Postmortem Redistribution of Fentanyl in Blood

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Abstract

Fentanyl concentrations were measured in postmortem specimens collected in 20 medical examiner cases from femoral blood (FB), heart blood (HB), heart tissue, liver tissue, and skeletal muscle. Unique was a subset of 7 cases in which FB was obtained at 2 postmortem intervals, shortly after death (FB1) and at autopsy (FB2). The mean collection times of FB1 and FB2 after death were 4.0 and 21.6 hours, respectively. Fentanyl concentrations for FB1 and FB2 ranged from undetectable to 14.6 μg/L (mean, 4.6 μg/L) and 2.0 to 52.5 μg/L (mean, 17.3 μg/L), respectively. Corresponding mean HB, liver tissue, and heart tissue fentanyl concentrations were 29.8 μg/L, 109.7 mg/kg, and 103.4 mg/kg, respectively. The fentanyl HB/FB1 ratio (mean, 8.39) was higher compared with the corresponding HB/FB2 ratio (mean, 3.48). These results suggest that postmortem redistribution of fentanyl can occur in FB.

The cause and manner of death established by forensic authorities in cases involving single or mixed drug ingestion must take into consideration postmortem redistribution (PMR). PMR is defined as the variation of drug concentration in blood samples taken from different sites (heart blood [HB] and femoral blood [FB]) and can be affected by the postmortem interval, or the time of death to specimen collection. As the interval between death and collection of blood becomes longer, drugs from tissues and organs that contain high drug concentrations redistribute owing to cadaver decomposition, leading to increased drug concentration in the blood. During the postmortem interval, drugs from the gastrointestinal tract, lungs, heart, or liver may travel via diffusion through blood vessels or via direct diffusion into other organs or vessels.1-3 Drugs that are highly concentrated in the liver, lungs, or myocardium redistribute quickly into HB, causing concentrations to increase. Examples of these drugs include tricyclic antidepressants, morphine, and flecainide.4-9 Other factors, such as blood coagulation and hypostasis or movement of the cadaver before sampling, may cause PMR.7,3 Mechanisms of PMR may also be affected by a particular drug’s characteristics, such as lipophilicity, volume of distribution, and pH status (acidic, basic, or neutral). For example, basic, highly lipophilic drugs with a volume of distribution greater than 3 L/kg (fentanyl, 3-8 L/kg) are particularly susceptible to PMR.3

The most common postmortem samples used for forensic toxicologic analysis include FB (peripheral), HB (central), urine, liver tissue, gastric contents, and vitreous humor. For most drugs, peripheral blood is regarded as the optimal sample for interpretation based on its greater distance from organs that may be influenced by PMR mechanisms.3,4 However, studies have emerged that demonstrate alternative samples as

Upon completion of this activity you will be able to:

- describe the mechanisms that influence postmortem redistribution of drugs and the most common characteristics of a drug that cause it to redistribute after death.
- state the specimen types that may be most accurate for determination of fentanyl concentration in forensic toxicology.
- describe why interpretation of blood fentanyl concentrations in cause of death determinations can be difficult.

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more accurate in reflecting perimortem drug concentrations for particular drugs. For example, it has been shown that liver concentrations better represent true body burden of tricyclic antidepressants, which are highly concentrated in tissues that may break down after death and cause falsely increased concentrations of the drug in blood.\(^5\)\(^6\) In the same way, the unique PMR patterns of each type of drug must be taken into account in creating a complete picture of the perimortem conditions.

Fentanyl is a high-potency, rapid-onset synthetic opioid drug prescribed for the treatment of chronic pain and used as a surgical anesthetic.\(^10\) In the past decade, fentanyl misuse and abuse have steadily been on the rise.\(^11\)\(^-\)\(^16\) Fentanyl toxicity causes decreased respiratory rate and depth, delirium, hypotension, bradycardia, and decreased body temperature, leading to death if untreated. As with all opioids, long-term fentanyl use often leads to tolerance, and increasing amounts of the drug are required to maintain its effects, putting its users at risk for developing a dangerous dependence. Because of these tendencies, fentanyl contribution to the cause and manner of death in medical examiner cases is complicated by the high amounts of the drug present in long-term users. This makes the decision of the medical examiner or coroner more difficult when determining whether increased fentanyl blood concentrations beyond what was expected from a patient’s clinical history contributed to the patient’s death. Considerable overlap exists between blood fentanyl concentrations in deaths attributed to fentanyl alone and those attributed to mixed drug overdose, along with the concentrations found in fentanyl-related overdose deaths compared with hospitalized patients being treated for chronic pain.\(^14\)

The possibility of a drug concentration falsely increasing in a blood sample owing to the lengthening of a postmortem interval makes defining therapeutic, toxic, and lethal ranges more difficult. A study of 25 cases by Anderson and Muto\(^15\) on the postmortem distribution of fentanyl led to their conclusion that liver concentrations less than 31 \(\mu\)g/kg appear to represent therapeutic use, and liver concentrations greater than 69 \(\mu\)g/kg seem to indicate overdose situations. The primary purpose of the present study was to determine whether PMR of fentanyl occurs in FB.

