

ESSENTIAL FORENSIC BIOLOGY BRD EDITION

ALAN GUNN





Essential Forensic Biology

Essential Forensic Biology

Third Edition

Alan Gunn School of Natural Sciences & Psychology Liverpool John Moores University UK

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To Sarah, who believes that no evidence is required in order to find a husband guilty.

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Introduction

The word 'forensic' derives from the Latin *forum* meaning 'a market place': in Roman times, the forum was where people conducted business transactions and some legal proceedings. For many years, the term 'forensic' had a restricted definition and denoted a legal investigation. However, nowadays it applies to any detailed analysis of past events, i.e. when one looks for evidence. For example, tracing the source of a pollution incident is now a 'forensic environmental analysis', determining past planetary configurations is 'forensic astronomy', whilst 'forensic musicology' refers to the comparison of two pieces of music in cases of alleged copyright infringement. For the purposes of this book, 'forensic biology' is defined broadly as 'the application of biological sciences to legal investigations' and therefore covers human anatomy and physiology, viruses to vertebrates and topics from murder to the trade in protected plant species.

Although forensic medicine and forensic science only became specialised areas of study within the last 200 or so years, their origins are traceable back to the earliest civilisations. The first person in recorded history to have medico-legal responsibilities was Imhotep, Grand Vizier, Chief Justice, architect and personal physician to the Egyptian pharaoh Zozer (or Djoser). Zozer reigned from 2668 to 2649BC and charged Imhotep with investigating deaths occurring under suspicious circumstances. The Sumerian king Ur-Nammu (ca 2060 BC) began the first codification of laws with the eponymous 'Ur-Nammu Code', in which penalties of various crimes were stipulated. The first record of a murder trial appears on clay tablets inscribed in 1850 BC at the Babylonian city of Nippur.

In England, the office of coroner dates back to the era of Alfred the Great (871–899), although his precise functions at this time remain uncertain. It was during the reign of Richard I (1189–1199) that the coroner became an established figure in the legal system. The early coroners had widespread powers and responsibilities that included the investigation of crimes ranging from burglary to murder and suspicious death. The body of anyone dying unexpectedly had to be kept for inspection by the coroner, even if the circumstances were not suspicious. Failure to do so meant that those responsible for the body were fined, even though it might have putrefied and created a noisome stench by the time he arrived. It was therefore common practice for unwanted bodies to be dragged away at night to become another village's problem. The coroner's responsibilities changed considerably over the centuries, but up until 1980, he was required to view the body of anyone dying in suspicious circumstances.

Although the coroner was required to observe the corpse, he did not undertake an autopsy. In England and other European countries, dissection of the human body was considered sinful and was banned or permitted only in exceptional circumstances until the nineteenth century. Most Christians believed that after a person died, their body had to be buried 'whole'. If it was not, the chances of material resurrection on Judgement Day were slight. The first authorised human dissections took place in 1240, when the Holy Roman Emperor Frederick II decreed that a corpse could be dissected at the University of Naples every five years to provide teaching material for medical students. Subsequently, other countries followed suit, albeit slowly. In 1540, King Henry VIII became the first English monarch to legislate for the provision of human dissections by permitting the Company of Barber Surgeons to examine the corpses of four dead criminals per annum. In 1663, King James II increased this figure to six per annum. Subsequently, after passing the death sentence, judges had the option of decreeing the body of the convict to be buried (albeit without ceremony), or exposed on a gibbet or dissected. Nevertheless, the lack of bodies and an eager market among medical colleges created the trade of body snatching. Body snatchers usually left behind the coffin and the burial shroud, because taking these counted as a serious criminal offence – which was potentially punishable by hanging. Removing a body from its grave was merely a 'misdemeanour'. The modern-day equivalent is the Internet market in human bones of uncertain provenance. A notorious case arose in 2004 when the body of the eminent journalist Alistair Cooke was plundered whilst 'resting' in a funeral parlour in New York. Despite being 95 years old at the time of his death and suffering from cancer, his arms, legs, and pelvis were surreptitiously removed. These were then sold to a tissue processing company for use in surgery or as dental filler. The trade in human bones is legal provided the correct protocols are followed, but it is also highly lucrative and this tempts some people into criminal behaviour.

Although the ancient Greeks performed human dissections, Julius Caesar (102/100–44 BC) has the dubious distinction of being the first recorded murder victim in history to undergo an autopsy. After the assassination, the physician Antistius examined his body. He concluded that although Julius Caesar was stabbed 23 times, only the second of these blows, struck between the first and second ribs, was fatal. The first recorded post mortem to determine the cause of a suspicious death took place in Bologna in 1302. A local man called Azzolino collapsed and died suddenly after a meal and his body quickly became bloated whilst his skin turned olive and then black. Azzolino had many enemies and his family believed that he had been poisoned. A famous surgeon, Bartolomeo de Varignana, was called upon to determine the cause of an accumulation of blood in veins of the liver and that the death was therefore not suspicious. Although this case set a precedent, there are few records from the following centuries of autopsies to determine the cause of death in suspicious circumstances.

The first book on forensic medicine may have been written by the Chinese physician Hsu Chich-Ts'si in the sixth century CE but this has since been lost. Subsequently, in 1247, the Chinese magistrate Sung Tz'u wrote a treatise entitled '*Xi Yuan Ji Lu*' that is usually translated as 'The Washing Away of Wrongs', and this is generally accepted as the first forensic textbook. Sung Tz'u would also appear to be the first person to apply an understanding of biology to a criminal investigation. He relates how he identified the person guilty of a murder by observing the swarms of flies attracted to bloodstains on the man's sickle. In Europe, medical knowledge advanced slowly over the centuries and forensic medicine really only started to be identified as a separate branch of medicine in the 1700s (Chapenoire and Benezech 2003). The French physician Francois-Emanuel Foderé (1764–1835) wrote a landmark 3-volume publication in 1799 entitled *Les lois éclairées par les sciences physiques: ou Traité de médecine-légale et d'hygiène publique* that is recognised as a major advancement in forensic medicine. In 1802, the first chair in Forensic Medicine in the UK was established at Edinburgh University and in 1821 John Gordon Smith wrote the first book on forensic medicine in the English language, entitled '*The Principles of Forensic Medicine*'.

Today, forensic medicine is a well-established branch of the medical profession. Clinical forensic medicine deals with cases in which the subject is living (e.g. non-accidental injuries, child abuse, rape), whilst forensic pathology deals with investigations into causes of death that might result in criminal proceedings (e.g. suspected homicide, fatal air accident). Pathology is

the study of changes to tissues and organs caused by disease, trauma, toxins, and other harmful processes. Theoretically, any qualified medical doctor can perform an autopsy. However, in practise, at least in the UK, only those doctors who have received specialist training in post-mortem pathology conduct autopsies.

The majority of deaths are not suspicious, so an autopsy is unlikely to take place. Indeed, even if a doctor requests an autopsy, the relatives of the dead person must give their permission. Some religious groups are opposed to autopsies and/or require a person to be buried within a short period of death so an autopsy may be refused. For example, many Muslims, orthodox Jews and some Christian denominations oppose autopsies. Some doctors are concerned about how few autopsies take place. This is because some estimates suggest that 20–30% of death certificates in the UK incorrectly state the cause of death. The errors are seldom owing to incompetence or a 'cover-up', but result from the difficulty of diagnosing the cause of death without a detailed examination of the dead body.

