# Toothache of Cardiac Origin

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*Pain referred to the orofacial structures can sometimes be a diagnostic challenge for the clinician. In some instances, a patient may complain of tooth pain that is completely unrelated to any dental source. This poses a diagnostic and therapeutic problem for the dentist. Cardiac pain most commonly radiates to the left arm, shoulder, neck, and face. In rare instances, angina pectoris may present as dental pain. When this occurs, an improper diagnosis frequently leads to unnecessary dental treatment or, more significantly, a delay of proper treatment. This delay may result in the patient experiencing an acute myocardial infarction. It is the dentist's responsibility to establish a proper diagnosis so that the treatment will be directed toward the source of pain and not to the site of pain. This article reviews the literature concerning referred pain of cardiac origin and presents a case report of toothache of cardiac origin.*

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Many different types of pain disorders are felt in the orofacial structures. The clinician managing orofacial pain must always be aware of the many possibilities so that a proper diagnosis is established. Too often an improper diagnosis leads to ineffective and unnecessary therapy. This is the greatest challenge for the clinician. One of the most common pain complaints routinely seen in the dental office is toothache. Toothaches of dental origin, such as pulpal and periodontal, are routinely managed well in the dental office. Some toothaches, however, are nonodontogenic, meaning that although the patient reports pain in the tooth, the actual source is not dental but instead originates from another location. These types of pains are called *heterotopic pains*. Heterotopic pains need to be identified before treatment is begun, since the success of a therapy depends upon locating the true source of the pain, not the site of the pain. One possible source of heterotopic toothache is cardiac pain. This type of pain needs to be identified immediately, not only to avoid inappropriate dental therapy, but also to ensure that the patient is referred to the proper health care professional for appropriate therapy. This paper will highlight the pathophysiology of cardiac pain referral and the clinical presentation of toothache of cardiac origin.

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## The Pathophysiology of Cardiac Pain

In recent years, researchers have tried to better understand the origin and mechanisms that underlie cardiac pain, such as angina pectoris, and the common complaints of associated pain referral. The contributions of cardiac nociceptors, chemical mediators, afferent pathways, and central neural mechanisms are some of the most important topics that have been investigated. Although the underlying mechanisms of cardiac pain are still poorly understood, most data suggest that ischemia of the heart muscle is the main cause of cardiac pain.[1–3] In experimental conditions, coronary artery occlusion activates cardiac afferent nerves.[4–6] The peripheral biochemical changes that occur during cardiac ischemia include alterations in tissue concentrations of bradykinin, serotonin, adenosine, potassium, and prostaglandins.[7–11]

It is still unknown which specific chemical mediators released during coronary occlusion are responsible for evoking painful sensations. Attempts to identify a single mediator causing nociceptive stimulation have failed[3]; therefore, it is likely that multiple nociceptive mediators are released simultaneously. Bradykinin is believed to be the most important mediator of cardiac pain.[12,13] Veelken et al[14] showed that cardiac bradykinin elicits a sympathoexcitatory reflex, supporting the idea that this chemical mediator may increase sympathetic activity during cardiac ischemia. It is interesting to note that bradykinin alone does not produce pain in experimental animals,[15–17] suggesting that it may play only a supportive role in cardiac pain.

Serotonin has also been studied as an important chemical mediator of cardiac pain. Intracoronary injections of serotonin in lightly anesthetized dogs cause pseudo-affective responses indicative of pain.[18] Serotonin blood concentrations increase after experimental coronary occlusion or after angina attacks,[19] suggesting its involvement as a mediator of cardiac pain. There is evidence that adenosine is also a mediator in angina. Coronary sinus concentrations of adenosine are elevated following myocardial ischemia, and after intravenous administration of adenosine, angina pectoris–like pain was reported in healthy volunteers.[20,21] The existing data suggest that this mediator can sensitize afferent cardiac nerves during painful cardiac ischemia.[22] Potassium has also been shown to participate in angina pectoris mechanisms. Extracellular potassium concentration in myocardial tissue increases rapidly after cardiac ischemia.[23] However, the potassium release is often within physiologic ranges during ischemia and may not be sufficient to induce angina.

