مجله پزشکی قانونی نوین
فهرست

سر برند اسپیلسری ۲

اهمیت بررسی آنتروپولوژیک صحن جرم در موارد پیمانای سوخته در خودرو ۱۳

گزارش یک مورد خودکشی با چهار چنگی تفگه نیزه انداز ۲۰

پارگی و آگوار با شکستگی مهره گردنی پس از تلاش ناموفق برای خودکشی با دارآویختنگی ۲۵

تروموو آمبولی سیستمیک پس از تروموز ورید عمیق ناشی از میوم رحمی ۲۸
سر برند اسپینسیری

یک بررسی بر موارد کالبدگشایی او از اسناد کتابخانه ولکام لندن

چکیده: سر برند اسپینسیری در طول حیاتش به عنوان "پدر پزشکی قانونی" شناخته می‌شد. این عنوان به واسطه چندین پرونده معروف به او داده شده بود. چندین مقاله در مورد زندگی و کار او نوشته شده است. اما به دلیل فقدان دسترسی به اسناد اینجا شده توسط خود اسپینسیری انجام یک بررسی عینی مشکل است. میراث اصلی از مجموعه‌ای از کارتهای است که سالانه متمادی در دسترس پژوهشگر نیستند. در سال 2008 مجموعه‌ای شامل 4000 کارت متعلق به اسپینسیری توسط کتابخانه ولکام لندن خریداری شد و در نتیجه در اختیار عموم قرار گرفت. در این مقاله ما گزارشی از بررسی انجام شده بر روی 650 عده از این کارتهای ارائه می‌دهیم. ما گزارش‌های موجود در کار اسپینسیری و چندین مورد خاص را دقیق تر بررسی خواهیم کرد این کارتها نمای عینی بر از کار روزمره اسپینسیری را نشان داده و تصویر می‌کنیم اکنون موقعیت ارزیابی مجدد میراث او است.
Sir Bernard Spilsbury
A Survey and Catalogue of His Autopsy Case Cards
From the Wellcome Library, London

Megan L. Walmsley, BSc and Matthew John Almond, DPhil

Abstract: During his lifetime, Sir Bernard Spilsbury was referred to as the “father of forensic medicine.” He became a household name as a result of several famous cases. Several articles have been written about his life and work, but an objective assessment has proved difficult because of the lack of available material that Spilsbury himself produced. His main legacy has been a series of case cards, but for many years these were unavailable to the researcher. In 2008, a collection of some 4000 of Spilsbury’s case cards was bought by The Wellcome Library in London and therefore entered the public domain. In this article, we report our study of 650 of these cards. We discuss trends in Spilsbury’s work and several specific cases in more detail. These cards allow an objective view to be taken of Spilsbury’s everyday work, and we feel that some reappraisal of his legacy is now timely.

Key Words: forensic pathology, forensic science, autopsy, Spilsbury, Wellcome Library

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The life of Sir Bernard Spilsbury has been well documented elsewhere. However, it is perhaps useful to mention here some of the major events of his life and to note that his more famous cases have previously been reviewed in this journal. These cases led him becoming a household name at the height of his career and to being referred to as the “father of forensic medicine—the greatest forensic expert of all time.”

He was born on May 16, 1877, at 35 Bath St, Leamington Spa, the eldest of 4 children of James Spilsbury—a manufacturing chemist—and his wife Marion. In 1899, he graduated with a bachelor of arts degree in natural science from Magdalen College, Oxford. He enrolled as a medical student at St Mary’s Hospital, Paddington, and graduated MBBCh (Bachelor of Medicine, Bachelor of Surgery) in 1905. He obtained his master of arts degree from Magdalen College in 1908. Upon qualifying in 1905, he was appointed resident assistant pathologist at St Mary’s Hospital. This post required him to perform autopsies following sudden deaths—in line with the request that had very recently been made by London County Council that 2 pathologists should be appointed at each hospital in the region to perform such work. He was strongly influenced during this time by his colleague Dr Pepper, who had encouraged Spilsbury to focus on the field of pathology while he was still a medical student.

Spilsbury’s career was as lecturer in forensic medicine at University College Hospital, London, School of Medicine for Women and St Thomas’ Hospital. He also acted as home office pathologist. He was knighted at Buckingham Palace on February 15, 1923, and in 1933, he served as president of the Medico-Legal Society. On September 3, 1908, Spilsbury married Edith Caroline Horton with whom he lived at 31 Marlborough Hill, London NW8 from 1912 until the bombing of London in 1940, during which one of his sons, Peter, was killed. A second son, Alan, died of tuberculosis in 1945. It is thought to be depression over the deaths of his sons and concerns regarding his financial position that led to his suicide—by carbon monoxide (CO) poisoning—in his laboratory at University College London on December 17, 1947.

Some 30 years ago, in this journal, Eckert reviewed Spilsbury’s career and his famous cases, and this report emphasizes that Spilsbury’s impact on medicolegal science was immense. Eckert pointed out that Spilsbury’s work contributed greatly to the regaining of confidence in expert witness testimony by both the judiciary and the public in England, although his international reputation was never so strong, partly because of his reluctance to collaborate widely. During his career, Spilsbury certainly introduced methods that nowadays would be regarded as essential in any forensic investigation. He developed the use of “murder bags” to store evidence and strongly encouraged the use of gloves, tweezers, bags, and test tubes for collecting evidence to try to avoid contamination. Spilsbury published little, although such writings as he left have been discussed in previous articles in this journal, which describe Spilsbury’s lectures on “The Medico-Legal Significance of Bruises,” “Some Medico-Legal Aspects of Shock,” and 2 short case reports on “Sudden Death From Inhibition” and “A Hat From a Fatal Case of Shooting.” Although these works allow some aspects of Spilsbury’s work and personality to be considered, they describe very specific topics and do not provide a truly balanced overview of his career. To date, most of the information on Spilsbury’s career has come from a number of biographies with more personal information being given in a 1961 address to the Royal Society of Medicine by Keith Simpson who had worked with Spilsbury during the last 12 years of his career. In this address, Simpson refers to the fact that Spilsbury was seen by many to have become, in his later career, inflexible and uncompromising—perhaps even to believe himself infallible. Certainly, Spilsbury’s reputation has suffered since his death. It is said that he would never admit to mistakes and often came to judgments that were felt to be beyond those justified by the evidence. Questions regarding his objectivity have been raised, and his “virtuoso” performances both in the mortuary and courtroom have been criticized. Recent concerns have focused on his insistence on working alone and his failure to submit articles for peer review, to engage in academic research, or to train students. This has led to accusations that it was his reputation rather than objective
science that led juries to give so much credence to his evidence. He also left no textbook and published very little in the academic sphere. These points are explored by Burney and Pemberton\(^1\) in a recent article that focuses on the “Thorne” case of 1925, where the defendant, Thorne, upon conviction for murder, commented that he was a “martyr to Spilsburyism.” In Burney and Pemberton’s article, Spilsbury is referred to as “a new creature on the forensic landscape—the ‘celebrity pathologist.’” It is certainly true that even today Spilsbury’s cases can provoke comment and debate in the UK national press.\(^{12-14}\)

This approach to forensic science is contrasted with the modern approach of trace-oriented crime-scene analysis and team-based pathology, which has displaced the authoritative status of the lone pathologist. Until very recently, however, it has been difficult to make a truly objective assessment of Spilsbury’s career because of the lack of primary source material.

Spilsbury’s main legacy is in fact a series of case cards.\(^{15,16}\) noting the autopsies that he carried out between 1905 and 1946. These cards are each 12.5 x 7.5 cm, written front and back in Spilsbury’s “almost impenetrable handwriting.”\(^2\) A small collection of these, describing his more famous cases, has been held at the Galleries of Justice in Nottingham.\(^3\) However, in 2008, a much more substantial collection of around 4000 of these cards was discovered in a lost cabinet and was sold to The Wellcome Library in London. These cards detail cases from the Home Counties and London County for the period 1905 to 1932. A gift of a further tranche of cards, covering the years 1933 to 1944 (with gaps for 1939 and 1940) with a very few cards for 1945 and 1946, was made to The Wellcome Library in 2009.\(^4\)

These cards are perhaps the only resource remaining that can give a key to the work of this controversial figure. They allow for the first time a more objective assessment of Spilsbury’s work to be made and for his wider contributions to pathology to be assessed. The recent possibility for public access to these case cards also makes a study of this material very timely. Accordingly, in this work, we have made a full catalogue of all of the cards from the Wellcome collection for the years 1911, 1916, 1921, and 1926. In this article, we comment upon our findings in terms of both Spilsbury’s career and the development of forensic pathology in the early 20th century.

**MATERIALS AND METHODS**

It was clear at the outset that to read, interpret, and catalogue every one of the 4000 cards available at The Wellcome Library would be beyond the scope of our survey. As such, some degree of selection was necessary. We decided therefore to fully catalogue cards at 5-year intervals between 1911 and 1926 and to carry a general survey of the cards from 1931. This starting point (1911) was chosen as it was the first year that Spilsbury became a household name following the “Crippen” case in 1910.\(^3,17-19\) By cataloguing cards at 5-year intervals, it was hoped that an overview of Spilsbury’s work would emerge and that changes in the type of case studied or the conclusions drawn by Spilsbury would become apparent. In Figure 1, a series of Spilsbury’s case cards relating to the autopsy of E. Gerard in the case of *Rex v Voisin and Roche*, 1917, are reproduced. These cards have been reproduced by the kind permission of the Galleries of Justice, Nottingham. It may be seen that some problems present themselves in interpreting the data on these cards. First is the matter of reading Spilsbury’s handwriting. Second, there is the problem of interpreting the abbreviations used by Spilsbury, which are unique to him and which he did not publish. We have produced a list of all of the abbreviations that we encountered, and these are reproduced in Table 1. We hope that this list will be of use to future scholars of Spilsbury’s work. In Table 2, we reproduce 1 page of the catalogue (January to March 1911) that we produced to illustrate the information that we obtained from the cards and how we documented this information and to show the type of case that Spilsbury investigated. It is not possible here to reproduce the whole of our catalogue, but this is available to readers from the authors on request.

