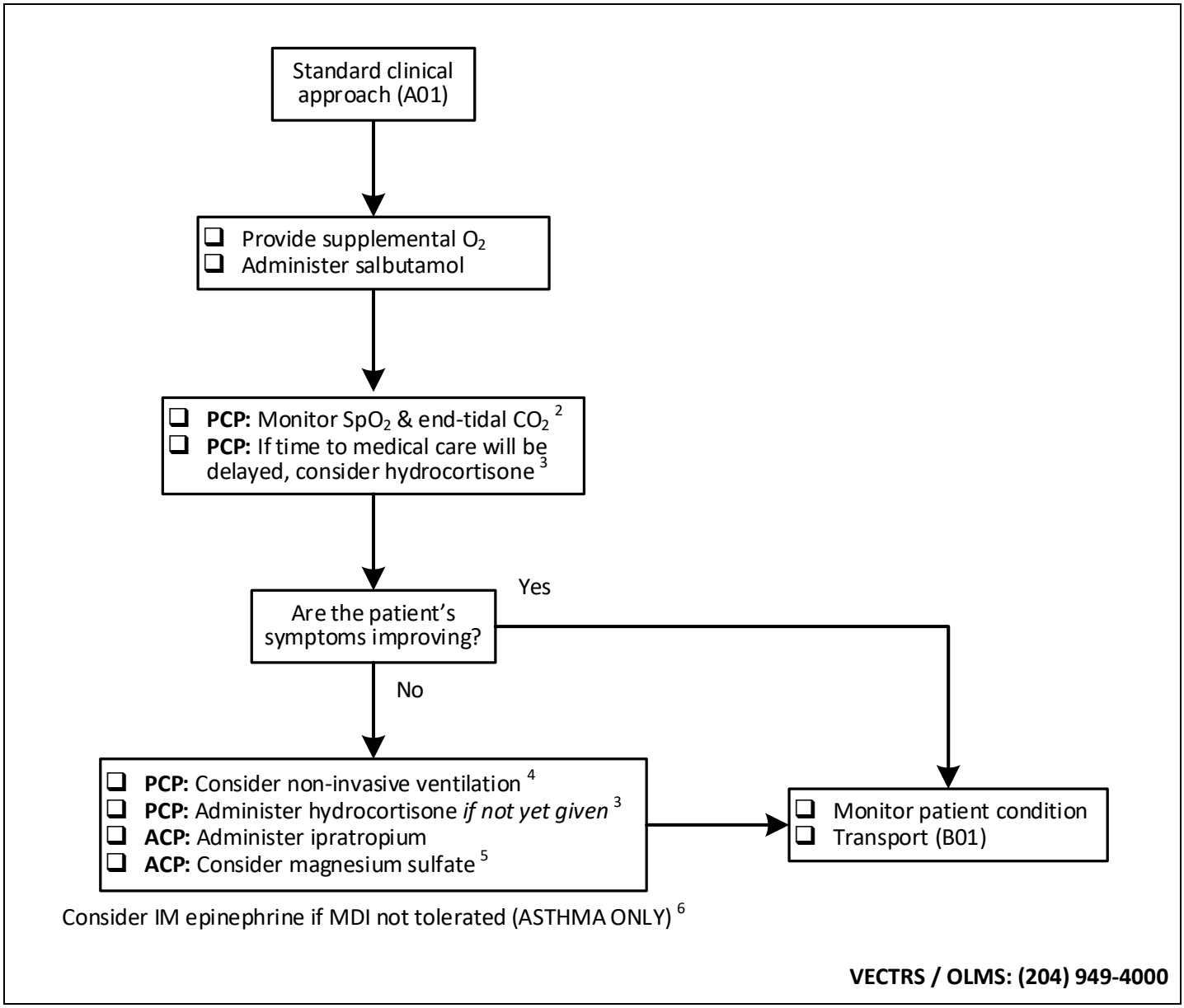
Shared health Soins communs Manitoba	E07 - ASTHMA & COPD (ALL AGES)	
	Version date: 2025-03-25	Effective date: 2025-04-30 (07:00)
PCP = PCP - ACP	ICP = ICP & ACP	ACP = ACP only	None = EMR - ACP



INDICATIONS
• Patients with dyspnea, respiratory distress, or respiratory failure known or suspected to be due to asthma or chronic obstructive pulmonary disease (COPD)

WARNINGS
• Not applicable

NOTES

1. The presence of certain physical findings indicates a severe asthma exacerbation, including the inability to speak in full sentences, the use of accessory muscles, and a pulsus paradoxus (i.e. a fall in systolic blood pressure during inspiration) or more than 12 mmHg. However, many patients will not show obvious clinical signs of severe airflow obstruction. Airflow measurements are one of the best ways to determine the severity of an attack, but are not usually available to paramedics.