It is widely accepted that the pathway of cardiac nociception produced by ischemia is by way of the cardiac sympathetic afferent system.[24–26] However, the idea that only sympathetic fibers are responsible for this transmission has been challenged by several reports. These studies have shown that only 50 to 60% of patients who underwent sympathectomy reported complete relief from angina pectoris, while 40% reported partial relief and 10 to 20% reported no relief at all.[3]

Vagal afferent involvement in pain transmission from the ischemic heart is not clear, but a review of clinical and experimental reports[27–29] suggests that it can play an important role. After consideration of the anatomic distribution of the sympathetic and vagal fibers, it has been speculated that angina may be primarily the result of sympathetic afferent activation in the anterior surface of the heart, while vagal afferent could be activated on the inferior-posterior surface.[3]

Angina pain is a common clinical manifestation of coronary disease. Myocardial ischemia, however, should not be considered synonymous with angina pectoris.[30] More than 25% of myocardial infarctions may be asymptomatic, and in some cases "silent" infarctions are detected only by pathologic findings on a routine electrocardiogram (EKG).[31] Patients who experience "non-silent" acute myocardial ischemia may present with a variety of clinical symptoms.

## Cardiac Pain Referral

The central processing of nociception arising from the heart during ischemia involves complex mechanisms. Several reports have shown that cardiac afferents and somatic inputs from the upper limbs, chest, and face converge on spinothalamic tract neurons in the central nervous system (CNS).[13,32,33] Convergence mechanisms and central sensitization in the trigeminal complex have also been suggested as an explanation for the referral of pain to the orofacial structures (Fig 1). As cardiac nociceptive input enters the CNS and ascends to the higher centers for evaluation and interpretation, adjacent central neurons in the region of convergence can also become activated. This results in the stimulation of these adjacent nociceptive neurons, which are not directly involved with the primary source of pain. As the information ascends, the cortex can misinterpret this data as pain in another site (heterotopic pain). Thus, pain can be felt in the region