**RESULTS AND DISCUSSION**

From our catalogue of Spilsbury’s cases from 1911 to 1931, we first report some general trends; we will then look in more detail at a number of specific individual cases. The first trend that we investigated was the most common cause of reported death (Table 2). In 1911, this is anesthetic death—with 35% of all deaths arising from this. Nowadays, anesthetic death is rare. By 1931, coronary heart disease has taken over as the most common cause of death, and this, of course, is still a major killer. In other words, the 1931 data have a much more “modern” look to them than do the 1911 data. Second, we calculated the average and noted the oldest ages of the deceased of the cases studied by Spilsbury. This is listed in Table 3 and illustrated in Figure 2. Both show a steady general increase over the period 1911 to 1931 in line with known life expectancy trends. In studying the reference cards, we also noted that several cases give “stat. lymph.” as the cause of death. This is Spilsbury’s abbreviation for “status lymphaticus,” thought to be constitutional abnormalities including hyperplasia of the lymphoid tissues and thymus gland that could place pressure on the trachea.
and cause sudden death. This condition has been discussed at some length and is now known to be non-existent.20 Many of these cases occur where an anesthetic has been administered, and they can probably be added to the list of anesthetic deaths, emphasizing the unreliability of early anesthetics. In Table 4, we summarize the most common causes of death reported by Spilsbury for the cases that he investigated in the years 1911, 1916, 1921, and 1926.

It is also of interest to note the number of cases studied by Spilsbury and how variable this is from year to year. For example, for 1926, there are only 18 case cards, whereas for 1931 the collection consists of 12 boxes of cards or some 350 cases. Moreover, the amount of detail given on the case cards is very variable. In some cases, for example, the cards include annotated diagrams of injuries, whereas others are very brief. In 1931, in particular, perhaps because of the volume of work, hardly any of the cards include a “history” section, where Spilsbury noted details of the deceased’s life indicating possible contributory factors to their death. In general, the cards are written in a casual, almost informal style, certainly not in the style of a formal pathologist’s report.

In Table 5, we have reproduced our findings from a number of Spilsbury’s reference cards for the years 1911, 1916, 1921, and 1926. Again this is not a comprehensive list. It is intended to illustrate the range of cases that came to Spilsbury’s attention and to show trends and developments that occurred during these 20 years. We feel, however, that this gives a fascinating insight into the work of a forensic pathologist in the early years of the 20th century.

In 1911, it is probably the huge number of anesthetic deaths that stand out most prominently to the modern reader. Chloroform is particularly hazardous as an anesthetic, especially to children whose body mass is low as it is prone to cause cardiac arrhythmia. The action of chloroform can also be delayed as noted, for example, in the cases of A. W. Hamer (4/12/1911), T. F. Foster (4/19/1911), and J. Chandler (4/20/1911). These children were aged 3 and 2 months, 5 and 11 months, and 6 years, respectively, and their autopsies were carried out by Spilsbury in a period of just 8 days. All had had operations on their noses and/or throats. New anesthetics were being introduced, but these were themselves not without hazard. H. Przedecki (4/13/1911) died of the use of veronal (a barbiturate). There were also many other hazards associated with medical treatment at that time. A case of mercury poisoning is noted (F. Hawker: 4/11/1911). Although Spilsbury does not elaborate, mercury compounds were often used to treat sexually transmitted diseases. Another aspect of note that emerges from Spilsbury’s work is that what would nowadays be termed “forensic science” is clearly beginning to be developed for the reconstruction of crimes. The case of A. I. Linfold (4/29/1911) is interesting because it shows that in this murder investigation Spilsbury is considering the angle and position of a wound to determine that the murderer stood behind the victim.

In 1916, we note the 5 females who died of TNT (trinitrotoleuene) poisoning: E. Deebank, reported as the first fatality from handling TNT powder (3/17/1916); D. Willis (10/17/1916); A. Marsh (8/22/1916); L. E. Gibson (10/11/1916); and E. Mead...
In 1916, World War I was at its height, and the production of high explosives including TNT had enormously expanded. The munitions factories were, of course, largely staffed by women. Women also suffered terribly from attempted abortions, many of which caused death. Some of these were investigated by Spilsbury, and some led to prosecutions (eg, L. F. Ellender [5/5/1916], Hamilton [6/15/1916], G. L. Patterson [4/29/1916]). There is terrible human suffering behind the few lines written on the cards by Spilsbury: “Criminal abortion, probable injection of scalding fluid into uterus.”

Rex v Holmes, posed as doctor (G. L. Patterson [4/29/1916]); Rex v Olley, criminal abortion, insertion into cervix of pieces of gum elastic catheter (Hamilton [6/15/1916]). As in 1911, there are several cases where arsenic or mercury compounds have been ingested as attempted cures for sexually transmitted diseases. A. M. Goodwin (8/11/1916) died of use of the arsenic compound Kharsivan—dioxydiamido-arseno-benzoldihydrochloride. H. Jackson (8/22/1916) and A. Moss (10/27/1916) also died, and L. Roberts (10/21/1916) fared no better from use of neosalvarsan, which had been introduced in 1912 as a less toxic alternative to other arsenic treatments. Spilsbury notes that he discovered arsenic in all major organs and 9 mg of neosalvarsan in total in the body. We note also the 2 who were killed (M. Coventry and B. Stockwell [3/23/1916]) when they were given strychnine rather than butyl chloride hydrate by their chemist and E. M. Beckerson (7/29/1916) who died of starvation after being recommended to fast by H. Bundec who claimed to be a hydropathist. Bundec was subsequently convicted of manslaughter. We also note B. A.

This table shows cases that Spilsbury examined between January 16, 1911, and March 28, 1911.

### TABLE 2. Page 1 of the Catalogue That Has Been Produced in This Work Following Inspection of Sir Bernard Spilsbury’s Case Cards for the Years 1911, 1916, 1921, 1926, and 1931

<table>
<thead>
<tr>
<th>Date of Autopsy</th>
<th>Name</th>
<th>Sex</th>
<th>Age</th>
<th>Cause of Death</th>
<th>Other Notes</th>
<th>Author’s Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/16/1911</td>
<td>Britten, W.</td>
<td>M</td>
<td>35 yr</td>
<td>Anesthetic death</td>
<td>From tracheostomy wound</td>
<td></td>
</tr>
<tr>
<td>1/28/1911</td>
<td>Boize, L. W.</td>
<td>M</td>
<td>35 yr</td>
<td>Toxic goiter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/28/1911</td>
<td>Saunders, E.</td>
<td>M</td>
<td>14 yr</td>
<td>CO poisoning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/30/1911</td>
<td>Potter, B.</td>
<td>F</td>
<td>4 yr</td>
<td>Misadventure and stat. lymph.</td>
<td>Given anesthetic</td>
<td>Stat. lymph. now disregarded</td>
</tr>
<tr>
<td>2/3/1911</td>
<td>Lamb, K.</td>
<td>F</td>
<td>49 yr</td>
<td>Choked on regurgitated vomit material while under anesthetic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2/6/1911</td>
<td>Penney, E. M.</td>
<td>F</td>
<td>17 yr</td>
<td>Pneumonia and pleurisy</td>
<td></td>
<td>No ptomaine poison</td>
</tr>
<tr>
<td>2/9/1911</td>
<td>Larrance, A.</td>
<td>F</td>
<td>26 yr</td>
<td>Rupture of right frontal lobe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2/13/1911</td>
<td>Alleyne, M.</td>
<td>F</td>
<td>48 yr</td>
<td>Anesthetic death</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2/20/1911</td>
<td>Barne, R.</td>
<td>M</td>
<td>57 yr</td>
<td>Acute intestinal obstruction</td>
<td>Gallstone operation</td>
<td></td>
</tr>
<tr>
<td>2/21/1911</td>
<td>Teague, E.</td>
<td>F</td>
<td>29 yr</td>
<td>Heart failure</td>
<td>Gallstone operation</td>
<td></td>
</tr>
<tr>
<td>3/4/1911</td>
<td>Eames, S. W.</td>
<td>M</td>
<td>14 yr</td>
<td>Anesthetic death</td>
<td></td>
<td>Acute appendicitis</td>
</tr>
<tr>
<td>3/7/1911</td>
<td>Mason, G.</td>
<td>M</td>
<td>5 mo</td>
<td>Pneumonia and emphysema</td>
<td></td>
<td>Emaciated</td>
</tr>
</tbody>
</table>

- **TABLE 3. Most Common Cause of Death, Average and Oldest Ages at Death of Individuals Whose Autopsies Were Performed by Spilsbury for the Years 1911, 1916, 1921, 1926, and 1931**

<table>
<thead>
<tr>
<th>Year</th>
<th>Most Common Cause of Death</th>
<th>Percentage of Deaths Recorded as Most Common Cause, %</th>
<th>Average Age of Deceased, yr</th>
<th>Oldest Deceased, yr</th>
<th>Percentage of Autopsies Carried Out on Children (&lt;18 yr), %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1911</td>
<td>Anesthetic death</td>
<td>35</td>
<td>31</td>
<td>68</td>
<td>54</td>
</tr>
<tr>
<td>1916</td>
<td>Septicemia and peritonitis</td>
<td>13</td>
<td>29</td>
<td>76</td>
<td>29</td>
</tr>
<tr>
<td>1921</td>
<td>Poisoning</td>
<td>14</td>
<td>34</td>
<td>74</td>
<td>21</td>
</tr>
<tr>
<td>1926</td>
<td>Pulmonary embolism</td>
<td>33</td>
<td>38</td>
<td>78</td>
<td>22</td>
</tr>
<tr>
<td>1931</td>
<td>Coronary artery disease</td>
<td>22</td>
<td>46</td>
<td>87</td>
<td>13</td>
</tr>
</tbody>
</table>
Pantrey (5/1/1916) who died in childbirth and Spilsbury’s subsequent autopsy, which led to a successful manslaughter prosecution of the doctor—Dr Heskin—who, Spilsbury notes, separated the child but did not attempt to remove the afterbirth and was drunk at the time. Outside the munitions factories, there were other industrial accidents including the worker (E. Norvell [1/27/1916]) who died of chronic lead poisoning after cleaning lead batteries. These cases emphasize the lack of regulation in particular health and safety regulation at the time. In 1916, Spilsbury begins to investigate a number of cases that arise from motor accidents. For example, J. Sloughgrove (11/7/1916) dies as a result of being knocked down by a van with fractured ribs and spine. Cases of electrocution also begin to be seen such as J. B. Edward (8/27/1926) who died following a fall caused by electrocution; J. B. Edward (8/27/1926) died of a fractured skull following the administration of anesthetics. F. W. Graham (8/23/1926) died of a fractured skull following a fall caused by electrocution; J. B. Edward (8/27/1926) died of a fractured skull following a fall caused by electrocution.

In a case of potassium cyanide poisoning, tests for hydrogen cyanide (HCN) have been made, and it is noted that the stomach contents were alkaline. In the case of I. M. Hall (8/15/1921), a suicide case who had died of CO poisoning, Spilsbury notes the mumification and presence of maggots. This type of observation has developed into an area of study—forensic entomology—which is now of wide importance. In 2 murder cases (S. Seabrooke [1/29/1921] and H. J. Blackmore [3/18/1921]), Spilsbury gives detailed notes about the nature of the weapon used from his investigation of the wounds, and in the case of Blackmore, annotated diagrams of the injuries are given.

