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The clinical use of cocaine in rhinosurgery: A case-report and a review

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SUMMARY

Three patients with adverse reactions of the nasal mucosa after topical anaesthesia with crystalline cocaine in combination with adrenaline are described. The current knowledge of the pharmacology of cocaine is summarized. The systemic and local reactions and side-effects of cocaine as topical anaesthetic in rhinosurgery are reviewed.

CASE REPORTS

In 1988 three cases of serious toxic reactions of the nasal mucosa after local anaesthesia with topical applied crystalline cocaine in combination with adrenaline were observed in our department.

All three patients were young, healthy adults (age: 22–33 years) with serious complaints of persistent nasal obstruction caused by a deviation of the cartilaginous septum. None of the patients had a positive history of atopic diseases. In all three cases reconstructive surgery of the cartilaginous septum was performed under local anaesthesia by three different surgeons. Topical anaesthesia was obtained by 210 mg cocaine crystals on cotton-wool applicators, which were soaked in a solution of adrenaline 1 : 1000. The applicators were placed in the nasal cavity at the pterygopalatine foramen, the anterior ethmoidal nerves and the pterygopalatine nerves. One to three days postoperatively a progressive tenderness of the nose had developed. Examination of the internal nose showed an extremely swollen septal mucosa with an intense pale and edematous appearance. Locally a tendency to necrosis of the mucous membrane was found. The body temperature was normal to subfebrile. Routine laboratory investigations did not show any abnormalities. After re-opening of the hemitransfixion no signs of a septal haematoma or septal abscess were found. All patients were treated for several days with nasal packs soaked in Terracortril® suspension. Systemic antibiotics were prescribed to prevent secundary infection. Within three to six weeks postoperatively complete recovery of the septal mucous membrane was observed. The long-term postoperative results were satisfactory in all three patients.

Retrospective pharmacological analysis of the applied cocaine crystals revealed no abnormalities explaining the complications. An extremely potentiative interaction between the topical applied cocaine in combination with adrenaline could be the etiological factor responsible for the toxic reactions of the nasal mucous membrane, although the incidence rate of these complications is very rare.

Cocaine is an alkaloid which occurs in the leaves of Erythroxylon coca, a tree indigenous to Peru and Bolivia. For centuries coca leaves have been chewed by South American Indians to reduce hunger and to increase work tolerance and resistance to the cold. The active alkaloid was first isolated from the coca leave by Niemann in 1857. The first clinical use of cocaine in medicine was reported in 1884 by Karl Koller (a colleague of Sigmund Freud), who introduced cocaine into ophtalmology as a topical anaesthetic. In 1947 Moffett described a method of providing local anaesthesia of the nasal mucosa using a solution of cocaine and adrenaline.
Until today cocaine has been widely used in otorhinolaryngology as a topical anaesthetic because of the excellent anaesthetic properties as well as the strong vasoconstriction and decongestion of mucous membrane surfaces (Huizing et al., 1973; Schenck, 1974; Johns, 1977; Pearman, 1979; Verlander et al., 1981; Hashisaki et al., 1987; Jonathan et al., 1988). In this article the pharmacological properties and the possible toxic reactions of cocaine as a local anaesthetic in rhinosurgery are reviewed.

PHARMACOLOGY

Cocaine (benzoylmethylecgonine) is an alkaloid prepared from the leaves of Erythroxylon coca. The cocaine alkaloid is a colourless, transparent crystalline substance, which is insoluble in water. Cocaine hydrochloride is a water-soluble salt, which is available for medical application.
The local anaesthetic action of cocaine is due to a direct effect on the nerve cell membrane (Hertting, 1961; Goodman et al., 1975; Verlander et al., 1981; Cregler et al., 1986). Cocaine blocks the generation and conduction of the nerve impulses by preventing the transient increase of the permeability to sodium ions during depolarization. Cocaine is the only local anaesthetic that is known to interfere with the uptake of noradrenaline by the adrenergic nerve terminals. Cocaine also prevents the uptake of exogenously administered adrenaline. The blockage of the uptake of noradrenaline leads to increased levels of circulating catecholamines and produces sympathetic nerve stimulation (vasoconstriction, mydriasis, tachycardia).

ABSORPTION

Application of cocaine to intact skin has no anaesthetic effect (Goodman et al., 1975). Systemic absorption does occur when cocaine is applied to mucosal surfaces. Absorption was found to be most rapid from the tracheal mucosa (Campbell et al., 1958). A survey of otorhinolaryngologists in the United States showed more toxic reactions after the application of cocaine to the tracheobronchial mucosa then to any other mucous membrane (Johns et al., 1977). After intranasal administration of 1.5 mg/kg of cocaine serum levels of the drug persist in the circulation for four to six hours (Van Dyke et al., 1976).

