Systemic Emergencies and their Management in Dentistry: Complications Independent of Underlying Disease

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If a systemic complication occurs in dental practice, the dentist is obligated to make a prompt diagnosis and provide emergency treatment as soon as possible. Therefore, the dentist must be fully aware of medical complications that may occur in dental practice. Emergencies during dental treatment can be classified by etiology into two major groups: complications associated with an underlying disorder and those independent of pre-existing disease. This paper reviews the causes, pathophysiology, symptoms, signs, and emergency treatment of complications unrelated to underlying disease. The main systemic complications include (a) neurogenic shock or syncope; (b) hyperventilation syndrome; (c) local anesthetic intoxication; and (d) allergic reaction to local anesthetics.

NEUROGENIC SHOCK OR SYNCOPE

Neurogenic shock or syncope, also called vasovagal syncope and acute cerebral anemia, is the most common systemic complication in dentistry.[1,2] Some investigators may call it syncope in mild cases and neurogenic shock in severe instances, as the patients manifest signs and symptoms of varying intensity.

Cause and Pathophysiology

Noxious stimuli due to dental intervention, such as the injection of local anesthetics, can provoke the trigeminovagal reflex.[3] This reflex causes transient hypotension and bradycardia by the activation of parasympathetic nerves. If this reflex takes place under intense emotional stress, such as fear and anxiety for dental treatment, these responses could be intensified, and neurogenic shock or syncope may develop.

In the attack, increased vascular capacitance induced by peripheral vasodilatation causes a relative deficiency of circulating blood volume and a decrease in venous return to the heart. A decreased venous return then leads to a decreased stroke volume. The cardiac output is further reduced by bradycardia due to parasympathetic hyperactivity, and hypotension results. With a systolic blood pressure below 50 mm Hg, cerebral blood flow would become insufficient to maintain cerebral functions, and loss of consciousness would ensue.[4]

Although there are still many details to be clarified regarding neurogenic shock, it was suggested in a recent study[5] that hypotension is related to changes in blood flow in skeletal muscle. The blood flow in skeletal muscle is increased by emotional stress through stimulation of the hypothalamic defense area. Sudden reflexive vagal stimulation by pain under this situation could bring about marked reduction of cardiac output and lead to profound hypotension with the additional simultaneous pooling of blood in skeletal muscle.

Symptoms and Signs

Pallor, forehead sweating, nausea, lethargy, weakness, bradycardia, hypotension, and loss of consciousness are progressive manifestations of syncopy.

Treatment

Dental treatment should be discontinued immediately and any foreign objects in the mouth such as gauze and cotton should be removed at once. Vital signs should be checked carefully. At first, pulse check is performed at the radial artery. In checking the pulse, special attention should be paid to the tension of the palpating artery and its rhythm. In neurogenic shock, bradycardia less than 60 beats/min and a weak pulse are commonly seen. If

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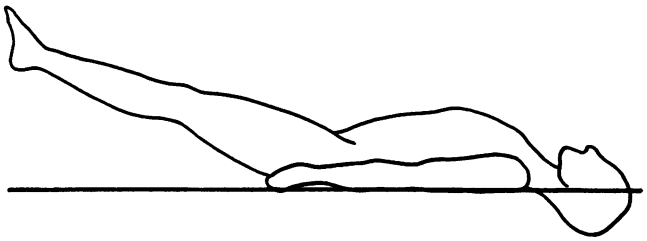

Figure 1. Trendelenburg position (head down, legs elevated).

the systolic blood pressure is reduced to less than 50 mm Hg, pulses are often difficult to palpate.

When hypotension is confirmed, the patient should be placed in the supine position and the legs should be elevated (Figure 1). Oxygen is administered by face mask at a flow rate of 4 to 6 L/min, with the clothes loosened to facilitate ventilation. A face mask with a reservoir bag can provide an inspiratory oxygen concentration of more than 70%. These treatments are usually all that is required. Use of an ammonia inhalant may stimulate ventilation and return the patient to consciousness when the syncope is not immediately reversed by positional change.

If there is no improvement in the patient with these treatments, intravenous access must be established for rapid infusion and drug administration.

Veins in the dorsum of the hand are the preferred initial site of cannulation, because at this site venipuncture can be performed easily even in obese patients, and there are many veins to cannulate (Figure 2). Cannulation of the antecubital vein should be avoided initially because this vein is normally more difficult to find in obese patients. Lactated Ringer's or normal saline solution should be used at an infusion rate of 250 to 500 ml/hr.