There are rogue elements in all professions and the GP Dr Harold Shipman murdered over 200 mostly elderly patients over the course of many years. He did this through administering morphine overdoses and then falsifying the death certificates (Pounder 2003). Dr Shipman's victims suffered from a range of chronic ailments and because of their age and infirmities, nobody questioned the certificates he signed. He also falsified his computer patient records. It therefore appeared that the patient suffered from the condition that Dr Shipman claimed caused their death. He sometimes did this within hours of administering a fatal dose of morphine. Ultimately, suspicions were aroused and several of his victims who had been buried were disinterred and autopsied. The findings indicated that although they may have been infirm, they had not died because of disease. Their bodies did, however, contain significant amounts morphine. Providing the tissues do not decay too much, morphine residues are detectable for several years after death. Dr Shipman had therefore, surprisingly for a doctor, chosen one of the worst poisons in terms of leaving evidence behind. Dr Shipman was found guilty of murdering 15 of his victims in January 2000 and subsequently committed suicide whilst in prison.

In England and Wales, when a body is discovered in suspicious circumstances, the doctor issuing the death certificate or the police will inform the coroner. They can then request that an autopsy be performed regardless of the wishes of the relatives. In this case, the autopsy is undertaken by one of the doctors on the Home Office List of pathologists: as of 23 June 2017, there were 36 of them. Each of the Home Office Pathologists covers one of seven regions of England and Wales. The name is a bit of a misnomer because although the Home Office accredits them, the Home Office does not employ them. Scotland has its own laws and the Procurator Fiscal decides whether a death is suspicious and whether one or two pathologists should conduct the autopsy. The situation in Northern Ireland is slightly different again, with pathology services provided by The State Pathologist's department. Other countries have their own arrangements.

Animals and plants have always played a role in human affairs, quite literally in the case of pubic lice, and have been involved in legal wrangles ever since the first courts were convened. There is a long history of disputes over ownership, the destruction of crops and the stealing or killing of domestic animals. For example, Hammurabi, who reigned over Babylonia during 1792–1750 BC, codified many laws relating to property and injury that subsequently became the basis of Mosaic Law. Among these laws, it states that anyone stealing an animal belonging to a freedman must pay back 10-fold, whilst if the animal belonged to the court or a god, then he had to pay back 30-fold. Animals have also found themselves in the dock accused of various crimes. In the Middle Ages, several cases are recorded in which pigs, donkeys and other animals were executed by the public hangman following their trial for murder or sodomy. The judicial process was considered important, the animals were appointed a lawyer to defend them, and they were tried and punished like any human. In 1576, the hangman brought shame

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on the German town of Schweinfurt by publicly hanging a pig in the custody of the court before due process took place. He never worked in the town again and his behaviour gave rise to the term '*Schweinfurter Sauhenker*' (Schweinfurt sow hangman) to describe a disreputable scoundrel (Evans 1906). Sadly, the phrase has now fallen out of fashion. Today, the owner of a dangerous animal is prosecuted when it wounds or kills someone, although it may still face the death penalty.

During the nineteenth century, a number of French workers made detailed observations on the sequence of invertebrate colonisation of human corpses in cemeteries, and attempts were made to use this knowledge to determine the time since death in murder investigations. Thereafter, invertebrates were used to provide evidence in a sporadic number of murder investigations, but it was not until the 1980s that their potential was widely recognised. Part of the reason for the slow development is the problem of carrying out research that can be applied to real case situations. Pigs, and in particular foetal pigs, are the forensic scientists' usual choice of corpse, although America has a 'Body Farm' in which dead humans can be observed decaying under a variety of 'real life (death?) situations' (Bass and Jefferson 2003). Leaving any animal to decay inevitably results in a bad smell and attracts flies – so it requires access to land far from human habitation. It also requires protecting the body from birds, dogs, and rats that would drag it away. Consequently, it is difficult both to obtain meaningful replicates and to leave the bodies in a 'normal' environment. Even more importantly, in EU countries, these types of experiments conflict with European Union Animal By Products Regulations, which require the bodies of dead farm and domestic animals to be disposed of appropriately to avoid the spread of disease – and leaving a dead pig to moulder on the ground clearly contravenes these.

The use of animals other than insects as forensic indicators has proceeded slowly and that of plant-based evidence has been slower still. The first use of pollen analysis in a criminal trial appears to have taken place in 1959 (Erdtman 1969). Although not widely used in criminal trials since then, its potential is increasingly recognised. By contrast, the use of plants and other organisms in archaeological investigations has been routine for many years. Microbial evidence seldom features in criminal trials, although this is likely to change with the development of new methods of detection and concerns over bioterrorism.

The use of molecular biology in forensic science has expanded rapidly since the landmark Colin Pitchfork case in 1988, and it is now an accepted procedure for the identification of individuals. Indeed, the use of DNA in forensic science is in danger of becoming a victim of its own success. For example, some commentators have voiced concerns that juries (and lawyers) might consider that DNA evidence is mandatory for a successful prosecution and ignore all other sources of information. In addition, the latest DNA sequencing methods can now detect extremely small amounts of DNA. This means that there is an increased risk of detecting contaminating transfer DNA. In addition, new methods of analysing DNA are revealing information about us that is potentially valuable to law enforcement agencies but also poses privacy issues. On a more positive note, the techniques required to analyse non-human DNA are advancing rapidly. This will lead to increasing use of evidence from animals and plants in legal proceedings.

A major obstacle to the use of biological evidence in English trials is the nature of the legal system. In a criminal prosecution case, the court must be sure 'beyond all reasonable doubt' before it can return a guilty verdict. The court therefore requires a level of certainty that science rarely provides. Indeed, science is based upon hypotheses and a scientific hypothesis is one that can be proved wrong – if one can find the evidence. Organisms are affected by numerous internal and external factors and therefore the evidence based upon them usually has to have qualifications attached to it. For example, suppose the pollen profile found on mud attached to a suspect's shoe was similar to that found at the site of a crime. This suggests a

possible association but it would be impossible to state beyond reasonable doubt that no other sites have similar profiles – unlikely perhaps, but not beyond doubt. Lawyers are, quite correctly, experts at exploiting the weaknesses of biological evidence. In particular, it is seldom possible for one to state there is no alternative explanation for the findings or an event would never happen. Within civil courts, biological evidence has greater potential, since here the 'burden of proof' is based upon 'the balance of probabilities'.

Although all biological evidence has its limitations, it is often useful in answering many of the questions that arise whenever a body is found under suspicious circumstances. The first question is, of course, are the remains human? This might be obvious if the body is whole and fresh or even if there is just a skull but sometimes there may be no more than a single bone or some old bloodstains. Assuming that the remains are human, biological evidence can also help to answer the subsequent questions (Table I.1).

Similar questions arise in wildlife crime (e.g. killing of/trade in protected species), neglect of humans and domestic animals, mis-selling of animal products, and food contamination. This book intends to demonstrate how an understanding of biology can answer all these questions. It is designed for undergraduates who may have a limited background in biology and not the practicing forensic scientist. I have therefore kept the terminology simple, whilst still explaining how an understanding of biological characteristics provides evidence. Descriptions of potential sources of biological evidence and tests continue to grow at a bewildering rate. Therefore, it is essential to distinguish between approaches that will be useful in the real world and those that will never proceed further than the laboratory pilot study. To be truly useful any test/source of evidence must be accurate, simple, affordable, and deliver results within an acceptable period (Table I.2). With such a large subject base, it is impossible to cover all topics

Table I.1 Questions arising when a body is found in suspicious circumstances.

Are the remains of human origin?

Who is the victim?

What was the cause of death?

How long ago did the victim die?

Did the victim die immediately or after a period – and if so, how long?

Did the person die at the spot where their body was found?

Did the person die of natural causes, suicide, an accident, or a criminal act?

If the person was killed as a result of a criminal act, who was responsible?

Table I.2 Characteristics of an ideal forensic test.