Adrenaline is frequently added to solutions of cocaine to intensify the vasoconstriction and thus limit the absorption of cocaine in the systemic circulation. The effectiveness of this vasoconstrictor effect is uncertain. In a study of Campbell and Adriani (1958) topically applied cocaine to the pyriform sinus showed no significant difference in the absorption of cocaine regardless of the presence of adrenaline. Bromley and Hayward (1988), however, found that the absorption of cocaine is significantly reduced by the inclusion of adrenaline 1 : 1000 in solution applied to the nasal mucosa. The combination of adrenaline and cocaine may intensify the shrinkage of mucous membranes and may increase the possibility of hypertension and cardiac arrhythmias (Schenck, 1975; Verlander et al., 1981).

SYSTEMIC EFFECTS OF COCAINE

Central Nervous System

The central nervous system is stimulated by cocaine from above downwards (Goodman et al., 1975; Verlander et al., 1981; Cregler et al., 1986). The first manifestations are garrulousness, euphoric excitement and restlessness, followed by tremors and convulsive movements. Increase of the respiratory rate and stimulation of the vomiting centre occurs as the medulla is affected. The stimulation of the central nervous system is followed by depression, eventually resulting in death from respiratory failure.

Sympathetic Nervous System

The sensitization to catecholamines by cocaine produces activation of the sympathetic nervous system resulting in vasoconstriction, mydriasis and tachycardia (Goodman et al., 1975; Verlander et al., 1981).

Cardiovascular System

Small doses of cocaine given systemically produce bradycardia as a result of central vagal stimulation. Moderate to large doses cause an increased cardiac action by sympathetic stimulation as well as a central effect. The increased cardiac rate in combination with the vasoconstriction causes a prominent rise of the blood pressure. Cardiac arrhythmias may occur after cocaine application including ventricular premature contractions, ventricular tachycardia and fibrillation, and asystole. A case of myocardial ischemia following the application of cocaine and adrenaline during septoplasty is reported in literature (Littlewood et al., 1987). Large doses of cocaine given intravenously have a direct effect on the heart muscle, which may result in an immediate death from cardiac failure.

Gastro-Intestinal System

A dose of oral cocaine is at least as effective as intranasally applied cocaine. Oral administration of large doses of cocaine may induce intestinal ischemia and gangrene of the bowel (Nalbandian et al., 1985).

Body Temperature

Cocaine causes a marked increase in body temperature by peripheral vasoconstriction, increased muscle activity and stimulation of the heat-regulating centre (Goodman et al., 1975; Verlander et al., 1981).

LOCAL EFFECTS OF COCAINE ON THE NASAL MUCOSA

Habitual sniffing of cocaine in powder form is known to cause rhinitis, nasal septal ulcerations and perforations (Messinger, 1962; Gollom, 1968; Van Epen, 1981; Vilensky, 1982; Rutka et al., 1984). The septal pathology is the result of mucoperichondrial necrosis and ulceration due to the intense vasoconstriction. Mechanical irritation of the nasally applied cocaine and stasis of mucociliary activity may potentiate the septal changes (Rutka et al., 1984; Ukai et al., 1985). Total necrosis of the cartilaginous septum with saddle-nose deformity and osteolytic sinusitis due to cocaine abuse has been described (Schweitzer, 1986). Nasal insufflation of cocaine may result in massive necrosis of midline structures of the upper respiratory tract simulating midline granuloma (Becker et al., 1988). Severe local reactions of the nasal mucosa after intranasal application of cocaine as topical anaesthetic have never been reported in literature. In this report three cases with serious adverse reactions of the nasal mucosa after local anaesthesia with cocaine in combination with adrenaline are described.

DOSAGE

Cocaine is frequently used as topical anaesthetic in rhinosurgery in a 3–10% solution of cocaine hydrochloride salt or as a crystalline cocaine powder of the alkaloid base. In the literature 200–300 mg is regarded to be the maximal safe limit for cocaine as a surface anaesthetic, although this is only based on clinical experience and not on experimental data (Adriani et al., 1964; Huizing et al., 1973; Johns et al., 1977; Pearman, 1979; Verlander et al., 1981).

CONCLUSIONS

Cocaine is a valuable topical anaesthetic in rhinosurgery providing strong vasoconstriction and excellent topical anaesthesia. The incidence rate of complications secondary to the topical application of cocaine to the nasal mucosa is extremely rare (Johns et al., 1977; this report).

The combination of cocaine and adrenaline appears unadvisable for the potentiative interaction between cocaine and catecholamines (Goodman et al., 1975; Johns et al., 1977; Verlander et al., 1981).

Patients with a pseudocholinesterase deficiency or patients treated with cholinesterase inhibitors do not metabolize cocaine as rapidly as normal individuals and therefore are probably more sensitive for cocaine (Verlander et al., 1981; Hashisaki et al., 1987).

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