For continuing hypotension, 125 to 1000 mg of methylpredonisolone or 2–10 μg/kg/min of dopamine may be given intravenously. For bradycardia, 0.4 to 0.6 mg of atropine is administered intravenously. The same dose is repeated until an adequate response is obtained or a full vagolytic dose of 2 mg is administered.

If loss of consciousness occurs, the upper airway is liable to be obstructed by the sagging tongue. When airway obstruction occurs, an airway-opening maneuver is necessary (Figure 3). For cardiac or respiratory arrest, cardiopulmonary resuscitation must be performed.[5]

Prevention

Emotional and physical stress reduction is most important to prevent this complication. Conscious sedation is indicated by the inhalation, intravenous, and/or oral routes.

In order to diminish the pain due to the injection of local anesthetics, it is recommended to use topical anesthesia at the insertion point and then to anesthetize the tissue just in front of the needle tip by injecting a drip of anesthetic before advancing the needle. During dental treatments, strict attention to adequate local anesthesia should be given to prevent further pain, anxiety, and stress response.

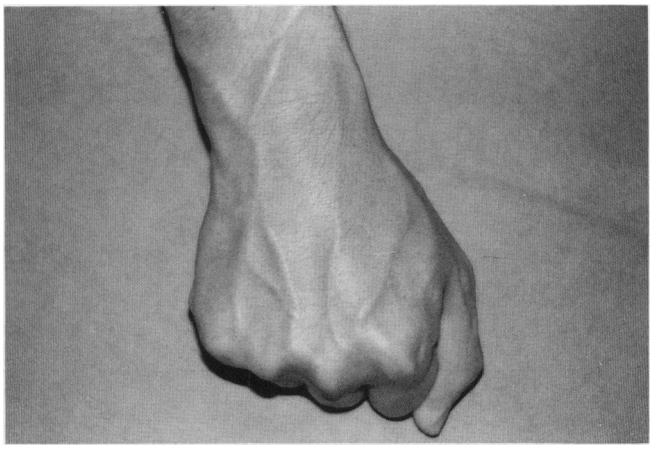

Figure 2. The dorsum of the hand is usually the best site for the establishment of an intravenous infusion.

As the patient with a history of neurogenic shock is characterized by uncontrollable anxiety about the recurrence of the attack, the operative procedures should be as brief and painless as possible. On the other hand, patients can be trained to control themselves and to feel confident in having dental treatments performed. It is a matter of course to perform conscious sedation according to necessity.

Although patients with neurogenic shock can improve easily and in most cases the condition rarely results in a fatal outcome, it may produce dangerous responses in a medically compromised patient. Sugiyama et al[7] reported severe hypotension and bradycardia by neurogenic shock in the patient with essential hypertension who has received beta-blocker and calcium channel blocker therapy. Therefore, the dentist has to pay proper attention to prevent neurogenic shock or syncope.

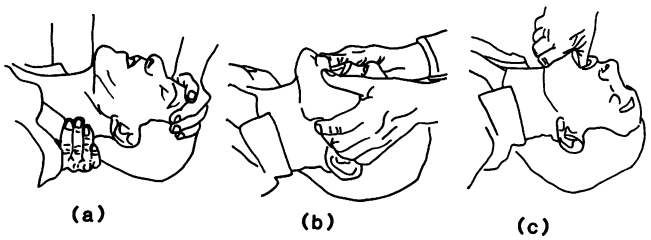

Figure 3. Airway opening maneuver (a) backward tilt of the head with neck lift; (b) jaw thrust; (c) jaw lift.

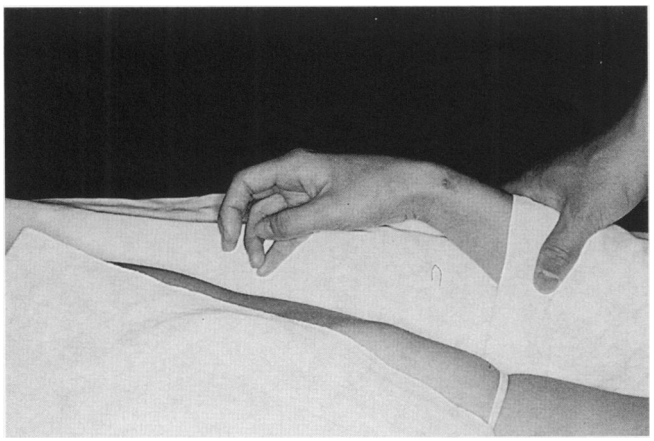

Figure 4. Obstetrician's hand.