   A lack of wheezing ("silent chest") may indicate severe airflow obstruction and is an ominous sign. Many patients with chronic obstructive pulmonary disease (COPD) will have severe fixed obstruction to airflow and do not move enough air to produce wheezing.

   For practical purposes, a lack of response to inhaled bronchodilators should raise suspicion of a severe attack and warrant aggressive therapy and close monitoring in the prehospital setting.

2. Patients with severe asthma can rapidly progress to respiratory failure despite initial normal readings. Significant hypoxemia (oxygen saturation less than 90%) or hypercapnia (end-tidal CO_2 greater than 40 mmHg) are infrequent findings during uncomplicated exacerbations. Their presence indicates a life-threatening situation and possible complication by pneumonia, atelectasis, or mucous plugging.

   Constant monitoring of oxygen saturation (SpO_2) and end-tidal carbon dioxide ($ETCO_2$) as well as continuous cardiac monitoring and frequent blood pressure measurement is essential (appendix A). Equally important, paramedics should frequently reassess the patient's work of breathing (WOB) and level of consciousness (LOC).

   Decreasing WOB and LOC may be signs of hypercapnia due to worsening respiratory failure. Agitation may indicate worsening hypoxemia. Do not sedate a patient with agitation who has a low oxygen saturation.

3. Systemic steroids hasten improvement in patients with severe airway obstruction and early administration is indicated if initial bronchodilator treatment is ineffective.

4. Non-invasive ventilation by continuous positive airway pressure (CPAP) is an aerosol generating medical procedures. Appropriate personnel protective equipment (PPE) is required (A09).

5. Intravenous magnesium sulfate is a short-acting bronchodilator that is well established to be beneficial in asthma. Recent evidence suggests it is *equally* effective in acute exacerbations of COPD (AECOPD).

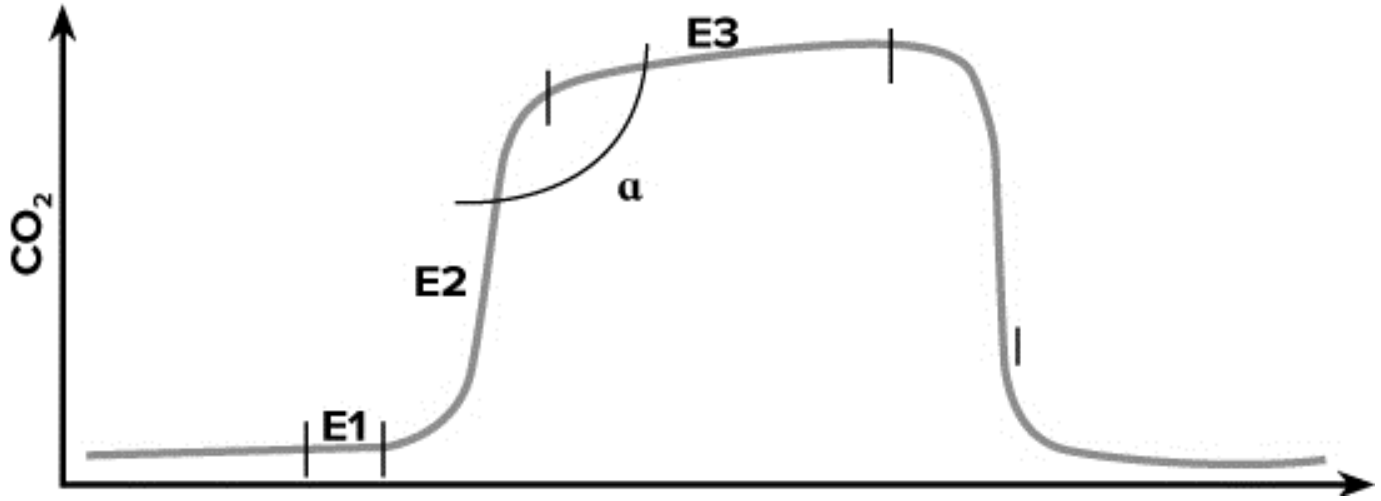
6. Intramuscular (IM) epinephrine can be used for patients who cannot tolerate or cannot cooperate with inhaled bronchodilators. It may be lifesaving in patients with impending respiratory arrest due to asthma, but there is no evidence to support its use in COPD, and it may precipitate cardiac arrhythmias or myocardial ischemia, especially in patients who are already hypoxemic or acidotic.

