Journal of Respiratory Medicine

Case Report Open Access

Medications Commonly Used in the Treatment of Pulmonary Disorders

Mike Saunders*

Department of Epidemiology and Public Health, University of Nottingham, UK

Abstract

Medications that stimulate adrenergic receptors are frequently referred to as sympathomimetic adrenergic, and those that inhibit adrenergic receptors are referred to as Sympatholytic. Similarly, medications that stimulate the cholinergic receptors are referred to as para-sympathomimetic, and those that inhibit cholinergic receptors are called para-sympatholytic. The actions and adverse effects of the principal bronchodilators, specifically, the autonomic-active agents and methyl-xanthines, will be reviewed next.

Keywords: Methyl-xanthines; Sympathomimetic drugs; Epinephrine; Alpha-receptor; Isoproterenol

Introduction

Additionally, two types of anti-inflammatory medications corticosteroids and cromolyn sodium will also be considered in this section because of their positive effect on bronchial intraluminal diameter. Sympathomimetic drugs may be selective or non-selective in their activity. Selective medications react with specific receptors, whereas non-selective medications react with several receptors [1]. As with all drugs, the response elicited by sympathomimetic agents depends on the relative intensity of the receptor reaction, the route of administration, and the dosage of the particular drug. Recall that alpha-receptors are distributed within peripheral and bronchial smooth muscle, the myocardium, and mucosal blood vessels, but they are most abundant in the peripheral smooth muscles, beta-receptors are more abundant in cardiac tissue, although they are also present in mucosal blood vessels, and beta-receptors predominate in the bronchial smooth muscles, although they are also found in peripheral smooth muscle and skeletal muscle. Epinephrine and ephedrine typify general, non-selective sympathomimetic [2]. Epinephrine has a short duration of action, demonstrating moderate alpha-receptor activity, strong beta,-receptor activity, and moderate beta-receptor Ephedrine has a long duration of action, exhibiting mild alpha-receptor activity and moderate beta, and beta, activity. Thus, in the treatment of bronchoconstriction, peripheral vascular constriction and accelerated cardiac responses may result from the alpha and beta, receptor stimulation [3]. Other adverse reactions include agitation, sweating, headache, and nausea. It should be noted that the route by which a particular medication is administered significantly influences the extent of any adverse reactions. For example, sympathomimetic medications administered by an inhalational route produce less profound deleterious side effects than when they are administered systemically [4]. As long as infusion Continues Clearly, drugs that elicit no alpha receptor activity and more specific beta-receptor activity would be desirable for bronchodilator therapy. Unfortunately, however, no purely beta-specific sympathomimetic medications have been identified. Isoproterenol, a drug with very weak alpha-receptor activity and strong beta-and beta, receptor activity, is the most commonly prescribed nonspecific beta sympathomimetic [5].

Methodology

Selective stimulation of beta-receptors is preferable, because beta,-specific agents would affect the lungs without affecting the heart. Beta-specific agents produce bronchiolar dilatation by relaxing the bronchial smooth muscle through facilitation of increased Cyclic adenosine monophosphate levels. The most commonly prescribed beta-specific-type sympathomimetic include isoetharine and terbutaline, which elicit weak beta-receptor activity and moderate beta activity, as well as albuterol and bitolterol, which elicit weak beta, receptor activity and strong beta,-receptor activity [6]. Although the adverse effects of the currently available beta, specific sympathomimetic are similar to, but usually less profound than, those of the nonspecific beta-sympathomimetic, their severity affects the dosage of the drug administered. The adverse effects that are associated with beta sympathomimetic medications include tremor, palpitations, headache, nervousness, dizziness, nausea, and hypertension. In addition, nonspecific beta-sympathomimetic drugs may also elicit inotropic and chrono-tropic effects. Because alpha-adreno-receptor stimulation produces both vasoconstriction and bronchoconstriction, the pharmacologic reduction of alpha-adrenergic activity via alpha-adreno-receptor antagonists is certainly warranted for patients with pulmonary disease [7]. Alpha-sympatholytic action inhibits the decrease of' CAMP associated with an antigen-antibody reaction, allowing broncho-dilation to occur. The most common side effects associated with alpha sympatholytic administration are nausea and dizziness. The lungs receive a rich supply of parasympathetic innervation through the vagus nerve. The role of the para-sympatholytic is similar to that of alpha-sympatholytic in that it is the lytic action that produces the desired effect of bronchodilation [8]. Blocking parasympathetic stimulation prevents an increase in Cyclic adenosine monophosphate, thereby producing a relative increase in the amount of Cyclic adenosine monophosphate and promoting bronchial smooth muscle relaxation. The agent most commonly used for this purpose was the muscarinic antagonist atropine. Because atropine is readily absorbed into the systemic circulation, however, it is frequently associated with adverse reactions, including central nervous system stimulation with low dosages or depression with high dosages, delirium, hallucinations, and decreased gastrointestinal activity [9].

