CASE REPORT

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Absorption of Intubation-Related Lidocaine from the Trachea during Prolonged Cardiopulmonary Resuscitation

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ABSTRACT: The purpose of this study was to determine whether lidocaine is absorbed from the trachea during the artificial circulation of cardiopulmonary resuscitation. The tissue distribution of lidocaine was investigated in eight individuals (Cases 1–8) who underwent cardiopulmonary resuscitation before being pronounced dead.

In Cases 1–4, there was no restoration of heart beat during cardiopulmonary resuscitation. Heart massage had been continued for 5 min in Cases 1 and 2, and for 60 min in Cases 3 and 4. Relatively high concentrations of lidocaine (more than 0.1 mg/L) were detected in the blood left in the heart and/or in the large thoracic vessels in the four cases. In Cases 1–3, a large proportion of the lidocaine detected in these blood samples may have diffused from the trachea after cessation of cardiopulmonary resuscitation since no lidocaine was detected in the cerebrospinal fluid, cerebrum, liver, right kidney, and/or right femoral muscle. In Case 4, however, tracheal lidocaine was thought to have been absorbed during cardiopulmonary resuscitation because 0.167–0.340 mg/L or mg/kg lidocaine was detected in the cerebrospinal fluid, liver, right kidney, and right femoral muscle. This was substantiated in experiments performed in rabbit carcasses given 50 μL/kg Xylocaine™ jelly (a 2% lidocaine hydrochloride preparation) intratracheally, followed by rhythmical thoracic compressions (100–150 times per minute) for 60 min. A possible reason for lack of absorption of lidocaine from the trachea of Case 3 during a 60-min cardiopulmonary resuscitation procedure may have been that effective blood circulation was not obtained during cardiopulmonary resuscitation because of bleeding and pulmonary collapse.

Cases 5–8 survived for 3 h to 10 days after successful cardiopulmonary resuscitation; it was obvious that lidocaine was distributed to the tissues under the influence of the natural circulation. The kidney to liver lidocaine ratio in Case 4 (0.8) was much lower than that in Cases 5–8 (1.3–4.6), although the lidocaine ratio in the blood in the left ventricle when compared to blood in the right ventricle was similar in the five cases. The kidney to liver lidocaine ratio may be helpful in judging whether the lidocaine detected was absorbed during the artificial circulation of cardiopulmonary resuscitation or naturally. Additionally, postmortem diffusion of tracheal lidocaine into the blood in the left ventricle was much greater than into the blood in the right ventricle due to their anatomical location during a supine position. The pattern of tissue distribution of lidocaine gives useful information on the state of decedents during cardiopulmonary resuscitation.

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Forensic pathologists are often asked to examine accident- or crime-related individuals who have undergone emergency medical treatment before being pronounced dead. In these patients, there is no doubt that testing for drugs and chemicals contributes greatly in clarifying not only the circumstances at the time of the accident, but also the state of the victims during the medical treatment. Xylocaine™ jelly, a 2% lidocaine hydrochloride preparation, is often in Japan used to facilitate endotracheal intubation for cardiopulmonary resuscitation. Of all the forensic autopsy cases seen at the Department of Legal Medicine of Kochi Medical School, 20–30% were patients who had undergone cardiopulmonary resuscitation with endotracheal intubation. Lidocaine was detected in about 42% of the patients. In a previous study, we (1) reported that a significant amount of lidocaine applied to the endotracheal tube diffuses into the cardiac chambers from the trachea after death in individuals in whom cardiopulmonary resuscitation was unsuccessful. Drug concentrations in postmortem blood are often used for evaluating whether an individual was under the influence of a drug at the time of death, since basic and clinical information on the relationship between drug concentrations in blood and their toxicity is available (2). Blood taken from the heart is most frequently the specimen of choice when determining drug concentrations in non-decomposed bodies because it can usually be taken in large enough volumes. Thus, postmortem diffusion of lidocaine from the trachea into the heart blood should be taken into account as a factor affecting postmortem toxicological analysis in patients given medical treatment. It is well documented that drug concentrations in heart blood are erroneously elevated postmortem due to drug redistribution from the lung, liver and/or myocardium; drugs are distributed at much higher concentrations in these tissues than in blood (3–6), and there may be drug diffusion from a stomach containing large amounts of unabsorbed drugs (7–9). Comparison of the lidocaine concentrations in heart blood with those in peripheral blood or in limb skeletal muscles may be helpful in establishing the extent to which lidocaine may have diffused into the heart blood from the trachea postmortem (1,10). However, in the first instance, it is important to establish whether tracheal lidocaine is absorbed during cardiopulmonary resuscitation and the extent of its absorption.

