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Site of Action of Intravenous Regional Anesthesia

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The principal site of action of intravenous regional anesthesia was studied using both prilocaine HCl 0.5% and technetium pertechnetate to define their distribution in the upper limb during this method of anesthesia. Using a single upper arm tourniquet and injecting technetium pertechnetate into a cubital fossa vein, the isotope spread to the finger tips. When a double tourniquet system was used to isolate the hand from the forearm, the following results were obtained: for up to 20 min after injection of the 40 ml of normal saline and radioisotope there was no leakage into the general circulation nor into the hand; after injection of 40 ml prilocaine HCl 0.5% into a cubital fossa vein, there was no anesthesia in the hand except for a small area on the dorsum corresponding to the area of sensory distribution of the radial nerve; while the tourniquets were inflated there was cramping pain in the hand. The results indicate that the initial analgesia obtained with the intravenous regional technique was due to blockade of small nerves or possibly nerve endings and not of the major nerve trunks at the elbow as has been suggested previously. (Key words: Anesthesia techniques: regional, intravenous. Anesthetics, local: prilocaine.)

FOLLOWING THE REINTRODUCTION of intravenous regional anesthesia,[1] it has been shown to be a safe and reliable method of anesthesia for short procedures involving the extremities.[2–5] However, there has been some disagreement on the site of action of the administered local anesthetic. Initially it was believed that this was on the small nerve and/or peripheral nerve endings.[6,7] However, subsequent reports have stated that the local anesthetics exert their effect on the main nerve trunks.[8–10] In response to this difference of opinion, a radioisotope and double-blind placebo controlled study was conducted in healthy volunteers.

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Method

This study, approved by the institutional ethics committee, was conducted using five consenting normotensive healthy male volunteers, whose ages ranged from 31 to 45 yr and physical characteristics were weights from 70 to 91 kg, and heights from 172 to 188 cm. Physical examination revealed no contraindication to the procedure. Throughout the study, continuous ECG monitoring was employed and blood pressure measurements were recorded every 5 min by sphygmomanometry.

The studies were conducted in five stages with at least a week between each stage.

STAGE 1

A tourniquet ("Zimmer Inflatomatic Cuff" 60 cm long, 5 cm wide) was placed around the upper arm on the nondominant side. Intravenous cannulae (22 gauge) were replaced in a vein of the cubital fossa of the arm to be studied and in the contralateral arm to provide iv access if necessary. The limb to be studied was elevated for 60 s while occlusive digital pressure was applied over the brachial artery, and the tourniquet was inflated to 300 mmHg, using a constant pressure pneumatic inflator. The limb then was lowered to the horizontal, and 40 ml physiologic saline with 1 mC_i technetium pertechnetate–^{99m}Tc was injected intravenously via the cannula in the cubital fossa. The arm was screened continuously with a gamma camera to identify any leakage past the tourniquet. The distribution of the technetium in the forearm and hand was recorded. The tourniquet was released after 20 min.

STAGE 2

A second tourniquet, 8.5 cm wide, was placed around the wrist in addition to the upper arm tourniquet (fig. 1). Following the same techniques described earlier, this cuff was inflated to 300 mmHg at the same time as the arm tourniquet. Forty milliliters of saline and technetium solution were injected into the cubital fossa vein. The forearm and hand were screened for 20 min. The cuffs then were deflated.

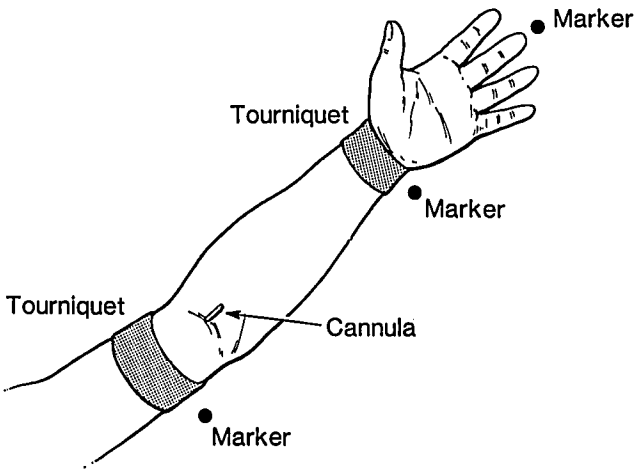

FIG. 1. Diagram of the position of the cannula and markers in Stage 2.

STAGES 3 AND 4

These stages consisted of an injection of 40 ml prilocaine HCl 0.5% or 40 ml physiologic saline in a double-blind fashion, while the wrist and arm tourniquets were inflated to 300 mmHg. The presence and distribution of cutaneous anesthesia were determined using pin prick every 2.5 min in the upper one-third of the forearm on the medial, lateral, and posterior aspects, while the hand was tested in the areas of distribution of the median, ulnar, and radial nerves. All subjective sensations were reported and recorded. The tourniquet was released at 20 min and the observations continued for a further 20 min.