### Materials and Methods

**Specimens**

The experimental design used in this study was approved by the institutional review board for human subjects research. FB (FB2), HB, heart tissue, skeletal muscle tissue, and liver tissue were collected at autopsy in a series of 20 fentanyl-related death cases from the Hennepin County (Minneapolis, MN) and Ramsey County (St Paul, MN) Medical Examiners’ Offices in 2007 and 2008. Data from 9 cases reported in 2008\(^16\) (comprising 25% of the data reported in this study) are included in the current analysis. Blood samples were collected in EDTA or sodium fluoride tubes, and liver (right lobe) tissue, heart (left ventricle) tissue, and skeletal muscle tissue were stored without preservative in plastic vials. Unique to the current study was a subset of 7 cases that had FB specimens collected both shortly after death (FB1, at entry to the morgue) and again hours later at autopsy (FB2). The FB1 and FB2 samples were collected by placing a syringe in the femoral vein after anatomic dissection for visual localization of the femoral vein. At each time point, 5 mL of EDTA whole blood (purple-top tube) was collected without removing the syringe between draws. The bodies were moved after the FB1 draw and stored refrigerated until autopsy and FB2 draw. Samples were stored at 4°C and analyzed by gas chromatography–mass spectrometry (GCMS) within 24 to 72 hours of collection. Otherwise they were frozen at \(-20°C\) until analysis.

**Materials**

A fentanyl standard (100 \(\mu\)g/mL) and a deuterated fentanyl internal standard (100 \(\mu\)g/mL) were obtained from Cerilliant (Round Rock, TX). Bond Elut solid-phase extraction columns were purchased from Varian (Harbor City, CA). All solvents used were analytic grade.

**Instrumentation**

All fentanyl analyses were performed using a bench-top GCMS that consisted of a Hewlett-Packard (HP) 6890 series GC, interfaced with an HP 5973 quadrupole MS (Agilent Technologies, Santa Clara, CA). The GCMS was operated with a transfer line temperature of 280°C and a source temperature of 230°C. The MS was tuned on a daily basis using perfluorotributylamine. The electron multiplier voltage was set at 700 eV above the tune value.

Chromatographic separation was achieved using an Agilent HP-5MS 5% phenyl methyl siloxane capillary column (30 m \(\times\) 0.25 mm internal diameter; 0.25 \(\mu\)m film thickness; Agilent Technologies). The HP 7683 6890 autosampler (Agilent Technologies) was used to inject 3 \(\mu\)L of extract into the GCMS. The GC was equipped with a split/splitless injection port operated at 225°C in the splitless mode with the purge time of 2.00 minutes. The oven temperature profile was established as follows: 100°C to 270°C at 30°C/min and a final hold time of 5.50 minutes, resulting in a total run time of 11.17 minutes. The MS was operated in the selected ion-monitoring mode, and the following ions were monitored: quantitating ion 245 and qualifier ions 146 and 189 for fentanyl and quantitating ion 250 and qualifier ions 151 and 194 for fentanyl-d5. The GC was equipped with a 30-m DB-5 capillary column (Agilent Technologies), with helium as the carrier gas set at a flow rate of 1.8 mL/min.
Procedures

Whole blood and liver, heart, and skeletal muscle tissue specimens were quantitated for fentanyl as described previously. Internally prepared whole blood low (4 μg/L) and high (8 μg/L) controls were reanalyzed in each run along with the case specimens. The extraction consisted of 1 mL of whole blood or 1 mL of homogenate (5 g of tissue in 5 mL of water thoroughly homogenized with a blender) mixed with 10 μL of fentanyl-d5 internal standard (10 mg/L) and 3 mL of water and vortex mixed. After sitting for 5 minutes, it was centrifuged at 1950g for 15 minutes, and the pellet was discarded. To the supernatant, 3 mL of 100 mmol/L phosphate buffer was added and the pH adjusted to 6. The specimen was transferred onto a solid-phase extraction column preconditioned with methanol, water, and 100 mmol/L phosphate buffer. Following treatments with water, 100 mmol/L acetate buffer, and methanol, the column was eluted with methylene chloride, isopropanol, and ammonium hydroxide (78:20:2). The eluent was evaporated at 30°C to 40°C with nitrogen, reconstituted with 0.05 mL of ethyl acetate, and transferred for analysis into the autosampler for injection on the GCMS.