In this study, we have investigated whether absorption of intubation-related lidocaine occurs from the trachea during cardiopulmonary resuscitation, and whether this varies in relation to the duration of the procedure. Additionally, we also present the findings of animal experiments using rabbits.

Materials and Methods

Human Autopsy Cases

From September 1, 1994 to April 30, 1997, 21 patients who were dead-on-arrival at hospital and who underwent cardiopulmonary resuscitation before being pronounced dead, underwent autopsy at the Department of Legal Medicine of Kochi Medical School. Of these individuals, eight (Cases 1–8) were examined for tissue distribution of lidocaine. In Cases 1–4, the heart never resumed beating during cardiopulmonary resuscitation; heart massage had been continued for 5 min in Cases 1 and 2, and for 60 min in Cases 3 and 4. No lidocaine preparation other than Xylocaine™ jelly was used for endotracheal intubation; antemortem use of lidocaine preparations by patients was also excluded. Cases 5–8 survived for 3 h to 10 days following cardiopulmonary resuscitation and were used in this study as controls to determine the antemortem absorption of lidocaine. Of the four cases, one was given endotracheal lidocaine only, while the other three received both endotracheal and intravenous lidocaine. Cases 1–8 are summarized in Table 1.

Chemicals

Xylocaine™ jelly (2%), Novo heparin™ (sodium heparin: 1000 units/mL), and Nembutal™ (sodium pentobarbital: 50 mg/mL) were purchased from Fujisawa Pharmaceutical Co. (Osaka, Japan), Kodama Co. (Tokyo, Japan), and Dainippon Pharmaceutical Co. (Osaka, Japan), respectively. The other reagents were of analytical grade.

Apparatus

A Shimadzu GC-14B (Kyoto, Japan) equipped with a TC-1 capillary column [dimethyl silicone, 15 m by 0.53 mm ID, 1-5 μm film thickness (GL Sciences Inc., Tokyo, Japan)], a TC-17 capillary column [50% phenylmethyl silicone, 15 m by 0.53 mm ID, 1.5 μm film thickness (GL Sciences Inc., Tokyo, Japan)] and a flame thermionic detector (FTD) was employed for screening and quantification of lidocaine. The temperature of the injection port and detector was 280°C for the TC-1 capillary column and 260°C for the TC-17 capillary column. The column temperatures were programmed as follows: an initial temperature of 150°C was maintained for 2 min, and then increased to 280°C for the TC-1 capillary column and to 260°C for the TC-17 capillary column at a rate of 10°C/min. The final temperatures were maintained for 10 min. The carrier gas was nitrogen, with a flow pressure of 15 kPa.

A GC/MS system, consisting of a Shimadzu GC-9A (Kyoto, Japan) equipped with a 2 m by 0.26 cm ID glass column packed with 2% OV-1 on 60–80 mesh Chromosorb W AW DMCS, and a Shimadzu QP 1000 D (Kyoto, Japan) was employed for confirmation of lidocaine. The temperatures of the injection port and column were identical to those for the GC with the TC-1 capillary column. The temperature of the separator was 280°C. The electron impact ionization energy and accelerating voltage were 70 eV and 3 kV, respectively. The carrier gas was helium, with a flow rate of 40 mL/min.

Animal Experimentation

Six male rabbits (3.30–3.75 kg) were given 1000 units of sodium heparin, intravenously, to prevent blood coagulation during experiments, and were then anesthetized with intravenous sodium pentobarbital (30 mg/kg). The animals were sacrificed by an intravenous injection of 2 mmol/kg potassium chloride. The rabbit carcasses were then divided into two groups (Groups I and II) and 50 μL of Xylocaine™ jelly per kilogram of body weight (1 mg/g as lidocaine hydrochloride) was administered into the trachea just above the

TABLE 1—*Summary of eight individuals in whom cardiopulmonary resuscitation (CPR) was performed and medical treatment-related lidocaine was detected.*

