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SUCCESSFUL TREATMENT OF DELAYED ARTERIAL SPASM IN THE RHESUS MONKEY AFTER SUBARACHNOID HAEMORRHAGE

CHRISTOPHER W. NORWOOD, G. JOSEPH POOLE, DIXON MOODY, and EBEN ALEXANDER, JR (Winston-Salem) noted that a successful treatment for delayed cerebral vasospasm after subarachnoid haemorrhage had been reported. In their Rhesus monkey population subjected to subarachnoid haemorrhage 62.5% developed delayed cerebral vasospasm. Seven were treated with a beta-adrenergic drug alone, and five of the seven responded to the drug, representing a 71% response rate. Four other monkeys with delayed cerebral vasospasm were treated with a combination of a phosphodiesterase inhibitor and a beta-adrenergic stimulator. One hundred per cent of these animals responded with complete relief of the delayed cerebral vasospasm. When the two groups of monkeys were treated as one group, nine of 11 animals with delayed cerebral vasospasm had relief of the vasospasm, representing an 81% response rate. The pharmacology of beta-adrenergic stimulators and phosphodiesterase inhibitors was discussed and a rationale for their synergistic effect was postulated.

STUDY OF CEREBRAL ARTERIAL SPASM. *IN VITRO* CONTRACTILE ACTIVITY OF VARIOUS VASOACTIVE AGENTS ON THE HUMAN BASILAR AND ANTERIOR CEREBRAL ARTERIES

GEORGE S. ALLEN, SHELLEY N. CHOU, and L. A. FRENCH (Minneapolis) had previously reported *in vitro* experiments in which a small volume chamber was used to determine the contractile activity of various vasoactive agents on canine basilar and middle cerebral arteries. They described similar experiments carried out on human basilar and anterior cerebral arteries. Segments of these vessels were removed within one hour of death from patients dying of a variety of causes, including subarachnoid haemorrhage from aneurysms. Cumulative dose-response curves were obtained from most of the agents tested, including serotonin, noradrenaline, and F_2 prosta-

glandin. These human arterial segments reacted to these agents in concentrations which were sometimes as low as 1×10^{-12} molar. Serotonin produced a 90% maximal contraction of these arterial segments at a concentration 10-30 times less than that known to be present in blood. The cumulative dose-response curves for the human arteries were similar to those for the canine arteries. The canine cerebral artery must therefore be a good experimental model for the study of the aetiology of cerebral arterial spasm.

INFLUENCE ON THE HYPOTHALAMUS OF INTRACRANIAL ARTERIAL SPASM

ROBERT H. WILKINS (Temple, Texas) postulated that hypothalamic injury plays a role in the development of arterial spasm associated with ruptured intracranial aneurysms in humans. The evidence for this hypothesis was, firstly, that cerebral vasospasm in humans did not have the characteristics that might be expected if it were solely due to the exposure of the arteries to blood, as in animal models. There was, for example, a delay varying from hours to days after subarachnoid haemorrhage before spasm develops, even though the arteries at the base of the brain were immediately surrounded by extravasated blood. Not all patients developed vasospasm and, when it did occur, it seemed to follow preferentially rupture of aneurysms near the hypothalamus and involving vessels which supplied the hypothalamus. Secondly, there was postmortem evidence that patients with ruptured aneurysms had destructive lesions in the anterior hypothalamus where they might be expected to cause overactivity of the sympathetic nervous system, an effect that had been shown by electrocardiography and other investigations to occur. This sympathetic overactivity seemed to occur in the same time sequence as did intracranial arterial spasm. Thirdly, a study of postoperative and post-traumatic cerebral vasospasm had provided analogous and supporting data. At least three mechanisms might be involved in the relationship between hypothalamic injury and the development of intracranial arterial spasm.