to selective depression of inhibitory synaptic control by lidocaine.

Summary and Conclusions

Five to 25 mg/kg lidocaine were given intravenously to 25 decerebrate or spinal cats, mechanically ventilated with oxygen. A volley to a lumbar dorsal root elicited a compound reflex action potential in the ipsilateral homologous ventral root. Lidocaine greatly enhanced the monosynaptically transmitted reflex potential in the 17 survivors, and simultaneously depressed the polysynaptic response. Maximal height of the monosynaptic spike was 7½ times when 5 mg/kg, 10 times when 10 mg/kg, and 25 times the control height when 20 mg/kg lidocaine were given.

The effect of lidocaine on the reflex potential was related neither to blood pressure nor to level of transection. Hence, the changes were attributed to selective depression of segmental inhibitory neural elements. Marked enhancement of monosynaptic transmission suggests that the spontaneous seizure discharges set up in the brain by convulsant doses of lidocaine find at the same time a greatly facilitated spinal motoneuron pool. The generalized seizures accompanying high blood levels of local anesthetic may result from selective depression of the inhibitory restraint normally imposed on excitatory neural input.

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Anesthesia

UMBILICAL CIRCULATION The continuing growth and development of the unborn infant depend upon the ability of its heart to propel an ever-increasing amount of blood through the umbilical circulation. This consists of vessels in the cord and placenta which are not innervated and seem virtually incapable of dynamic adjustment. In perfusion experiments it has been shown that normal fetal blood gas tensions produce near-maximal dilatation in this vascular bed. As its *in-vivo* oxygen requirement is considerable (up to 30 per cent of that supplied to fetus and placenta), the placenta competes with the fetus for the available supply of oxygen. In response to asphyxia, innervated fetal vascular beds in parallel with the placenta compensate by constriction and enable the fetus to redistribute its cardiac output and maintain placental perfusion. When the umbilical vein is progressively constricted, left ventricular output falls more gradually than does right ventricular output. Thus, blood flow crucial to the brain and myocardium may be maintained for a time. (*Goodwin, J. W.: The Impact of the Umbilical Circulation on the Fetus, Amer. J. Obstat. Gynec. 100: 461 (Feb.) 1968.*)