THE AGE OF A WOUND IN THE LIVING
A medical practitioner may be required to estimate the period that has elapsed between the infliction of a wound and the time of examination of a complainant or an accused, on the basis of the naked-eye appearances of the wound. An exact determination of the age of a wound by this method is not possible as the intensity of the local inflammatory reaction varies. Under average conditions, however, the edges of a wound are red and swollen after a lapse of about 12 hours. A small wound may show scab formation after approximately 24 hours, and when a wound has become infected, pus may be seen after a period of about 36 hours.
Epithelium begins to grow at the edges of a wound after about 24 hours and epithelialisation of small clean wounds may be complete in 4 to 5 days.

The naked-eye appearances of a wound depend to a large extent upon whether healing is proceeding with or without infection. Once infection has supervened, healing may be delayed and it is usually impossible to determine the age of the wound with any degree of accuracy.

DISTINGUISH BETWEEN ANTE-MORTEM AND POST-MORTEM WOUNDS AND THE ESTIMATION OF THE APPROXIMATE AGE OF AN ANTE-MORTEM WOUND

It may be possible on naked-eye examination to state that a wound is ante-mortem in origin if it shows evidence of a marked inflammatory reaction. In cases of doubt an ante-mortem wound must be distinguished from a post-mortem wound by a microscopic examination for evidence of tissue reaction. Although margination and a limited emigration of leucocytes may occur in tissues in response to injury after somatic death, marked cellular exudation and reactive changes in the tissue cells are seen in ante-mortem wounds only. The absence of tissue reaction, however, does not necessarily indicate that a wound was post-mortem in origin. There may have been insufficient time before death for the development of tissue reaction, or, in the case of small wounds, the reaction may have terminated in resolution. In small wounds such as small contusions, the degree of cellular injury may have been insufficient to elicit an appreciable leucocytic exudation, while in severe injuries the associated circulatory failure may have interfered with the normal reaction.

The intensity of the local reaction to an injury depends upon many factors, such as the severity of the injury, the vascularity of the injured tissue, and the presence or absence of infection or foreign bodies. The course of tissue reaction is therefore variable. For this reason it is never possible to determine the age of an ante-mortem wound within narrow limits, but an estimate of its approximate age may be made by comparing the microscopic reactive changes in the wound with the course of tissue reaction as it has been observed experimentally. When the age of an ante-mortem wound has to be determined for medico-legal purposes, medical practitioners should submit portions of the excised wound, preserved in formol-saline, to a pathologist for histological examination.

According to Moritz the time of occurrence of the reactive changes of inflammation to an aseptic mechanical injury under experimental conditions is as follows: dilatation of the capillaries and margination of the leucocytes may be seen within a few minutes of the injury. Emigration of leucocytes is usually observed within an hour of the injury; the first leucocytes to pass into the tissues are the polymorphonuclear neutrophils. Monocytes appear at a later stage and are seldom seen in the exudate before the lapse of about 12 hours. In traumatic aseptic inflammation the exudation usually reached its maximum intensity within 48 hours of the injury. Reactive changes in the tissue histiocytes and
swelling of the vascular endothelium may be observed within an hour of the injury. The fibroblasts at the site of injury show reactive changes within a few hours and these cells begin to undergo mitotic division about 15 hours after the injury. The rate of proliferation of the fibroblasts and the formation of new capillaries varies, but it usually takes at least 72 hours for vascularized granulation tissue to develop. The time taken for the formation of collagen varies, but new fibrils may be seen in the injured tissue within 4 to 5 days of the injury. In small wounds a fibrous tissue scar may be apparent at the end of one week. The presence of infection leads to considerable modification in the time of occurrence and the duration of the changes which have been described. Once infection has supervened it may be maintained for days or weeks and the age of a wound cannot be determined reliably in these circumstances.

The microscopic evidence relied upon for the determination of the age of a wound includes histochemical as well as conventional histological techniques. Raekallio,\textsuperscript{14,15} in a study of human autopsy material claims that the microscopic demonstration of enzyme reactions in wounds provides evidence that they were sustained before death. The medico—legal application of such new techniques to the determination of the age of an ante-mortem injury requires further evaluation.

Robertson and Hodge,\textsuperscript{16} using conventional histological techniques, classified the changes which occur in ante-mortem abrasions during the healing process into the following age groups:

1. Dead on arrival at hospital or surviving less than 4 hours;
2. Survival period: 4 to 12 hours
3. Survival period: 12 to 48 hours
4. Survival period: 2 to 4 days
5. Survival period: 4 to 8 days
6. Survival period: 8 to 12 days
7. Survival period: over 12 days.

The foregoing classification provides reasonable confidence limits within which the ante-mortem age of an abrasion can be determined. Their work confirms the earlier observations by Robertson and Mansfield\textsuperscript{17} demonstrating the difficulty of determining the age of an ante-mortem bruise with any accuracy.