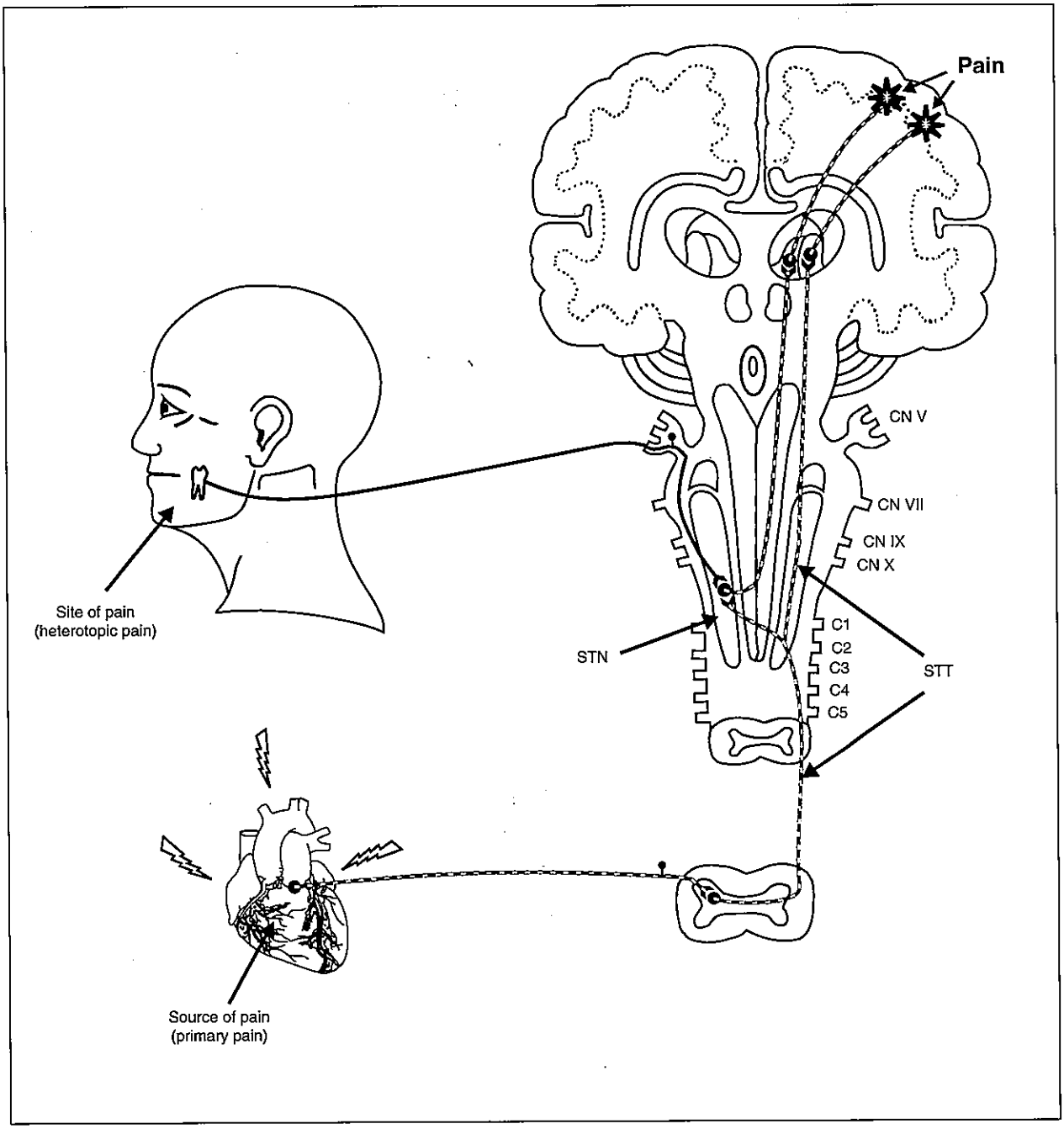



Fig 1 This figure simplistically illustrates a possible mechanism responsible for pain referral from cardiac ischemia to a tooth. Nociceptive input originates in the cardiac muscle. This input is carried by the primary visceral afferent neurons to the CNS, and then the input ascends by way of the spinothalamic tract (STT). Note that when the input passes by the spinal tract nucleus (STN), it converges on interneurons in the medullary dorsal horn of the spinal tract nucleus. This convergence centrally excites second-order trigeminal neurons that normally carry sensory input from a tooth. As this information ascends to the cortex, it is perceived by the cortex as pain felt coming from the tooth. The tooth represents a site of pain (heterotopic pain), while the primary source of the nociception is the cardiac muscle (primary pain). CN = cranial nerve. Adapted from Okeson[36p72] with permission of the publisher.

of the mandible secondary to cardiac ischemia. More specifically, as depicted in this article, pain can actually be felt in the region of a tooth when the primary source of nociception is the cardiac muscle.

Deep pain input from visceral structures has been demonstrated to converge in specific lamina of the dorsal horn and trigeminal spinal tract nucleus, with other primary afferent neurons carrying input from trigeminal structures.[34] This convergence of input leads to the possibility that nociceptive input from visceral structures, such as the heart, can lead to pain referral in the trigeminal region.

There is still scientific debate regarding the peripheral and central neural mechanisms involved in referred pain arising from the heart. Sympathetic fibers are clearly involved, but the role of the parasympathetic system is not as clear. Cardiac inputs enter the CNS through the spinal cord and terminate mainly in the thoracic dorsal horn, where they synapse with spinothalamic tract neurons. The information is then projected to the thalamus. Convergence mechanisms into the trigeminal brain stem complex and/or in the thalamus can explain referred pain to the face. In anginal occipital headache, it has been proposed that thoracic input ascends via Lissauer's tract to converge with the upper cervical input.[35]

Cardiac pain can be referred to the trigeminal region without the typical clinical presentation of angina pectoris. In some rare instances, jaw and/or tooth pain can be the only clinical complaint. In these situations it is the dentist's responsibility to establish the appropriate diagnosis and immediately refer the patient to the appropriate health care professional.

## Clinical Characteristics of Cardiac Pain Referred to the Orofacial Structures

The clinician should always assess the patient's pain complaint by evaluating the pain location and its quality, intensity, and duration. The clinician should also consider any aggravating, relieving, and radiating factors.[36] Typically, cardiac pain is localized in the sternal region and left side of the chest. The pain can frequently radiate to the neck, left arm, shoulder, jaw, teeth, eyes, and head.[37,38]

The duration of the attack can vary from a few minutes to 1 or 2 hours. The primary precipitating factor is usually physical effort, but the patient may not associate the pain with any cardiac source. Rest and sublingual nitroglycerin tablets are the most common relieving factors.[37] The presence and intensity of pain are variable and do not indicate different levels of the disease. In fact, the cardiac hemodynamic and mechanical changes experienced by individual patients during both symptomatic and asymptomatic episodes can be quite similar.[39]

Several reports[40–45] have demonstrated a clinical relationship between cardiac ischemia and orofacial pain. Toothache, mandibular pain, ear pain, and headache are the most commonly related symptoms. Tzukert et al[40] reported 3 cases in which orofacial pain was the initial chief complaint. A 56-year-old woman complained of bilateral sharp pain in the anterior maxillary area that radiated to the infraorbital region, neck, and shoulder. Clinical examination showed no evidence of oral or dental pathology. Marked dyspnea alerted the clinician to the possibility of cardiovascular involvement, and the patient was referred to a cardiologist. During a stress test, the patient developed the facial pain complaint associated with EKG abnormalities. Coronary obstruction was demonstrated with an arteriographic examination. A coronary bypass procedure was performed, and the facial pain complaint was resolved.