Case No.	Age (sex)	Duration of CPR	Restoration of Heart Beat	Survival Time	Postmortem Interval	Origin of Lidocaine	Main Pathological Findings (Cause of Death)
1	3.5 mo. (♀)	5 min	No	—	20 h	Endotracheal intubation	Petechiae on the thymus, epicardium and lungs; congestion of organs (sudden infant death syndrome)
2	44 yr. (♂)	5 min	No	—	20 h	Endotracheal intubation	Basal fractures of the skull; a subarachnoid hemorrhage; a cerebral contusion; edematous brain (brain swelling)
3	38 yr. (♂)	60 min	No	—	20 h	Endotracheal intubation	A stab wound in the left thigh; partial severing of the left femoral artery; subcutaneous emphysema, multiple fractures of the sternum and ribs, and pulmonary collapse due to CPR (bleeding)
4	4 mo. (♂)	60 min	No	—	12 h	Endotracheal intubation	Petechiae on the conjunctiva of the right eyelid, thymus and epicardium; slight lung emphysema and hemorrhages due to CPR (sudden infant death syndrome)
5	52 yr. (♀)	<5 min	Yes	3 h	12 h	Endotracheal intubation	Stab wounds in the thorax and liver; partial severing of the portal vein; 2500 mL of blood in the abdominal cavity (bleeding)
6	42 yr. (♀)	5 min	Yes	1 d	9 h	Endotracheal intubation and IV injection*	A stab wound in the left thigh; complete severing, which was anatomosed, of the left femoral artery and vein (hemorrhagic shock)
7	53 yr. (♂)	<5 min	Yes	3 d	14 h	IV injection	Subdural hematomas; cerebral hernia; edematous brain; hemorrhages of the brainstem (brain swelling)
8	8 yr. (♂)	5 min	Yes	10 d	3 d	IV injection ?	Brain-dead state; extensive malacia of the cerebellum (brain swelling)

*Intravenous injection.

bifurcation. The rabbit carcasses in Group I were given cardiac massage immediately by rhythmical compression of the thoraces (100–150 times per minute) in the supine position at an ambient temperature. The rabbit carcasses in Group II were left in the supine position at an ambient temperature. The pulmonary vein blood, blood in the cardiac chambers (left and right ventricles), inferior vena cava blood, cerebrum, hilar regions of the lungs, myocardium, right lobe of the liver, right kidney and right femoral muscle were procured 1 h after treatment with lidocaine. The volume of each blood specimen collected was approximately 0.5 mL.

GC Quantification of Lidocaine in Various Fluids and Tissues

The lidocaine concentration in the various fluids and tissues was determined as described previously (1). Briefly, 2 mL of each body fluid (each rabbit blood diluted to 1/5 with distilled water) or 2 g of each tissue homogenate (tissue: distilled water = 1:3) was mixed with carbinoxamine maleate in methanol (100 μL of 12 mg/L; internal standard) and carbonate buffer (2 mL, 1M, pH 9.7). Each mixture was extracted with n-chlorobutane/isoamyl alcohol (98:2; 8 mL), and the organic phase was back-extracted with HCl (1 mL, 0.1 N). The resulting aqueous phase was washed with 2-methylbutane/toluene/isoamyl alcohol (94:5:1; 4 mL) and mixed with carbonate buffer (1 mL). The mixture was re-extracted with 2-methylbutane/toluene/isoamyl alcohol (94:5:1; 4 mL). The organic phase was reduced to approximately 100 μL, and then aliquots (1 μL) of the concentrated extract were injected into the GC.

Results

Human Autopsy Cases

The lidocaine concentrations in Cases 1–8 are shown in Table 2. Relatively high concentrations of lidocaine, more than 0.1 mg/L, were detected in blood taken from the heart and/or the large thoracic vessels in all of the four cases (Cases 1–4) in whom the heart beat was never restored by cardiopulmonary resuscitation. In Cases 1–3, no lidocaine was detected in the cerebrospinal fluid, cerebrum, liver, right kidney and/or right femoral muscle. In contrast, Case 4 showed high concentrations (0.167–0.340 mg/L or mg/kg) of lidocaine even in these samples. In the control cases (Cases 5–8) where the heart beat was restored by cardiopulmonary resuscitation, lidocaine was detected in all of the specimens examined. The lidocaine ratio in blood taken from the left ventricle when compared to that taken from the right ventricle in Case 4 was relatively similar to that observed in Cases 5 and 6, but much lower than that in Cases 1 and 2. The kidney to liver lidocaine ratio was lower than 1 in Case 4, but higher than 1 in Cases 5–8 (Table 3).