HYPERVENTILATION SYNDROME

Hyperventilation syndrome is commonly seen during dental treatment. This syndrome seems to occur more in female than in male patients.[8,9] Although it has been said that the highest incidence of this syndrome occurs in the second decade of life, in our recent study the highest incidence proved to occur in middle age.[8]

Cause and Pathophysiology

Intense emotional stress, such as hysterical anxiety about dental treatment, gives rise to deep and rapid breathing. Increased respiratory minute volume leads to a fall of the arterial carbon dioxide level. Consequently, the blood pH rises and respiratory alkalosis develops. Because respiratory alkalosis can affect the availability of oxygen to the brain by decreasing the cerebral blood flow from cerebral vasoconstriction, dizziness and fainting occur as a disturbance of the central nervous function. Paresthesia and numbness in the lip and the extremities occur as a disturbance of the peripheral nervous function. Muscular rigidity, hypocalcemic tetany, and convulsion develop as a muscular disturbance. The first sign of tetany is adduction of the thumb toward the palm of the hand, which is called "obstetrician's hand" (Figure 4). In addition to these manifestations, several cardiovascular and gastrointestinal disturbances usually occur. Cardiovascular disturbance is manifested by palpitation, chest oppression and chest pain, and gastrointestinal disturbance by stomach ache and abdominal inflation.

Because patients with hysteric anxiety are under the illusion that the air is thin, they breathe more deeply and rapidly. As a result, various symptoms such as paresthesia, muscular rigidity, and dizziness develop. The patient feels more serious anxiety and feels a premonition of death. Thus, the vicious circle is established.

Although hypocapnia is associated with vasoconstriction in the heart and brain, this phenomenon itself is not a life-threatening event. However, the dentist may well interpret it as a grave situation—acute myocardial infarction for example—because the complaints of dyspnea, suffocating sensation and chest pain are so striking. We should keep our presence of mind and watch the patients' vital signs.

Treatment

Dental treatment should be discontinued immediately and any foreign objects in the mouth such as gauze and cotton must be removed at once. The patient should be placed in a semi-sitting position and the clothes should be loosened. The dentist should instruct the patient to breathe slowly or to hold his or her breath. In order to reassure the restless patient, the dentist should explain that the patient's condition is not life-threatening. This treatment is effective in most cases, but if it is ineffective, rebreathing in a paper bag will quickly reverse the symptoms, as this will elevate the arterial carbon dioxide level. Although this method is theoretically likely to be effective, it is not actually always successful.[9] In the patient with intense anxiety and serious difficulty in breathing, masking the mouth with a paper bag may further intensify his or her anxiety. In extreme cases, the patient may refuse this treatment or become angry.

In such cases, intravenous diazepam reverses the symptoms rapidly. An intravenous tranquilizer is recommended. However, we must be careful about intravenous diazepam because it is often accompanied by angialgia and phlebitis.

Prevention

Stress reduction and reassurance are important for the prevention of hyperventilation syndrome as well as that of neurogenic shock. Intravenous sedation with tranquilizers is especially effective.

LOCAL ANESTHETIC INTOXICATION

Cause and Pathophysiology

Local anesthetic intoxication arises when the levels of local anesthetics in the blood rise too rapidly. The toxic effect of local anesthetics is primarily directed to the central nervous system and the cardiovascular system. Several symptoms and signs occur as disturbances in each system.

Because the site of injection is vascularly rich in dental anesthesia, the rate of absorption of local anesthetics is rapid. Therefore, local anesthetic intoxication may oc-