GCMS parameters were as follows: Linear standard curves were derived for each analysis using 2.0-, 5.0-, 10.0-, and 20.0-μg/mL whole blood calibrators, and area ratios for unknowns were used to calculate the corresponding analyte concentration. Quantitation of fentanyl was based on ratios of integrated ion areas to the corresponding deuterated internal standard. Analytes were identified based on comparison of relative retention times (within 1% of retention time of calibrators) and ion ratios with the corresponding values of calibrators assayed in the same run. Ion ratios were calculated by dividing the area of the qualifier ion by the area of the quantitative ion and were required to be within ±20% of calibrators. Limits of detection (LoD), quantitation, and linearity were experimentally found to be 2.0, 2.0, and 100 μg/L, respectively. Any concentration that was less than 2.0 μg/L (LoD and limit of quantitation) was considered undetectable. Any sample greater than 20 μg/L was diluted and reanalyzed to achieve a quantitation concentration within the calibration curve. Intra-assay precision was determined to be 4.9% by assaying 5 replicates of fentanyl-fortified blood samples at 2.0 μg/L. Interassay precision was determined to be 10.6% during a 6-month period using the blood low control (n = 22). Several drugs, including opiates, benzodiazepines, cocaine, antidepressants, amphetamines, and antiepileptic drugs reported in the present study, showed no interference with the assay used to quantitate fentanyl.

Data Analysis

The mean and range of fentanyl concentrations by specimen type and time of collection and the ratios between specimen types and time of collection were determined. Cases were separated and evaluated based on whether fentanyl was the sole cause of death or part of a mixed drug overdose. For calculations that used fentanyl concentrations reported below the limit of detection as less 2.0 μg/L (ND, none detected or zero), a conservative value of 1.9 μg/L was used instead of zero, so as not to skew the data analysis when changes over time in the 7 paired FB collection cases were analyzed.

Results

Table 1 describes fentanyl concentrations for FB1 (n = 7), FB2 (n = 20), HB (n = 17), liver tissue (n = 19), heart tissue (n = 10), and skeletal muscle (psoas) tissue (n = 2). Qualitative and quantitative (when available) information regarding other drugs that were detected in HB samples is also noted. Information pertaining to the route of fentanyl administration, cause of death, and manner of death obtained from medical examiner case notes is included as available. All cause and manner of death decisions were determined by the medical examiner. The time from declaration of death to collection for all autopsy samples ranged from 3 to 53 hours (mean, 16.4 hours). Three cases (cases 6, 19, and 20) had fentanyl as the sole drug detected. In 7 cases, fentanyl toxicity was the cause of death (cases 1-7), and 9 cases (cases 8-16) were mixed drug overdoses. In 4 cases, deaths were of natural causes (cases 17-20). Most of the cases (n = 16) involved transdermal patches, the majority through application to the skin. Two cases involved probable intravenous administration after removing fentanyl from a patch (found at the scene; cases 6 and 16), and 1 case (case 7) involved oral ingestion of fentanyl from a patch. The majority (n = 14) of deaths were accidental, with the remaining cases classified as natural (n = 4) or undetermined (n = 2).

For the 7 cases that had both FB1 and FB2 collections, the time from death to blood collection ranged from 2.5 to 6.0 hours (mean, 4.0 hours) and 7.0 to 53 hours (mean, 21.6 hours), respectively. There was a mean difference of 17.6 hours between FB1 and FB2 collection times. FB1 and FB2 fentanyl concentrations ranged from ND to 14.6 μg/L (mean, 4.6 μg/L) and 2.0 to 52.5 μg/L (mean, 17.3 μg/L), respectively. As shown in Figure 1 and Table 2, 4 of 7 cases (cases 5, 8, 16, and 17) had fentanyl concentrations that increased from ND in FB1 to quantitatable in FB2, 1.5 to 50 hours later. HB concentrations ranged from 5.0 to 58.8 μg/L (mean, 29.8 μg/L). The HB/FB fentanyl ratio was always higher for the FB1 collection compared with the FB2 collection, ranging from 1.00 to 17.26 (mean, 8.39) for HB/FB1 compared with 0.94 to 14.91 (mean, 3.48) for HB/FB2 (Table 2).