TABLE 3—*The patterns of tissue distribution of medical treatment-related lidocaine in cases where cardiopulmonary resuscitation (CPR) was performed.*

Case No.	Main Route of Lidocaine Entrance into Tissues	Mean (Range) of Lidocaine Concentration Ratio	
		Left Ventricular Blood/Right Ventricular Blood	Kidney/Liver
1–3	Postmortem diffusion from the trachea	4.2 (3.4–4.9)*	—
4	Absorption under artificial circulation during CPR	1.5†	0.8
5–8	Absorption under natural circulation	1.9 (1.8–2.0)†‡	2.9 (1.3–4.6)

*Cases 1 and 2; no blood was contained in the cardiac chambers in Case 3.

†Left ventricular blood might have been affected by postmortem diffusion from the trachea and/or lungs to some degree, because the postmortem interval was 9–12 h.

‡Cases 5 and 6; lidocaine concentrations in the left ventricular blood were not determined in Cases 7 and 8.

TABLE 2—*Lidocaine concentrations in various fluids and tissues of eight patients who underwent cardiopulmonary resuscitation (CPR) with endotracheal intubation.*

Specimen	Lidocaine Concentration (mg/L or mg/kg)							
	No Restoration of Heart Beat				Restoration of Heart Beat			
	CPR for about 5 min		CPR for about 1 h					
	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Blood								
Pulmonary artery	—	—	—	1.80	0.262	0.217	—	—
Pulmonary vein	—	—	—	1.26	0.371	0.278	—	—
Left ventricle	0.349	1.02	—	0.569	0.231	0.220	—	—
Right ventricle	0.102	0.209	—	0.373	0.117	0.121	0.029	0.145
Thoracic aorta	—	—	0.642	0.419	—	—	—	—
Superior vena cava	—	—	0.746	—	—	—	—	—
Inferior vena cava	0.195	0.163	0.133	0.225	0.078	0.088	—	—
Right iliac vein	—	0.074	0.057	—	—	—	—	—
Right femoral vein	—	0.015	ND	0.171	0.036	—	—	—
Pericardial sac fluid	0.193	0.097	0.171	0.449	0.164	0.240	—	—
Cerebrospinal fluid	ND	—	—	0.268	0.136	0.339	—	—
Cerebrum (parietal region)	ND	ND	ND	—	0.157	0.393	0.070	0.014
Lung								
Left hilus	—	10.9	1.37	—	0.281	0.413	—	—
Right hilus	—	2.65	1.41	—	0.354	0.461	—	—
Liver (right lobe)	ND	ND	ND	0.213	0.036	0.064	0.026	0.075
Right kidney	—	ND	ND	0.167	0.047	0.295	0.091	0.150
Right femoral muscle	ND	ND	ND	0.340	0.036	0.179	—	—

TABLE 4—*Lidocaine concentrations in various blood and tissues of rabbit carcasses given 1 mg/kg lidocaine hydrochloride, intratracheally, and placed at an ambient temperature for 1 h with (Group I) or without (Group II) rhythmical compression (100–150 times per minute) to their thoraces.*

Specimen	Mean (Range) of Lidocaine Concentration (mg/L or mg/kg)	
	Group I (n = 3)	Group II (n = 3)
Blood		
Pulmonary vein	21.8 (12.2–40.8)	42.0 (28.1–60.9)
Left cardiac chambers	11.2 (10.4–16.5)	17.5 (8.74–30.7)
Right cardiac chambers	2.89 (1.36–3.79)	1.45 (0.287–3.23)
Inferior vena cava	2.43 (1.93–3.40)	1.21 (0.316–2.94)
Cerebrum	0.229 (0.105–0.358)	ND
Myocardium	6.72 (4.33–9.35)	2.92 (1.64–4.92)
Lung		
Left hilus	7.85 (7.34–8.12)	87.1 (0.192–190)
Right hilus	45.0 (21.9–81.0)	100 (19.0–144)
Liver (right lobe)	0.426 (0.054–0.627)	0.057 (ND–0.170*)
Right kidney	0.136 (ND–0.232†)	ND
Right femoral muscle	0.223 (ND–0.385†)	ND

*Of the three carcasses, one was positive for lidocaine.
†Of the three carcasses, two were positive for lidocaine.