An interesting point to note is that between 1911 and 1916, more accurate scientific methods are being used. In CO poisoning cases, blood concentrations of CO are now being reported by Spilsbury, although the “Haldane” method used may in fact give results that are not completely accurate because of the erroneous belief at that time that CO had no reaction in the body except that with hemoglobin. A homicide case—that of V. Rainlow (11/12/1916)—where the cause of death is given as shock from immersion in water is highly reminiscent of the “brides in the bath” case on which Spilsbury had worked a year earlier. Spilsbury’s autopsy showed no trace of poison in the body, and he discounted death by drowning because there was no watery fluid or froth in the lungs.

In 1921, we note again the precise scientific nature of many of Spilsbury’s investigations. Carbon monoxide concentrations are given for CO poisoning cases (C. L. Busby [12/2/1921], E. F. Stead [12/14/1921]), the exact mass of morphine (13.05 g) found in the body of R. M. Leslie (4/1/1921) is stated. In a case of potassium cyanide poisoning, tests for hydrogen cyanide (HCN) have been made, and it is noted that the stomach contents were alkaline. It is also noted that the stomach contents were alkaline. In the case of I. M. Hall (8/15/1921), a suicide case who had died of CO poisoning, Spilsbury notes the mumification and presence of maggots. This type of observation has developed into an area of study—forensic entomology—which is now of wide importance. In 2 murder cases (S. Seabrooke [1/29/1921] and H. J. Blackmore [3/18/1921]), Spilsbury gives detailed notes about the nature of the weapon used from his investigation of the wounds, and in the case of Blackmore, annotated diagrams of the injuries are given.

Medical treatment remained hazardous in 1921. There are a number of anaesthetic deaths including E. Laker (12/22/1921) who died when Stovaine—the first synthetic local anaesthetic—was injected into her spine. W. A. Overin (6/10/1921), M. R. Hare (7/6/1921), and G. B. Grave (12/23/1921) also died following the administration of anaesthetics. C. King (5/25/1921) died after oil of Chenopodium was used to treat amebic dysentery. W. M. Bolton (9/26/1921) died having taken HgCl2 tablets. J. Bird (2/4/1921) died of shock following the removal of his teeth. E. Whittle (5/26/1921) died of a tumor; we note here the use of radium, presumably as a treatment, which had been introduced into the tumor.

Modern life was also presenting new hazards, and in 1921, we note the case of P. C. Sweeney (10/1/1921), who was trapped in a lift and crushed. In 1926, we note also 2 cases of electrocution. F. W. Graham (8/23/1926) died of a fractured skull following a fall caused by electrocution; J. B. Edward (8/27/1926) died of a fractured skull following a fall caused by electrocution.

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### Table 4. Statistical Summary (Expressed as Percentages of Total Deaths) of the Most Common Causes of Death of Individuals Whose Autopsies Were Performed by Spilsbury for the Years 1911, 1916, 1921, 1926, and 1931

<table>
<thead>
<tr>
<th>Year</th>
<th>Anesthetic death</th>
<th>Heart/cardiovascular disease</th>
<th>Poisoning</th>
<th>Peritonitis</th>
<th>Septicemia</th>
<th>Choking/asphyxia</th>
<th>Head injury (murder)</th>
<th>Stomach lymphatic</th>
<th>TNT poisoning</th>
<th>Failed abortion</th>
<th>Cancer</th>
<th>Burns</th>
<th>Hemor</th>
</tr>
</thead>
<tbody>
<tr>
<td>1911</td>
<td>35</td>
<td>11</td>
<td>8</td>
<td>7</td>
<td>5</td>
<td>12</td>
<td>4</td>
<td>39</td>
<td>39</td>
<td>39</td>
<td>39</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>1916</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>1921</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1926</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**Note:**
- The values represent the percentage of deaths attributed to each cause.
- The table does not include percentages for cases not listed.

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**Graph Illustrating the Average and Oldest Age of the Deceased from Spilsbury’s cases for the years 1911, 1916, 1921, 1926, and 1931.**

**Figure 2.** Graph illustrating the average and oldest age of the deceased from Spilsbury’s cases for the years 1911, 1916, 1921, 1926, and 1931.
<table>
<thead>
<tr>
<th>Date of Autopsy (Month/Day/Year)</th>
<th>Name</th>
<th>Sex</th>
<th>Age</th>
<th>Cause of Death</th>
<th>Other Notes</th>
<th>Author’s Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/28/1911</td>
<td>Saunders, E.</td>
<td>M</td>
<td>14 yr</td>
<td>CO poisoning</td>
<td>Spilsbury noted an “overpowering suffocating smell”</td>
<td></td>
</tr>
<tr>
<td>2/21/1911</td>
<td>Teague, E. F</td>
<td>F</td>
<td>29 yr</td>
<td>Heart failure</td>
<td>Full-term pregnancy, puerperal fever</td>
<td></td>
</tr>
<tr>
<td>3/7/1911</td>
<td>Mason, G. M</td>
<td>M</td>
<td>5 mo</td>
<td>Pneumonia and emphysema</td>
<td>Emaciated</td>
<td></td>
</tr>
<tr>
<td>4/11/1911</td>
<td>Hawker, F.</td>
<td>F</td>
<td>18 yr</td>
<td>Mercury poisoning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4/12/1911</td>
<td>Hamer, A. W.</td>
<td>M</td>
<td>3 and 2 mo</td>
<td>Delayed chloroform poison</td>
<td>Operation on nose and throat</td>
<td></td>
</tr>
<tr>
<td>4/13/1911</td>
<td>Przedecki, H.</td>
<td>F</td>
<td>27 yr</td>
<td>Veronal poisoning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4/19/1911</td>
<td>Foster, T. F.</td>
<td>M</td>
<td>5 and 11 mo</td>
<td>Delayed chloroform poison</td>
<td>Tonsillectomy</td>
<td></td>
</tr>
<tr>
<td>4/20/1911</td>
<td>Chandler, J.</td>
<td>F</td>
<td>6 yr</td>
<td>Delayed chloroform poison</td>
<td>Tonsillectomy and adenoidectomy</td>
<td></td>
</tr>
<tr>
<td>4/29/1911</td>
<td>Linfold, A. I.</td>
<td>F</td>
<td>22 yr</td>
<td>Murder—cut throat</td>
<td>Rex v Pateman murder; diagram of injuries included. Spilsbury noted from the angle and position of the injury that it was a right cutting motion, from which he interpreted that the assailant stood behind the victim to inflict the wound. Chlorine used.</td>
<td></td>
</tr>
<tr>
<td>5/16/1911</td>
<td>Bonden, C.</td>
<td>M</td>
<td>40 yr</td>
<td>Acute ammonia poisoning</td>
<td>Misdventure; stomach contents examined</td>
<td></td>
</tr>
<tr>
<td>11/24/1911</td>
<td>Hooper, E.</td>
<td>M</td>
<td>38 yr</td>
<td>Laudanum poisoning—suicide</td>
<td>Letters and checks found</td>
<td></td>
</tr>
<tr>
<td>1/27/1916</td>
<td>Norvell, E.</td>
<td>M</td>
<td>57 yr</td>
<td>Chronic lead poisoning</td>
<td>laudanum—10% opium, 1% morphine</td>
<td></td>
</tr>
<tr>
<td>2/2/1916</td>
<td>Braddock, J. H.</td>
<td>M</td>
<td>38 yr</td>
<td>Peritonitis</td>
<td>Worked with lead, cleaning batteries, for example</td>
<td></td>
</tr>
<tr>
<td>2/10/1916</td>
<td>Bourne</td>
<td>NB</td>
<td></td>
<td>Hemorrhage and fractured skull</td>
<td>Perforated stomach lining</td>
<td></td>
</tr>
<tr>
<td>2/12/1916</td>
<td>Barry, E. M.</td>
<td>F</td>
<td>49 yr</td>
<td>Blow in the pit of the stomach</td>
<td>Body found in brown paper parcel under seat of railway carriage</td>
<td></td>
</tr>
<tr>
<td>3/17/1916</td>
<td>Deebank, E.</td>
<td>F</td>
<td>49 yr</td>
<td>TNT poisoning</td>
<td>Page of lavatory floor read in her hand “I have burst a blood vessel in my brain”</td>
<td></td>
</tr>
<tr>
<td>3/23/1916</td>
<td>Convent, M.</td>
<td>F</td>
<td>28 yr</td>
<td>Strychine poisoning</td>
<td>1st fatal case of handling powder</td>
<td></td>
</tr>
<tr>
<td>3/23/1916</td>
<td>Stockwell, B.</td>
<td>F</td>
<td>34 yr</td>
<td>Strychine poisoning</td>
<td>Chemist’s mistake, given instead of butyl chloride hydrate</td>
<td></td>
</tr>
<tr>
<td>4/11/1916</td>
<td>Minton, A. T.</td>
<td>M</td>
<td>54 yr</td>
<td>Gas gangrene</td>
<td>As above, died together</td>
<td></td>
</tr>
</tbody>
</table>