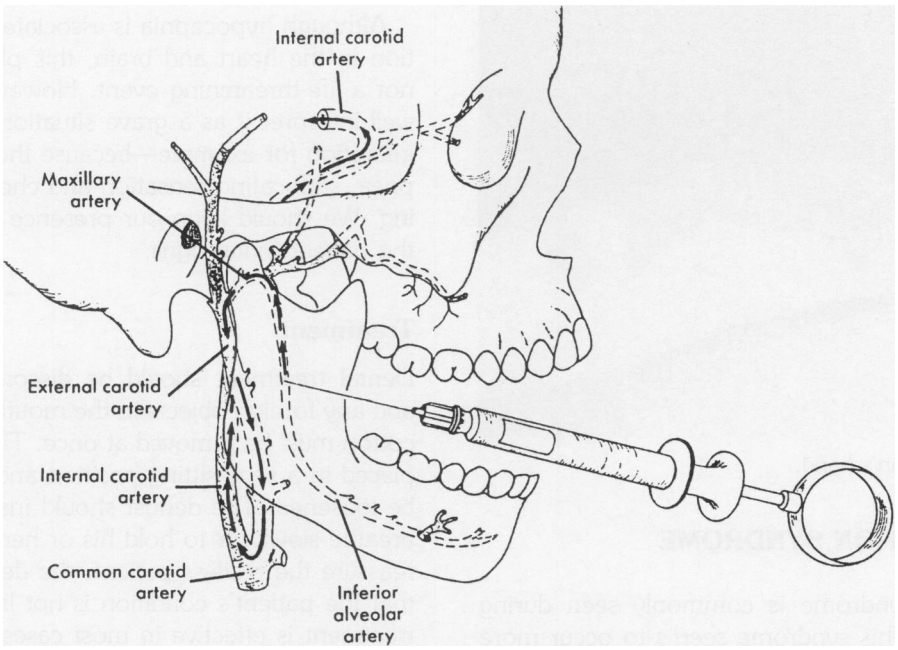

Figure 5. Proposed retrograde flow of anesthetic solution after accidental injection into the inferior alveolar artery.

cur at a low dose of anesthetic. Intravenous injection of local anesthetics often leads to toxic reaction.[10] Local anesthetic intoxication seems to occur more often in block anesthesia than in infiltration anesthesia. If local anesthetics are mistakenly injected rapidly into the inferior alveolar artery in block anesthesia for mandibular foramen, they could flow back to the external carotid artery and the common carotid artery, finally reaching cerebral circulation through the internal carotid artery[11,12] (Figure 5). In such cases, even small doses of local anesthetic could be enough to give rise to a toxic level in the central nervous system.

Symptoms and Signs

When plasma concentration of lidocaine exceeds 5 to 10 μg/ml, the toxic effects are reported to be present in central nervous and cardiovascular systems.[13]

Central Nervous System (CNS) Intoxication. Generally, excitatory responses of CNS occur as early signs of intoxication. The patient becomes loquacious, restless, and excited. In addition, the patient speaks quickly, stutteringly, and inarticulately. In the meantime, muscular twitching ensues in the face and extremities, leading to convulsion. In an advanced CNS intoxication, loss of consciousness, coma, and respiratory arrest could take place as a result of CNS depression.

Cardiovascular System (CVS) Intoxication. Local anesthetics suppress myocardial contractility and relax vascular smooth muscle as a direct action. While these responses can be indirectly countered through the central stimulation of autonomic nervous system, the net effect on CVS is an increase in blood pressure and heart rate. In advanced intoxication, depression of myocardial contraction and peripheral vasodilatation become marked, leading to vascular collapse and cardiac arrest.

Toxic manifestations appear gradually, within 5 to 10 min after the injection of local anesthetics. However, if local anesthetics are injected intravenously, the severe toxic responses may occur abruptly.

Treatment

When excitatory response of CNS is manifested as an early sign of intoxication, dental treatment should be discontinued immediately and oxygen administered. Intravenous access must be established as soon as possible. It is also important to reassure the restless patient. If excitatory response is marked, intravenous diazepam of 0.1 to 0.2 mg/kg is effective.

If consciousness is lost because of advanced intoxication, airway management is required. For local anesthetic-induced convulsion, the administration of oxygen by bag and mask is necessary. When convulsion interferes with ventilation, the administration of an anticonvulsant drug is indicated. The administration of 0.1 to 0.2 mg/kg of diazepam is effective. The danger of convulsion has much to do with cerebral hypoxia, because cerebral metabolism and oxygen requirement are mark-

edly increased by cerebral hyperactivity.[14] If convulsion interferes with ventilation, cerebral hypoxia could be further exacerbated.

Airway obstruction due to the aspiration of vomitus is one of the likely causes of death during convulsion. If the patient is vomiting, the mouth should be opened, the patient should be placed in a lateral position, and vomitus should be removed with the finger, gauze, or a suction machine. It is also important to prevent bodily injury during convulsion.

For circulatory collapse, the treatment is an elevation of legs, rapid intravenous infusion and vasopressor therapy. Three to 10 μg/kg/min of dopamine is often used as a vasopressor. For bradycardia, 0.25 to 0.5 mg of atropine or 0.5 to 5.0 μg/min of isoproterenol should be given. Large doses of epinephrine may be necessary to support the heart rate and blood pressure.

