# Accidental Intra-Arterial Injection

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## *INTRODUCTION*

When an animal is given an injection, whether intravenously (IV), intramuscularly (IM), subcutaneously or by any other parenteral route, intra-arterial (IA) injection may occur. Since arteries run parallel to many veins, the chances are greater when IV routes are used.

Reports of IA accidents are infrequent in veterinary and medical literature, but some authors, such as Gabel, mention that many of their colleagues can recall such incidents (7). Because of the legal consequences of IA accidents, it may be assumed that most are unreported and an accurate analysis of their incidence is therefore difficult.

## *DRUGS INVOLVED*

The drugs involved in IA accidents are generally tranquilizers, sedatives, and general anesthetic agents, possibly because these drugs are usually administered IV and are thus being injected near an artery. Veterinary and medical literature lists the following drugs:

1. promazine (7, 19)
2. propiopromazine (7, 19)
3. acetopromazine maleate (7, 19)
4. trifluoropromazine (7, 19)
5. thiopental (7, 19)
6. chloral hydrate (7)
7. chloral hydrate with $MgSO_4$ (7)
8. oxalic and malonic acid (7)
9. calcium gluconate (7)
10. pyribenzamine (7)
11. vitamin $B_{12}$ (7)
12. myanesin (7, 19)
13. ethyl ether (7, 19)
14. tubocurarine (7, 19)
15. meperidine-diparcal (7, 19)
16. stropanthin (7, 19)
17. pentobarbital (7, 19)
18. 2-methoxy-4-allylphenoxy-acetic acid-N, N-diathylamid (7)
19. sclerosing agents (7)
20. ethanolamine (3)
21. sodium iodomethomate (50%) (11)
22. iodopyracet (70%) (11)
23. sodium acettrizoate (50%) (11)

## *TYPE OF REACTION*

Reactions to IA injections range from minor degrees of anesthesia to gangrene, ischemic constricture of the muscles, and death (14). Other writers have reported convulsive reactions, general muscular twitching, and incoordination appearing from a few minutes to an hour after injection. Clonic convulsions may follow in any of these cases (11). If the artery supplies a superficial area, intense local vasospasm leads to a blanching of the skin which may be followed by necrosis. In cases where the subject has been tranquilized, the reaction, if it occurs, is often less severe (7, 12).

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### *FACTORS INVOLVED*

The concentration, volume, rate of injection, and degree of sedation determine the degree of response obtained (4, 6, 14). The concentration is the most critical of these factors. For example, the degree of necrosis in thiopental injection into the ventral artery of a rabbit's ear is proportional to the concentration of the solution (14). Undiluted solutions of radio-opaque compounds have been shown to cause damage to the blood-brain barrier, which increases the toxicity of the compound. Related to the concentration are the volume and the rate of injection. Small volumes and slow injection allow the blood to dilute the chemical before it reaches a toxic level (11).

### *MECHANISMS IN RESPONSE*

The importance of the degree of sedation in determining the response indicates that pain may be a factor. Physiologists making intra-carotid artery injections in anesthetized rats do not mention any response (12). Horses treated with IV promazine do not react to IA promazine injected 1–2 hours later (7). The burning pain along the artery following IA injection of thiopental, acetylcholine, etc. may be due to the caustic action of these materials on the sensory nerve endings which supply the arterial walls (5, 10). The type of tranquilizer and the period of time since its administration affect the response (7).

A second possible cause of the response to IA injection is vasospasm as a result of local irritation by the drug. Vasospasm, in turn, is due to the vasoconstricting action of norepinephrine which is released from the vessel walls by the action of the drug (3). The initial spasm retards the circulation in the area, leading to prolonged exposure to a drug which normally would be diluted by the blood (9, 17). Concommitant with this reduced blood flow are cyanosis, temperature depression, and necrosis of the area supplied by the affected vessel. In support of this theory, Gabel noted that IA chlorpromazine (6.3 mg/100 lbs) does not cause a reaction (7). This drug is reported to be a vasodilator (12). Reserpin, administered subcutaneously around the artery before injection, delays the response to an IA injection, possibly by effecting the disappearance of norepinephrine from the vessels (2). The protection afforded by prior promazine (see above) may be due to this same action (7).

Stone and Donnelly (19), in their extensive review of IA accidents with thiopental, state that vasodilation follows the initial constriction. This resuults in destruction of endothelial and subendothelial tissues, and, in severe reactions, the inner muscle layers (14). Such damage will naturally be increased if a temporary vasoconstriction leads to prolonged exposure to the drug (9). Certain contrast media have been shown to disrupt cerebral circulation with stasis, hemorrhage, and multiple thrombi (19). This intimal injury may be a major factor in the pathology of IA injection responses (11). All clinical signs can be shown to result from vascular lesions and their sequelae (7).

### *PATHOLOGY*

The initial response to IA injection is edema, followed by congestion of the vessels and swelling of the endothelial linings. Damage to the intima, stasis of the blood, and thrombus formation have also been demonstrated (19). When the carotid artery of the horse is involved, dilation of the perivascular lymph spaces of the brain and formation of a status spongiosus is seen. Ganglion cells degenerate and small hemorrhages my occur in areas of severe vascular change (7, 19). Sheep exhibit the mildest chemical response—a slight cortical edema (7). The speed with which total vascular occlusion occurs is indicated by the lack of inflammatory elements in the area and lack of edema in the degenerated muscle (19).

### *THERAPY*

Therapy is primarily directed at prevention of thrombus with anticoagulants such as heparin (7) and at increasing the blood flow with vasodilators (14). Sympathetic block or denervation causes a decrease in norepinephrine release and thus an increase in vasodilation (2, 14, 19). This has been shown to decrease the area of necro-

sis, but does not bring about complete recovery. Anticoagulant therapy gives similar results (14).

General anesthesia has also been used to relieve the reactions (1, 3, 8). This does not abolish terminal convulsive phenomena at death, but it raises the dosage necessary to bring about convulsions.

## *PREVENTION*

Recommended measures for the prevention of IA accidents include the use of a large gauge needle and polyethylene tubing or a tom cat catheter. If a large gauge needle is threaded into the vessel, bright red, pulsating blood will be visible if an artery has been entered. The vein may be depressed distal to the needle. The continued flow of blood indicates strongly that the needle is in an artery.

Knowledge of regional anatomy is essential, but occasional anomalies make threading especially advisable. Adequate lighting and good restraint are important. In some cases, part of the dose should be injected and the rest withheld until it has been determined that no raction will occur.

## *SUMMARY*

Because of their consequences, IA injections are seldom reported but are numerous enough to be of concern to every practitioner. The most severe results of such accidents are large areas of skin necrosis, convulsive seizures, and death. The drugs involved are usually tranquilizers, sedatives, and others which are normally administered IV. The concentration, volume, rate of injection, and degree of sedation affect the type and extent of the response. Possible immediate causes of reactions are pain, prolonged arterial spasm, and intimal damage leading to thrombosis. The reaction is so rapid that inflammatory elements are not able to aggregate before blood flow is stopped. Recommended therapy includes general anesthesia, heparinization, sympathetic nerve block or denervation, vasodilators, and removal of thrombi by arteriotomy. Good venipuncture technique, particulary threading the needle into the vessel, is the best means of prevention.

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