**DISTINCTION BETWEEN ANTE-MORTEM AND POST-MORTEM BRUISES**

It is usually accepted that small bruises resembling ante-mortem bruises can be produced if marked force is applied to a body within a few hours of death. If the force applied after death is sufficiently great, the capillaries in the affected area may be ruptured and blood may be extravasated into the tissue spaces, with the production of a bruise which is similar in structure to an ante-mortem bruise.

It may be impossible to distinguish an ante-mortem bruise from a post-mortem bruise if the death occurs rapidly after the injury, but if the death is delayed, these bruises may be differentiated on microscopic examination. Moritz has described the microscopic changes which a bruise undergoes during life, and
has drawn attention to the criteria upon which an estimate of the approximate age of a bruise may be based. These criteria include the rate of disintegration of the red cells and the extent and character of the tissue reaction. If the red cells have lost their shape and staining characteristics, and if iron-containing pigment is demonstrable either at the site of injury or in the regional lymph nodes, it is probable that at least 12 hours have elapsed since the injury. The presence of tissue reaction of a degree beyond a margination and limited emigration of the white cells would indicate that the bruise was probably ante-mortem in origin.

The differentiation between ante-mortem bruises and post-mortem dissection artefacts of the neck has been dealt with at page 84.

HAEMORRHAGE FROM ANTE-MORTEM AND POST-MORTEM WOUNDS

Evidence of profuse haemorrhage from a wound usually indicates that it was received before death. The absence of haemorrhage, however, even when a relatively large blood vessel has been injured, does not necessarily indicate that the wound was post-mortem in origin. This condition may be seen in ante-mortem wounds where shock has been a major factor in causing the death.

External haemorrhage may occur from post-mortem wounds, but such bleeding is usually slight in amount unless a large blood vessel or a vein in a dependent portion of the body has been injured. It should be noted that ante-mortem wounds may continue to bleed externally after death, particularly if they are situated in a dependent portion of the body.

Problems which arise in determining the amount of bleeding which can occur into a pleural cavity before death and after death may be illustrated by the findings in a case reported by one of us:18

In a murder trial an unmeasured volume of blood (estimated at 1-1½ pints for the first time by the medical practitioner concerned 4 months after he had performed the autopsy) was found in the right pleural cavity of the deceased, who had been shot in the back of the chest and the back of the head.

The bullet wound in the chest shattered the 5th rib posteriorly. The intercostal vessels were torn across and the bullet wound traversed the upper lobe of the right lung. It made its exit through the second intercostal space in front.

At the preparatory examination medical experts for the Crown expressed the view that it would have taken at least half an hour for 1-1½ pints of blood to accumulate in the pleural cavity as a result of the bullet wound in the chest. They regarded the head wound as almost immediately fatal and drew the inference that the deceased had been shot in the chest in another part of the city and then brought to the place where he was ultimately found and where the fatal head wound was administered.

On this basis two accused persons were indicted with murder.

The Crown's medical hypothesis raised several interesting points for consideration:

1. How rapidly during life can 1 to 1½ pints of blood accumulate in the chest cavity following a bullet wound of the type described?
2. To what extent can post-mortem bleeding account for the quantity of blood found at autopsy?

It was submitted that the Crown's contention that it would have taken at least half an hour for 1 to 1½ pints of blood to accumulate in these circumstances, was unacceptable and provided no basis for the view that the chest wound was inflicted before the head wound.

The impropriety of guessing the volume of blood in the chest cavity from inspection, instead of measuring the amount, was a matter for criticism, especially in view of the grave inferences which the Crown sought to draw on the basis of this observation.
During life bleeding will continue until arrested by the usual processes of clotting and contraction of the vessel; but death may occur before this happens. The amount of bleeding may be considerable within the short space of 3 or 4 minutes. This depends, *inter alia*, on the size of the vessels injured.

It is often overlooked that vessels of substantial calibre take a course close to the periphery of the lung and that they may be a source of considerable bleeding from lung wounds.

The absence of clots in cases of intrapleural bleeding does not assist in determining whether the bleeding occurred *ante mortem* or *post mortem*. Sellors has pointed out that blood shed in the pleural cavity in most cases does not appear to have clotted. He attributes this phenomenon to the rapid defibrination of the blood as it is being shed. The blood is subjected to violent agitation; the collapsed lung splashes about in the blood, bouncing with each transmitted cardiac pulsation. There is little chance for any firm clot to form. Fibrin shreds may be deposited in the course of this process.