Animal Experimentation

Table 4 shows the results of the animal experiments. Low to high concentrations of lidocaine were detected in the pulmonary vein blood, heart blood, inferior vena cava blood, myocardium, and hilar regions of the lungs in all of the rabbit carcasses in Groups I and II. In Group I, the cerebrum and liver contained detectable amounts of lidocaine; the right kidney and right femoral muscles of the two rabbit carcasses also showed an increased concentration of lidocaine. In Group II, no lidocaine was detected in the cerebrum, right kidney and right femoral muscle; a small amount of lidocaine was detected in the liver of a rabbit carcass. As shown in Table 5, the mean lidocaine ratio in blood taken from the left ventricle when compared to that taken from the right ventricle in Groups I and II was 3.9 and 12.1, respectively. The kidney to liver lidocaine ratios were lower than 1.

Discussion

Drug concentrations in postmortem blood tend to be site-dependent, and are affected by several factors including: 1) postmortem metabolism of drugs by bacteria and any residual enzyme activity (11,12); 2) postmortem production of drugs or chemicals by bacteria (7,13); 3) postmortem redistribution of drugs from the lung, liver and/or myocardium, organs in which drugs accumulate at concentrations much higher than in the blood (3–6); 4) postmortem diffusion of drugs from the stomach containing large amounts of unabsorbed drugs (7–9); and 5) postmortem diffusion of drugs through the trachea associated with agonal aspiration of vomitus (14,15). In a previous study, we (1) demonstrated that postmortem diffusion of intubation-related lidocaine occurred through the trachea in patients who underwent cardiopulmonary resuscitation before being pronounced dead. Therefore tracheal lidocaine may be the source of lidocaine detected in the blood in the heart and the large central vessels.

TABLE 5—*The patterns of tissue distribution in rabbit carcasses given 1 mg/kg lidocaine hydrochloride, intratracheally, and placed at an ambient temperature for 1 h with (Group I) or without (Group II) rhythmical compression (100–150 times per minute) to their thoraces.*

Rabbit	Mean (Range) of Lidocaine Concentration Ratio	
	Left Ventricular Blood/ Right Ventricular Blood	Kidney/Liver
Group I (n = 3)	5.6 (1.9–12.1)	0.2 (0.0–0.4)
Group II (n = 3)	18.6 (9.5–30.5)	—

However, it is not known whether a significant amount of lidocaine is absorbed from the trachea during cardiopulmonary resuscitation itself. In order to answer this question, we have measured the concentrations of lidocaine in various bodily fluids and tissues in eight individuals who were dead-on-arrival at hospital but had cardiopulmonary resuscitation with endotracheal intubation performed. In Cases 1–4, the heart never resumed beating during cardiopulmonary resuscitation, and no lidocaine preparation other than Xylocaine™ jelly was used for endotracheal intubation. In Cases 1 and 2 where heart massage had been continued for 5 min, a large proportion of the lidocaine detected in the various blood samples may have diffused from the trachea after being pronounced dead, since no lidocaine was detected in the cerebrospinal fluid, cerebrum, liver, right kidney and right femoral muscle (1). However, in Case 4 where cardiopulmonary resuscitation had been continued for 60 min, relatively large amounts of lidocaine were detected in all of the specimens including the brain, liver, right kidney and right femoral muscle. Hence, a large proportion of tracheal lidocaine had been absorbed while the patient was receiving resuscitation. To substantiate this phenomenon, we performed experiments using rabbit carcasses administered with 50 μL/kg of Xylocaine™ jelly intratracheally.

Although the amount of Xylocaine™ jelly used in the present cases was undocumented, 2–3 mL of Xylocaine™ jelly is usually applied to the endotracheal tube. Thus, the dosage of lidocaine in the rabbit carcasses roughly corresponds to that in a patient weighing 50 kg. The rabbit carcasses given rhythmical thoracic compression for 60 min showed an increase in the concentration of lidocaine within the brain and liver; two of the rabbit carcasses also showed high drug concentrations within the right kidney and right femoral muscle. When thoracic compression was not performed, lidocaine could not be detected in the brain, right kidney or right femoral muscle. Thus, it seems that prolonged cardiopulmonary resuscitation can cause absorption of substantial amounts of tracheal lidocaine. Additionally, postmortem diffusion of tracheal lidocaine into the surrounding fluids and tissues can occur very quickly if the blood remains uncoagulated. In Case 3, there was no increase in lidocaine concentration in the right femoral vein blood, brain, liver, right kidney and right femoral muscle despite 60-min of cardiopulmonary resuscitation. This apparent discrepancy between Cases 3 and 4 may have been due to the lethal bleeding and pulmonary collapse which occurred in Case 3, but not in Case 4 (Table 1). The pulmonary collapse may have occurred at an early stage of the cardiopulmonary resuscitation procedure and no blood was present in the cardiac chambers. Thus, since there was no effective blood circulation during the cardiopulmonary resuscitation procedure in Case 3, most of lidocaine which was detected may have been the result of postmortem diffusion from the trachea.