Accurate: The results must stand up to intense scrutiny in court.

Sensitive: Many forensic samples are extremely small and are finite (i.e. one cannot collect more material once it used up).

Specific: If the test also cross-reacts with other materials, then its accuracy will be compromised. **Quick:** Investigations must not drag on. If there is a chance that a criminal might offend again, they must be apprehended and charged as soon as possible. It is also unfair to keep a suspect in a state of anxiety and/or deprive him/her of his/her liberty for long periods whilst time-consuming tests are conducted.

Simple: The more complex a test becomes the more opportunity there is for mistakes to occur. It also becomes expensive to train people to conduct the tests.

Reliable and repeatable: It is essential that a test is replicable by workers at other laboratories.

Affordable: Financial considerations are important. One cannot employ an exceedingly expensive test on a routine basis.

Equipment and reagents are readily available: The effectiveness of a test is compromised if equipment cannot be used through lack of spare parts or the reagents it requires are difficult to obtain.

in depth and readers wishing to identify a maggot or undertake PCR analysis should consult one of the more advanced specialist texts or review articles mentioned at the start of each chapter. Where information would not otherwise be easily accessible to undergraduate students, I make use of web-based material, although the usual caveats apply to such sources.

This is the third edition of Essential Forensic Biology and although the basic structure is similar, all the chapters have been re-written, updated, and include many new illustrations. Some of the chapters in the second edition are now divided into two, in order to provide greater focus and in-depth coverage of topics. There is also a new chapter on Wildlife Forensics. This is in recognition of the scale of the problem and the consequences it is having on both the environment and human societies. The illegal trade in wildlife is a global problem and often involves other illegal activities and organised criminal gangs. There is also an expanded Companion Website. This includes multiple choice questions (and answers) and short answer questions associated with each chapter. In addition, there are interpretative questions that require the reader to utilise information gained from several chapters. The website now includes numerous photographs that could not be included in the book without increasing its size and cost. I provide some ideas for project work that do not require access to complex laboratory facilities. Because the usefulness of biological material as forensic evidence depends on a thorough understanding of basic biological processes and the factors that affect them, there is plenty of scope for simple projects based upon identifying species composition or that measure growth rates. Obviously, for the majority of student projects, cost, time, and facilities are serious constraints. Although DNA analysis is extremely important in many aspects of forensic biology, it can be expensive and requires specialist equipment. Similarly, the opportunities to work with human tissues or suitably sized dead pigs may not exist. However, one can undertake worthwhile work using the bodies of laboratory rats and mice or meat and bones bought from a butcher as substitute corpses with plants and invertebrates as sources of evidence.

At the start of each chapter, I list a series of 'objectives' to illustrate the material covered. These take the form of examination essay questions, so that the reader might use them as part of a self-assessment revision exercise. I divide the book into a series of conventional chapters but because topics are inter-related, the reader will find certain subjects picked up, put down, and then returned to later. This is also a good way of learning, since it is better to take in bitesized chunks of information and return to them frequently, rather than attempt to grasp all aspects of a topic in a single sitting. The book begins with a discussion of how the human body decays and how one discovers and recovers a dead body. There is then an in-depth consideration of how one conducts DNA and RNA analysis and how this contributes to forensic biology. This area of science is advancing at an incredible speed, but in the process of providing a wealth of information that can help solve crimes, it is also throwing up serious practical and ethical issues. The book then deals with body tissues and fluids as forensic indicators. We then consider how wound analysis can help establish whether a suspicious death was a result of an accident, suicide, or homicide. Film and TV depictions of forensic pathology often suggest a degree of certainty when diagnosing the cause of death that is not always possible. This chapter emphasises the importance of proceeding cautiously and keeping an open mind. There is then a consideration of the animal kingdom as forensic indicators. This begins with the invertebrates. These are used primarily to determine the minimum time since death, although their importance in other scenarios is also considered. We then deal with the vertebrates and, as mentioned previously, there is now a new chapter on wildlife forensics.

The chapter on plants as forensic indicators is now longer because until recently this source of forensic evidence was often overlooked. In addition, there is now more information on plant poisons, such as ricin, because of the concern about their use by terrorists and hostile governments targeting unwelcome critics. There is also more information on the illegal trade in plants, because it is equally important (and profitable) as illegal animal trafficking but seldom receives attention in the popular press. The final chapter on microbes and viruses now includes a detailed consideration of the prospects of microbiomes as forensic indicators. To its supporters, microbiome analysis offers the prospects of revolutionising our understanding of many disease processes and the prospects of new therapeutic approaches. As a spin-off from this, microbiome analysis is increasingly proposed as a new means of answering forensic questions such as the time since death, individual identity, and geographical origin. This chapter considers the strengths of these claims. There is also new information on the transmission of a disease as a criminal act and the use of microbes and viruses in bioterrorism.

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About the Companion Website

The companion website for this book is at

www.wiley.com/go/Gunn/Forensic



The website includes:

- PPTs of all figures in the book
- MCQs as per 2e BCS
- Projects
- Short Answer Questions
- Interpretative Questions
- Interpretative Questions Answers
- Website Images

Scan this QR code to visit the companion website.



Part I

Decay and the Discovery and Recovery of Human Remains

The Decay of Human Bodies

OBJECTIVES

1

Compare the chemical and physical characteristics of the different stages of decomposition. Explain how a body's rate of decomposition is affected by the way in which death occurred and the environment in which it is placed. Compare the conditions that promote the formation of adipocere and of mummification and how these

Compare the conditions that promote the formation of adipocere and of mummification and how these processes preserve body tissues.

1.1 Introduction

The time before a person dies is the *ante-mortem* period, whilst that after death is the *post-mortem* period. The moment of death is the 'agonal period' – the word being derived from 'agony', because it used to be believed that death was always a painful experience. Either side of the moment of death is the *peri-mortem* period, although there is no consensus about how many hours this should encompass. It is important to know in which of these time periods events took place in order to determine their sequence, the cause of death, and whether or not a crime might have been committed. Similarly, it is important to know the length of the postmortem period, referred to as the *post-mortem* interval (PMI). This is because by knowing exactly when death occurred it is possible, among other things, to either include or exclude the involvement of a suspect. The study of what happens to remains after death is 'taphonomy' and the factors that affect the remains are 'taphonomic processes.

When investigating any death, it is essential to keep an open mind as to the possible causes. For example, if the partially clothed body of a woman is found on an isolated moor, there are many possible explanations other than she was murdered following a sexual assault. First, she may have lost some of her clothes after death, through them decaying and blowing away or from them being ripped off by scavengers. Second, she may have been a keen rambler who liked the open countryside. Most people die of natural causes and she may have suffered from a medical condition that predisposed her to a heart attack, stroke, or similar potentially fatal condition whilst out on one of her walks. Another possibility is that she may have committed suicide: people with suicidal intent will sometimes choose an isolated spot in which to die. Another explanation for the woman's death would be that she had suffered an accident, such as tripping over a stone, landing badly, and receiving a fatal blow to her head. And, finally, it is possible that she was murdered. All of these scenarios must be considered in the light of the evidence provided by the scene and the body.

1.2 The Stages of Decomposition

After we die, our body undergoes dramatic changes in its chemical and physical composition and these provide an indication of the PMI. The changes also influence the body's attractiveness to detritivores (organisms that consume dead organic matter) and their species composition and abundance. These also act as indicators of the PMI. Furthermore, the post-mortem events may preserve or destroy forensic evidence, as well as bring about the formation of artefacts. For example, the discharge of bloody fluids from the mouth and nose or the bluish discoloration of the skin, which are perfectly normal consequences of decay, can be mistaken for signs of assault or poisoning. An understanding of the decay process, and factors that influence it, is therefore essential for the interpretation of dead human and animal remains.