The possibility that the autopsy dissection has contributed to the quantity of blood found in the chest cavity must be excluded. This source of contamination may be considerable and rapid. We have demonstrated that when clear fluid is aspirated out of the chest cavity (for measurement of the quantity) bleeding from divided vessels can colour the pleural fluid within a matter of seconds. In cases of this nature, therefore, the dissection technique described by Gonzales *et al.* is to be commended. They state:

The sternoclavicular joint is disarticulated by cutting downward from the top of the joint and then cutting outward at right angles; these joints and the first costal cartilages are not cut until the pleural cavities are inspected, for fear of contaminating them with blood from severed innominate vessels.

The extent of bleeding after death depends basically on the fact that in almost all circumstances the blood is liquid *post mortem* in most parts of the body. This applies particularly to the peripheral vessels and the capillaries.

Other factors include the patency of the divided vessels, e.g. the lack of obstruction to the lumen by firm clot, the influence of gravity and the size and nature of the vessels concerned, i.e. the importance of veins as opposed to arteries, which are generally almost empty.

In the chest cavity, damage to the intercostal vessels may have a special significance. The right intercostal vessels drain into the vena azygos, which receives the superior and inferior hemiazygos veins from the left side. The vena azygos itself forms an anastomosis between the superior vena cava and the inferior vena cava.

If, therefore, the blood is liquid and if gravity assists drainage, for purely anatomical reasons there will be a vast reservoir of liquid blood which may be drained through even a small incision.

In view of the absence in the literature of quantitative data on the amount of post-mortem bleeding from lung wounds, experiments were conducted by Shapiro and Robertson on recently dead bodies. The results are shown in Table 5.

These experiments indicate the very considerable amount of bleeding which may occur after death from wounds of the lung tissue.
DISTINCTION BETWEEN SUICIDAL, ACCIDENTAL AND HOMICIDAL WOUNDS

In most deaths the circumstances are such that it is usually possible to state whether the fatal injury was of accidental, suicidal or homicidal origin. Difficulties may arise, however, when bodies are found in places such as fields or on the roadside or in deserted dwellings. In such cases medical practitioners are usually called to the scene before the body is moved and they should carefully note the position of the body, the state of the clothing, the position of blood stains and the condition of the surroundings. As a general rule the police take a photograph of the body in the position in which it was found. The nature of the injury in these cases must be determined from the character of the wounds taken in conjunction with a knowledge of the circumstances of the injury.

THE CHARACTER OF THE WOUNDS

The presence of a large number of wounds is usually suggestive of homicide if an accident can be excluded, but multiple wounds may occasionally be self-inflicted. Suicidal wounds may be found in any part of the body which can be reached by the person concerned, but certain sites such as the front of the neck,

---

Table 5. Results of Experiments on Post-mortem Bleeding of Lung Wounds (After Shapiro and Robertson).  

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Case History</th>
<th>State of Body</th>
<th>Time between Infliction of Wound and Opening of Body</th>
<th>Cause of Death and Main Findings</th>
<th>Amount of Blood in Right Pleural Cavity</th>
</tr>
</thead>
<tbody>
<tr>
<td>59/60</td>
<td>Female, aged 35 years Sudden collapse</td>
<td>Warm to the touch; tissues appear fresh</td>
<td>20 minutes</td>
<td>Hypertensive cardiac disease; left ventricular hypertrophy; left lung slightly congested; no fluid</td>
<td>375 c.c.</td>
</tr>
<tr>
<td>40/60</td>
<td>Male, aged 22 years Collapsed at work; died in ambulance en route to hospital</td>
<td>Warm to the touch; tissues appear fresh</td>
<td>15 minutes</td>
<td>Cerebral haemorrhage; old head injury with cortical scarring</td>
<td>50 c.c.</td>
</tr>
<tr>
<td>87/60</td>
<td>Male, aged 16 years Knocked down by motor-car</td>
<td>Slightly warm to the touch; tissues appear fresh</td>
<td>15 minutes</td>
<td>Multiple fractures involving all the bones of the vault of the skull with cerebral contusion; no thoracic injury</td>
<td>50 c.c.</td>
</tr>
<tr>
<td>98/60</td>
<td>Male, aged 35 years Drowned in car which drove over the jetty</td>
<td>Slightly warm to the touch; post-mortem interval 6 hours</td>
<td>30 minutes</td>
<td>Drowning; lungs oedematous but not markedly congested; no injuries present</td>
<td>800 c.c. (Clots present)</td>
</tr>
<tr>
<td>192/60</td>
<td>Male, aged 28 years Collapsed at work</td>
<td>Post-mortem interval 5 hours</td>
<td>7 minutes</td>
<td>Subendocardial fibrosis; pyelonephritis with marked scarring and contraction of the kidneys</td>
<td>175 c.c.</td>
</tr>
<tr>
<td>149/60</td>
<td>Male, aged 62 years Collapsed whilst sitting at home; died en route to hospital</td>
<td>Post-mortem interval 6 hours</td>
<td>30 minutes</td>
<td>Slight emphysema of upper lobes only; vascular atherosclerotic disease marked; no injury</td>
<td>1000 c.c.</td>
</tr>
</tbody>
</table>