*TABLE 5. Catalogue of Selected Autopsies Carried Out by Sir Bernard Spilsbury for the Years 1911, 1916, 1921, and 1926*
<table>
<thead>
<tr>
<th>Date</th>
<th>Name</th>
<th>Age</th>
<th>Gender</th>
<th>Disease/Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>4/12/1916</td>
<td>Crozier, E. P.</td>
<td>M</td>
<td>35 yr</td>
<td>Leprosy</td>
</tr>
<tr>
<td>4/25/1916</td>
<td>Collins, A. M.</td>
<td>F</td>
<td>NK</td>
<td>Placenta praevia—placenta grows over cervix</td>
</tr>
<tr>
<td>4/29/1916</td>
<td>Patterson, G. L.</td>
<td>F</td>
<td>24 yr</td>
<td>Criminal abortion, probable injection of scalding fluid into uterus. Rex v Holmes, posed as doctor</td>
</tr>
<tr>
<td>5/1/1916</td>
<td>Re: Pantrey, B. A.</td>
<td>F</td>
<td>NK</td>
<td>Manslaughter Rex v Dr Heskin; drunk doctor separated child but did not attempt to remove after birth</td>
</tr>
<tr>
<td>5/5/1916</td>
<td>Re: Ellender, L. F.</td>
<td>F</td>
<td>29 yr</td>
<td>Misdemeanor Rex v M. Fedderman, supplying noxious substances with intent to procure miscarriage</td>
</tr>
<tr>
<td>5/10/1916</td>
<td>Brissonetti, A.</td>
<td>M</td>
<td>25 yr</td>
<td>Intestinal obstruction Diaphragmatic hernia caused by previous passage of a bullet; French Canadian soldier</td>
</tr>
<tr>
<td>5/12/1916</td>
<td>Coward, L.</td>
<td>F</td>
<td>29 yr</td>
<td>Chronic Bright disease Evidence of criminal abortion</td>
</tr>
<tr>
<td>5/27/1916</td>
<td>Gleig, J. R.</td>
<td>M</td>
<td>40 yr</td>
<td>Meningeal hemorrhage Consequence of a fracture to base of skull and laceration of mid meningeal artery; knocked down</td>
</tr>
<tr>
<td>5/31/1916</td>
<td>Unknown</td>
<td>M</td>
<td>NK</td>
<td>Decomposition too advanced; body in box at Fenchurch St Station Paget disease of the bone—one leg longer than the other</td>
</tr>
<tr>
<td>6/14/1916</td>
<td>Williams, C. C.</td>
<td>M</td>
<td>51 yr</td>
<td>Paget disease; while excavating in Falklands, rock landed on his head, death attributed to this</td>
</tr>
<tr>
<td>6/15/1916</td>
<td>Re: Hamilton</td>
<td>F</td>
<td>NK</td>
<td>Shock Rex v Olley; criminal abortion, insertion into cervix of pieces of gum elastic catheter Gum-elastic catheters were made by dissolving plant gums onto woven silk tube</td>
</tr>
<tr>
<td>6/19/1916</td>
<td>Blanchet, M.</td>
<td>F</td>
<td>29 yr</td>
<td>CO poisoning Suicide; CO in blood 68%, H6 Using Haldane gas analysis apparatus, created 1889</td>
</tr>
<tr>
<td>7/7/1916</td>
<td>Unknown</td>
<td>F</td>
<td>NB</td>
<td>Brain hemorrhage Fractured skull; found in box</td>
</tr>
<tr>
<td>7/9/1916</td>
<td>Abrahams, E. A.</td>
<td>F</td>
<td>31 yr</td>
<td>CO poisoning CO in blood 68% Haldane method, as above</td>
</tr>
<tr>
<td>7/10/1916</td>
<td>Willis, D.</td>
<td>F</td>
<td>22 yr</td>
<td>TNT poisoning Worked with TNT; jaundice</td>
</tr>
<tr>
<td>7/13/1916</td>
<td>Glenville, E.</td>
<td>F</td>
<td>36 yr</td>
<td>Acute peritonitis Septic infection of uterus; passed crochet hook in herself and took pills</td>
</tr>
<tr>
<td>7/13/1916</td>
<td>Alison, C.</td>
<td>M</td>
<td>15 yr</td>
<td>Pulmonary embolism Appendicectomy</td>
</tr>
<tr>
<td>7/29/1916</td>
<td>Beckerson, E. M.</td>
<td>F</td>
<td>49 yr</td>
<td>Starvation Rex v H. Bundee—convicted. Recommended victim to fast from food, said he was a hydropathist—cures disease by internal and external use of water</td>
</tr>
<tr>
<td>8/2/1916</td>
<td>Jackson, H</td>
<td>M</td>
<td>40 yr</td>
<td>Cerebral syphilis Accelerated by injection of Kharsivan Kharsivan=dioxydiamido-arseno-benzoldihydrochloride</td>
</tr>
<tr>
<td>9/26/1916</td>
<td>Jackson, E.</td>
<td>F</td>
<td>50 yr</td>
<td>Exanguination And shock as a consequence of injuries to chest and pelvis, road accident</td>
</tr>
<tr>
<td>10/6/1916</td>
<td>Jones, A. E. J.</td>
<td>M</td>
<td>2 and 6 mo</td>
<td>Blunt force trauma to head Concussion and head injuries, several bruises. Rex v Mrs Jones for murder</td>
</tr>
<tr>
<td>10/17/1916</td>
<td>Smith, F. W.</td>
<td>M</td>
<td>45 yr</td>
<td>Hemorrhage Of ulcer in duodenum and mitral stenosis and chronic lead poison</td>
</tr>
<tr>
<td>10/18/1916</td>
<td>Unknown</td>
<td>F</td>
<td>NB</td>
<td>PDA Almost full term, found wrapped in garments</td>
</tr>
<tr>
<td>10/19/1916</td>
<td>Snell</td>
<td>F</td>
<td>NB</td>
<td>PDA Stillborn</td>
</tr>
<tr>
<td>Date of Autopsy (Month/Day/Year)</td>
<td>Name</td>
<td>Sex</td>
<td>Age</td>
<td>Cause of Death</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>------------</td>
<td>-----</td>
<td>-----------</td>
<td>--------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>10/20/1916</td>
<td>Theobald, M. A.</td>
<td>F</td>
<td>2 and 5 mo</td>
<td>Exsanguination</td>
</tr>
<tr>
<td>10/21/1916</td>
<td>Roberti, L.</td>
<td>F</td>
<td>26 yr</td>
<td>Neosalvarsan poisoning</td>
</tr>
<tr>
<td>10/27/1916</td>
<td>Moss, A.</td>
<td>M</td>
<td>62 yr</td>
<td>Arsenic poisoning</td>
</tr>
<tr>
<td>10/21/1916</td>
<td>Methafer, F. R.</td>
<td>F</td>
<td>9 yr</td>
<td>Cirrhosis of liver</td>
</tr>
<tr>
<td>10/31/1916</td>
<td>Wilson, C.</td>
<td>F</td>
<td>41 yr</td>
<td>Breast cancer</td>
</tr>
<tr>
<td>11/7/1916</td>
<td>Sloughgrove, J.</td>
<td>M</td>
<td>68 yr</td>
<td>Shock and blood loss</td>
</tr>
<tr>
<td>11/12/1916</td>
<td>Rainlow, V.</td>
<td>F</td>
<td>21 yr</td>
<td>Shock from immersion in water</td>
</tr>
<tr>
<td>11/16/1916</td>
<td>Pearce, A. E.</td>
<td>M</td>
<td>41 yr</td>
<td>Exophthalmic goiter—shock</td>
</tr>
<tr>
<td>11/17/1916</td>
<td>Jackson, C. R.</td>
<td>M</td>
<td>67 yr</td>
<td>Hemorrhage of stomach cancer</td>
</tr>
<tr>
<td>11/27/1916</td>
<td>Marsh, L. G.</td>
<td>M</td>
<td>22 yr</td>
<td>Electrocution</td>
</tr>
<tr>
<td>11/29/1916</td>
<td>Hopkinson, M.</td>
<td>F</td>
<td>41 yr</td>
<td>Graves disease</td>
</tr>
<tr>
<td>1/8/1921</td>
<td>Bishop, E. W.</td>
<td>F</td>
<td>16 yr</td>
<td>Stat. lymph.</td>
</tr>
<tr>
<td>1/29/1921</td>
<td>Seabrooke, S.</td>
<td>F</td>
<td>71 yr</td>
<td>Blood loss—murder</td>
</tr>
<tr>
<td>2/4/1921</td>
<td>Bird, J.</td>
<td>M</td>
<td>45 yr</td>
<td>Shock</td>
</tr>
<tr>
<td>3/19/1921</td>
<td>Shaw, L. D.</td>
<td>M</td>
<td>38 yr</td>
<td>Poison—suicide</td>
</tr>
<tr>
<td>3/23/1921</td>
<td>Miles, A. C.</td>
<td>M</td>
<td>16 yr</td>
<td>Abscess left frontal lobe of brain</td>
</tr>
<tr>
<td>3/25/1921</td>
<td>Hoydonk, H. F. G.</td>
<td>M</td>
<td>20 yr</td>
<td>Poison</td>
</tr>
<tr>
<td>Date</td>
<td>Name</td>
<td>Gender</td>
<td>Age</td>
<td>Cause</td>
</tr>
<tr>
<td>------------</td>
<td>-----------------</td>
<td>--------</td>
<td>-----</td>
<td>------------------------</td>
</tr>
<tr>
<td>4/1/1921</td>
<td>Leslie, R. M.</td>
<td>M</td>
<td>54</td>
<td>Poison</td>
</tr>
<tr>
<td>5/25/1921</td>
<td>King, C.</td>
<td>M</td>
<td>38</td>
<td>Poison</td>
</tr>
<tr>
<td>5/26/1921</td>
<td>Whittle, E.</td>
<td>F</td>
<td>38</td>
<td>Chest tumor</td>
</tr>
<tr>
<td>5/31/1921</td>
<td>Murphy, V. S.</td>
<td>M</td>
<td>24</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>6/10/1921</td>
<td>Overin, W. A.</td>
<td>M</td>
<td>60</td>
<td>Anesthetic death</td>
</tr>
<tr>
<td>6/11/1921</td>
<td>Moores, P. M.</td>
<td>F</td>
<td>20</td>
<td>Shock and blood loss</td>
</tr>
<tr>
<td>6/29/1921</td>
<td>Coffey, J.</td>
<td>M</td>
<td>38</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>7/6/1921</td>
<td>Hare, M. R.</td>
<td>F</td>
<td>39</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>7/14/1921</td>
<td>Edwards, F. G.</td>
<td>M</td>
<td>48</td>
<td>Anesthetic death</td>
</tr>
<tr>
<td>8/15/1921</td>
<td>Hall, I. M.</td>
<td>M</td>
<td>46</td>
<td>CO poisoning—suicide</td>
</tr>
<tr>
<td>8/19/1921</td>
<td>Cartor, N. L.</td>
<td>F</td>
<td>41</td>
<td>Acute septicemia</td>
</tr>
<tr>
<td>8/19/1921</td>
<td>Moss, E. J.</td>
<td>M</td>
<td>10</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>8/20/1921</td>
<td>Tong, A.</td>
<td>F</td>
<td>35</td>
<td>Poison</td>
</tr>
<tr>
<td>9/26/1921</td>
<td>Bolton, W. M.</td>
<td>F</td>
<td>36</td>
<td>Poison—misadventure</td>
</tr>
<tr>
<td>10/1/1921</td>
<td>Sweeney, P. C.</td>
<td>F</td>
<td>64</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>10/2/1921</td>
<td>Busby, C. L.</td>
<td>F</td>
<td>70</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>12/14/1921</td>
<td>Stead, E. F.</td>
<td>F</td>
<td>26</td>
<td>CO poison</td>
</tr>
<tr>
<td>12/22/1921</td>
<td>Laker, E.</td>
<td>F</td>
<td>49</td>
<td>Anesthetic death</td>
</tr>
<tr>
<td>2/23/1926</td>
<td>Grave, G. B.</td>
<td>M</td>
<td>53</td>
<td>Anesthetic death</td>
</tr>
<tr>
<td>8/23/1926</td>
<td>Graham, F. W.</td>
<td>M</td>
<td>35</td>
<td>Fractured skull and brain injury</td>
</tr>
<tr>
<td>8/27/1926</td>
<td>Edward, J. B.</td>
<td>M</td>
<td>18</td>
<td>Electrocution</td>
</tr>
<tr>
<td>9/8/1926</td>
<td>Margenson, G. F.</td>
<td>M</td>
<td>16</td>
<td>Bullet wound to head</td>
</tr>
<tr>
<td>9/28/1926</td>
<td>Chapman, G. W.</td>
<td>F</td>
<td>18</td>
<td>Shock</td>
</tr>
<tr>
<td>10/12/1926</td>
<td>Manwin, N. E.</td>
<td>F</td>
<td>24</td>
<td>Shock</td>
</tr>
</tbody>
</table>

