthalmol. 72: 773–781, 1971.*)

DRUG-INDUCED PULMONARY EDEMA An episode of acute pulmonary edema in a heroin addict is attributed to alleged intravenous injection of the contents of three capsules (10 mg each) of Librium (chlordiazepoxide). Diffuse rales and rhonchi, frothy pink sputum, and severe arterial hypoxemia ($pH = 7.10$, $P_{O_2} = 28$ torr, $P_{CO_2} = 44$ torr) were present. On the patient's admission to the hospital, the chest x-ray showed diffuse, poorly defined infiltrates throughout both lungs, and a normal heart. The edema cleared after 72 hours following treatment with mechanical ventilation, antibiotics, steroids and alkalinization. No measurements of serum drug levels are reported. (*Richman, S., and Harris, R. D.: Acute Pulmonary Edema Associated with Librium Abuse, Radiology 103: 57–58, 1972.*) EDITOR'S COMMENT: The incidence of pulmonary edema secondary to intravenous self-administration of drugs is likely to increase. We hope that some of these cases will be studied in detail regarding their hemodynamic state (cardiac output, pulmonary artery and capillary wedge pressures, etc.) in order to clarify the cause of the edema. Although the direct "toxic" effect of heroin and other drugs on the pulmonary capillaries has been suggested as a cause, there is little sound evidence to support it. The effect of the drug on ventilation and the ensuing hypoxia are more likely to be responsible. The affected individuals provide an ideal model for studying hemodynamic responses following and during recovery from severe, acute hypoxemia.