Procedure: A scalpel was passed through the rib interspace 2½ inches to the right and 3 inches below the spine of the seventh cervical vertebra. The body was opened in the usual way, after lying recumbent for an interval. The blood in the right pleural cavity was measured with precautions to avoid contamination by the dissection technique.
the front of the left wrist, the upper portion of the left thigh and the front of the chest are commonly selected for these injuries. Wounds of the back and the back of the neck are generally homicidal in origin, but suicidal wounds in these regions have been described. Wounds of the front, back and sides of the head may be accidental or homicidal, but wounds on the top of the head are usually of homicidal origin. Suicidal and homicidal wounds of the neck are considered at page 252. Multiple incised wounds of the forearms, palms and fingers are suggestive of an assault in which the victim has attempted to defend himself. The recognition of self-inflicted fabricated wounds has been dealt with at page 201.

A combination of fractures of the bones of the legs and lumbar and head injuries is suggestive of a pedestrian traffic injury (p. 286).

THE CIRCUMSTANCES OF THE INJURY

A weapon found firmly grasped in the hand of the deceased is strong presumptive evidence of suicide. The finding of hair or a portion of clothing in the hands of the deceased, and not belonging to him, may be indicative of homicide (p. 16). The finding of a weapon beside the body is not necessarily indicative of suicide as a murderer may leave a weapon at the scene of the crime to simulate suicide. Conversely, a person committing suicide may have had sufficient time to dispose of the weapon.

Evidence of a struggle or the presence of drag marks at the scene is suggestive of homicide. As it is unusual for a person committing suicide to injure himself through his clothes, the presence of a wound in a region ordinarily covered by clothing without any corresponding damage to the overlying clothes is suggestive of suicide. Recent tears in clothing and the loss of buttons may be indicative of a struggle and homicide.

A weapon is often produced as an exhibit at an inquest or criminal trial and a medical witness may be asked whether the injuries found could have been caused by that particular weapon. In certain circumstances it may be possible to show that the pattern of an injury corresponds to the form of the weapon with which it has been produced, but this is exceptional (p. 196). As a general rule it is impossible to show that a particular wound has been caused by a specific weapon, and the medical witness should therefore confine himself to an opinion as to whether the injuries could or could not have been produced by the exhibit weapon.

It will be apparent that the manner in which an injury is produced cannot be determined on the basis of any general rules; every case has to be considered on its own merits in relation to the particular circumstances obtaining at the time of the injury.

PERIOD OF SURVIVAL AFTER INJURY

In cases where death has occurred from haemorrhage, a medical witness may be asked to estimate the length of time that the deceased could have lived after the infliction of the injury. This period will depend upon the nature of the injury, the degree of associated shock and the rate of bleeding. The degree of shock and the rate of bleeding cannot be determined from the autopsy findings. Wounds of the heart, the lungs and the great blood vessels often lead to death within a few
minutes of the injury, but cases have been recorded where persons have lived for many hours with large wounds of these structures. A medical witness should be cautious before expressing an opinion in this type of case.

VOLUNTARY ACTIVITY BEFORE DEATH IN RAPIDLY FATAL INJURIES

In cases where the injuries are presumed to have caused rapid death, a medical witness may be asked whether it would have been possible for the deceased to have performed some voluntary act, such as walking, after the infliction of the injury. Individuals vary in their reaction to injuries. A punctured wound of the heart, for instance, may lead to instantaneous death in one person, while another person with a similar wound may be capable of walking or running a considerable distance before collapsing. In the same way, although it is unusual, a person may remain conscious for several minutes before dying from a severe intracranial injury. Unless it can be shown that an injury would have been immediately incompatible with life, it is seldom possible to state that a deceased person could not have performed some activity before his death.

REFERENCES

13. Some of the data in this section have been obtained from A. R. Moritz, *The Pathology of Trauma*, Lea & Febiger, Philadelphia, 1942, pp. 19–35.
13. Firearm Wounds

An appreciation of the nature of firearm wounds depends upon a knowledge of the structure of firearms and ammunition, and upon an understanding of the mechanism of discharge of projectiles.

THE STRUCTURE OF FIREARMS AND CARTRIDGES

A firearm consists of three parts—the chamber, the leed, and the bore. The chamber is situated at the back or breech end of the weapon and contains the cartridge before it is fired. The chamber is connected by a short cone, known as the leed, to the bore of the weapon, and the bore extends from the leed to the muzzle or front end of the weapon. In shotguns the inner surface of the bore is smooth from end to end. In the revolver, automatic pistol, and rifle the bore is cut longitudinally into a series of spiral grooves and these weapons are therefore termed rifled weapons.

