INTRODUCTION

In the early 1960s α_2 adrenoceptor agonists were initially synthesized for intended use as anticongestants, because it was expected that a locally acting α adrenergic vasoconstrictor agent would provide relief by shrinking swollen nasal membranes, thereby producing an unobstructed nasal passage. A small sample of the new compound (now named clonidine) was supplied to the medical department of Boehringer to test its nasal decongestive properties in humans. A secretary who has a cold was allowed to administer to herself a few drops in her nostrils. However, there was some surprise and embarrassment when the lady fell asleep for 24 hours. She also developed rather low blood pressure, marked Bradycardia and dryness of the mouth. The dose amounted as determined latter, to the equivalent of approximately 20 tablets of clonidine. During subsequent testing, it becomes clear that clonidine is a potent antihypertensive drug and it was introduced to the market in 1966 ***(Stahle et al., 1982).***

Today the therapeutic objective for administration of α_2-adrenoceptor agonists has shifted from reduction of high blood pressure to various other application, including the management of myocardial ischemia and withdrawal symptoms in drug addicts. The development of highly specific α_2- adrenoceptor agonists with profound effects on vigilance and haemodynamics has created new interest for the use of α_2-adrenoceptor agonists for use in anaesthesia and intensive care medicine. α_2- adrenoceptor agonists posses a variety of pharmacological properties that render them desirable as adjuncts in anaesthesia. Clonidine, an imidazoline, is the prototypal α_2- adrenoceptor agonists. It has a relatively slow onset (0.5 h) and an elimination half – life of 9-12 h.

A second – generation α_2- adrenoceptor agonists has been developed; these have shorter duration of action and possess full agonist properties with a high selectivity for α_2- compared to α_1 – adrenoceptors. Such compounds include mivazerol and dexmedetomidine, which have elimination half – lives of approximately 4h and 2.3h, respectively. The distribution volume and clearance of dexmedetomidine are similar to those of fentanyl, and its α_2/ α_1 selectivity ratio is seven to eight times greater than that of clonidine (1620 Vs 220, respectively) ***(Judeth et al., 2000).***

Alpha2- adrenoresceptor agonists administrated into intrathecal and epidural space have been found to be effective in treatment of chronic pain. More over, it was shown that they increase the analgesic effects of local anaesthetics and provide sedation, anxiolytic and haemodynamic stability ***(Konkci et al., 2007)*** intrathecal administration of clonidine prolong motor blockade induced by local anaesthetic. Since the affinity of dexmedetomidine (DEX) to alpha-2 adrenoreceptors is eight – times greater than clonidine, it is expected that DEX could be advantageous in clinical anaesthesia ***(Calasan, Maia et al., 2005).***

AIM OF THE WORK

The purpose of this study was to compare the onset and duration of sensory and motor block, as well as hemodynamic changes and level of sedation, following intrathecal and epidural bupivacine supplemented with low dose dexmedetomidine.