NB indicates newborn; NK, age not known; PDA, patent ductus arteriosus (a type of congenital heart defect in which blood vessels that bypass the lungs fail to close as the infant is born).
died when he fell while running and grasped an electric fence. In 1926, there are still deaths as a result of criminal abortions, for example, G. W. Chapman (9/28/1926) and N. E. Manwin (10/12/1926). Although World War I had ended some 8 years previously, its legacy lives on in the case of G. F. Margenson (9/8/1926) who was killed, reportedly accidentally, with a German Mauser pistol. In this case, Spilsbury shows his knowledge of gunshot wounds in that he concludes that the weapon was not touching the wound because there was no singeing or blackening. He also notes the blood spatter—again a field that has now developed into a highly important part of many murder investigations.

CONCLUSIONS

It is perhaps inevitable that Spilsbury’s reputation has largely been based on his famous cases. These made him a household name during his lifetime, but have also led to questions about the scientific accuracy of his work in more recent times. It has been the lack of scientific papers or textbooks written by Spilsbury and the fact that he did not train students in his methods that has meant that an objective review of his work has been very difficult. The public availability of his case cards at The Wellcome Library changes this situation. It is now possible to monitor how his work progressed on an almost daily basis, and by looking at these more routine cases that did not impinge on the public consciousness, perhaps a more balanced picture of Spilsbury’s work begins to emerge. We have made only a preliminary survey of these case cards. In total, we have looked at about 650 of the 4000 case cards that were sold to The Wellcome Library in 2008. Of these, we have studied about 300 in detail. Even with this incomplete sample, some clear conclusions emerge. First, we note Spilsbury’s immense capacity for hard work. In 1931, for example, he reports on no less than 350 cases in the year. He introduced rigorous scientific methodology to his cases. Concentrations of blood CO and masses of poisons found within the body are reported. Areas of forensic investigation are utilized such as studying the growth of maggots on a body, studying blood spatter, and noting the presence or absence of scorch marks around gunshot wounds. Perhaps more importantly, his work drew attention to various areas of poor and sometimes illegal medical practice and poor industrial safety in an age when processes were not so regulated as in ours. Without clearly establishing the cause of death in many of these cases, it would not have been possible to pursue prosecutions of those who had acted illegally, for example, those carrying out illegal abortions, drunken doctors, and pharmacists accidentally giving out poisons rather than medicines. Moreover, without such work, the dangers of anesthetics may not have been properly recognized, and industrial hazards may not have been identified and corrected. Clearly, Spilsbury was sometimes unclear about cases (and often he admits this), and he must sometimes have been wrong. Some of his medical diagnoses would not stand the test of time, for example, his belief in *stat. lymph*. He attributes some causes of cancer and the autoimmune disease, Graves disease, that nowadays would not seem plausible. But we believe that it is timely to reappraise the work of Spilsbury, as with such a large amount of reference material now in the public domain it is possible to make an appraisal based on objective evidence.

ACKNOWLEDGMENTS

The authors thank Chris Hilton from The Wellcome Library (London) and Bev Baker from the Galleries of Justice Library (Nottingham) for help in accessing source material.

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اهمیت بررسی آنتروپولوژیک صحتن جرم در موارد بقاوی سوءخته در خودرو

چکیده: بررسی صحتن جرم یک مرحله تعیین کننده در پزشکی قانونی است، و حتی روش‌های آموزشی شده از آنترپولوژی قانونی نیز ضروری هستند درحالیکه یک بررسی کامل می‌تواند اطلاعات تعیین کننده‌ای به دست دهد. یک بررسی غیر دقیق می‌تواند به‌قاعده‌ای صحتن جرم را ۲۰۰۹ یافته شده بودند را گزارش نموده است. در هر سه مورد. یک مرحله به ضرب گلوله کشتی شده بود و جسد در درون خودرو به آتش کشیده شده بود تا ضایعات اسکلتی از بین بروند و مانع شناسایی شوند. در تمام موارد درخواست کمک از آنترپولوژیست قانونی به عمل امده بود، اما تنها یک از بررسی جسد در کالبدگشایی مشخص شده بود که بقاوی ناقص هستند. نتایج نشان داد که در موارد صحتن جرم با روش‌های دقیق آنترپولوژی و باستان شناسی توسط آنترپولوژیست قانونی انجام شد تا بازیابی کامل‌تری انجام شده و این موارد به وضعیت اهمیت یک بزرگان مناسب و کاربرد روش‌های آنترپولوژی قانونی در انجام به شدت سوءخته و زیغای شده و اهمیت بزرگی کامل‌تری اطمینان استخوانی را نشان داد. چرا که حتی کوچکترین قطعه استخوانی می‌تواند اطلاعات مهمی از ارتباط دهد. بنابراین یک همگامی دقیق یک بازیابی دقیق و کامل قطعات استخوانی و یک روش‌کرده آنترپولوژیک برای بررسی از موارد از جمله تحلیل صحتن جرم بارزاسی جسد و حوادث پیش از مرگ، تعیین کننده هستند.
The Importance of an Anthropological Scene of Crime Investigation in the Case of Burnt Remains in Vehicles

3 Case Studies

Davide Porta, BSc, Pasquale Poppa, BSc, Valeria Regazzola, BSc, Daniele Gibelli, MD, Daniela Roberta Schillaci, MD, Alberto Amadasi, MD, Francesca Magli, MSc, BA, and Cristina Cattaneo, MD, PhD

Abstract: Inspection of a crime scene is a crucial step in forensic medicine, and even the methods taught by forensic anthropology are essential. Whereas a thorough inspection can provide crucial information, an approximate inspection can be useless or even harmful. This study reports 3 cases of burnt bodies found inside vehicles between 2006 and 2009 in the outskirts of Milan (Italy). In all 3 cases, the victim was killed by gunshot, and the body was burnt in the vehicle to destroy signs of skeletal injury and prevent identification. In every case, the assistance of forensic anthropologists was requested, but only after the inspection of the body at autopsy showed that the remains were incomplete, thus making it more difficult to determine the identity, cause, and manner of death. A second scene of crime inspection was therefore performed with strict anthropological and adapted archeological methods by forensic anthropologists to perform a more complete recovery, proving how much material had been left behind. These cases clearly show the importance of a proper recovery and of the application of forensic anthropology methods on badly charred bodies and the importance of recovering every fragment of bone: even the smallest fragment can provide essential information. Thus, a precise coordination, a correct and thorough recovery of bone fragments, and an anthropological approach are crucial for many issues: analysis of the scene of crime, reconstruction of the corpse, and reconstruction of the perimortem events.

Key Words: forensic sciences, forensic anthropology, crime scene investigation, carbonization, charred remains


Some of the most difficult cases forensic pathologists usually have to deal with are charred bodies. As a matter of fact, this is a fairly usual method used to conceal a body or make it unrecognizable. However, the high temperatures reached within a fire are usually not capable of completely destroying a body, but may lead to significant alterations of the morphology and structure of human tissues.1-5 In this context, the radical morphostructural alterations, which are often caused by heat, may represent a serious hindrance to the development of many aspects of the investigation: first, for the reconstruction of the cause and manner of death, as well as for personal identification, due to the loss of the morphological facial features and the alterations of features and marks on the body.6,7 Many parts of the human body are likely to survive even at extremely high temperatures, especially the denser bones and those covered by a thick layer of soft tissues.8 A modern adult cremation leaves an amount of incinerated bones of about 1600 to 3600 g with an average of 3000 g.9

For all these reasons, the role of the scene of crime investigation with a complete recovery of bone fragments becomes crucial, more than in other contexts: a scrupulous analysis of the remains is certainly very useful in cremation cases not only for the identification of the victim but also to define the cause and the manner of death. In particular, at the time of the inspection, the accurate collection and sorting of the material are critical and often essential steps for the success of the entire investigation. This concerns mainly bones: in fact, whereas soft tissues are often completely destroyed by fire, the bony tissue can survive even at extreme temperatures (although with relevant morphological alterations) and may still preserve macroscopically and microscopically specific signs related to the subject’s biological profile, to pathological deformations, to surgery or even lesions.10-17 Among the debris one can find during the scene of crime investigation, every bone or fragment of bone can be crucial and can provide relevant information.

The following cases concern 3 different “cremations” that took place inside cars in the outskirts of Milan, Italy, in a time interval between 2000 and 2009, to show the importance of an adequate analysis of the crime scene and recovery in cases of charred bodies. In these cases, lack of anthropological expertise on site by legal authorities led to an incomplete recovery. Only upon the second examination were all the human remains collected after appropriate search and sieving.

CASE A

During the summer of 2007, a burnt car (Alfa Romeo 147) was found in a field in the outskirts of Milan. During the investigation, a charred body was found inside, in a prone position and on the back seats of the car (Fig. 1A).

During the first inspection, only some articulated parts of the body, mostly burnt and calcined (Crow-Glassman scale level 4),18 were recovered: precisely, the entire splanchnocranium and parts of the neurocranium (calcined, in fragments, and with many parts of the cranial vault missing), the entire spinal column, sternum, part of the left thorax, left clavicle and scapula, the entire pelvic girdle, proximal epiphysis and proximal part of the diaphysis of both humeri, right and left femurs, proximal epiphysis, and proximal part of the diaphysis of tibiae and fibulae. This inspection led to the recovery of approximately 70% of the entire skeleton. The trunk was still covered by completely charred soft tissues, with exposition of the rib cage. Only the

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larger and more visible parts of upper and lower limbs were recovered from the car.