In a rifled weapon, the portions of the bore between the grooves are known as lands. The lands project into the bore between the grooves, and the calibre of the weapon is measured across the bore between a pair of lands. When a bullet is fired it passes through the bore and its surface comes into contact with the projecting spiral lands. In this way a spinning motion is imparted to the bullet and this motion keeps the projectile stable in its flight.

The general features of the structure of bullet and shotgun cartridges are shown in Figs. 13.1 and 13.2. It will be seen that both types of cartridge contain a charge of propellant powder. In the bullet cartridge this powder fills the cylinder up to the level of the base of the projectile and in the shotgun cartridge the powder is separated from the pellets by wads. The propellant powders used in bullet and shotgun cartridges are composed of black powder or smokeless powder. Black powder consists of a mixture of potassium nitrate, charcoal, and sulphur. Smokeless powder consists of cellulose nitrate either alone or in combination with nitro-glycerine. Apart from their difference in chemical composition, smokeless powders also vary in physical characteristics. The differences in the size, shape, and colour of the grains of some of the commoner smokeless powders are shown in Fig. 13.3. All cartridges have a percussion cap which is set into the base of the cartridge cylinder. This percussion cap contains a small amount of explosive mixture, known as the priming.

MECHANISM OF DISCHARGE OF PROJECTILES

When a weapon is fired, the priming is detonated by the striker or firing pin and the explosion ignites the powder in the cartridge case. This ignition results in
the evolution of a relatively large volume of gas within the confined space of the cylinder, and the pressure of this gas forces the bullet or shot out of the cartridge and through the bore of the weapon.

The release of the projectile is accompanied by the discharge of flame, of hot compressed gases and of unburned or partially burned grains of powder from the weapon. The flame, discharged gases and powder grains produce effects on the skin in the region of the entrance wound if the weapon is fired at close range. The flame burns the skin and often singes the hair, while the deposit from the gases blackens the skin surface. The deposit of unburned powder grains on the skin gives rise to the condition known as tattooing. Tattooing by black powder is readily recognized on naked-eye examination but a hand-lens may be necessary to demonstrate the tattooing of smokeless powder. The amount of tattooing diminishes as the range of fire increases (Fig. 13.4).

Low power rim fire cartridges such as the 6 mm BB cap and the 6 mm CB cap, particularly as manufactured in Western Europe, may not contain propellant gun powder, but only a primer compound. These cartridges, therefore, cannot
produce the tattooing which is found after the discharge of cartridges containing propellant powder.

Fatal bullet wounds may be produced by rifles, revolvers, or automatic pistols. In all bullet injuries there is usually an entrance wound, a bullet track in the tissues, and an exit wound. An exit wound is absent in cases where the bullet becomes lodged in the tissues after its penetration.

FIG. 13.2. Shotgun cartridge showing (from above downwards) pellets, wads and powder charge.

REVOLVER AND AUTOMATIC PISTOL BULLET WOUNDS

Revolver and automatic pistol bullet wounds are the most important in medico-legal practice. There are practical advantages in considering together the wounds caused by these weapons. The calibres of revolver and pistol bullets are approximately the same (Figs. 13.5 and 13.6), and both bullets produce entrance wounds which are essentially similar.

Revolver bullets are composed of lead which has been hardened with a small percentage of metal alloy, while most automatic pistol bullets consist of a central core of soft lead encased in a hard coating such as nickel or cupro-nickel.
FIG. 13.3. Propellant powders.
Revolvers have a muzzle velocity of about 600 to 900 ft. per sec. and are termed low-velocity weapons, while automatic pistols are higher-velocity weapons with a muzzle velocity up to 1400 ft. per sec. The power of penetration of the tissues is greater with pistol bullets than with revolver bullets.

FIG. 13.4. Variation in the amount of powder grain deposit at ranges of 3 in., 6 in., 9 in. and 12 in.
Information about the following facts may be obtained from an examination of bullet wounds:
1. The Range of Fire.
2. The Direction of Fire.
3. The Kind of Weapon and the Type of Ammunition used.
4. The Nature of the Wound, i.e. whether it was of Accidental, Suicidal or Homicidal Origin.

FIG. 13.5. Revolver cartridges with calibres ranging from 0.455 in. to 0.22 in. Note the projecting rims around the edges of the bases of the cylinders.

FIG. 13.6. Automatic pistol cartridges with calibres ranging from 0.455 in. to 0.25 in. The third cartridge from the right is a blank cartridge (0.32 in.). Note the grooves around the bases of the cylinders.