Animal decomposition in terrestrial environments can be divided into four stages: fresh, bloat, putrefaction, and putrid dry remains. However, these stages merge into one another and it is impossible to separate them into discrete entities. Indeed, bloat results from the process of putrefaction and therefore is dealt with as a sub-section of putrefaction in this chapter. In addition, a body seldom decays in a uniform manner. Consequently, part of the body may be skeletonised, whilst another part retains fleshy tissue.

1.2.1 Fresh

Once the heart stops beating, the blood pressure drops and blood is no longer moved through the body. The blood within the vessels therefore settles under gravity to the lowermost dependent regions. Consequently, shortly after death, the skin and mucous membranes appear pale. Once the circulation ceases, tissues and cells no longer receive oxygen and nutrients and they begin to die. Different cells die at different rates, so, for example, brain cells die within 3–7 minutes, while skin cells can be taken from a dead body for up to 24 hours after death and still grow in a laboratory culture. Contrary to folklore, human hair and fingernails do not grow after death, although shrinkage of the surrounding skin makes it seem as though they do.

1.2.1.1 Temperature Changes

Because normal metabolism ceases after death, our body starts to cool in a process known as *algor mortis*: literally, the coldness of death. For many years, measurements of body temperature were the principal means of determining the PMI. However, the technique suffers from a variety of shortcomings. To begin with, the skin surface usually cools rapidly after death and the mouth often remains open. Therefore, measurements recorded from the mouth or under the armpits would not reflect the core body temperature. In living persons, one way of determining core body temperature is with a rectal thermometer. However, this approach is not always appropriate in forensic cases. This is because inserting a rectal thermometer often requires moving the body and removing the clothing. This potentially interferes with evidence collection in cases where anal intercourse before or after death occurred.

Nowadays, the body temperature of living humans and many domestic animals is often determined from the temperature in the external auditory canal, measured using a custom-designed electronic digital ear thermometer. This has the advantage of being quick, non-invasive, and does not risk cross contamination or breakage of the thermometer in the body. The external auditory canal temperature correlates well with the brain temperature and it is useful for recording the temperature of dead bodies in hospital settings (Baccino *et al.* 1996). Unfortunately, in forensic scenarios, there are often complications that make the measurement of the ear temperature either difficult or the interpretation of the results questionable (Rutty 1997); for example, if the body is submerged or water enters the ear canal

Table 1.1 Factors affecting the rate at which a body cools after death.

Factors that enhance the rate of cooling

Small body size Low fat content Body stretched out Body dismembered Serious blood loss Lack of clothes Wet clothes Strong air currents Low ambient temperature Rain, hail Cold, damp substrate that conducts heat readily (e.g. damp clay soil) Body in cold water Dry atmosphere

Factors that delay the rate of cooling

Large body size High fat content Foetal position (reduces the exposed surface area) Clothing – the nature of clothing is important because a thin, highly insulated layer can provide more protection than a thick poorly insulated material. Insulated covering (e.g. blanket, dustbin bags, paper, etc.) Protection from draughts Warm ambient temperature Warm microclimate (e.g. body next to a hot radiator) Exposed to the sun Insulated substrate (e.g. mattress) High humidity

from rain or condensation, if there is bleeding into the ear canal following a skull fracture, and if there is traumatic damage to the ears from blows to the head.

A second major problem with using body temperature as a measure of the PMI is that the rate of cooling depends upon a host of complicating factors. These start with the assumption that the body temperature at the time of death was 37 °C. In reality the body temperature may be higher (e.g. owing to infection, exercise, or heat stroke) or lower (e.g. hypothermia or severe blood loss). In addition, the rate of temperature loss depends upon numerous factors (Table 1.1). For example, subcutaneous fat acts as an insulator that reduces the rate at which heat is lost from the body. Adult women tend to have a higher fat content than men, and therefore the bodies of a woman and a man of the same weight cool down at different rates. Similarly, the body of a fat man who dies inside a car on a hot sunny day may not lose heat to any appreciable extent; indeed, his body temperature may even increase.

Various formulae relate body temperature to the length of time since death, but these are mostly too simplistic to be reliable. Clauss Henßge designed a sophisticated nomogram that accounts for body weight and environmental temperature and allows application of corrective factors according to the individual circumstances of the case (Henssge and Madea 2004). A nomogram is a graphical calculator that usually has three scales (Figure 1.1). Two of these scales record known values (rectal and environmental temperature) and the third scale is the one from which the result is read off (time since death). Unfortunately, even this approach has limitations – for example, it is not reliable if the body was left exposed to the sun or if there is reason to believe that it was moved after death. In the latter situation, the body experiences

PERMISSIBLE VARIATION OF 95 % (± h)

TEMPERATURE TIME OF DEATH

RELATING NOMOGRAM



Figure 1.1 Clauss Henßge's nomogram for the determination of time since death from body temperature. *Source:* Reproduced from Henssge and Madea (2004), © Elsevier, with permission. The nomogram works as follows: (a) One draws a straight line between the rectal temperature and the ambient temperature. In this case, one draws a line from 27-15 °C. (b) The 'standard' is a naked body lying in an extended position in still air and therefore 'corrective factors' are applied for any situations other than this. Henssge and Madea (2004) list these factors. In this example, the body wears three thin layers of dry clothes in still air and therefore the corrective factor is 1.3. One multiplies the weight of the body by the corrective factor. The body weighs 70 kg and therefore $70 \times 1.3 = 91$ kg. The nomogram goes up in units of 10 and therefore 91 kg rounds down to 90 kg. (c) One draws a second straight line from the centre of the circle that is found at the left-hand side of the nomogram, so that it hits the intersection of the nomogram's diagonal line and that drawn between the rectal temperature and the ambient temperature in step (a). The line then continues until it hits the outermost circle. (d) Where the line drawn in step (c) hits the 90 kg semi-circle is the time since death (17.2 hours). Where the line hits the outermost circle, one can read off the 95% confidence limits (2.8 hours). Therefore, the person was dead for 17.2 ± 2.8 hours (95% CI).

at least two different environments and therefore spends time cooling at two or more rates. This is not to say that temperature measurements are of limited value, but one should be aware of possible complicating factors.

Body temperature, like most biological measurements of the PMI, is a 'rate method'. Rate methods initiate or cease at the time of death and the subsequent rate of change provides an estimate of elapsed time. Other examples include the increase in the potassium ion concentration in the vitreous humour of the eye, the development of *rigor mortis* and the growth of maggots on the dead body. Rate methods become increasingly inaccurate the longer the PMI, because they are influenced by a wide variety of biotic and abiotic factors. However, as long as their limitations are recognised, they can be extremely useful and concordance between several different methods means the time of death is predictable with a fair degree of confidence. Furthermore, in the absence of any other evidence, an indication is more useful to a police investigation than nothing at all. The other methods of determining the time since death are 'concurrence methods'. They work by evaluating the occurrence of events that happened at known times at or around the time of death. Typical concurrence events are finding that the victim's watch stopped at a particular time as a consequence of being smashed (e.g. following a fall or during a struggle) or that mobile phone records indicate that a victim was alive until at least a certain date and time.

