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The study was conducted in the department of medicine of M.L.B Medical College, Jhansi from the period August 2003 to September 2004. The cases were patients coming to medicine OPD and in the emergency being admitted in the medicine ward as cases of stroke.

One hundred and nine cases of stroke were included in the study. These were aged between 20 to 95 years, average age 60.95 ± 14.32 years; the maximum number of cases were in 7th and 8th decade thus reflecting an increase in the incidence of stroke with age which was similar to that found by Maria AK et al. The male preponderance and higher association with hypertension (65%) was consistent with other studies 77(70.6%) were males and 32 (29.35%) females, M:F 2.4:1 which increased to 3.78.1 in stroke in elderly (>60 yrs). The ratio of stroke in young (<40 yrs) was M:F 1.3

The mean systolic blood pressure on admission was 158±42.3 mmHg and the mean diastolic blood pressure was 90.5±19.8 mmHg. The higher association of hypertension with PICH (100%) than in CI (47.36%) was comparable with a study conducted in AIIMS and a study in Himachal Pradesh entitled Stroke in High Altitude by Prashar.
BS et al past H/O stroke/TIA (12.8%) was comparable with Kaur et al. Incidence of diabetes mellitus 10(9 17%) in my study was similar to three studies including the one in Kashmir valley. Incidence of rheumatic heart disease 8 (7 3%) was also similar to other studies done in the northern region. Incidence of coronary artery disease 7(6%) was higher than stroke at high altitude.

On CT scan 76(69.7%) were infarcts, 16(14%) intracerebral hemorrhage, 3 (2.75%) subarachnoid hemorrhage and 14 (13%) were undetermined. Lesions of right side of brain were more common (51R, 33L) Parietal lobe (50%) was most commonly involved site, extensive lesions (>2 areas involved) in 41% was next most common. Cerebellum (3%), temporal (3%) and frontal (2%) was other area involved.

Eighty-four (77%) had hemiplegia/hemiparesis, 42 (38%) 7th nerve palsy, 35 (32%) speech disorder, 19 (17%) were unconscious, 4 (3.66%) had convulsions and 3 (2.75%) had headache and vomiting. Hemiparesis, 7th nerve palsy and speech disorders were equally distributed between CI and PICH, whereas headache, vomiting and altered sensorium more common in PICH.

Dyslipidemia was seen in 32 (29.35%), and previous H/O hypertension in 13 (11.9%).
The admission blood pressures as per JNC VII were as follows. 28 (25.68%) normal, 9 (8.25%) prehypertensive, 9 (8.25%) stage 1, and 63 (57.59%) stage 2.

During 30 days following stroke the outcome was that 26 of the patients improved with only mild residual disability, while 48 of them showed no signs of improvement and there disability was stationary. Fifty-two of them deteriorated and worsening course during their stay in hospital and there after 10 of the patients died of which 8 during the first 24–72 hours of their admission.

The second part of my study involved pharmacological elevation of blood pressure in patients who developed a fall in MABP within 48 hrs of admission along with worsening neurological deficit as adjudged by Mathew score.

Thirty-four patients were selected who showed a fall in their blood pressure when compared to that at admission. Seventeen (every alternate starting from the first) comprised of study group 1 who underwent elevation of blood pressure. MABP of this group was 88.63±13.49 that increased to 98.98±7.6 after elevation of blood pressure for five days. In the control group the MABP after 48 hours was 90.78±12.2 and after 5 days it was 89.37±9.77 (p<0.01). In the study group after 5 days 13 improved while in the control group this figure was only one, 2 of them showed no change one of them
deteriorated while 1 one of them died. The control group showed results quite opposite to study group, in this improvement was seen in only 1 of the patients, 9 showed no change, 4 deteriorated while 3 died during the study. This was consistent with similar study undertaken by Guy Rordorf, Steven C, Cramer in their study of pharmacological elevation of blood pressure in acute stroke. This suggests that, elevation of blood pressure can be performed without undue morbidity and mortality in acute stroke patients. Furthermore, in selected patients raising the BP was consistently associated with rapid improvement in neurological deficits.

These associations suggest that patients requiring pressor agents to preserve neurological function might have more extensive brain regions with maximally dilated vascular beds in which local CBF is sensitive to BP.

The length of time during which group I patients depended on hypertensive therapy to maintain there level of neurological function varied considerably. Natural thrombolysis or improved collateral flow may have occurred to eventually obviate the need for increased BP.

Parallel clinical observations have been observed in patients with vasospasm after SAH. In vasospasm after SAH, induced hypertension is thought to improve leptomeningeal collateral flow and
improve CBF in the maximally dilated vascular bed.\textsuperscript{45} Patients with focal brain ischemia treated with vasopressor drugs have been described.\textsuperscript{40,42,86} The largest cohort of patients treated with hypertensive therapy was reported by Wise et al\textsuperscript{42} in 1972. Thirteen patients were treated with pressors, 5 showed improved neurological status immediately after increased BP, in 3 of the 5, significant recovery was maintained after the immediate postischemic period. Other series reported improved neurological function after BP was raised in association with volume expansion after LMW dextran\textsuperscript{87,88}

Indeed phenylephrine-induced hypertension has been reported to improve oxygen metabolism on PET in a stroke patient\textsuperscript{89} and CBF in patients with vasospasm after SAH\textsuperscript{45} Hypertensive therapy may also act by preventing postischemic hypoperfusion\textsuperscript{90-97}

The paucity of differences seen in baseline and in morbidity/mortality between groups using the drug and those not suggest that phenylephrine use is safe in the setting of acute stroke. However, deleterious effects have been observed in some instances,\textsuperscript{98-100} including increased blood-brain barrier permeability\textsuperscript{101} and vasogenic edema.\textsuperscript{99} In my study there was no increased risk for development of neurological complications due to the use of systemic hypertensive therapy.
Phenylephrine was selected as vasopressor agent of choice in our patients because (1) cerebral vessels have low density of alfa1 receptors so that phenylephrine does not produce significant direct cerebral vasoconstriction, and (2) as a pure alfa 1 agonist does not cause tachycardia or tachyarrhythmias. However, it can cause direct vasoconstriction in the coronary artery circulation and increased afterload and may contribute to congestive heart failure, cardiac ischemia, renal insufficiency, and gastrointestinal ischemia.\textsuperscript{102}

Seventeen patients were included in the group 2 which showed an elevation or had blood pressure >220/110 mmHg. All 17 patients underwent lowering of blood pressure. Lowering of blood pressure in acute ischemic stroke (p<0.01) although very significant produced changes that could not be labeled as significant in any direction. Following lowering of blood pressure by nimodipine and other antihypertensive drugs produced no change in outcome of 11 of the patients, 1 of them improved, 2 deteriorated, and 3 died during the five days.