1. THE RANGE OF FIRE
The range of fire may be determined from the character of the entrance wound.

Close-range and Contact Wounds
When a revolver or automatic pistol is discharged very close to or in contact with the surface of the skin, the gases produced by the explosion pass into the tissues with the bullet and cause considerable laceration of the skin and sub-
cutaneous tissues. Under these conditions the bullet entrance wound has a cruciate appearance (Fig. 13.7).

At close range there is usually some burning, blackening, and tattooing of the skin around the bullet entrance opening, while the hair in the region of the wound is often singed. In contact wounds, the whole of the discharge passes into the tissues through the bullet entrance opening and the burning, blackening, and powder grain deposit are found in the depths of the wound. The burning, blackening, and tattooing effects are relatively less marked with smokeless powder than with black powder.

FIG. 13.7. Suicidal firearm wound showing point-blank revolver bullet entrance wound in region of right temple. Note cruciate appearance of wound.

**Range up to About 6 in.** When a revolver or automatic pistol is discharged at a range of about 6 in., the lacerating and burning effects of the gases are usually lost owing to the dispersion and cooling of the gases before they reach the skin. Under these conditions the bullet entrance wound is circular in shape and is surrounded by a narrow zone of desquamation and bruising of the skin. Blackening of the skin is sometimes seen, while tattooing is invariably found at this range. The general appearance of a homicidal close-range revolver bullet entrance wound is shown in Figs. 13.8A and 13.8B.
FIREARM WOUNDS

FIG. 13.8 A. Homicidal firearm wound. The arrow points to the bullet entrance wound between the nose and right eye; B. Same case as Fig. 13.8A. Enlarged photograph of bullet entrance wound. The arrow points to particles of cordite indicating that the deceased was shot at close range.

Range Beyond 6 in. Beyond a range of 6 in. all trace of blackening usually disappears, while the bullet opening remains circular in shape. Tattooing is still present, and is usually seen up to ranges of about 16 to 20 in. with medium-calibre weapons, but the limit within which powder tattooing can occur varies with different weapons and different cartridges.
Factors Governing the Maximum Range of Tattooing
The amount of tattooing and the maximum distance to which the powder grains can be discharged depend upon the calibre and barrel length of the weapon, and upon the rapidity and degree of combustion of the powder in the cartridge case. Under average conditions, the larger the calibre the greater the distance to which the powder is discharged, e.g. tattooing may occur up to ranges of about 30 to 36 in. with heavy-calibre weapons such as the 0.45 in. revolver.

Tests to determine the maximum possible distance to which the powder can be discharged should be performed in all cases where it is important to ascertain the range of fire. If possible, these tests should be carried out with the weapon used at the crime and with similar ammunition.

Range of Fire beyond the Range of Tattooing. The range of fire of a revolver or automatic pistol cannot be scientifically determined in cases where the weapon is discharged beyond the range of powder tattooing.

2. THE DIRECTION OF FIRE

The direction of fire can often be determined from the nature of the entrance wound and from the direction of the bullet track in the tissues. A bullet track is situated between the entrance wound and the exit wound, or between the entrance wound and the site of lodgement of the bullet where through-and-through penetration of the body has failed to occur.

The Direction of Fire as determined from the Entrance Wound
Bullets usually strike the skin at right angles and the entrance wound has a cruciate or circular appearance according to the range of fire. In certain circumstances, however, a bullet entrance wound may be irregular in shape even though the weapon was fired at right angles. The spin which is imparted to a bullet when it is fired exerts a gyroscopic effect on the bullet and its motions are therefore comparable to those of a spinning top. Like a spinning top, a bullet has a slight wobble when first fired, it then rights itself, and may commence to wobble again when it slows down. If the skin is struck by a wobbling bullet the entrance wound may be irregular in shape and it may resemble the type of wound produced when the skin is struck obliquely.

When a bullet strikes the skin obliquely the entrance wound is usually oval. As the obliquity of fire is increased the wound becomes elongated in shape, and if the skin is struck at a tangent penetration may fail to occur and only a slight linear furrowing of the skin is produced.

The Direction of Fire as determined from the Bullet Track
A bullet usually travels through the tissues in a straight line so that the direction of fire may be determined from its track. Under certain conditions, however, a bullet may be deflected from its course and this occurs most commonly when it strikes bone in its passage through the tissues. For instance, in one of our cases a
bullet struck a rib over the front of the chest and was deflected in the subcutaneous tissues to the back of the chest without penetrating the thoracic cavity. In addition, a bullet may be deflected from its course if it commences to wobble when it meets the resistance of the tissues. In these circumstances the direction of fire cannot be determined from the course of the bullet track.

3. THE KIND OF WEAPON AND THE TYPE OF AMMUNITION USED

If a weapon is discharged beyond contact or very close range at right angles to the skin surface, the approximate calibre of the bullet may be determined from the diameter of the entrance wound. Under these conditions the diameter of the opening is slightly smaller than the calibre of the bullet because the skin is stretched at the moment of penetration. As a general rule the calibre of a bullet cannot be determined if it strikes the skin surface obliquely.