1.2.1.2 Chemical Changes

Owing to the lack of oxygen, after death cellular processes switch from aerobic to anaerobic, and there are dramatic increases and decreases in specific metabolites. Furthermore, as membrane integrity is lost, metabolites redistribute within and between tissues. These changes do not take place uniformly throughout the body at the same time. For example, energy metabolism ceases more rapidly in the blood than the vitreous humour of the eye. Unfortunately, few studies compare changes in different chemicals or compare chemical changes with other existing techniques for determining the PMI. In addition, most studies to date lack field data. More work is also needed on the influence of taphonomic factors, such as temperature, and ante-mortem factors, such as age, drug use and disease, on post-mortem chemical changes. The most commonly used chemical measurement of PMI is the determination of potassium ion concentration in the vitreous humour of the eye. However, there are marked discrepancies between authors concerning its reliability.

1.2.1.3 Hypostasis

Between 20 and 120 minutes after death, hypostasis (also called *livor mortis* and post-mortem lividity) occurs – it develops in all bodies, but may be difficult to observe. Hypostasis is a purple or reddish purple discoloration of the skin caused by the blood settling in the veins and capillaries of the dependent parts of the body (Figure 1.2a). Blood plasma also settles to the dependent regions and this causes oedema (fluid accumulation) and the formation of blisters on the surface of the skin. If the person is lying on their back, hypostasis will develop in the back and those body surfaces adjacent to the ground, whilst if the person is hanging by their neck, pronounced hypostasis will develop in their hands, forearms, and lower legs. It starts as a series of blotches that then spread and deepen in colour with time. Initially, blood remains in the blood vessels, but eventually the red blood cells haemolyse (break down and rupture) and the pigment diffuses out into the surrounding tissues. Here it becomes metabolised to sulphaemaglobin that gives rise to a greenish discoloration. Sulphaemoglobin is not present in normal blood, although it may be formed after exposure to drugs such as sulphonamides. This emphasises that normal decomposition processes may mimic those induced before death or by the action causing death.

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The rate of development of hypostasis varies from body to body and is also influenced by underlying medical conditions, such as circulatory disease. Consequently, there is variation in the literature about when events begin and when they reach their maximal effect. Indeed, hypostasis may not develop at all in infants, the elderly or those suffering from anaemia. Some of the literature suggests that after about 10–12 hours of a body remaining in a set position, the discoloration caused by hypostasis becomes 'fixed'. Furthermore, if the body is then moved and left in a different position, a second area of discoloration forms. Two or more distinct patterns of discoloration therefore indicate movement of the body. However, according to Saukko and Knight (2015), there is so much variation in the time it takes for 'fixation' to develop, if it develops at all, that it is not a reliable forensic indicator of the PMI or evidence of movement after death.

Pressure, whether from tight fitting clothes such as belts and bra straps, a ligature around the neck, ropes used to bind hands together, or corrugations in the surface on which the body is resting, prevent underlying blood vessels from filling with blood. Therefore, these regions appear paler than their surroundings – this is 'pressure pallor' or 'contact pallor'. Whilst the body is fresh, *ante mortem* bruising and hypostasis are distinguishable, because bruising results from the leakage of blood out of damaged blood vessels into the surrounding tissues and the consequent formation of clots. By contrast, in hypostasis, the blood is restricted to dilated blood vessels. However, as time passes and tissues decay, blood begins to leak out of the vessels and it becomes more difficult to distinguish between the two.

Initially, blood remains liquid within the circulatory system after death, rather than coagulating, because of fibrinolysins released from the capillary walls. These destroy fibrinogen and therefore prevent clots from forming. However, wounds inflicted after death do not bleed profusely, because the heart is no longer beating and therefore blood pressure is zero. Therefore, blood from even a severed artery trickles out because of gravity, rather than spurting out as it would during life. A common question on finding a body that had fallen several metres at the base of a building is whether the victim was alive before hitting the floor. This is important because a murderer may attempt to mask wounds caused by an assault within the much greater trauma caused by a fall from a great height. That is, the crime would be mistaken for an accident or suicide. A dead body would bleed less on impact than a live body. Furthermore, a bleeding body would leave stains at the point 'take-off' and cast off stains during the fall.

Unlike the situation on land, a dead body floating on water may suffer considerable loss of blood from wounds. After initially sinking, a dead body rises to the surface owing to the accumulation of gas from the decay process and then floats face downwards. Consequently, the blood pools in the facial and dependent regions and wounds affecting these areas bleed profusely. Bodies floating in the sea frequently suffer extensive post-mortem wounds from dashing against rocks, boats, and other maritime structures. Boat propellers cause serious lacerations to floating bodies and potential dismemberment, whilst seagulls cause stab-like wounds.

1.2.1.4 Changes in Muscle Tone

Immediately after death, muscles usually become flaccid, and the joints relax such that a person's height may increase by as much as 3 cm. Furthermore, the body may be found in a posture that would be highly uncomfortable in life. Once consciousness is lost, a standing individual collapses without attempting to break their fall, whilst a seated individual slumps forwards (usually) and may fall to the floor unless supported. Consequently, the body may receive injuries which might themselves have been life-threatening had the person not already been dead. The relaxation of muscles can lead to the sphincters loosening, and the release of urine and faeces or the regurgitation of gut contents at or shortly after the moment of death. Suffocation can lead to the victim urinating involuntarily, but this may also happen naturally at the time of death. Therefore, it would be unwise to make too much of such findings, unless there was other evidence to indicate that criminal activity was involved. By contrast, when a person is in a coma, the volume of urine in the bladder can increase markedly, because they are not responsive to stimuli that would normally wake them up. Consequently, an unusually distended bladder is an indication that a person was comatose for several hours before they died.

1.2.1.4.1 Rigor Mortis

About 20 minutes after death, *rigor mortis*, the stiffening of muscles and limbs, manifests itself in the eyelids and small muscles of the face. The stiffening then spreads to the other muscle groups over the subsequent hours. There is some discrepancy in the literature about how long it takes for the whole body to become rigid, with various figures of between 2 and 12 hours being quoted. This is because the speed of development of rigor mortis and its duration are both heavily influenced by environmental temperature, with onset commencing earlier and duration shorter at high environmental temperatures. By contrast, onset is delayed at low temperatures and at a constant 4°C may last for at least 16 days, with partial stiffening still detectable up until 28 days after death (Varetto and Curto 2005). Children tend to develop rigor mortis sooner than adults, whilst onset is delayed if death was owing to asphyxiation or to poisoning with carbon monoxide. The extent and degree of rigor mortis is therefore not an especially accurate measure of the PMI.

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Rigor mortis is breakable by pulling forcefully on the affected limbs but, depending upon the time since death, may subsequently re-set. This provides a crude estimate of the time since death. A common assumption is that rigor will re-establish if the person was dead for less than eight hours. However, Anders *et al.* (2013) show that this figure is a considerable underestimate and that while the percentage of instances in which rigor mortis re-establishes declines with time, it can still happen up to 19 hours after death.

Rigor mortis affects both the skeletal and the smooth muscles. When it affects the *arrector pili* muscles, it results in 'goose bumps' and the scalp and body hairs standing on end. This can make it look as though a person died in a state of shock. The *arrector pili* are smooth muscles running from the superficial dermis of the skin to the side of the hair follicles. Normally, our hair emerges at an angle to the skin surface, but when the *arrector pili* are stimulated to contract – for example, as a consequence of the body's response to cold or stress – the hair is pulled into a more upright position. This also gives rise to the phenomenon of 'goose bumps'.

Rigor mortis results from the rise in the intracellular concentration of calcium ions in muscle cells following death. This happens because the membranes around the sarcoplasmic reticulum and the cell surface become leaky. Consequently, calcium ions move down their concentration gradient into the cytoplasm of the muscle cells. This rise causes the regulatory proteins troponin and tropomyosin to move aside, thereby permitting the muscle filaments actin and myosin to form cross bridges. This is possible because the heads of myosin molecules already contain adenosine triphosphate (ATP). However, actin and myosin, once bound, cannot detach from one another, because this process requires ATP – and this is no longer being formed. Thus, the actin and myosin filaments remain linked together by immobilised cross bridges, resulting in the stiffened condition of dead muscles.