For respiratory and cardiac arrest, cardiopulmonary resuscitation must be performed.

Prevention

The minimum necessary doses of anesthetics should be used for dental procedures. The vasoconstrictor should be contained to local anesthetics to delay absorption to the neighboring tissues and vessels. Suggested maximum safe dose of lidocaine is 500 mg with epinephrine and 200 mg without vasoconstrictors.[15] Although local anesthetic toxicity depends on the amounts of anesthetics administered, the site of injection, the procedure of injection, and the capacity of each patient, it is more important to keep the fundamental technique—that is, injection should be performed slowly with frequent aspiration for blood.

It is said that the risk of acute intoxication from administering too much drug is relatively low in dental anesthesia and that most true overdoses to local anesthetics in dentistry occur in young children.[16] However, even small doses may cause intoxication in cases of intra-arterial or intravenous injection, and a medically compromised patient is much more sensitive to overdose with local anesthetics.[17] Therefore, the dentist must be completely familiar with local anesthetic intoxication.

ALLERGIC REACTION TO LOCAL ANESTHETICS

True allergic reactions to local anesthetics are extremely rare.[18] Many patients visit our department of dental anesthesiology in order to further examine a diagnosis of allergy to local anesthetics.[19] Most patients have experienced some systemic complication after the injection of local anesthetics in dental treatment or another surgical operation and are often labeled as allergic to local anesthetics. However, after careful examination, the initial diagnosis of local anesthetic allergy proved to be false in more than 90% of the patients.[19] In most cases, the true etiology was found to be a psychogenic adverse response, such as neurogenic shock or hyperventilation syndrome.[19]

Cause

Although the immune system provides a host defense, it can also respond inappropriately and cause life-threatening untoward allergic reaction. Allergic responses are classified into four basic types.[20] In dental practice, the type one reaction, which is known as anaphylactic or immediate type of hypersensitivity reaction, is important. Anaphylaxis is the most severe form of allergic reaction, leading to severe, life-threatening shock. Although anaphylactic reaction to local anesthetics is rare, within a short period of time the symptoms can worsen and cardiac arrest may develop.

True allergy to amide-type local anesthetics such as lidocaine and prilocaine is so rare that most dentists may not encounter it even once in their lifetime. Allergy to ester-type agents is fairly common compared to amide-type agents.[21–23]

Methylparaben is usually contained in local anesthetic solutions as a preservative agent. Some patients, once thought to be allergic to local anesthetics, proved to be allergic to the preservative methylparaben after more careful examination.[24,25]

Pathophysiology

Immunoglobulin E (IGE) is a circulating immunoglobulin and mediates allergic reactions of the immediate hypersensitivity type (anaphylactic response). When an IGE antibody is fixed to a mast cell or basophil combines with the antigen, the resulting complex disrupts the mast cell or basophilic membranes, releasing histamine and the slow reacting substance of anaphylaxis. Histamine produces increased capillary permeability and vasodilatation, which are followed by loss of intravascular fluid into the interstitial space, leading to tissue edema and urticaria. In addition, histamine causes smooth muscle contraction and activates gastric secretion. As a result, circulatory collapse, anasarca, including laryngeal edema, and bronchospasm could ensure.

Symptoms and Signs

Signs and symptoms of anaphylactic reaction are varied and become worse in short order. As an early symptom, the patient complains of chest discomfort and itching in

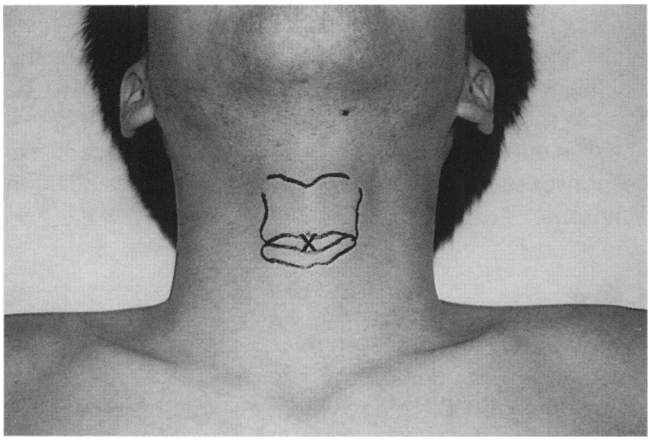

Figure 6. Location of the cricothyroid membrane (X) between the thyroid and cricoid cartilages.

