



Cardiac dilatation index as an indicator of terminal central congestion evaluated using postmortem CT and forensic autopsy data



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ABSTRACT

Previous studies demonstrated possible application of postmortem quantitative CT data analysis of the heart and lung *in situ* to investigate terminal cardiopulmonary pathophysiology. The present study analyzed virtual CT morphometric and autopsy data of the heart to investigate terminal central congestion in forensic autopsy cases ($n = 113$, within 3 days postmortem); the virtual total heart weight *in situ* was estimated using CT morphometry, and the difference from and ratio to the measured weight at autopsy were calculated as indicators of heart blood pooling and the cardiac dilatation index (CDI) before dissection, respectively. There were substantial differences between the estimated heart blood pooling *in situ* and volume recovered at autopsy, including a characteristic decrease in drowning, alcohol/sedative-hypnotic intoxication and sudden cardiac death (SCD), possibly due to blood redistribution after thoracic dissection. The estimated *in situ* heart blood pool and CDI values were higher in SCD but lower in fatal hemorrhage and hemopericardium, as well as in acute mechanical asphyxiation and hyperthermia (heatstroke). In addition, there was a significant difference in heart blood pooling between mechanical asphyxiation or drowning and SCD. The CDI was significantly lower in fatal hyperthermia (heatstroke) than in drowning, fatal methamphetamine abuse, alcohol/sedative-hypnotic intoxication and SCD. These findings suggest the usefulness of applying the CDI and postmortem heart blood volume *in situ* as supplementary indicators of terminal central congestion, especially for investigating deaths from hemorrhage, hemopericardium, hyperthermia (heatstroke) and SCD.

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1. Introduction

Postmortem imaging using computed tomography (CT) and magnetic resonance imaging (MRI) provides a noninvasive procedure for the documentation of whole-body data on routine autopsy and the detection of bone and visceral lesions, as well as various possibilities to investigate the status of viscera before autopsy [1–5]. Most investigations have been devoted to detecting pathologies for determining the cause of death to supplement or minimize the autopsy procedure [6–10], or for use in forensic identification [11,12]; however, several studies demonstrated the possible application of postmortem CT (PMCT) data to analysis of the status of the heart and lungs *in situ* before opening the body

cavity at autopsy for investigating terminal cardiopulmonary pathophysiology in diagnosis of the cause and mode of death [13–16]; these quantitative data are not available with conventional autopsy.

In the conventional autopsy procedure, cardiac enlargement and the blood volume recovered on removal of the heart at autopsy are considered to be indicators of cardiac congestion or a reduced circulating blood volume [17–19]; however, the measurements may be modified by blood redistribution after death and on opening the body cavity. Meanwhile, virtual measurement of blood pooling or clotting in the heart is difficult using postmortem plain CT data, but the difference between the virtual whole weight and measured weight of the heart may represent the heart blood volume at autopsy; thus, combined analysis of autopsy and PMCT data, using quantitative indicators, may provide useful information for the precise investigation and objective interpretation of terminal cardiac dysfunction.

On the basis of the aforementioned hypothesis, the aim of the present study was to investigate the virtual whole-heart weight *in*

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situ, including pooling blood, as estimated using CT morphometry, and the heart weight measured at autopsy for evaluating terminal central congestion in forensic autopsy cases. Ratio of the virtual whole-heart weight *in situ* as estimated by CT morphometry to the measured weight at autopsy was calculated as the cardiac dilatation index (CDI) and examined with regard to the cause of death.

2. Materials and methods

2.1. Subjects and autopsy protocol

2.1.1. Subjects

Forensic autopsy cases of >16 years of age ($n = 113$) within 3 days postmortem, in which the causes of death had been clearly established, were used in the present study retrospectively, excluding cases involving significant ante- or postmortem body destruction or decomposition, destruction or deformity of the chest due to injury, burns or surgical intervention, hemo-/pneumothorax, massive pleural effusions, and putrefactive changes with gas formation, as well as cases with significant complications of disease or trauma involved in other cause of death groups, prolonged deaths in hospital after temporary recovery by cardiopulmonary resuscitation, or under critical terminal care. The cases partly included those in previous reports [13–15], comprising those of fatal hemorrhage from sharp instrument injury, mechanical asphyxiation, drowning, drug intoxication, including fatal methamphetamine (MA) abuse and alcohol/sedative-hypnotic intoxication, fire fatalities due to burns with blood carboxyhemoglobin level (COHb) of <60% and carbon monoxide (CO) intoxication with blood COHb of >60%, hyperthermia (heatstroke), fatal hypothermia (cold exposure), hemopericardium and sudden cardiac death due to acute ischemic heart disease (SCD). Details are shown in Table 1. SCD cases included a 16-year-old boy who died suddenly during playing basketball, due to acute cardiac dysfunction resulting from a right coronary anomaly. Pathological data including heart and lung weights and the heart blood volume were collected from autopsy documents.