The body was then subjected to autopsy, but many parts were obviously missing. One never knows where useful information for identification and reconstruction of manner of death may come from (eg, a distal phalanx with a cut mark), so one must attempt to recover the entire skeleton. In fact, the help of forensic anthropology became obvious for a correct recovery of the entire skeleton. The team managed by the forensic anthropologist started with a second inspection of the car, dividing the vehicle into 4 parts: the area of the driver's seat was identified as “1,” and the other areas with the following numbers up to 4 in a clockwise direction. During the recovery, the middle portion of the car (around the gear lever) and the area under the posterior seats were further divided, because of the high amount of material found (Fig. 1B). After a thorough sieving of all the materials recovered inside the car, this inspection led to the identification of several bony remains: they were totally calcined, mostly in fragments and scattered on the floor of the car. The following bony parts were recovered during the inspection: parts of the frontal and cranial vault (maximum size 6 × 6 cm, minimum size 3 × 1.5 cm), fragments of many left and right ribs, the distal epiphysis of both humeri, the proximal epiphysis of right and left radii and ulnae, some metacarpals and phalanges of the hands, right patella, distal epiphysis of right tibia and fibula, right talus, right calcaneus, and metatarsals and phalanges of the right foot (Fig. 2, A–C). The material recovered was about 25% of the skeleton. The thorough investigation of the scene led to the discovery of a fragment of fabric (that could have been used as a ligature) near some disarticulated feet bones, and several bony fragments (recognized as portions of a left shinbone) melted with the bottom of the car. More importantly, the recovery of fragments of the cranial vault allowed for a reconstruction of the skull. In particular, the recovery of a part of the frontal bone (5 cm in width) made it possible to reconstruct a particular and roughly round lesion, suggesting a gunshot entry wound, on the glabella, with a diameter of 6 mm (Fig. 3). A second similar lesion was found on the right parietal bone, near the sagittal suture, with a diameter of 9 mm. Thus, the collection of further parts of the skull during the second inspection (eg, pars frontalis) allowed for an identification of at least 2 gunshot entry wounds.

**CASE B**

During the summer of 2000, a completely charred body was found lying inside a partially burnt Mercedes Vito van. The
body was found in a supine position, behind the anterior seats and in a diagonal line with respect to the major axis of the vehicle (Fig. 4). The corpse consisted mainly of an apparent vertebral column, thorax, hip bones, and charred soft tissue (Crow-Glassman scale level 4). During the first inspection, the clearly visible body parts were recovered. The corpse was severely damaged by the fire, with exposition of the rib cage and cervical vertebrae, amputation of upper and lower ribs, and absence of most of the skull. The body parts still covered by charred tissues were less damaged, whereas all the parts coming from upper and lower arms or from the head were totally calcined.

The material recovered during the first inspection on which the autopsy was performed was composed of 32 calcined fragments of the skull, partly from frontal, parietals, temporals, and occipital (maximum size 4 × 6 cm, minimum size 2 × 2 cm) without any anatomical relationship, almost the entire splanchnocranium; both clavicles, right scapula, manubrium of the sternum, all the right ribs, the entire spinal column and the pelvic girdle; of the upper limbs, both entire humeri, the proximal epiphysis and the diaphysis of the right femur, the diaphysis of the left femur, both calcined patellae, parts of the charred diaphysis of both tibiae and fibulae, and many charred and fragmented parts of the feet (Fig. 6, A–C). Globally, forensic anthropologists found a further 25% of the skeleton. Many fragments of burnt and calcined human bones could not be referred to specific body parts because of the bad preservation of the remains.

At this point, it was possible to compare the data obtained from anthropological investigations with antemortem data from an identity suspect. As a matter of fact, the comparison of antemortem dental radiographs and postmortem radiographs of the prosthesis (recovered during the second investigation) led to a positive identification (Fig. 5, B and C). Furthermore, because of the presence of the bullet jacket found inside the van during the second inspection, the victim was presumed to have been shot.

These procedures added many elements to the reconstruction of the victim’s body, making it more complete. In fact, the inspection led to the recovery of many further bony parts: the missing part of the maxilla; many calcined parts of the left ribs; calcined parts of the diaphysis and the distal epiphysis of both radii and ulnae and many parts of charred carpals, metacarpals, and phalanges; and the medial part of the charred diaphysis of the right femur, the diaphysis of the left femur, both calcined patellae, parts of the charred diaphysis of both tibiae and fibulae, and many charred and fragmented parts of the feet (Fig. 6, A–C). Globally, forensic anthropologists found a further 25% of the skeleton. Many fragments of burnt and calcined human bones could not be referred to specific body parts because of the bad preservation of the remains.

At this point, it was possible to compare the data obtained from anthropological investigations with antemortem data from an identity suspect. As a matter of fact, the comparison of antemortem dental radiographs and postmortem radiographs of the prosthesis (recovered during the second investigation) led to a positive identification (Fig. 5, B and C). Furthermore, because of the presence of the bullet jacket found inside the van during the second inspection, the victim was presumed to have been shot.

**CASE C**

This case concerns a burnt body found in a car (Volkswagen Golf), left in a woody area in 2005. The body appeared seated in the driver’s position, largely destroyed by fire, with many charred and calcined parts, particularly the cranium, upper and lower limbs, and exposition of charred intestines (Crow-Glassman scale level 3). The first inspection led to the recovery of the main body parts and the clearly visible remains (Fig. 7).

The material recovered during the first inspection and subjected to autopsy was about 75% of the skeleton and was composed of large portions of the splanchnocranium and neurocranium partially charred and calcined; the complete thoracic, abdominal, and pelvic regions, with both clavicles and scapulae, the entire sternum, and all the ribs, vertebrae and pelvic girdle;
of the upper limbs, the proximal epiphysis of the right humerus, the proximal epiphysis and the diaphysis of the left humerus, and the diaphysis of the left radius and ulna; and of the lower limbs, the proximal epiphysis and the diaphysis of both femora (still articulated with the pelvic girdle and covered by charred tissues), the proximal epiphysis and the diaphysis of the left tibia and fibula, and the tarsal bones of the left foot. As in the previous cases, because of the incompleteness of the remains, a further inspection by forensic anthropologists was considered strictly necessary, to collect more information about the victim's identity, the cause, and the manner of death.

The second inspection was structured in 2 phases: a first step of recovery and sieving of the material found on the scene and then the complete study of the collected bones in the laboratory. The former was organized by dividing the vehicle into 3 sections: respectively, anterior, middle, and posterior. The anterior section was further divided into 3 subsections: one near the body, the second including the driver's seat, and the last including the passenger's seat. The middle section included the posterior seats, and the posterior section, the luggage van (Fig. 7).

The sieving of all the collected material brought to the recovery of 2 monoradicular dental roots, 2 projectiles, and further bony fragments (Fig. 8, A–C), that is, many charred and calcined parts of the skull, including parts of frontal, parietals, temporals, occipital, and mandible (mandibular symphysis, left condyle, and left ascending ramus) with a maximum size of 8.5 × 6 cm and a minimum size of 2 × 2 cm; in the upper limbs, the diaphysis and distal epiphysis of the right humerus, the entire right ulna, the distal epiphysis of the left humerus,
proximal and distal epiphysis of right radius and ulna, and many parts of carpals, metacarpals, and phalanges. In particular, all 10 distal phalanges were recovered, and the second metacarpals, the lunates, and the right capitate were clearly recognized; and in the lower limbs, both patellae, the diaphysis and the distal epiphysis of the right tibia, the entire right fibula, the distal epiphysis of left tibia and fibula, both tali, and proximal and distal phalanges. Many fragments of burnt and calcined human bones were not referable to specific body parts because of their small dimensions. The second inspection led to an accurate recovery of about 20% of the skeleton.

The second scene of crime investigation was important for the bullets found but especially for another issue: an informant, supposed to be involved in the case, had said that the victim’s hands had been chopped off at the wrist before burning the car. To verify the reliability of this witness, the judge wanted to know whether this were true. The remains told a different story: there were no signs of cut marks on the distal portions of radii and/or ulna (recovered the second time around), which would have inevitably been visible. In fact, cut marks can be seen even after charring.\textsuperscript{11–13}

In this case also, the second systematic inspection permitted the forensic anthropologist to recover several human fragments and important elements, useful for the reconstruction of some of the perimortem events.

**DISCUSSION**

The 3 cases clearly show how adequate recovery and forensic anthropology may be essential in cases where the pathologists have to face badly preserved remains and where the inspection of the crime scene is a critical step: the presence of burnt bodies is a clear example.

In every case, recovery of small fragments left behind at the first scene of crime inspection was crucial for specific aspects of the case: finding gunshot wounds or elements crucial for identification or the absence of cut marks.

These cases underline how, because fire can lead to extreme alterations such as charring of the soft tissues, shrinkage, and fragmentation of the bones, the analysis of the scene has to be conducted with a complete, precise, and standardized method, which includes the collaboration with experts in human forensic osteology. Ideally, forensic anthropologists should
always take part in the recovery and selection of the remains and apply their abilities to recognize fragmented human remains or to distinguish between human and animal remains or between human remains and environmental contamination. Therefore, the inspection of a crime scene, especially in these cases, has to be conducted by a team of specialists along with forensic anthropologists because even the smallest bony fragment, which may appear unimportant to the eyes of an inexperienced observer, can bring fundamental information to a case.

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گزارش یک مورد خودکشی با چهار شلیک تفنگ نیزه‌نداز

چکیده: در متأسفانه تعدادی از مرگ‌مرتبه‌های سال‌های گذشته گرمی بوده، و چندین نمونه در این موارد وجود دارد. خودکشی، یکی از مهم‌ترین عوامل در مشکلات سلامتی انفیلدرای دنیا در حال کاهش است.

در مورد این امر، شهد دو نويسنده، یک مورد نادر از خودکشی را گزارش کرده‌اند. شامل ۳ شلیک به سینه و یک شلیک به سر می‌شود.

در بررسی کامل یکی از قانونی مشخص شده، مربوط به دلیل نارسایی حاد قلبی ناشی از هموبریکاردی به نبضات آسیب شویان آلوری چپ بوده است.

بررسی صحت و یافته‌های کلیدنگشایی به نویسنده اجازه داد تا این فرضیه را مطرح کند که آسیب به سر آخرین تلاش برای خودکشی بوده است. که در زمان پیشرفت تامورین قلبی رخ داده است.

نویسنده در مقاله به اشتباه گذاشتن این موضوع به جامعه پزشکان قانونی درباره آسیب‌ها ناشی از تفنگ‌های نیزه‌نداز است که باعث تأیید آنها از ضایعات بسیار مشابه ایجاد شده توسط جامعه علوم می‌شود.