The second patient reported by Tzukert was a 79-year-old man who complained of severe pain in the jaw. The clinical history also revealed episodes of chest pain. The facial pain responded immediately to sublingual administration of isosorbide dinitrate. An EKG showed evidence of acute inferolateral myocardial infarction. The last case was that of a 67-year-old man with bilateral paroxysmal jaw pain. The pain was associated with physical effort. It usually appeared when he walked back to work after lunch. The patient described no other complaints, and no source of facial pain was identified. A cardiologist treated the patient with propanolol and nitrites, which resolved all facial pain symptoms.

Batchelder et al[41] reported a case of anginal pain limited to the mandible, in which a misdiagnosis resulted in unnecessary dental treatment and a delay in appropriate management. This was a 71-year-old male who complained to his dentist of dental pain. The pain was initially diagnosed as odontogenic, secondary to pulp disease in a mandibular first premolar. Endodontic treatment was performed, but the pain did not resolve. The patient was then referred for diagnostic consultation, which revealed a history of chest pain and possible cardiac involvement. The patient also reported left shoulder and clavicular region pain that was relieved by rest. An EKG showed severe

injury to the anterior wall and ischemia when compared with an EKG taken 1 year earlier. Coronary angiography showed 90% occlusion of the left anterior descending coronary artery. The patient reported that nitroglycerin medication took away the pain.

In a report by Penarocha Diago et al, 2 cases of left mandibular pain were presented.[42] Both patients were finally diagnosed as suffering from ischemic cardiopathy, which was referring pain to the face.

Headache and ear pain can also be correlated with coronary disease and should be included in the differential diagnosis. Takayanagi et al[43] reported 2 fatal cases of angina pectoris in patients who complained primarily of headache. Grace et al[35] reported a case of angina manifesting as vertex and occipital headache provoked by exercise and relieved by rest. After bypass surgery the patient became pain-free. Ishida et al[44] reported a 64-year-old male who complained of headache, without chest pain, at the onset of a myocardial infarction. The authors concluded that the headache was due to referred pain rather than a generalized vasospastic disorder. At this time, the underlying mechanisms of headache related to myocardial ischemia are still not clear. Rothwell[45] reported 2 cases in which cardiac ischemia presented with pain confined to the ear. In both cases the diagnosis was missed and the treatment delayed.