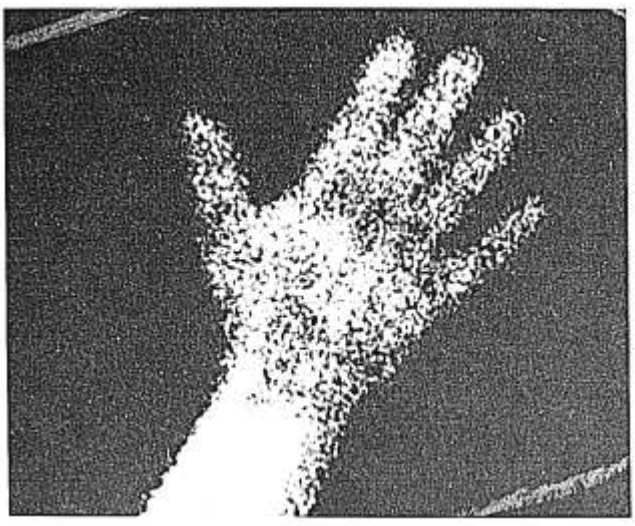

FIG. 2. Distribution of technetium in hand. Isotope can be seen spreading into the finger tips in Stage 1 following injection into the cubital fossa vein.

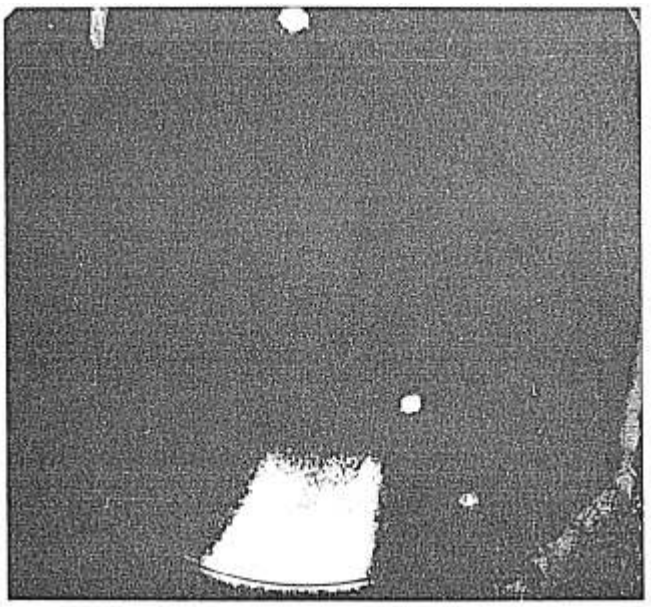

FIG. 3. Wrist tourniquet. The upper marker is situated at the tip of the third finger and the lower right marker at the center of the wrist tourniquet. No radioisotope is seen leaking under the tourniquet.

STAGE 5

This phase of the study was carried out using the same technique as stage 2 except that 40 ml prilocaine HCl 0.5% was injected into the cubital fossa vein after inflation of the upper arm tourniquet to 300 mmHg.

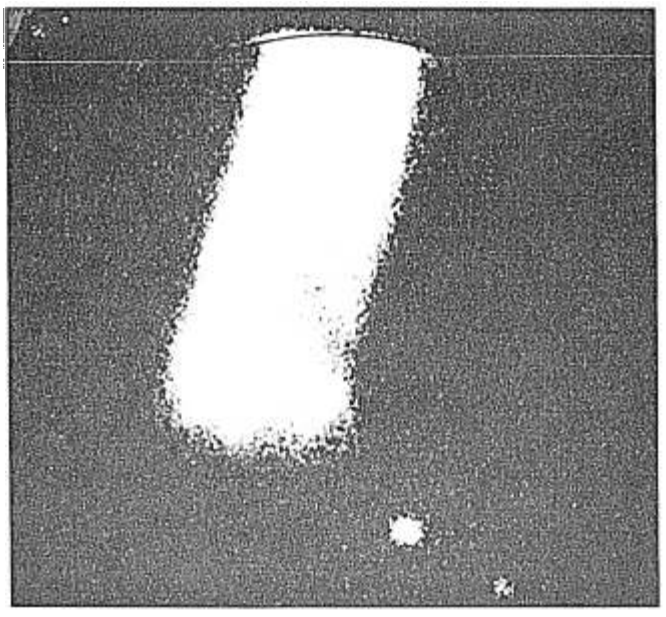

FIG. 4. Upper arm tourniquet. The marker is situated next to the centre of the upper arm tourniquet. No radioisotope is seen leaking under the tourniquet.

TABLE 1. Distribution of Block in the Hand and Forearm with Placebo and Prilocaine with Wrist Tourniquet Inflated

	Forearm						Hand					
	Medial		Lateral		Posterior		Median		Radial		Ulnar	
Subject	PRIL	N/S	PRIL	N/S	PRIL	N/S	PRIL	N/S	PRIL	N/S	PRIL	N/S
1	L	—	L	—	L	—	—	—	L	—	—	—
2	—	—	—	—	—	—	—	—	—	—	—	—
3	L	—	L	—	L	—	—	—	L	—	—	—
4	L	—	L	—	—	—	—	—	L	—	—	—
5	L	—	L	—	—	—	—	—	L	—	—	—

PRIL = active (prilocaine HCl 0.5%); N/S = placebo (normal saline); L = loss of pin-prick sensation; — = no change in sensation.

