

1. ACUTE RESPIRATORY DEPRESSANT EFFECTS OF INJECTED HEROIN

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Abstract

Opioids are respiratory depressants and heroin/opioid overdose is a major contributor to the excess mortality of heroin addicts. The individual and situational variability of respiratory depression caused by intravenous heroin is poorly understood. This study used advanced respiratory monitoring to follow the time course and severity of acute opioid-induced respiratory depression. 10 patients (9/10 with chronic airflow obstruction) undergoing supervised injectable opioid treatment for heroin addiction received their usual prescribed dose of injectable opioid (diamorphine or methadone) (IOT), and their usual prescribed dose of oral opioid (methadone or sustained release oral morphine) after 30 minutes. The main outcome measures were pulse oximetry (SpO₂%), end-tidal CO₂% (ETCO₂%) and neural respiratory drive (NRD) (quantified using parasternal intercostal muscle electromyography). Significant respiratory depression was defined as absence of inspiratory airflow >10s, SpO₂% < 90% for >10s and ETCO₂% per breath >6.5%. Increases in ETCO₂% indicated significant respiratory depression following IOT in 8/10 patients at 30 minutes. In contrast, SpO₂% indicated significant respiratory depression in only 4/10 patients, with small absolute changes in SpO₂% at 30 minutes. A decline in NRD from baseline to 30 minutes post IOT was also observed, but was not statistically significant. Baseline NRD and opioid-induced drop in SpO₂% were inversely related. We conclude that significant acute respiratory depression is commonly induced by opioid drugs prescribed to treat opioid addiction. Hypoventilation is reliably detected by capnography, but not by SpO₂% alone. Chronic suppression of NRD in the presence of underlying lung disease may be a risk factor for acute opioid-induced respiratory depression.

Keywords opioid overdose: respiratory depression