همچنین نشان می‌دهد که چگونگی ممکن است تا مدتی یکی از اصابات چنین سلاحی زنده ماند.
Suicide Due to Four Speargun Shots

A Case Report

Alessandro Bonsignore, MD, Luca Vallega Bernucci, MD, Marco Canepa, MD, and Francesco Ventura, MD, PhD

Abstract: In the literature, only a few cases of deaths related to the use of atypical firearms are present and even more rare are cases of suicide due to multiple lesions.

In the present case, the authors report a rare occurrence of suicide due to 4 speargun shots, 3 to the chest and 1 to the head.

A complete forensic approach led to attribute the death to acute cardiac failure due to hemopericardium after the injury of the left coronary artery.

Scene investigation and autopic findings allowed authors to hypothesize that injury to the head was a last attempt of suicide, elapsed during the progression of cardiac tamponade.

With this report, the authors would like to share knowledge with the forensic community about speargun-related lesions distinguishing them from the very similar ones produced by cold steels.

It also shows how it is possible to survive for some time after being shot by such a weapon.

Key Words: suicide, atypical firearms, speargun, multiple wounds

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In evaluating the methods used in producing cuts and stab wounds, along with their distinguishing features, forensic pathologists should pay particular attention in cases of death occurring through the use of unusual weapons.

In the literature, only a few cases of murder, suicide, or accidental death involving atypical firearms such as darts1-3 or harpoons4-10 are described.

In particular, only a small number of reports concerning the use of crossbows and spearguns are present and even more rare are cases of suicide due to multiple lesions.11,12

These events are extremely uncommon as the victim typically has to reload the weapon several times before firing the lethal shot.

In the present case, the authors report a rare occurrence of suicide due to 4 speargun shots, with multiple wounds to the head and chest.

CASE REPORT

A 75-year-old man fought with a colleague at work, then locked himself inside his office. The police arrived on the scene and ordered him to open the door but, moments later, they heard muffled, confused noises and then silence.

When firemen knocked down the door they found a 45-cm speargun in his right hand with the weapon’s 40-cm harpoon lodged in his right temporal region, next to the pericranium; there were also multiple wounds to the chest. The emergency service performed several resuscitation attempts without any result.

Afterward, during scene investigation, the man’s body was found lying on his back near a pool of blood (Fig. 1, A and B).

Detectives, owing to the features of the lesions, hypothesized that the man had repeatedly loaded and fired the weapon.

An autopsy was performed 48 hours after the exitus to evaluate the cause of death. Gross examination revealed a break of the scalp on the right temporal region 2 cm upper-front the auricle; beneath this first lesion, there was a linear and horizontal puncture wound of the skull, 1.5 cm long with extended edges (Fig. 2A).

On the left side of the chest, there were 3 circular puncture wounds (diameter, 0.7 cm each) rounded by an ecchymotic ring (Fig. 2, B and C), respectively, located in the following:

-0.5 cm from the marginosternal line, in the second infracostal space (A);

-1 cm from marginosternal line, in the third infracostal space (B) -close to the preceding one, 1 cm laterally (C).

Examination of the skull showed 1 puncture wound of the scalp with hemorrhagic infiltration of pericranium on the right temporal region (Fig. 2D).

Subdural and subarachnoid hematoma, edema and serum-hematic intraventricular liquid were also present ipsilaterally.

At the second infracostal space, beneath wound A, the harpoon had chipped the upper edge of the third left rib. On the left side of the chest, next to the external lesions, soft tissues showed hemorrhagic infiltration (Fig. 3A); once the sternum was removed, left hemothorax (1500 mL) was discovered.

Deep below the second wound (B), the harpoon had perforated the left lung at the intralobal fissure, generating a small 0.5-cm hole surrounded by hemorrage (Fig. 3B).

After perforating the skin, by the third wound (C), the harpoon passed through the upper edge of the lower lobe of the left lung.

Lesion A continued through pericardium, left auricle (Fig. 3C), puncturing the anterior wall of the left coronary artery, causing a massive hemopericardium (Fig. 3D).

The death was attributed to acute cardiac failure due to hemopericardium after the injury of the left coronary artery;
FIGURE 1. Scene investigation (A) after emergency service resuscitation attempts; (B) detail of (A) showing the 3 wounds on the chest; (C) detail of the harpoon after being removed from the skull; and (D) Mares Speargun next to the pool of blood.

FIGURE 2. Linear and horizontal puncture wound of the skull, (A) 1.5 cm long with extended edges, in the right temporal region; (B and C) left side of the chest with 3 circular puncture wounds (diameter, 0.7 cm each) rounded by an ecchymotic ring at different magnification; and (D) hemorrhagic infiltration of pericranium on the right temporal region.
addition, other significant remarks were left hemothorax, subdural, and subarachnoid hematoma.

Perforating injuries to the chest and the head were made by sharp-end items, compatible with the harpoon found on the crime scene.

**DISCUSSION**

With this report, the authors would like to share knowledge with the forensic community about speargun-related lesions.

It is necessary to describe the weapon used before proceeding to an analysis of the injury pattern.

The speargun is an atypical firearm which uses an air-compressed or propelled elastic mechanism. Unlike normal firearms, it causes cutting and stabbing wounds which are more similar to cold steels.

The Mares Speargun (Fig. 1D) found on the scene had a pneumatic mechanism; it was 50 cm long including the harpoon (Fig. 1C), the latter having around 1 cm maximum diameter.

There were multiple wounds, 3 of which were at the left chest and 1 at the head.

After hitting the third left rib (A), the harpoon was deflected through pericardium, left auricle and it ended in a lesion of the anterior wall of the left coronary artery, leading to hemopericardium.

Two more shots perforated the pulmonary parenchyma (B–C).

The fourth shot to the right temporal region perforated the skull, coming close to the brain and tearing vascular structures, causing mild leptomeningeal hematoma.

Feature of injuries to the chest suggested that lesion A caused hemopericardium and cardiac tamponade.

Therefore, it seemed that injury to the head was a last attempt of suicide, elapsed during the progression of cardiac tamponade; in fact, there was insufficient subarachnoid bleeding and no brain lesions to lead to immediate death.

In other cases presented in the literature, it has been shown how it is possible to survive for some time after being shot by a similar weapon.

A speargun shot has less kinetic energy than a normal firearm bullet, so it is possible that more shots are required to achieve death, especially if vital centers are not involved.

Moreover, due to poor handling of the speargun, it is difficult for those who attempt suicide to succeed at the first attempt. In this case, considering that speargun needs of a manual reload for every shot, man survived enough to get 4 shots.

On the basis of crime scene and autopsy findings, Public Prosecutor’s reconstruction consisted in a 3-times shot to the chest and a last fourth 1 to the head, while cardiac tamponade was going on.

**CONCLUSIONS**

Despite spearguns are classified between firearms, even if unusual ones, it is important to know that injuries can be very similar to those produced by cold steels.

Characteristics of the weapon (ballistics and mechanisms), associated with the shape of harpoons, influence the potential related damage.
To study unusual firearms and their lesions, an accurate description is fundamental to define specific parameters, and distinguish them from common firearms injuries.13

Moreover, as well as other similar cases described in the literature,14 behind small and mild wounds can be hidden more serious and acute damage, so that detectives may suspect which kind of weapon have been used, especially if it is not found during the scene investigation.

A thorough examination can be useful for a fast identification of the weapon involved and its potential damage even for surgeons in case of emergency that requires immediate operation.

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پارگی والکولار با شکستگی مهره گردنی پس از تلاش ناموفق برای خودکشی با دارآویختگی

چکیده: دارآویختگی روش رابطه برای خودکشی است. ما یک مورد پارگی والکولار و شکستگی مهره گردنی بدون زخم خارجی را پس از یک تلاش ناموفق برای خودکشی با دارآویختگی را گزارش نمودادیم. درمان جراحت شامل اتصال خلفی C3 به همراه ترمیم پارگی والکولار با یک روبکرد خارجی بود. بیمار بدون هیچ عارضه باقی مانده جسمی با ذهنی بهبود یافت.
Vallecular Rupture With Cervical Spine Fracture After a Failed Hanging Suicide Attempt

Joong Keun Kwon, MD, PhD, Seong Rok Lee, MD, Ho Min Lee, MD, Jung Min Lee, MD, and Jong Cheol Lee, MD, PhD

Abstract: Hanging is a common method of suicide. We present a case of vallecular rupture and cervical spine fracture without an external wound after a failed hanging suicide attempt. Surgical treatment involved posterior fusion of C2 to 3, followed by repair of the vallecular rupture via an external approach. The patient recovered with no residual physical or mental sequelae.

Key Words: suicidal hanging, hanging injury

Hanging is a common method of suicide. We present a case of a failed hanging suicide. The resultant injuries were vallecular rupture and a cervical spine fracture. We describe the management and postoperative outcome for this patient.

CASE REPORT

A 38-year-old male patient was admitted after an attempted suicide by hanging. The incident occurred at the patient’s workplace. The patient had tied a rope to a banister of the fifth floor and jumped into a stairwell. However, coworkers quickly grabbed the rope such that the length of rope from the banister to the running noose was only 1.5 m. He was taken to the emergency department of the Ulsan University Hospital, Ulsan, Republic of Korea. On arrival at the emergency department, he was conscious, had no voice change, and had a blood pressure of 117/77 mm Hg, respiratory rate of 22 breaths per minute, pulse was stable without any external wounds. The blood pressure was stable.

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The resultant injuries were vallecular rupture and cervical spine fracture. We describe the management and postoperative outcome for this patient.

Previous studies of hanging neck injuries report that the most frequent injuries are muscle hemorrhage due to direct pressure and indirect stretching of the muscles. The reported frequency of solid neck structure fractures varies from 0.8% to 75.3%. The most commonly fractured structures are the hyoid bone and thyroid cartilage. The occurrence of fractures is greatly affected by the age of the subject as calcification reduces the flexibility of the hyoid bone. The most common larynx ruptures are laryngotracheal separation, supraglottic rupture (thyrohyoid membrane), and subglottic rupture (cricothyroid membrane).

The present report describes a rare case of a failed hanging suicide. The resultant injuries were vallecular rupture and cervical spine fracture. We describe the management and postoperative outcome for this patient.

In the United States, hanging suicide is the second most common method of suicide, accounting for 14% of such events.

Suicide hanging injuries include hyoid bone fractures, laryngotracheal fractures, carotid injuries, and cervical spine fractures. The most common larynx ruptures are laryngotracheal separation, supraglottic rupture (thyrohyoid membrane), and subglottic rupture (cricothyroid membrane).

The present report describes a rare case of a failed hanging suicide. The resultant injuries were vallecular rupture and cervical spine fracture. We describe the management and postoperative outcome for this patient.