In certain cases small portions of a bullet may be found in the wound and these portions should be removed and handed to the investigating officer for transmission to the ballistics expert for analysis. By suitable chemical and spectroscopic methods it may even be possible to identify small fragments of bullets adherent to the margins of a wound. In close-range wounds the type of ammunition used may also be determined if unburned powder grains are found on the skin or in the wound. Most of the smokeless powders have distinct chemical and physical properties and such powders may be readily recognized by the ballistics expert.

4. THE NATURE OF THE WOUND

As in the case of wounds in general, the nature of a firearm wound has to be determined from the circumstances of the injury taken in conjunction with the character of the wound. In most firearm wounds the circumstances are such that it is usually possible to state whether the injury was of accidental, suicidal or homicidal origin.

If a revolver or pistol is found firmly grasped in the hand of the deceased it is strong presumptive evidence of suicide. The finding of a weapon beside the body, however, is not necessarily indicative of suicide as a murderer may leave a weapon at the scene of the crime to simulate suicide. Conversely a person committing suicide may have had sufficient time to dispose of the weapon provided that the wound was not immediately incompatible with life.

Suicidal bullet wounds usually occur at contact or close range. Hatcher, Jury and Weller state that 'more than two-thirds of the people who shoot themselves aim for the brain, either through the roof of the mouth, at the temple or through the forehead. Almost all the rest aim for the heart.' Homicidal wounds can occur at any range and may be situated in any region of the body. It is uncommon to find a suicidal bullet injury with an entrance wound in the back. The existence of several bullet wounds on a body suggests that they are of homicidal origin but does not exclude suicide.
ADDITIONAL POINTS OF MEDICO-LEGAL IMPORTANCE

BULLET EXIT WOUNDS

A bullet exit wound is usually irregular in shape with its edges everted and torn. Burning, blackening, and tattooing are not seen in relation to the wound. In the case of contact entrance wounds, exit wounds are smaller than such entrance wounds, but at longer ranges they are usually larger than entrance wounds. Wobbling bullets may emerge from the tissues sideways and produce considerable laceration of the skin.

THE WOUNDING POWER OF BULLETS

Callender\(^2\) has shown that the most important factor in the wounding power of a bullet is its striking velocity. For example, he states that if a 0.45 calibre rifle bullet weighing 220 gr. strikes a target at a velocity of 600 ft. per sec. it will deliver 176 ft.-lb. of kinetic energy. At the same velocity, a 0.30 calibre rifle bullet weighing 150 gr. will have a striking power of only 120 ft.-lb. But when the 0.30 calibre bullet is fired at a speed of 2000 ft. per sec., it has a kinetic energy of 1330 ft.-lb. or more than seven times that of the larger slower rifle bullet. Callender states that the wounding power of a bullet is proportional to its mass multiplied by the cube of its velocity.

Another factor which influences the wounding power of a bullet is the density of the tissues, e.g. destruction is greater in dense tissue such as bone than in soft tissues. The wobbling of a bullet within the body can also result in considerable damage to the tissues. In its passage through the body a bullet may become fragmented and the fragments, together with portions of bone or other tissues, may act as secondary missiles and be driven in all directions away from the wound track. These secondary missiles may leave the body through a series of exit holes.

The explosive effects of a high-velocity bullet are well illustrated in a case described by Stevenson.\(^3\)

The remains of a human body were found in dense bush on a farm. These remains are shown in Fig. 13.9A. The tissues showed extensive putrefactive changes. Most of the ribs, vertebrae, and long bones were found in a disarticulated condition. The bones of the skull had not been separated though the sutures and 41 fragments of fractured skull were recovered. Some of these fragments are seen in the foreground of Fig. 13.9A. The various fragments were numbered and pieced together and the vault of the skull was reconstructed (Fig. 13.9B). A portion of bone in the right parietotemporal region was missing but a small fragment of the right temporal bone (marked 15 in Fig. 13.9A) had a crescentic edge. This edge appeared to form part of a hole in the skull. A clean-cut appearance was observed in the outer table along the edge and a large hole was seen in the inner table. Deposits of nitrites and nitrates were found on the surface of the fragment 15, close to the crescentic edge. These findings suggested that a high-velocity bullet had entered the skull at close range through the right temporal bone. It was subsequently determined that the deceased had been shot at close range with a high-velocity service rifle.