Rigor mortis gradually subsides and disappears entirely after about 36 hours, although this happens sooner in warm conditions. The muscles then become extremely flaccid, because the cross bridges between the muscle fibres are broken and the muscle proteins are decaying. Consequently, the anal sphincter and muscles surrounding the anal canal become loose and therefore the anus spreads open. This condition is referred to as 'patulous' and can give rise to a suspicion that the person was subject to anal intercourse/interference. Checking for bruising and abrasions to the anal canal and the presence of sperm or foreign bodies could alleviate this suspicion. Similarly, in women, the vulva and vagina become more distensible. This should not be mistaken for evidence of sexual assault, especially in children below the age of consent.

Rigor mortis can indicate the relationship between a body and a situation. This is because once rigor mortis develops, it results in a body taking on a fixed position. Consequently, the body cannot adopt a new position until rigor is broken. For example, a body that is curled to fit into the boot of a car or large suitcase will keep this position after removal. Its position can therefore provide an indication as to whether a particular car or suitcase might have been the means of transport. Similarly, a body found with one or more limbs raised without an obvious means of support suggests that rigor set in whilst the body was in a different position that supported the limb(s). The relaxation of muscles that accompanies death means that a standing person almost always slumps to the floor. However, Pirch *et al.* (2013) describe an unusual case in which the body of a dead woman was found in a standing position. The situation arose because at the time of death she was leaning against supports that kept her body upright whilst rigor mortis set in.

Exposure to sub-zero temperatures causes the body to stiffen but prevents the onset of rigor mortis entirely. In this case, the body becomes flaccid when it warms up and then subsequently exhibits rigor mortis. In this way, a murderer may confuse a police investigation by storing his victim in a freezer immediately after death before disposing of it later. One of the best-known examples of this is the case of the mafia hitman, Richard Kurlinski (1935–2006). He earnt the

sobriquet 'the iceman', because he would sometimes store his victims in a commercial deep freeze and dispose of the bodies months or even years later. His *modus operandi* was discovered when he didn't let one of his victims defrost thoroughly and the body was discovered on a warm summer's night with a partially frozen heart (Zugibe and Costello 1993).

There is a considerable literature in the food science sector on means of distinguishing between fresh and frozen meat. However, there are far fewer studies on human tissues. Miras *et al.* (2001) suggest that it is possible to identify previously frozen muscle tissue by its higher levels of the enzyme short-chain 3-hydroxyacyl-CoA dehydrogenase. It is uncertain how effective this would be in practice and would presumably rely on discovering the body within a few hours of defrosting. Freezing results in ice crystal formation, both within cells and in extracellular fluids and osmotic damage owing to changes in ion balances. Consequently, histological changes such as extended extracellular spaces and cell shrinkage are observable in histological sections (Schäfer and Kaufmann 1999).

1.2.1.4.2 Cadaveric Rigidity

So-called 'cadaveric rigidity', 'instantaneous rigor' or 'cadaveric spasm' is a contentious term. Some forensic pathologists dispute its existence, whilst others consider it a genuine phenomenon, albeit rare (Bedford and Tsokos 2013; Fierro 2013). Unlike rigor mortis, 'cadaveric rigidity' allegedly sets in immediately after death and affects part or all of the body. A common belief is that cadaveric rigidity occurs in the bodies of people who die in a state of extreme emotional and physical stress. For example, cadaveric rigidity is often given as the reason a person who shoots himself is found tightly grasping his gun, whilst a murder victim is found with tufts of his assailant's hair clenched in his hands. However, most murder victims and many who die of painful medical conditions are highly stressed at the time of death. It is also surprising that cadaveric rigidity does not appear to be documented as a feature of those who underwent judicial executions in the UK during the nineteenth and twentieth centuries. The law demanded that a doctor be present at these executions and he would subsequently undertake a postmortem. Therefore, if extreme stress is a requirement for cadaveric rigidity, then one would expect it to be relatively common rather than a rare phenomenon.

To date, no physiological mechanism explaining how instantaneous rigor might occur is available. Most instances are probably a consequence of gravity and positioning retaining the object in a person's hand during the initial muscle relaxation phase. Afterwards, normal rigor mortis sets in and the object becomes firmly grasped again.

1.2.1.4.3 Heat Stiffening

Heat stiffening is distinct from rigor mortis and results from exposing a body to extreme heat. This causes the body to exhibit a 'pugilistic posture' and evidence of severe burning will inevitably be apparent.

1.2.1.5 Indications of Poisoning in a Fresh Body

Sometimes the cause of death results in striking changes to normal skin coloration. For example, deaths from carbon monoxide poisoning often result in a cherry red/pink coloration to the skin, lips, and internal body organs (Figure 1.2b). However, several hours after death, the coloration may not be immediately apparent, owing to the settling of the blood to the dependent regions. Carbon monoxide gas forms during the combustion of many substances and poisoning is a common feature of accidental deaths in which people are exposed to fumes from a faulty gas boiler or during fires and suicides, in which the victim breaths in vehicle exhaust fumes. Carbon monoxide poisoning may cause death in homicides resulting from arson or the deliberate blocking of the flue to a fire or gas boiler. Carbon monoxide has much greater affinity than

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oxygen for the haeme molecule of haemoglobin. Therefore, even at low atmospheric concentrations, it rapidly replaces oxygen and thereby reduces the oxygen carrying capacity of the blood. When carbon monoxide binds with haemoglobin in the blood or myoglobin in the muscles, it forms carboxyhaemoglobin and carboxymyoglobin respectively and they are responsible for the pink coloration. However, carbon monoxide poisoning does not always result in the formation of a cherry pink coloration (Carson and Esslinger 2001) and it can be difficult to spot when the victim is dark skinned – though it may be apparent in the lighter regions such as the palms of the hands or inside the lips or the tongue. There are big differences in susceptibility to carbon monoxide poisoning. This is partly a consequence of age, size, and general health. For example, children are more susceptible owing to their higher respiration rate.

Cyanide poisoning also causes cherry red skin coloration, although it is said to be somewhat darker than that caused by carbon monoxide. Cyanide may be ingested as a means of suicide and homicide, and it is sometimes a lethal component of smoke. Cyanide forms during the combustion of many substances (e.g. wool, plastics) and its effect in conjunction with carbon monoxide is additive, since they work by different mechanisms. Indeed, a person inhaling smoke may die of cyanide poisoning before there is a marked rise in the levels of carboxyhaemoglobin. Cyanide affects a variety of enzymes and cell processes, but has its principal effect through the inhibition of cytochrome oxidase and thereby prevents the production of ATP via oxidative phosphorylation. The cherry red coloration results from the increased oxygenation of the blood in the veins, as a consequence of the inability of cells to utilise oxygen for aerobic metabolism.

Cyanide poisoning also causes cyanosis – a bluish tinge to the skin, fingernails and mucous membranes. However, the term cyanosis derives from the blue-green colour cyan rather than the chemical cyanide. Cyanosis may be localised or widespread, and be found on its own or in conjunction with cherry red skin coloration. It results from a reduction in the level of oxygen in the blood and therefore darker deoxygenated blood imparts colour to the tissues, blood vessels, and capillaries, rather than the normal bright red oxygenated blood. Cyanosis is therefore a common symptom of a whole range of conditions that interfere with the supply of oxygenated blood to the tissues, including carbon monoxide poisoning, a heart attack, and asphyxia. Cyanide has a reputation for causing rapid, near instantaneous death. However, a lot depends on the nature of the cyanide and its means of delivery (e.g. breathing in gaseous hydrogen cyanide, ingestion of a salt in solid or liquid form, or absorption through the skin) and the dose. Death may occur within minutes of acquiring a lethal dose or take several hours. The longer the time the victim struggles to breathe the greater the probability that cyanosis will develop.