LINKS
• A01 - Standard Clinical Approach • A09 - Aerosol Generating Medical Procedures • B01 - Standard Destination & Redirection • M05 - Epinephrine • M13.1 - Hydrocortisone • M15.1 - Salbutamol • M15.2 - Ipratropium • M24 - Magnesium Sulfate

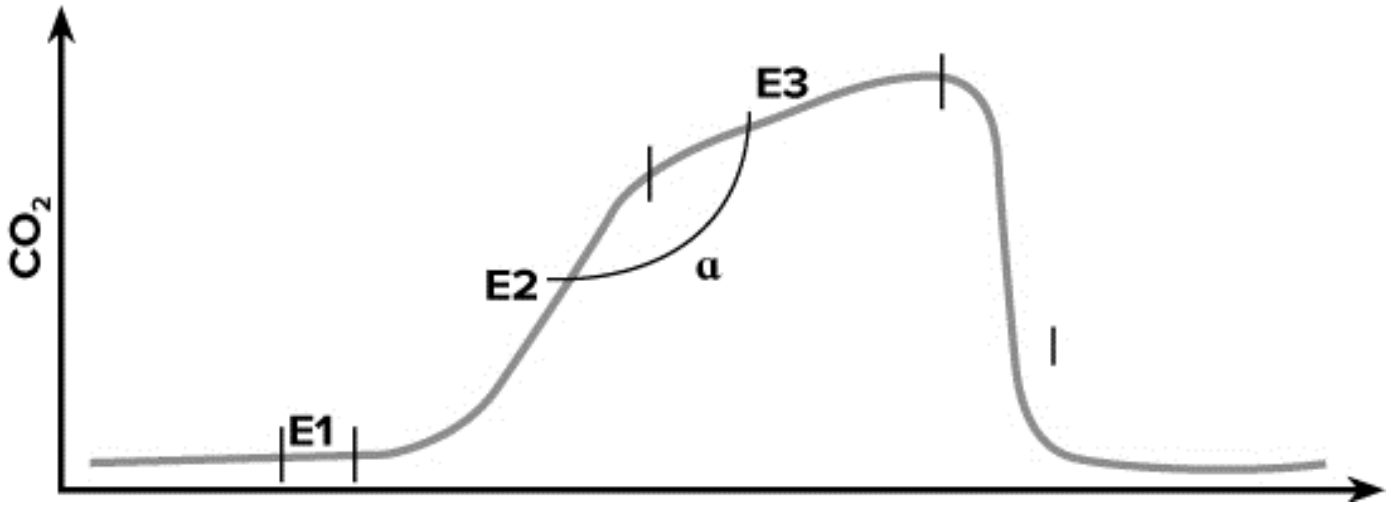
APPROVED BY	
	
EMS Medical Director	EMS Associate Medical Director

VERSION CHANGES (REFER TO X05 FOR CHANGE TRACKING)
• Addition of advanced work scope • Addition of appendix for capnometry

APPENDIX A: WAVEFORM CAPNOGRAPHY IN SEVERE ASTHMA



NORMAL TRACING



SEVERE AIRWAY OBSTRUCTION

With a severe asthma attack (lower tracing) the bronchoconstriction of small airways causes a decrease in ventilation that is unevenly distributed throughout the lung. Areas of the lung that have less constriction will have less CO2 and will empty first (phase E2). Areas of the lung that have a greater degree of obstruction (and thus more CO2) will empty later (phase E3). This desynchronization of alveolar emptying cause the typical “shark fin” waveform seen in severe airflow obstruction.