INTRODUCTION

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Friedman (1989) defined dysfunctional labor as any deviation in normal progress of labor either in cervical dilatation or in descent of the presenting part.

Wray (1993) showed that dysfunctional labor occur in approximately 3% to 8% of all deliveries. Also, ***Notzon et al. (1994)*** found that dysfunctional labor was responsible for about 12% of cesarean section rate. ***Sanchez-Ramos et al. (1996)*** mentioned that dysfunctional labor increased the cesarean section rate 4 times over the past four decades.

Wansbrough et al. (1968) stated that, the uterus (corpus and cervix) consists of smooth muscle layers that contain two types of adrenergic receptors, alpha and beta. Stimulation of alpha receptors causes an increase in uterine tone and contractions, whereas, stimulation of beta receptors has the opposite effect.

Soliman et al. (1993) stated that the incidence of dystocia and the use of oxytocin to treat this dysfunctional labour pattern have not subsantially changed over the past several years.

Mahon et al. (1967) concluded that the administration of the beta receptor blocking agent propranolol had no effect on uterine activity in pregnant women not in labour, while its administration during advanced labour resulted in increased uterine contractility.

Mitrani et al. (1975) were the first to evaluate the effects of propranolol on dysfunctional labor. They observed that the use of this beta blocking agent in cases of dysfunctional labor appeared to increase uterine contractility and accelerate the active phase of labor and mentioned that propranolol could play a role in the management of dysfunctional labor.

Sanche-Ramos et al. (1996), concluded that, propranolol administration in a small dose IV in patients with dysfunctional labor safely reduced the rate of cesarean section particularly among patients with inadequate uterine contractility.