Toxicological tests are required to confirm the presence of poisons. This can be problematic if there are no clues as to what the poison(s) might be. This is because many poisons induce non-specific pathologies, but they can only be identified through specific analytical tests – and there are thousands of potentially lethal chemicals. Some poisons are difficult to detect after death and this makes it difficult to implicate them as the cause. For example, Sastre *et al.* (2013) describe an interesting case in which a mother and child died in their flat as a consequence of suspected hydrogen sulphide poisoning. Hydrogen sulphide is an extremely toxic gas that inhibits mitochondrial cytochrome enzymes and therefore cellular respiration. Its repellent smell of rotten eggs means that people usually escape from the source before they breathe in a fatal amount. However, high levels are not detectable, because they induce olfactory paralysis. In this case, the gas emanated from a blocked sink and the victims' positions indicated they lost consciousness rapidly. The woman probably caused a sudden release of large amounts of gas when she attempted to unblock the sink. Immediately after discovering the woman and child, the rescuers opened all the windows because of the awful smell.

Consequently, subsequent measurements of the air in the flat were 'normal'. The investigators were surprised that hydrogen sulphide was not detectable in the blood of either the woman or the child. Therefore, they analysed the levels of hydrogen sulphide in the lungs and these proved to be at potentially fatal concentrations. This emphasises the importance of not relying solely on blood analyses when there are grounds for suspecting a poisonous gas was inhaled. During the late1990s, there were numerous cases of people committing suicide in Japan through manufacturing hydrogen sulphide using commercially available cleaning products and recipes downloaded from websites (Morii *et al.* 2010). The 'craze' has since spread to other parts of the world. It presents risks to those who treat the victims or handle their dead bodies, if this is done in a confined space with insufficient ventilation.

The heavy metal thallium is often referred to as the 'poisoner's poison', because its salts, such as thallium sulphate, are colourless, tasteless, highly toxic and have a reputation for being difficult to detect in foods and tissues. Thallium poisoning has various non-specific consequences, depending on the dose ingested and death may not take place until weeks after ingestion. Thallium ions are similar in size to potassium ions and therefore enter via the potassium pores found on all cell membranes. Once inside the cells, the precise mechanism by which thallium exerts its effects is/are not certain, but it is known to interfere with ATP production. Although older literature states that thallium is difficult to detect, new techniques such as inductively coupled plasma mass spectrometry (ICP-MS) are more effective. Li *et al.* (2015) provide a discussion of two cases of thallium poisoning.

The use of traditional poisons, such as cyanide and arsenic, for both homicide and suicide, has declined in developed countries. This is a consequence of restrictions on the sale of products containing such harmful products to the public. Instead, many cases of poisoning are nowadays associated with the intake of narcotics and pharmaceuticals. By contrast, in developing countries and especially those in which there are many peasant farmers, poisoning with insecticides and other agrochemicals is a major cause of mortality. For example, in Bangladesh during the year 1996–1997, 14% of all reported deaths of women aged 10–50 years were due to poisoning and, whilst the numbers have since declined, they remain remarkably high (Chowdhury *et al.* 2011; Yusuf *et al.* 2000). This is because agrochemicals can be purchased easily and these often include toxic chemicals that are banned in other parts of the world.

Case Study: The Poisoning of Alexander Litvinenko with Polonium

Alexander Litvinenko died in London, UK, of polonium poisoning on 23 November 2006. He was a former KGB agent who fled Russia and became a prominent dissident. He openly criticised the Russian president, Alexander Putin, accusing him and his government of corruption. On 1 November, he had various meetings, with other ex-KGB agents and people with connections with the Russian state security, in London restaurants. One of these men allegedly spiked Litvenko's tea with polonium-210. Some hours later, Litvinenko fell seriously ill and was admitted to hospital. The cause of his illness could not be ascertained, but he accused Putin of arranging for him to be poisoned. It was initially suspected that Litvinenko was poisoned with thallium, because it had been used by the KGB in the past and it causes similar pathology to that seen in Litvinenko. However, thallium was not present in his body and the presence of polonium was not discovered until after he died. Although polonium-210 is a radionucleotide, it emits only alpha particles and this was the reason for the delay in identification. Alpha particles have high energy and damage DNA and other biomolecules, but they lack the pene-trative abilities of gamma radiation. Therefore, once inside the body's tissues, they cannot be

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detected by Geiger counters. This also means that it is easy for polonium to be moved covertly within and between countries. Because it emits only alpha particles, it does not require heavy screening for safe transportation. In addition, radioactivity monitors at airports detect gamma radiation and therefore would not register its presence. Polonium is incredibly poisonous and weight for weight is about 2.5×10^{11} times as toxic as hydrogen cyanide (Haynes 2014). Consequently, a fatal dose is small enough to hide easily and safely among personal luggage.

Although polonium-210 has a relatively short half-life of 138.4 days, the traces of radioactivity meant that investigators could identify where Litvinenko was poisoned. Because of the manner in which Litvinenko was poisoned, others, such as restaurant workers and guests, were also exposed (e.g. from subsequently handling/drinking from the teapot and cup and from cross contamination). However, few laboratories are equipped to measure the presence of alpha particle emitting isotopes from biological samples. Consequently, screening was only offered to those who were deemed most likely to have been at risk (Maguire *et al.* 2010). Therefore, a telephone help line was set up to offer reassurance to members of the public and this received calls from 872 individuals, 7 of whom remained seriously worried because they suffered from health-related anxiety (Morgan *et al.* 2008). This emphasises that criminal acts often have consequences for the wider community, including many who have no association with the event.

Although polonium is widespread in nature and even detectable in cigarette tobacco (Radford and Hunt 1964), it is present in small amounts. Virtually all the polonium used in research and industry comes from Russian nuclear reactors – and it is extremely expensive. According to White (2008), the dose that killed Litvinenko would have cost over US\$ one million. It is baffling why anyone should choose such an elaborate and expensive means of murdering someone, unless it was to demonstrate that no expense or effort would be spared. The main suspects soon moved back to Russia and to date the Russian government refuses to cooperate with the investigation into Litvinenko's death.

1.2.2 Putrefaction

Some authors distinguish several stages of putrefaction (decay), but the usefulness of this is uncertain. As the body enters the bloat stage, it is said to be 'actively decaying'. During this time, the soft body parts disappear through the actions of autolysis and microbial, insect, and other animal activity. The body then collapses in on itself as gasses are no longer retained by the skin. At this point, the body enters 'advanced decay' and, unless the body is mummified, much of the skin is lost.

Obese people tend to decay faster than those of average weight. According to Campobasso *et al.* (2001), this is due to the 'greater amount of liquid in the tissues whose succulence favours the development and dissemination of bacteria'. At first sight, this is surprising, since fat has a lower water content than most other body tissues and obese individuals therefore have a lower than average water content. However, fat acts as a 'waterproofing' agent, preventing the evaporation of water and therefore the drying out of the corpse, whilst its metabolism yields large amounts of water. Fat is also an insulator and this reduces the rate at which the body cools down after death. Consequently, decomposition and bacterial growth proceeds faster in a person with a high fat content.

