



## INVESTIGATING THE EFFECTS OF ANALGESICS MEDICATIONS ON BLOOD PRESSURE

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### Abstract

Analgesics has commonly used in our culture. Its is one of the counter medicine (OTC). Analgesics are mostly used in geriatric population for the pain management of arthritis and other than arthritic pain. Mostly female used analgesics for psychogenic pain in daily life. In clinical practice, hypertension and pain commonly coexist because, like pain, hypertension is widely prevalent in the general and elderly population. Blood pressure (BP) readings are known to be impacted by pain and analgesic drugs, with presser effects differing depending on the length of discomfort and the drug class taken into consideration. Therefore, in hypertensive individuals, pain and analgesics may interfere with blood pressure regulation and may have an impact on the development of arterial hypertension.<sup>i</sup>

"An unpleasant sensory and emotional experience, associated with actual or potential tissue damage" is the definition of pain. Pain is typically divided into two categories based on how long it lasts: acute pain and chronic pain. Acute pain is a short-lived, abrupt physiological reaction to unpleasant stimuli that lasts for less than three months. Chronic pain lasts longer than the average recovery period, lasting three to six months or longer each time. One of the most prevalent health complaints among adults seeking medical attention is pain, especially in the elderly population. In primary care, acute pain is quite common, with headaches and low back pain being the most common causes. It is also typical in the hospital context, where 80% of patients report some form of pain, with 9–36% of those cases being classified as severe. Globally, 25–35% of persons have chronic pain, and the frequency of these cases rises with age. It is a primary cause of sadness, low life quality, limited mobility, and disability, all of which have a substantial negative impact on people's psychological wellbeing. In clinical practice, hypertension and pain often coexist because, like pain, hypertension is widely common in the general and elderly population.<sup>ii</sup>

Acute pain causes a stress reaction, which causes a brief rise in blood pressure. Impaired control of the cardiovascular and analgesic systems is linked to chronic pain, which may make a person more susceptible to ongoing blood pressure increase. Moreover, analgesics may have BP adverse effects

that differ depending on the drug class. While there is disagreement over data regarding paracetamol, some studies suggest that non-steroidal anti-inflammatory medicines (NSAIDs) may raise blood pressure, with celecoxib having the least effect.<sup>iii</sup> Opioid medications have been linked to hypotension. Adjuvants that potentiate adrenergic transmission include tricyclic antidepressants and serotonin-norepinephrine reuptake inhibitors, which may have a pro-hypertensive effect. Analgesics and pain can cause a clinically significant instability of blood pressure. Future research should examine the effects on the occurrence of hypertension and blood pressure regulation, as they are currently unclear.<sup>iv</sup>

### **Acetaminophen**

Among over-the-counter drugs, paracetamol is a first-line treatment for both acute and persistent pain. Because it is thought to have less of an effect on blood pressure than non-steroidal anti-inflammatory medications (NSAIDs), paracetamol is frequently regarded as a safer option for hypertensive persons. However, there is conflicting and inadequate data regarding how paracetamol affects blood pressure.

Patients who use paracetamol have a greater risk of incident hypertension than those who do not, according to earlier observational studies, and this risk appears to increase with frequency of use. In contrast, Kurth et al. found no increased risk in paracetamol users. Nonetheless, the interpretation of causal relationships is constrained by the observational nature of these investigations.<sup>v</sup>

### **COX-2 Inhibitors and Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)**

Non-steroidal anti-inflammatory medicines (NSAIDs) are known to raise blood pressure, especially in patients with hypertension. NSAID use was linked to a 3.3 mmHg increase in mean arterial pressure in 1324 patients (mean age 46, 92% hypertensive) according to a meta-analysis by Pope et al. Naproxen and indomethacin were linked to the most increases in blood pressure (+3.7 and +3.6 mmHg, respectively), while ibuprofen, piroxicam, and aspirin had very little presser effects. Similar findings were reported in a meta-analysis by Johnson et al, which indicated an increase in mean arterial pressure of 5 mmHg in hypertensive patients taking NSAIDs. Comparing piroxicam to a placebo, the drug caused the greatest increase in blood pressure (+6.2 mmHg), whereas aspirin had the least amount of an impact.<sup>vi</sup>

### **Opioids**

The majority of the data that is currently available on the hemodynamic effects of opioid medications relates to acute intravenous delivery during anesthesia or postoperative analgesia. In this situation, opioids may have pertinent cardiovascular side effects, such as bradycardia and hypotension, especially if benzodiazepines are being used at the same time. Hypotension, orthostatic hypotension, and syncope are frequently described as potential side effects of most opioid analgesics, including morphine, buprenorphine, fentanyl, oxycodone, and tapentadol. Data on long-term opioid treatment are scarce. Still up for discussion, though, is the mechanism behind opioid-mediated hypotension.<sup>vii</sup>

### **Adjuvant Painkillers**

Adjunctive therapy with antidepressants is becoming more and more common for chronic pain, especially in neuropathic pain, migraine, and fibromyalgia patients. Antidepressants are known to affect blood pressure (BP), however the effects vary depending on the drug class.

Serotonin-norepinephrine reuptake inhibitors (SNRI) and tricyclic antidepressants (TCA) have historically been linked to an elevated incidence of incident hypertension and are thought to have hypertensive effects. At supratherapeutic dosages, the BP effects of SNRI appear to be dose-dependent and more significant. That is demonstrated by the fact that some studies found no appreciable changes in blood pressure in patients receiving therapeutic doses of duloxetine or venlafaxine.<sup>viii</sup>

Anticonvulsants are a valuable adjunct to antidepressants in the treatment of pain, especially in those with fibromyalgia and neuropathic pain. The molecules having the most evidence for treating chronic pain are gabapentinoids, such as gabapentin, but carbamazepine works best for treating trigeminal neuralgia. Recent research has shown that gabapentin can cause bradycardia and vasodepression by activating the nitric oxide pathway. It is in fact known that gabapentin reduces the hypertensive reaction to tracheal intubation and laryngoscopy.

Given that chronic pain appears to have a negative impact on blood pressure management, it makes sense that analgesic pain alleviation would promote blood pressure control in hypertensive patients. As a result, those on efficient analgesic therapy ought to exhibit improved blood pressure control when compared to those with untreated or inadequately managed chronic pain. However, the previously mentioned direct effects of analgesics on blood pressure may function as a confounding factor, making it challenging to determine whether pain management helps hypertensive people's blood pressure control. Nevertheless, there is a dearth of precise evidence and the BP effects of pain treatment have not been studied to date.

Blood pressure and pain seem to be closely connected. Based on current data, blood pressure readings may be clinically significantly destabilized by both pain and analgesic medications. Future research should examine the ambiguous implications of this on blood pressure regulation and the incidence of hypertension.

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