

## CHAPTER VI

### CONCLUSION

This study investigated the role of NO and 5-HT<sub>1B</sub> receptor agonist on cerebral hyperemia evoked by CSD. L-NAME and naratriptan were used as NOS inhibitor and 5-HT<sub>1B</sub> receptor agonist, respectively. CSD was induced by topical application of solid KCl on the parietal rat's brain surface whereas NaCl was served as a control. The rCBF was monitored by the laser Doppler flowmetry and pial arteriolar diameter was measured by the fluorescent microscopic technique.

The results showed that KCl application could induce cyclical pattern of cerebral hyperemia, which was likely due to CSD. In the fluorescent microscopic study, the repeated pattern of vasodilation-vasoconstriction cycles was observed. The temporal pattern of these two measures was closely correlated. NaCl application did not changes rCBF and pial arteriolar diameter in any period. Furthermore, administration of L-NAME could decrease the amplitude of hyperemic peaks as well as minimize the maximal vasodilation of hyperemic cycles. The minimizing effect of L-NAME on cortical hyperemia depended on its dosage. Our findings suggest that CSD evokes cerebral hyperemia via activation of NO pathway. Administration of naratriptan at the dose of 0.1 mg/kg BW could not decrease cerebral hyperemia evoked by CSD.

Based on these findings, we suggest that the stimulation of nNOS activity play a significant role in CSD-evoked cerebral hyperemia and 5-HT<sub>1B</sub> receptor activation has minor effect on this process.