1.2.2.1 Bloat

Our intestines are packed with bacteria and other microbes and they do not die with us. After death, these microbes break down the dead cells of the intestines, while some, especially the *Clostridia* and the enterobacteria, start to invade the other body parts. At the same time, the

body undergoes its own intrinsic breakdown, known as autolysis. This results from the release of enzymes from the lysosomes (subcellular organelles containing digestive enzymes), thereby causing cells to digest themselves. In addition, acid from the stomach and proteolytic and hydrolytic digestive enzymes naturally present in the body's organs contribute to the decay process. Sometimes the stomach wall decomposes within a few hours of death and consequently its contents enter the peritoneal cavity. This can give the misleading impression that a ruptured stomach contributed to the person's death. Similarly, the pancreas is packed with digestive enzymes, and so rapidly digests itself. Autolysis is not solely a post-mortem phenomenon and may also occur on a more restricted scale in a living person as a consequence of certain diseases.

Decomposing tissues release pigmented chemicals and gasses which make the skin discoloured and blistered, starting on the abdomen in the area above the caecum. Indeed, the skin can be so darkened that a person who had pale skin in life is mistaken for being black, whilst the raised fluid-filled blisters can be mistaken for burns. The front of the body swells, the tongue may protrude, and bloody fluid from the lungs oozes out of the mouth and nostrils. This is accompanied by a terrible smell, as hydrogen sulphide and various sulphur-containing organic molecules (mercaptans) are produced as end products of bacterial metabolism. Methane (which does not smell) is also formed in large quantities and contributes to the swelling of the body. In temperate countries such as the UK, this stage is reached after about four to six days during spring and summer, but takes longer during colder winter weather.

The accumulation of gas results in dramatic rises in the internal body pressure, thereby causing the body to swell and the skin to stretch. This distorts facial features making recognition impossible and gives a misleading impression of obesity (Figure 1.3). The rise in internal pressure distorts and obscures wound sites and can cause the skin to split at weak points such as healing scar tissue. For example, Byard *et al.* (2006a) describe a case in which the body of a



Figure 1.3 Late bloat stage of decomposition. The body is about seven days old and exhibits pronounced swelling owing to accumulation of gas. Note discoloration of the skin and exudates from the mouth and nose. The swelling makes recognition of facial features impossible. Internal pressures force the tongue out, the eyeballs bulge, and the skin splits. *Source*: Reproduced from Payne-James *et al.* (2011), © 2011 Hodder Arnold, London, with permission.

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man who had been dead for about 10 days was found with three incised wounds to his lower abdomen and groin. This raised suspicions that he was cut either at the time of death or shortly afterwards. Closer observation of the wound sites revealed suture material and that the incisions did not extend to the abdominal cavity. In fact, the man died naturally of a heart attack. The accumulation of gas during decay process caused the opening of incisions made during bypass surgery four to six weeks previously. The rise in internal pressure is sometimes sufficient to cause the entire skin to split. A possibly apocryphal instance of this occurred in 1547, whilst the corpse of King Henry VIII was transported back to Windsor Castle for burial. His body allegedly swelled to such an extent that his coffin exploded overnight and dogs were found feeding on the exposed remains in the morning. This was deemed to be divine judgement on the king for his dissolution of the monasteries.

1.2.2.2 Putrefactive Rigor

Putrefactive rigor or putrefactive rigor mortis arises after rigor mortis has subsided and does not involve changes in muscle tone. It is associated with the accumulation of large amounts of gasses beneath the skin surface because of normal putrefaction. The gasses cause the body to balloon and this distends the limbs and appendages. The internal pressure on the joints results in the body adopting a default pose in which there is 'nearly full extension of the elbow and slight abduction and forward flexion of the shoulder' (Gill and Landi 2011). The upper limbs therefore become raised without any visible support, whilst the upper legs flex upwards. This gives the body the unsettling appearance of being about to deliver a hug or of someone falling backwards into a swimming pool. In males, putrefactive rigor results in a post-mortem artefact known as 'pseudo-priapism', in which the penis becomes erect. This has nothing to do with the normal physiological process of engorgement. Priapism is a medical condition in which the penis remains in a permanently erect state in the absence of sexual stimulation. This is far from desirable and is not only painful but results in serious pathology and potential impotence. Priapism results from a variety of conditions, including diseases such as sickle cell anaemia, scorpion stings, the consumption of aphrodisiacs, and damage to the spinal cord. Consequently, the body of a male found with an erect penis requires a forensic explanation to exclude the possibility of a criminal act.

It is important to distinguish between rigor mortis and putrefactive rigor, because finding a body in an odd position could indicate that it was moved after death. The two forms are distinguishable because rigor mortis occurs shortly after death and subsides before advanced decomposition begins. By contrast, putrefactive rigor occurs through putrefaction and there is marbling of the skin and the stench of decay. Putrefactive rigor may, however, appear soon after death in countries (or situations) with hot moist climates in which decay sets in rapidly (Tsokos and Byard 2012).

1.2.2.3 Adipocere

Adipocere (grave wax or corpse wax) is a fatty substance described as being whitish, greyish or yellowish, and with a consistency ranging from paste-like to crumbly. Extensive adipocere formation inhibits further decomposition and ensures that the body is preserved for many years (Figure 1.4). Adipocere formation is therefore a nuisance in municipal graveyards, because it prevents the authorities from recycling grave plots but useful to forensic scientists and archaeologists who wish to autopsy long-dead bodies.

The term 'adipocere' refers to a complex of chemicals rather than a single chemical compound and it results from the breakdown of body lipids. After death, autolysis and bacterial decomposition of triglycerides, which make up the majority of the body's lipid stores, results in



Figure 1.4 The formation of adipocere has preserved the body of this child, despite it being buried for about three years. *Source*: Reproduced from Payne-James *et al.* (2011), © 2011 Hodder Arnold, London, with permission.

the production of glycerol and free fatty acids. The free fatty acids comprise of a mixture of both saturated and unsaturated forms, but as adipocere formation progresses, the saturated forms predominate. The fatty acids lower the surrounding pH, thereby reducing microbial activity and further decomposition. Adipocere has a characteristic odour, the nature of which changes with time. This smell is used to train cadaver dogs to detect dead bodies. Extensive adipocere formation results in the body swelling. Consequently, the pattern of clothing, binding ropes or ligatures imprints on the body surface, whilst incised or puncture wounds close and become obscured.

Adipocere forms in bodies in many situations, including fresh water, seawater and peatbogs, shallow and deep graves, tightly sealed containers, and in bodies buried but not enclosed at all (Evershed 1992; Mellen *et al.* 1993). Some workers state that warm conditions $(21-45 \,^{\circ}C)$ speed its formation, but adipocere develops on bodies in seawater at $10-12 \,^{\circ}C$ and icy glaciers (Ambach *et al.* 1992; Kahana *et al.* 1999). Indeed, the preservation of the 5300-year-old 'Iceman' ('Ötzi'), discovered in the South Tyrol region, is at least partly because of adipocere (Bereuter *et al.* 1997).

Tissues with high lipid content are more prone to form adipocere. Clothing that absorbs and retains moisture facilitates adipocere formation in both terrestrial and aquatic conditions (Notter and Stuart 2012). Most authors emphasise the importance of anaerobic conditions and anaerobic bacteria such as *Clostridium* species for the formation of adipocere.

The time required for extensive adipocere formation is case-dependent, with reports citing durations ranging from days to months to over a year. Therefore, it is impossible to use adipocere formation as an estimate of the PMI. However, because adipocere leaks out of the body, its presence in the soil can indicate post-mortem movement. For example, if the amount of adipocere in the underlying soil is commensurate with the state of decay of the body, then it suggests that the body rested in its current position since death. By contrast, if there is extensive adipocere formation in the body but little adipocere in the soil, it is possible that the body was moved there recently. Alternatively, the presence of adipocere in the soil but the absence of a