The cases investigated in this study can be classified into three categories depending on the main route of lidocaine entry into the

tissues: (1) postmortem diffusion from the trachea (Cases 1–3); (2) absorption from the trachea during the artificial circulation of cardiopulmonary resuscitation (Case 4); and (3) absorption from the trachea during natural circulation and/or intravenous administration (Cases 5–8). As observed in Cases 1 and 2, and in the animal experiments, diffusion of lidocaine from the trachea into the left ventricular blood was greater than into the right ventricular blood. In Cases 5 and 6, tracheal lidocaine may have been almost completely absorbed, since the lidocaine concentration in the hilar regions of the lungs was not as high as that seen in Cases 2 and 3. Hence, in Cases 5 and 6, the higher concentration of lidocaine in the left ventricular blood when compared to the right ventricular blood may have been due to postmortem redistribution of pulmonary lidocaine (3); this effect is significant but much less so than the postmortem diffusion of lidocaine from the trachea. In Case 4, although a significant amount of lidocaine still remained in the trachea after cardiopulmonary resuscitation because the lidocaine concentrations in the pulmonary artery and vein were much higher than those in the blood within the heart, the ratio of lidocaine in the left ventricle when compared to the right ventricle was much lower than that observed in Cases 1 and 2. This may not only be because the postmortem interval in Case 4 was shorter than that in Cases 1 and 2, but also because the differences in the diffusion of tracheal lidocaine between the left and right ventricular blood may have been buffered by lidocaine already present in the heart blood; i.e., that which had been absorbed during cardiopulmonary resuscitation. In animal experiments, 60-min of cardiac massage lowered the lidocaine ratios between the blood present in the left ventricle compared to that present in the right ventricle.

From the pattern of tissue distribution of lidocaine in Cases 4–6, little information can be gained whether the lidocaine which was detected was a result of absorption during natural circulation, or whether it was absorbed during the artificial circulation of cardiopulmonary resuscitation. However, absorption of lidocaine from the trachea during the artificial circulation of cardiopulmonary resuscitation gave a kidney to liver lidocaine ratio of lower than 1; in contrast, absorption during natural circulation gave a ratio higher than 1. Rabbit carcasses given cardiac massage for 60 min also showed a kidney to liver lidocaine ratio of less than 1. Thus, the kidney to liver lidocaine ratio seems to be useful in determining the different patterns of absorption. The different pattern of antemortem and postmortem distribution of lidocaine in the liver and kidney may simply reflect the metabolic state of lidocaine in the liver; it is likely that in individuals who suffer from cardiopulmonary arrest, liver function will deteriorate markedly if the heart does not resume beating. Since lidocaine is extensively metabolized within the liver of living persons and animals, accumulation of parenterally administered lidocaine is likely to be greater within the kidney than in the liver (16–18). Additionally, the detection of lidocaine metabolites in individuals who are not successfully resuscitated may also be helpful in judging whether the accumulation of lidocaine in tissues is a result of its absorption antemortem or postmortem.

In conclusion, we have demonstrated that substantial amount of lidocaine is absorbed from the trachea during prolonged cardiopulmonary resuscitation procedures even if the heart beat is not restored. Whether there has been absorption of lidocaine from the trachea during unsuccessful cardiopulmonary resuscitation can be easily judged from the pattern of tissue distribution of lidocaine, since although tracheal lidocaine diffuses rapidly into the surrounding fluids and tissues, it never results in an increase in the lidocaine concentration within the brain, liver, kidney and femoral muscle. Additionally, the kidney to liver lidocaine ratio may be helpful in judging whether tracheal lidocaine was absorbed antemortem or postmortem in an individual who underwent cardiopulmonary resuscitation. The pattern of tissue distribution of lidocaine gives useful information regarding the state of the victims during cardiopulmonary resuscitation.

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