A poor correlation (r = 0.18; P = .696) was found between the change in concentration between FB1 and FB2 when compared with the time elapsed between these 2 collections.
Table 1
Fentanyl Concentrations in Blood and Tissue Samples in 20 Fentanyl-Related Medical Examiner Cases

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Blood (μg/L)</th>
<th>Tissue (μg/kg)</th>
<th>Other Drugs Detected</th>
<th>Route of Drug Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FB1</td>
<td>FB2</td>
<td>Heart</td>
<td>Liver</td>
</tr>
<tr>
<td>1</td>
<td>—</td>
<td>13.0</td>
<td>14.3</td>
<td>170.0</td>
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<tr>
<td>2</td>
<td>5.0</td>
<td>5.1</td>
<td>5.0</td>
<td>48.0</td>
</tr>
<tr>
<td>3</td>
<td>—</td>
<td>16.0</td>
<td>32.4</td>
<td>75.5</td>
</tr>
<tr>
<td>4</td>
<td>—</td>
<td>20.1</td>
<td>20.4</td>
<td>129.0</td>
</tr>
<tr>
<td>5</td>
<td>ND</td>
<td>26.4</td>
<td>24.7</td>
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</tr>
<tr>
<td>6</td>
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<td>7</td>
<td>14.6</td>
<td>27.6</td>
<td>52.0</td>
<td>157.6</td>
</tr>
<tr>
<td>8</td>
<td>ND</td>
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<td>—</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>—</td>
<td>4.8</td>
<td>—</td>
<td>57.2</td>
</tr>
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<td>—</td>
<td>5.0</td>
<td>32.6</td>
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<tr>
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<td>—</td>
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<td>28.2</td>
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<tr>
<td>17</td>
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<td>—</td>
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<td>137.0</td>
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<td>8.3</td>
<td>16.1</td>
<td>37.0</td>
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<tr>
<td>20</td>
<td>—</td>
<td>2.7</td>
<td>3.2</td>
<td>53.0</td>
</tr>
</tbody>
</table>

ASHD, atherosclerotic heart disease; BE, benzoylecgonine; FB1, femoral blood collection 1; FB2, femoral blood collection 2 (at autopsy); FO, fentanyl overdose; IV, intravenous; MDO, mixed drug overdose; ND, none detected; SCC, squamous cell carcinoma; TP, transdermal patch; —, specimen not obtained.

* Drug concentration not quantitated, but reported as qualitatively positive.

Case 6 was an example of FB PMR, showing an FB1 fentanyl concentration of 4.7 μg/L that increased to 52.5 μg/L 9 hours later at the time of the FB2 collection. In contrast, case 2 did not demonstrate any PMR, with FB1 and FB2 concentrations of 5.0 and 5.1 μg/L, respectively. The mean liver tissue/FB fentanyl ratio decreased during the postmortem interval from 40.7 (at FB1) to 17.8 (at FB2). The mean ratios for liver tissue/HB and heart tissue/HB were 6.97 and 6.37, respectively.

For all 20 cases, FB fentanyl concentrations ranged from ND to 52.5 μg/L (mean, 11.9 μg/L). HB fentanyl concentrations ranged from 3.2 to 58.8 μg/L (mean, 22.6 μg/L). Liver tissue fentanyl concentrations ranged from 37.0 to 179.0 μg/kg (mean, 96.5 μg/kg). Heart tissue fentanyl concentrations ranged from 24.8 to 216.0 μg/kg (mean, 105.2 μg/kg). The 2 skeletal muscle (psosas) specimens had fentanyl concentrations of ND for case 8 and 7.7 μg/kg for case 6, similar to the concentrations found in the FB1 collection for the same cases, ND and 4.7 μg/L, respectively. The HB/FB2 ratio ranged from 0.76 to 14.91 (mean, 3.48). The liver/FB2 ratio...
ranged from 0.86 to 66.82 (mean, 12.27). The heart tissue/FB2 ratio ranged from 1.91 to 12.11 (mean, 6.21). A poor correlation was found between the liver fentanyl concentration and the FB2 fentanyl concentration (r < 0.1; P = .802).

Discussion

The observations of the present study are unique in that they demonstrate PMR of fentanyl in FB. No other study has examined 2 blood samples collected during the postmortem interval from the same peripheral femoral site in fentanyl-related medical examiner death investigations. Table 2 and Figure 1 showed that PMR increases occurred in 4 (57%) of 7 cases (cases 5, 6, 7, and 17). Furthermore, 4 cases demonstrated increases from undetectable concentrations (below the LoD) in FB1 to measurable concentrations in FB2 of 26.4 μg/L (case 5), 2.0 μg/L (case 8), 2.2 μg/L (case 16), and 5.5 μg/L (case 17) during postmortem intervals of 20, 19, 1.5, and 50 hours, respectively. No relationship is drawn from the length of time between the FB1 draw and the FB2 draw. No apparent explanation as to why case 2, despite an 11-hour time difference between sample collections, did not exhibit PMR. Cases 8 and 16 were inconclusive.