Observations were made in the same areas and at the same times as in Stages 3 and 4 for 20 min and for 20 min after deflation of the tourniquet.

Results

In stage 1, activity of the isotope was seen right up to the finger tips at the completion of the injection, which was performed over 60 s (fig. 2). In stage 2, the radioisotope studies showed that there was no leakage under either the arm tourniquet or the wrist tourniquet (figs. 3 and 4). Scanning of the contralateral hand before release of the tourniquets showed that there was no detectable amount of isotope in the general circulation. If there had been a leak of more than 5% of the total dose, activity would have been detected.

Stages 3 and 4 demonstrated that when prilocaine was used there was no anesthesia in the hand except in the radial nerve distribution (table 1). The onset of this block, however, was delayed in three of the five subjects, compared with the block that was produced in stage 5 (table 2). Sensory block of the posterior aspect of the forearm also was absent in three out of the five subjects. When saline was used, there was no anesthesia in any areas tested (table 1). All subjects described moderate to severe cramping pain in the hand when both prilocaine and saline were used.

In stage 5, four subjects had good sensory anesthesia in all areas tested, while one had decreased sensation but no loss of pin prick in the area of distribution of the ulnar nerve in the hand (table 3). No subject experienced pain in the hand.

On the release of the tourniquets, there were no signs or symptoms of local anesthetic toxicity. Sensation quickly returned to those areas that were anesthetized within 2.5–10 min.

Discussion

There have been a number of clinical reports of poor analgesia associated with intravenous regional anesthesia if the local anesthetic was prevented from reaching the site of operation by swelling and edema.[11] Nerve conduction studies, using the ulnar nerve and the response in the adductor policis, showed there was an early diminution of response only when the local anesthetic reached the hand.[11] Other studies, using radio opaque dye/local anesthetic solution, noted that where there was no evidence of dye on x-ray of the hand, there was no block in the hand.[7] Other workers, however, using nerve conduction studies, concluded that the site of action was at the main nerve trunks.[8,10] It was postulated that the local anesthetic solution reached the core of the nerve via the small venules.[8,9]

With the tourniquet system used in the present study, the radioisotope studies showed that the solution was confined to the forearm and that no leak occurred into the hand or the general circulation. In stages 3 and 4, when prilocaine had been used, there was no anesthesia in the distribution of the ulnar and median nerves in the hand. If the block was at the main nerve trunks and local anesthetic traveled to the core of the nerve via small venules, we would have expected to observe a blockade of the median and ulnar nerves, as these are surrounded by vascular channels in the lower arm while the radial nerve is isolated from such channels and so would have been less likely to be blocked.[12] The blockade of the radial nerve in stages 3 and 4 could be explained by the fact that in the lower one-third of the forearm radial nerve is the smallest of the three nerves to the hand and would thus be more likely to be blocked than

TABLE 2. Time in Minutes of Onset of Block of the Radial Nerve in Stages 3 and 4 and Stage 5

Subject	Stage 3 and 4	Stage 5
1	2.5	2.5
2	—	2.5
3	7.5	2.5
4	7.5	2.5
5	10	2.5

TABLE 3. Distribution of Block in Hand and Forearm Using Prilocaine HCl 0.5% without Wrist Tourniquet (Stage 5)

	Forearm			Hand		
Subject	Medial	Lateral	Posterior	Median	Radial	Ulnar
1	L	L	L	L	L	L
2	L	L	—	L	L	L
3	L	L	L	L	L	L
4	L	L	L	L	L	—
5	L	—	—	L	L	L

L = loss of pin-prick sensation; — = no loss of sensation.

the larger nerves in the presence of lower concentrations of the local anesthetic agent.

The cramping pains in the hand experienced by all subjects in stages 3 and 4 were probably of ischemic nature. In fact, it was this pain that prevented the study from extending beyond 20 min, because some of the volunteers were distressed by it. If the predominant site of blockade was at the main nerve trunks, small myelinated and unmyelinated pain fibers should have been the first to be blocked and this pain would not have been perceived.[13]

In one subject (subject 2) who did not obtain blockade from either solution in stages 3 and 4, the experiment was repeated with the use of prilocaine HCl 1.0%, and a block was obtained in the forearm and not the hand. The reason why the original solution did not give a block is unknown to us, but we surmise that, in this subject, who was the largest in the group of volunteers, the solution may have been "diluted" excessively by the blood already present in the forearm. The area that was least likely to be blocked was the posterior aspect of the forearm and around the olecranon, and this has been noted in a study where only two out of five patients have a satisfactory block in this area.[9] The area around the olecranon has only a few veins, and, although the isotope studies showed good concentration around the elbow, these were only two-dimensional studies, and thus we were unable to determine the concentration around the posterior aspect of the elbow. From the above evidence it seems that the main site of action of the local anesthetic in intravenous regional anesthesia, in its initial phase (20 min in this study), is on the smaller nerves and possibly the sensory nerve endings. We are unable to say whether there is any block at the main nerve trunks after a longer time has elapsed, due to the short duration of our observations.

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