Discussion

The newer drug, ipratropium, is administered by inhalation and is, therefore, poorly absorbed into the systemic circulation, consequently, it has fewer side effects. The intracellular level of Cyclic adenosine

***Corresponding author:** Mike Saunders, Department of Epidemiology and Public Health, University of Nottingham, UK, E-mail: MikeSaunders@gmail.com

Received: 30-Nov-2023, Manuscript No.JRM-23-122008; **Editor assigned:** 02-Nov-2023, PreQC No. JRM-23-122008(PQ); **Reviewed:** 16-Nov-2023, QC No. JRM-23-122008; **Revised:** 22-Nov-2023, Manuscript No. JRM-23-122008(R); **Published:** 29-Nov-2023, DOI: 10.4172/jrm.1000188

Citation: Saunders M (2023) Medications Commonly Used in the Treatment of Pulmonary Disorders. J Respir Med 5: 188.

mono-phosphate can be enhanced if the process of its degradation by the enzyme phosphor-diesterase can be inhibited. By blocking the inactivation of Cyclic adenosine monophosphate, broncho-dilation is facilitated. The group of drugs that most significantly inhibits phosphor-diesterase is the methyl-xanthines. Methyl-xanthines are also believed to facilitate broncho-dilation by means of prostaglandn inhibition, adenosine receptor blockade, enhancement of endogenous catecholamine levels, inhibition of Cyclic adenosine monophosphate, and enhancement of the translocation of intracellular calcium [10]. Although still inconclusive, there is evidence to suggest that these actions enhance diaphragmatic contractility, reduce patient complaints of dyspnoea, and improve exercise tolerance and gas exchange. The most commonly used methyl-xanthines are theophylline and arninophylline, which are associated with a variety of side effects, including increased myocardial work load, heightened susceptibility to ventricular and supraventricular dysrhythmias, and possibly diuresis [11]. Fortunately, the identification of several phosphor-diesterase isoenzymes is spurring the development of selective Inhibitors that should minimize the systemic side effects associated with such non-selective Inhibitors as theophylline and aminophylline [12]. The potential benefits and undesirable side effects associated with methyl-xanthines are summarized. Cromolyn Sodium Research in England in the 1960s on an extract of a Mediterranean plant resulted in the discovery of cromolyn sodium, which is known by the brand names Intal, Nalcrom, Nasalcrom, or Hypertension Increased diaphragmatic strength/endurance [13]. Palpitations Improved ventilatory muscle resolves Chest pain increased minute ventilations Agitation Enhanced gas exchange dizziness Enhanced exercise tolerance Headache Decreased sensation of Dyspnoea Rynacrom. Cromolyn is used prophylactically in cases of chronic asthma because it prevents the immediate hypersensitivity reaction. Immediate hypersensitivity reactions are mast cell controlled via the release of specific mediators that produce bronchoconstriction and a variety of other signs and symptoms. Cromolyn sodium does not prevent the activation of mast cells and the subsequent release of specific mediators that results from the coupling with an allergen [14]. The late phase reaction in an acute asthma episode which can cause severe airway obstruction after initial bronchoconstriction can be prevented by cromolyn sodium. Cromolyn prevents the influx of calcium ions into the mast cells, blocking the degranulation process of these cells and thereby inhibiting the further release of mediators that cause bronchoconstriction. Thus, although cromolyn has no effect on the mediators released from the antigen-antibody reaction of an acute asthma attack, it prevents later bronchoconstriction by impairing the release of mediators Cromolyn has few side effects, the most common being dry mouth and throat, airway irritation, and possible bronchospasm. The adrenal cortex synthesizes cholesterol and secretes two major corticosteroids, the mineralocorticoids and the glucocorticoids. Although there is some controversy as to the exact mechanism, glucocorticoids are postulated to suppress the process of mediated bronchoconstriction and to block or inhibit a variety of mediator substances. Even though glucocorticoids are not classified as primary bronchodilators, they are known to have the following general effects that contribute to the enhancement of bronchial intraluminal diameter. inhibition of the migration of leukocytes and mast cells reduction of stickiness and margination of poly-morpho-leukocytes potentiation of catecholamine activity reduction of tissue stores of histamine and other mediators suppression of kinin activity, resulting in constriction of the microvasculature stabilization of mast-cell membranes reduction of phosphor-diesterase activity It is because of their profound anti-inflammatory actions, that glucocorticoids are considered by many to be the drugs of first choice during an acute attack of asthma. The most commonly used glucocorticoids in the treatment of broncho-constriction are identified. Although it influences all medications, the route of administration of cortico-steroids is of particular importance because of the tremendous impact it plays in the incidence of side effects-the more localized the area of administration, the less likely the chance of systemic reaction. During episodes of severe bronchoconstriction, glucocorticoids are usually administered intravenously. For virtually no data on which to base any comparisons of the efficacy. Common Glucocorticoids numerous classes of drugs used in the Drug Generic Route of Name Administration Prednisone Intravenous, oral Hydrocortisone Intravenous, oral Methylprednisolone, Intravenous Dexamethasone Inhaler, Beclomethasone Inhaler, Triamcinolone lnhaler, Flunisolide Inhaler, prolonged use, however, an oral or inhalational route of administration is used. The inhalational route is usually preferred because there are fewer side effects. Most of the adverse effects of glucocorticoid therapy are dosage dependent and take a few days or weeks to manifest themselves. Some side effects are unavoidable; reactions span the spectrum from merely unpleasant to dangerous. The primary side effects associated with steroid use include immunosuppression, gastrointestinal disturbance, emotional ability, insomnia, osteoporosis, retardation of growth, muscle weakness and atrophy, hyper-glycemia, sodium and water retention, and cushingoid effect. Regarding Bronchodilators and Anti-inflammatoy Agents Even a cursory review of the literature concerning asthma illustrates the complexity of the disease and a lack of consensus regarding the use of medications in the treatment of the various types of asthma. Little consensus could be expected because there are treatment of asthma. The same probably holds true for many of the drugs used to treat other kinds of lung disease. Although the National Institutes of Health has developed and promulgated guidelines for patient care and pharmacologic therapy in the management of asthma, there are several limitations to the guidelines. The primary limitation involves the method of defining the severity of asthma, frequency of acute episodes versus the intensity and duration of airway obstruction. Moreover, some authors contend that the virtual absence of comparative data for different pharmacologic therapies fosters an environment in which the choices for the use of inhaled steroids, cromolyn, sympathomimetic, or specific beta-agonists are made on the basis of personal preference and anecdotal communication between physicians and patients.