Injuries to cervical vessels such as carotid artery dissection or obstruction can occur as a result of hanging.
case, no such lesions were revealed by CT imaging. Carotid sinus stimulation by hanging may cause circulatory collapse. In addition, only 2 kg of tension on a ligature is needed to block jugular veins, and diminished brain perfusion can be seen in all hanging cases.

A study of hanging injuries demonstrated that nearly 90% of victims who reached hospital alive survived with a low incidence of poor neurologic outcome. Thus, aggressive resuscitation and management of hanging victims is justified. In contrast, delayed presentation to a medical facility, a low Glasgow coma score at presentation, and cardiac arrest at the scene are predictors of a poor outcome.

The present case was unusual because it is rare that a person attempting to commit hanging suicide from a great height is rescued. The patient had vallecular-hyoid complex fracture with cervical spine fracture. After appropriate management with staged operations for cervical spine fractures and vallecular rupture, the patient completely recovered without residual physical or mental sequelae during follow-up.

CONCLUSIONS

We report a rare case of failed hanging suicide attempt and its presentation of vallecular rupture and cervical spine fracture. This case may be benefit for the management of the hanging victims, even if they have multiple fractures and ruptures around neck.

REFERENCES


تروموآمبولی سیستمیک پس از ترومبووز ورد عمیقی ناشی از میوم رحمی

چکیده: یک ترومبوآمبولی سیستمیک پس از ترومبووز ورد عمیقی (DVT) ناشی از میوم رحمی بسیار نادر است. نویسندگان اخیرا این ارتباط را در یک زن مجرد 46 ساله تایوانی با سابقه سلامتی قبیلی و بدون هیچ عامل مشاهده کردند. متوالی قیль از دریچه دار DVT خطر شناخته شده دیگری برای همی پازی ناگهانی راست شده‌بود و 34 ساعت پس از پذیرش در بیمارستان به طور ناگهانی دچار کلایس قلبی و مرگ شده بود. کلایدکشایی نشان داد که مارگ او به دلیل ترومبوآمبولی و سیگر بیوی با آمبولیزاسیون سیستمیک به همراه باز بودن همزمان سوراخ بیضی پس از DVT در هر دو انداز تحتانی ناشی از میوم رحمی بود.
Systemic Thromboembolism After Deep Vein Thrombosis Caused by Uterine Myomas

Supawon Srettabunjong, MD, LLB, MSc, MTox

Abstract: A systemic thrombus embolization after deep vein thrombosis (DVT) caused by uterine myomas is very rare. The authors recently had experienced this association in a single 46-year-old Thai woman with previous healthy history and no other known risk factors for development of DVT. On arrival at a nearby small hospital, the deceased had presented with an abrupt onset of right hemiparesis, and 34 hours after admission to the hospital, she suddenly developed a cardiopulmonary collapse and was pronounced dead. Autopsy examination revealed that her death was attributed to massive pulmonary thromboembolism with systemic embolization through coexistent patent foramen ovale after DVT of her bilateral lower extremities caused by uterine myomas.

Key Words: deep vein thrombosis, foremen ovale, pulmonary thromboembolism, systemic thromboembolism, uterine myoma

Deep vein thrombosis (DVT), the most common form of venous thromboembolism, is a rare complication of uterine myoma. There have been few reports on this association published in English-language literature listed in MEDLINE and PUBMED databases. The blood clot may then be dislodged and carried in the bloodstream to the lungs, known as a pulmonary thromboembolism (PTE), and to other visceral organs (eg, brain, heart, and kidney), called a systemic thromboembolism (STE). Systemic thromboembolism is a very rare event and occurs only in certain individuals. Recently, the authors had experienced a sudden and unexpected death due to acute massive PTE with STE after lower extremity DVT secondary to 2 uterine myomas in a single middle-aged Thai woman.

CASE REPORT

A single 46-year-old nonsmoking Thai woman presenting with dyspnea and a sudden onset of right hemiparesis was readily sent to a nearby small hospital. Thirty-four hours after admission, she abruptly developed cardiopulmonary collapse and was pronounced dead. Her family was suspicious about her death. The medicolegal death investigation then commenced, and the deceased was subsequently taken to the Forensic Medicine Department, Faculty of Medicine Siriraj Hospital, for further examination.

At scene, mild congestion of the deceased’s face and conjunctiva was found. Mild central and peripheral cyanosis was also detected. Swelling and pitting edema of her right ankle and calf was evident. No evidence of injury was found. According to her family, she had been healthy except for leg swelling and pain, aggravated by standing or walking for some time. The only laboratory investigation on admission revealed a hemoglobin level of 10.0 g/dL with normal red blood cell morphology and white blood cell count and platelets of 563,000/mm3.

At autopsy, external examination of the deceased revealed a woman of moderate built, measuring 165 cm in height and 55 kg in weight, with mild congestion of her face and conjunctiva. Mild central and peripheral cyanosis was still present. There was no evidence of injury in the neck region. Her right ankle and calf were swelling and pitting edema. No signs of injury were detected. Internal examination disclosed normal scalp and skull. The brain weighed 1300 g. Cut surfaces of fixed brain showed recent ischemic infarction in the left thalamic region, consistent with 1 to 2 days’ duration of cerebral infarction. Gross cerebral arteries appeared normal. Examination of the chest cavity showed no evidence of trauma. The heart weighed 380 g with engorged coronary arteries. Examination of the heart revealed a patent foramen ovale (PFO) with a maximal aperture of 0.6 cm (Fig. 1).

There was a mild dilatation of right ventricle and tricuspid-valve ring that was consistent with acute right ventricular failure. Intracardiac thrombus was not observed. Gross sections of the heart demonstrated no appearances of old or recent infarction. The aorta and the coronary arteries revealed no atherosclerotic changes. No apparent thromboemboli were found in major pulmonary arteries. The right lung and the left lung weighed 650 g and 530 g, respectively. Cut surfaces of both lungs revealed marked congestion and edema with multiple fresh thromboemboli of various sizes filled in pulmonary vessels (Fig. 2).

FIGURE 1. Gross view of a PFO with a maximal aperture of 0.5 cm (arrow).
Examination of the abdominal cavity disclosed no signs of injury. Two large firm smooth-surfaced pelvic masses, sized 19 × 13 × 8.5 cm and 17 × 12 × 8 cm of the right and left mass, respectively, with combined weight of 1820 g were found (Fig. 3). The right mass was the enlarged uterus itself, and the other was contiguous to the uterus at its left side just above the cervix. The right kidney weighed 120 g, and the left kidney, 110 g. There were multiple areas of recent infarction appearing from external surfaces to internal regions of both kidneys. The remaining internal organs were otherwise unremarkable. No pelvic vein thrombosis was detected. On opening the deep veins of both legs, extensive venous thrombosis of right leg involving distal femoral, popliteal, posterior tibial, and peroneal veins was present, whereas the thrombosis of left leg was detected only in some parts of distal femoral and popliteal veins. No evidence of remote or recent deep vein injury of both legs was detected.

Histologic examination of the lungs disclosed multiple fresh thromboemboli of varying sizes filled in the small vessels with marked congestion and edema. Histologic appearance of the brain was consistent with 1 to 2 days’ duration of infarction. Multiple areas of recent infarction were also observed in both kidneys. Sections of both pelvic masses demonstrated uniform, benign spindle cells consistent with leiomyomata with no evidence of necrosis. Central vein necrosis in the liver and intense acute congestion in the spleen was detected. There were minimal changes of myocardial infarction with fresh thromboemboli seen in the heart vessels. Thromboemboli in the lungs, brain, and heart consisted of fibrin and red blood cells but not fibroblasts (Fig. 4).

The cause of death was attributed to massive PTE with systemic embolization through coexistent PFO after extensive lower extremity DVT caused by 2 uterine myomas.

**DISCUSSION**

Although there are only 14 cases on the association between DVT and uterine myomas published in the literature,1–12 such associations have been well recognized. Occasionally, a large uterine myoma can cause compression of the pelvic veins leading to persistent pressure on pelvic veins that induces chronic venous stasis in the pelvis and lower extremities. Blood stasis of the pelvic veins is therefore a major etiologic factor for DVT in the pelvic region and/or in the lower extremity, either unilateral5–11 or bilateral.1,2,4,7,12 Nearly all of unilateral DVT was confined in the left side, thus supporting the notion that uterus mass was often localized more in the left side of the uterus than in the right. The individual ages ranged from 35 to 51 years including the present case, and the least weight of uterine myoma caused DVT was 800 g.1,11

The present case had no other known risk factors of DVT, except 2 large uterine myomas. Each myoma had a long vertical shape, thus its fundus might have compressed one of the pelvic veins leading to DVT in her both legs. The extensive thrombus was found in the right leg, which may probably be the result of her sleeping position and the larger size of right myoma, suggesting that the lodged thromboemboli were most likely generated from DVT of the left leg. Although most individuals with lower extremity DVT are clinically silent, the symptoms presented when having extensive distribution include pain, edema, and heaviness aggravated by standing or walking, as in the present case.

**FIGURE 2.** Gross view of cut surfaces of both lungs (A, left lung, and B, right lung) revealing marked congestion and edema with multiple fresh thromboemboli (arrows) of various sizes filled in pulmonary vessels.

**FIGURE 3.** Gross view of 2 large firm smooth-surfaced myomatous masses, sized 19 × 13 × 8.5 cm and 17 × 12 × 8 cm of right and left mass, respectively (arrows), with combined weight of 1820 g. The right mass was enlarged leiomyomatous uterus itself, and the other was contiguous to the uterus at its left side just above the cervix.
Systemic thromboembolism is a very rare incidence and occurs only in certain individuals with concomitant PFO, a frequent remnant of the fetal circulation found in approximately one fourth of the general population.13 Having a PFO and a DVT thus places an individual at high risk for paradoxical embolism. Well-recognized paradoxical embolism of thrombus across a PFO causing cryptogenic stroke in the young also presented.14 Fresh thromboemboli are susceptible to fragmentation during transit through the contracting right ventricle, producing multiple smaller emboli, which can readily run across the passage of PFO to other remote organs such as brain, as in this case.

In conclusion, STE is a very rare complication of uterine myomas. To the author’s knowledge, such a case has been previously reported in the literature in only 1 case.12 The present case is probably the second case of STE secondary to uterine myomas reported in the literature. This case report not only illustrates the association of uterine myoma and DVT with subsequent PTE/STE but also raises awareness of possible symptomatic thromboembolic disease. Accordingly, a middle-aged woman with uterine myoma deserves special attention as a possible etiologic contributor to DVT with subsequent dislodgement that can cause a serious condition or even sudden death.

REFERENCES