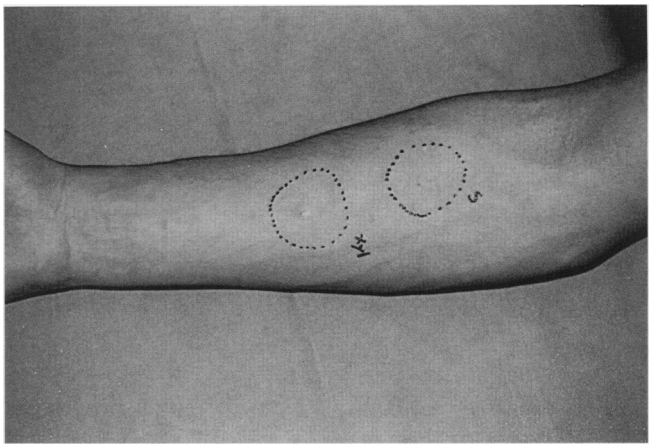

Figure 7. Skin testing.

the face and chest. Flushing and urticaria develop as a sign of cutaneous system irritation. Smooth muscle contraction in the gastrointestinal tract provokes stomachache, vomiting, diarrhea, and fecal or urinary incontinence. Retrosternal oppression and chest pain could be the cardiac manifestation. Wheezing and dyspnea become audible due to bronchospasm. Furthermore, acute airway obstruction occurs in association with laryngeal edema. Palpitation, tachycardia, arrhythmia, hypotension, loss of consciousness, and cardiac arrest would take place successively.

Treatment

Anaphylactic shock can be fatal within several minutes of onset. Therefore, the prognosis depends greatly upon initial treatment in the first 5 min. We should be fully aware of the premonition or early symptoms of anaphylaxis. As soon as anaphylaxis is suspected, the following treatments should be performed.

Position. The patient should be placed in the supine position and the legs elevated (Trendelenburg position).

Airway Maintenance with 100% Oxygen. If the patient complains of dyspnea, an airway patency maneuver should be performed. Assisted or controlled ventilation should be performed with a bag and mask to deliver 100% oxygen. If laryngeal edema develops, endotracheal intubation or tracheostomy is indicated to maintain the airway. In case of emergency, 14-gauge plastic catheters or needles are inserted through the cricothyroid membrane (Figure 6). In the patient with advanced bronchospasm, ventilation is difficult in spite of endotracheal intubation or tracheostomy, and in such cases, the use of a bronchodilator is indicated.

Intravenous Cannulation and Drug Administration. Intravenous access must be established as soon as possible.

Epinephrine is the drug of first choice in anaphylactic shock. At first, 0.3 to 0.5 mg of epinephrine is given intramuscularly. Additional doses of epinephrine are repeated every 5 to 10 min until a desirable response is obtained. Intravenously, 0.1 mg of a 1 : 10,000 solution is given. The effect of epinephrine is bronchodilatation, vasopressor, and inhibition of histamine release.

Antihistamines compete with released histamine at the H1 receptor and block the combination of histamine in the target organ. Intravenous administration of 2.5 to 5.0 mg of chlorpheniramine maleate is recommended.

It is well-known that a large dose of corticosteroid improves peripheral perfusion and inhibits the breakdown of lysosome membrane. For anaphylactic reaction, 500 to 1000 mg of hydrocortisone or methylpredonisolone is given intravenously.

Aminophylline is indicated in patients with persistent bronchospasm. Two hundred fifty milligrams of aminophylline should be given slowly.

For hypotension, 2 to 20 μg/kg/min of dopamine or 2.5 to 20 μg/kg/min of dobutamine is titrated until the desirable effects are obtained.

Hypovolemia rapidly ensues in anaphylactic shock because increased capillary permeability provokes shifts of intravascular fluid into the interstitial space. Therefore, volume expansion is extremely important and 500 to 1000 ml of lactated Ringer's solution should be given rapidly.

Transport. Finally, the patient should be transported to the hospital under strict systemic management.

Prevention

Medical history is extremely important to prevent allergic reactions to local anesthetics. When the patient has a history of adverse reaction to a drug, it is essential to

evaluate whether the reaction is a true allergy and whether the drug can be readministered.

Skin testing is a useful method to confirm specific sensitivity in the patient with a suspected history of local anesthetic adverse reaction (Figure 7). However, reliability of skin testing is questionable because of the possibility of false negative or false positive results.[26,27]

In performing skin testing, strict monitoring must be maintained and resuscitation equipment must be available because the testing creates the risk of producing severe and potentially fatal reactions in patients who are truly allergic.

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