Perhaps, physical therapists can take a part in limiting this dilemma by carefully documenting and reporting the functional ramifications associated with the withdrawal or titration of pharmacologic therapy in the treatment of patients with asthma. Because mast-cell stabilization is believed to reverse or prevent the acute physiologic and clinical aspects of asthma, there is an on-going quest to identify the most efficacious means of accomplishing such stabilization. Unfortunately, many investigations of the speech effects of cromolyn and corti-costeroids are highly theoretical and lack a large base of supportive data. In addition, the evidence suggesting that beta2-agonistsare more potent mast-cell stabilizers than cromolyn has not been widely substantiated.

Nonetheless, beta-agonists such as albuterol and terbutaline have been the most frequently prescribed drugs for the treatment of asthma since 1983.

Conclusion

We may need antibiotics to treat the infection. Remember that repeated exacerbations can cause bronchiectasis to worsen over time. All forms of airway clearance depend on good coughs to move loose mucus out. We can learn techniques such as huffing to improve your cough strength and effort.

Acknowledgement

None

Conflict of Interest

None

References

1. Mello RD, Dickenson AH (2008) Spinal cord mechanisms of pain. BJA US 101: 8-16.
2. Bliddal H, Rosetzsky A, Schlichting P, Weidner MS, Andersen LA, et al (2000) A randomized, placebo-controlled, cross-over study of ginger extracts and ibuprofen in osteoarthritis. Osteoarthr Cartil EU 8: 9-12.
3. Maroon JC, Bost JW, Borden MK, Lorenz KM, Ross NA, et al. (2006) Natural anti-inflammatory agents for pain relief in athletes. Neurosurg Focus US 21: 1-13.
4. Birnesser H, Oberbaum M, Klein P, Weiser M (2004) The Homeopathic Preparation Traumeel® S Compared With NSAIDs For Symptomatic Treatment Of Epicondylitis. J Musculoskelet Res EU 8: 119-128.
5. Gergianaki I, Bortoluzzi A, Bertsias G (2018) Update on the epidemiology, risk factors, and disease outcomes of systemic lupus erythematosus. Best Pract Res Clin Rheumatol EU 32: 188-205.]
6. Cunningham AA, Daszak P, Wood JLN (2017) One Health, emerging infectious diseases and wildlife: two decades of progress? Phil Trans UK 372: 1-8.
7. Sue LJ (2004) Zoonotic poxvirus infections in humans. Curr Opin Infect Dis MN 17: 81-90.
8. Pisarski K (2019) The global burden of disease of zoonotic parasitic diseases: top 5 contenders for priority consideration. Trop Med Infect Dis EU 4: 1-44.
9. Kahn LH (2006) Confronting zoonoses, linking human and veterinary medicine. Emerg Infect Dis US 12: 556-561.
10. Bidaisee S, Macpherson CNL (2014) Zoonoses and one health: a review of the literature. J Parasitol 2014: 1-8.
11. Cooper GS, Parks CG (2004) Occupational and environmental exposures as risk factors for systemic lupus erythematosus. Curr Rheumatol Rep EU 6: 367-374.
12. Parks CG, Santos ASE, Barbhaiya M, Costenbader KH (2017) Understanding the role of environmental factors in the development of systemic lupus erythematosus. Best Pract Res Clin Rheumatol EU 31: 306-320.
13. Barbhaiya M, Costenbader KH (2016) Environmental exposures and the development of systemic lupus erythematosus. Curr Opin Rheumatol US 28: 497-505.
14. Cohen SP, Mao J (2014) Neuropathic pain: mechanisms and their clinical implications. BMJ UK 348: 1-6.