## Case Report

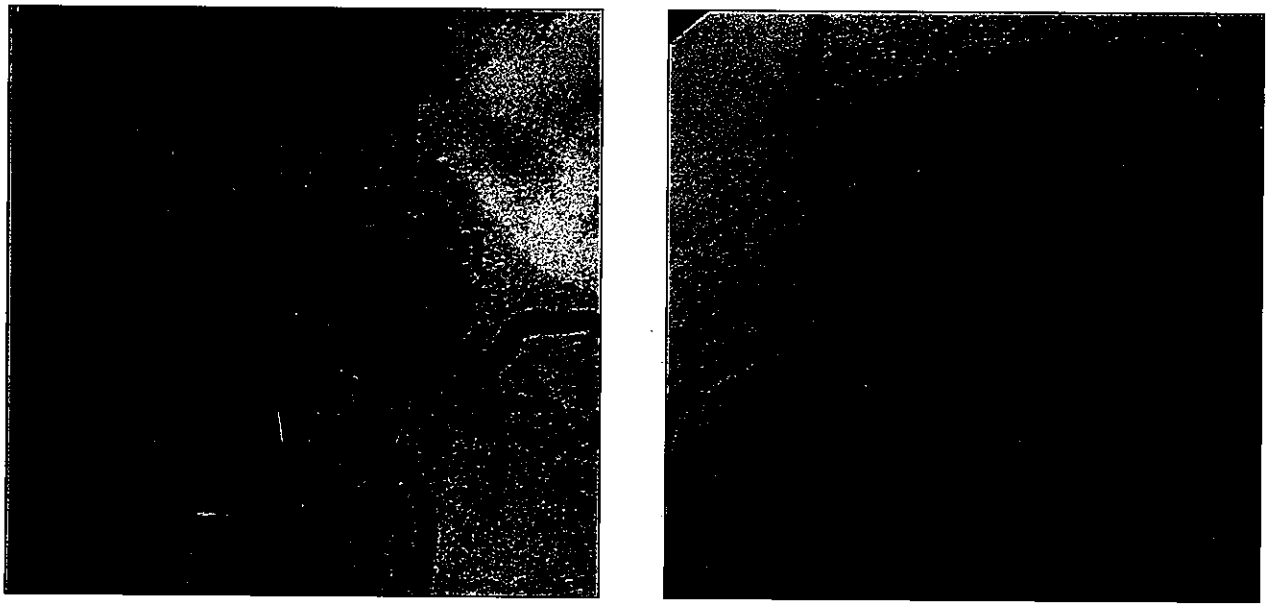
A 63-year-old man reported to the dental office with a complaint of moderate to severe pain in the left mandibular molar and premolar dentoalveolar area. The pain was episodic and had a consistent duration of 1 to 2 hours. The pain occurred every day in the evening and tended to decrease in intensity when the patient went to sleep. There was no pain upon awakening, and the patient remained pain-free until the next evening. In the same location as the pain was a periodontally compromised fixed partial denture with mobility. The local dental conditions made the diagnosis more difficult, since a local source of pain was certainly possible (Fig 2). Although dental pain was the chief complaint, upon questioning, the patient revealed a recent history of thoracic pain that irradiated to the left arm. The patient was immediately referred to a cardiologist, who performed an EKG. The result of this test was normal. Since the clinical characteristics of the pain were atypical for dental or periodontal sources and the recent history of possible angina pectoris had been reported, no dental therapy was performed immediately. Instead, the patient was asked to report to the emergency unit of the hospital the next time he felt any chest, mandible, or tooth pain. During the next pain episode, cardiologic examination revealed EKG abnormalities. Subsequently, angiographic studies were performed and revealed coronary occlusion. An angioplastic procedure was performed, which resolved both the coronary occlusion and the tooth pain (Fig 3).

Fig 2 Periapical radiograph of the region reported by the patient as painful. The periodontal condition and cantilever fixed partial denture add suspicion to a possible dental diagnosis. The history and clinical characteristics of the pain, however, did not confirm a dental etiology.

## Conclusions

The mechanisms underlying referred pain arising from the cardiac muscle are still not well understood. On occasion, ischemic heart disease can be felt primarily as an orofacial pain complaint. When this occurs, it poses a diagnostic challenge for the clinician. An understanding of the pathologic mechanisms and clinical features of cardiac pain and referred pain is essential for a correct diagnosis. An improper diagnosis can lead to unnecessary dental treatment or, even more significant, a delay of proper treatment that may lead to an acute myocardial infarction. The clinician must be able to differentiate the site of pain from the source of pain so that treatment will be properly directed toward the source of pain.[36]

Angina pectoris typically presents as substernal pain radiating to the left arm, shoulder, and/or neck. When this occurs, a differential diagnosis is



Figs 3a and 3b Photographs that reveal the patient's coronary arteries as depicted in angiograms. (*Left*) Angiogram before the angioplasty. Note the marked constriction of the coronary vessel before the procedure (*arrow*). (*Right*) Angiogram after the angioplasty. Note the return of the vessel to its normal size (*arrows*).

not extremely difficult. However, occasionally, a patient with angina may present with orofacial pain as the main complaint.[35,41,43,46] In such cases, a thorough history is the major clue in establishing the proper diagnosis. The quality of pain and its location, duration, intensity, and precipitating and ameliorating factors are the main clues that will lead to the correct diagnosis. A complete review of systems is also helpful.

When toothache is the primary pain complaint, local anesthesia of the tooth is often a helpful diagnostic aid. When profound anesthesia of the tooth is achieved without a reduction in pain, pain referral should be suspected. One possible source of this referral is the cardiac muscle. In a potential cardiac patient, local anesthetic without a vasoconstrictor should be used. Often the diagnosis can be confirmed by the development of pain concurrent with hemodynamic and electrocardiographic changes.[40] Sublingual administration of nitroglycerin can also be used as a diagnostic test, but this is best done by the cardiologist. The patient's age and sex are also important considerations, since cardiac pain is most common in males 50 to 70 years old. These conditions, however, can occur in younger patients.[46] When the clinician suspects that a toothache is of cardiac origin, the patient should be immediately referred for appropriate medical care.

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