2.1.2. Autopsy protocol

The heart was removed separately after opening the pericardial cavity, cutting the aorta at approximately 3 cm above the orifice and the other vessels at the posterior pericardial wall, to measure the heart weight excluding blood and blood clots after dissection as well as the heart blood volume during our routine dissection procedures. The heart blood volume was defined as the total volume including blood clots recovered in the pericardial cavity, including the inflow from great vessels, on removal of the heart after the collection of pericardial effusion.

Postmortem interval (the time from estimated time of death to autopsy) and survival time (the period from the onset of fatal insult to death) were estimated on the basis of autopsy findings in consideration of case history and circumstantial evidence; possible error ranges, up to hours, depended on the elapsed times in unwitnessed deaths. These data were extracted from autopsy documents, estimated based on pathological findings and circumstantial evidence.

2.2. PMCT data analysis

Whole-body PMCT scans were routinely performed immediately before forensic autopsy, employing a 16-row multidetector CT scanner (ECLOS, Hitachi Medical Co., Tokyo, Japan) within the framework of routine casework. The spiral CT scanning was performed under the following conditions: 120 kV; 250 mA; 16×1.25 -mm thickness; field of view, 500 mm. A CT data analysis system, Volume Analyzer SYNAPSE VINCENT (FUJIFILM Medical Co., Ltd., Tokyo, Japan), was used to reconstruct three-dimensional (3D) images of the heart *in situ* for virtual volumetry: the heart was extracted by manual cursoring from the ascending aorta up to 3 cm above the aortic valves to the bottom of the heart (Fig. 1). These analyses were performed by forensic pathologists and two radiographers in consultation with board-certified radiologists. The reproducibility of virtual reconstruction and measurement was checked by two independent observers. The whole-heart weight, including blood pooling, was estimated from the total volume and CT attenuation (Hounsfield units, HU), using the correlation of HU with tissue-specific gravity, as previously

Table 1
Case profile: autopsy data.

Cause of death	Case number (male/female)	Age range (median)	Survival time (h)	Postmortem time (h) range (median)	Heart weight (g) range (median)	Measured heart blood volume (mL) range (median)	Cardiac blood clots $-/+$	Combined lung weight (g) range (median)
Fatal hemorrhage from sharp instrument injury	8 (4/4)	55–91 (67)	<0.5	13–60 (33)	255–455 (313)	8–150 (31) ^b	4/4	375–940 (613)
Mechanical asphyxiation	16 (8/8)	26–87 (66)	<0.5–6.0	13–65 (31)	205–515 (363)	40–350 (95)	10/6	465–1800 (1008)
Drowning	14 (9/5)	16–87 (64)	<0.5	13–61 (34)	260–605 (328)	50–430 (140)	12/2	545–1565 (1328)
Fire fatality (COHb < 60%)	14 (10/4)	38–86 (63)	<0.5	10–32 (22)	200–660 (343)	50–450 (160)	10/4	515–1555 (1058)
Fire fatality (COHb > 60%)	11 (7/4)	33–95 (65)	<0.5	9–37 (17)	280–500 (360)	60–400 (180)	8/3	790–1210 (960)
Alcohol/sedative-hypnotic intoxication	8 (2/6)	26–43 (36)	<0.5–12.0	26–62 (38)	210–420 (265)	50–200 (100)	1/7	845–1570 (1065)
Methamphetamine abuse	9 (7/2)	35–52 (46)	<0.5–8.0	20–61 (33)	315–510 (375)	100–400 (250)	5/4	795–1865 (1440)
Hyperthermia (heatstroke)	7 (4/3)	60–92 (76)	<6.0–12.0	20–61 (30)	200–510 (385)	50–300 (100)	1/6	585–1480 (1075)
Hypothermia (cold exposure)	8 (3/5)	50–91 (83)	<6.0–24.0	23–62 (35)	190–400 (330)	80–500 (200)	2/6	350–960 (623)
Sudden cardiac death	11 (9/2)	16–91 (67)	<0.5–6.0	14–37 (21)	235–740 (465) ^a	60–550 (200)	8/3	535–1765 (1305)
Hemopericardium	7 (4/3)	29–88 (69)	<0.5–3.0	13–62 (22)	315–650 (370)	40–200 (70) ^c	6/1	550–1275 (995)
Total	113 (67/46)	16–95 (64)	–	9–65 (30)	190–740 (355)	8–550 (120)	67/46	350–1865 (1005)

COHb, carboxyhemoglobin.

^a Significantly greater in sudden cardiac death than in alcohol/sedative-hypnotic intoxication and hypothermia ($p < 0.01$) and fatal hemorrhage, mechanical asphyxiation and fire fatalities ($p < 0.05$) on individual comparisons using Mann–Whitney U -test.

^b Significantly smaller in fatal hemorrhage than in other groups except for hemopericardium ($p < 0.02$ – 0.01) on individual comparisons using Mann–Whitney U -test.

^c Significantly smaller in hemopericardium than in fire fatalities, hypothermia and sudden cardiac death ($p < 0.02$ – 0.01) on individual comparisons using Mann–Whitney U -test.

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