The literature in general suggests that drugs with a high ratio of HB to FB at autopsy (HB/FB2) have a greater potential.
for PMR. For fentanyl, studies have cited the HB/FB2 ratio ranging from 0.7 to 4.6 (mean, 1.6) in 13 deaths and 0.5 to 11.0 (mean, 2.7) in 80 deaths. For the 17 cases in the present study, the HB/FB2 ratio (mean, 2.36) was similar to the previous findings. Without case 16, the mean ratio was 1.57. However, the present study’s observations showed that the HB/FB ratio was influenced by the postmortem collection time in 57% of cases. A direct correlation of postmortem interval and ratio could not be established. In the 7 cases with 2 FB collections, the HB/FB1 and HB/FB2 ratios ranged from 1.00 to 17.26 (mean, 8.39) and 0.94 to 14.91 (mean, 3.48), respectively; a 241% decrease.

Multiple mechanisms of PMR have been discussed previously as to why HB obtained at autopsy should not be an acceptable forensic specimen for interpreting fentanyl blood concentrations. The influence of how fentanyl is administered (by patch, intravenously, or orally) has never been described. Additional studies are needed to address this issue. The present study demonstrated the following: (1) FB concentrations (4 of 7 cases demonstrated PMR) may increase during the postmortem interval, thereby more closely aligning with the HB fentanyl concentration (Table 2). (2) Liver and heart tissues may contain high fentanyl concentrations (mean, 96.5 and 105.2 μg/kg, respectively) that may contribute to PMR. (3) Tissue (heart or liver) to FB2 fentanyl ratios are very high (eg, 17.83 for liver/FB2) and are even higher with earlier FB collection (40.73 for liver/FB1).

Based on the current observations of FB PMR in 4 of 7 cases, the reliability of whether the fentanyl concentration measured postmortem can be assumed to be the same concentration that would have been present at the time of death is questioned. With the exception of case 8, the cases had a substantially higher liver or heart tissue fentanyl concentration compared with FB1 and FB2. Although the present study was not designed to validate the use of liver fentanyl concentrations in cause of death investigations, the suggested cutoff concentrations made by Anderson and Muto that therapeutic fentanyl use results in liver concentrations of less than 31 μg/kg, and that fentanyl overdoses give liver concentrations of more than 69 μg/kg leaving an intermediate range of 31 to 69 μg/kg, should be more carefully examined. Liver fentanyl concentrations are unlikely to be substantially influenced by PMR.

In the present study, liver fentanyl concentrations were quite variable, but all were greater than 31 μg/kg. Two natural death cases involving long-term use of prescribed 100 μg/h patches for treatment of terminal metastatic cancer (cases 17 and 18) revealed liver fentanyl concentrations of 99.4 and 137.0 μg/kg, respectively. In contrast, in case 19 with heart disease as the cause of death, following 6 days of prescribed 100 μg/h patches, the liver concentration was 37.0 μg/kg. Cases 17 and 18 both had a natural cause of death determination, but owing to long-term fentanyl use of 100 μg/h and 2 × 100 μg/h patches, had liver concentrations of more than 69 μg/kg. At the other extreme, cases 2 and 6, both certified as sole fentanyl overdoses, had liver concentrations of 48 and 45 μg/kg. Both were determined to be acute exposures in fentanyl-naive people. We have now implemented the routine toxicologic analysis of liver, along with FB, in all fentanyl-related cases.

The present study provides evidence that suggests that postmortem FB may not be reliable for the determination of the fentanyl concentration present at the time of death, ie, perimortem. Medical examiners and coroners need to be educated on the importance of this study’s observations before the use of blood fentanyl concentrations in cause of death interpretations. Forensic scientists must understand that FB concentrations may increase with postmortem interval and must consider this fact in their interpretation. The present study did not attempt to reclassify cause of death based on liver concentrations. We recommend measurement of FB fentanyl concentrations, along with the measurement of liver fentanyl concentrations, to improve the database to validate the initially proposed 31 and 69 μg/kg liver cutoffs for the therapeutic and potential overdose exposures. Each case needs careful individual determination of the route and extent of exposure to fentanyl and whether the use was in a naive or long-term user along with the toxicology findings. The fentanyl toxicology findings as described in the present study must be balanced with the entire death investigation.

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Dr Apple consults in medicolegal cases that involve fentanyl concentrations in postmortem blood and tissue.

References