BULLET WOUNDS OF THE HEAD

In most of these injuries the bullet passes completely through the skull and as it enters the skull from without it produces a clean-cut hole in the outer table and a
FIG. 13.9A. High velocity bullet wound of the head (see text). Note fragments of skull in foreground; B. High velocity bullet wound of the head (see text). Reconstructed skull. The arrow points to the entrance hole in the right temporal bone.
larger hole in the inner table (Fig. 13.10A). At its point of exit from within these conditions are reversed and the hole in the outer table is larger than the hole in the inner table (Fig. 13.10B). These facts are often of importance in determining the direction of fire in bullet injuries of the head. The track of a bullet through the brain tissues varies greatly. At long ranges a high-velocity bullet may pass through the skull in a straight line and produce little damage to the brain tissues away from its immediate track. On the other hand, considerable damage may be produced if the bullet is deflected from its course within the skull. At closer ranges a high-velocity bullet may produce explosive effects in the brain tissues. These effects result from the dispersion of the bullet's energy throughout the brain tissues.

FIG. 13.10A. Bullet entrance wound of the skull; on the left as seen from without, and on the right as seen from within.

FIG. 13.10B. Bullet exit wound of the skull; on the left as seen from within, and on the right as seen from without.

Fissured fractures often radiate through the vault and base of the skull from the bullet entrance and exit holes. In certain bullet injuries of the head the bullet may fail to emerge from the skull and in these cases an area of bony comminution is often found at the site of lodgment. When a bullet strikes the head at a
tangent penetration of the skull may fail to occur but the force of the bullet may be sufficient to fragment the inner table at the site of impact. Delayed pressure effects on the brain may be produced by these fragments.

**SHOTGUN WOUNDS**

Shotgun injuries are most commonly accidental in nature.

When a shotgun cartridge is fired the pellets begin to disperse soon after the cartridge has left the weapon. This dispersion increases with the range of fire but it also depends upon the degree of 'choking' of the barrel of the weapon. The term 'choking' refers to the constriction of the bores of certain shotguns at their muzzle ends. The degree of constriction is maximal in the ‘full choke’ weapon and is of lesser extent in the 'half choke' weapon. The purpose of the ‘choking’ device is to keep the charge of shot in a single mass for some distance before dispersion commences.

At close ranges (up to about 36 in.) the shot enters the body in a single mass with all types of shotguns. In these circumstances a single large irregular lacerated wound is produced (Fig. 13.11). Burning, blackening, and tattooing are seen around and in the depth of the wound. The wad is often found in the wound.

![FIG. 13.11. Accidental close range shot gun wound of the neck.](image)

As the range of fire increases to a few yards the burning, blackening, and tattooing disappear, while the charge of shot begins to spread so that small apertures, due to separate pellets entering the skin, appear around the central opening caused by the main mass of shot. In addition, an independent injury may be
caused by the wad. The dispersion of shot increases at greater ranges—the greater the dispersion the greater the range. The actual spread depends upon the type of boring of the weapon.

The direction of fire of a shotgun can only be determined from the entrance wound in close-range injuries.

The general appearance of entrance and exit wounds produced by shotgun pellets is shown in Fig. 13.12. In this case the deceased was shot by an assailant who stood in front and to the one side of him at a range of about 10 yards. Three pellets entered the skin of the left loin at an angle from before backwards and emerged a few inches behind the entrance wounds without penetrating into the abdominal cavity. The deceased then turned and received eight pellets from the next shot in the axilla. Two of these pellets emerged a few inches in front of the two upper entrance wounds and two pellets lodged in the tissues above and to the left of the left nipple.

FIG. 13.12. Homicidal shot gun wounds (see text). Three pellet entrance and exit wounds are seen in the region of the left loin. Eight pellet entrance wounds are seen in the axilla. Two pellet exit wounds are seen in front of the upper two entrance wounds, and two pellets are lodged in the tissues above and to the left of the left nipple.

who stood in front and to the one side of him at a range of about 10 yards. Three pellets entered the skin of the left loin at an angle from before backwards and emerged a few inches behind the entrance wounds without penetrating into the abdominal cavity. The deceased then turned and received eight pellets from the next shot in the axilla. Two of these pellets emerged a few inches in front of the two upper entrance wounds and two pellets lodged in the subcutaneous tissues to the left and above the left nipple. The four remaining pellets penetrated the left lung and the heart.

REFERENCES
15. Medico-legal Aspects of Acute Alcoholic Intoxication

The intoxicating component in alcoholic drink is ethyl alcohol (C₂H₅OH). It has a specific gravity of 0.79, i.e. 1 ml of alcohol weighs 0.79 g. The alcohol content of some common beverages is shown in Table 6.

When an alcohol-containing beverage is drunk, alcohol can be absorbed directly into the blood stream from the stomach (about 20 per cent), especially if this organ does not contain food. Such alcohol as has not been absorbed from the stomach (about 80 per cent) passes through to the next part of the digestive tract (the small gut) from which it is then absorbed into the blood stream.

The alcohol in the blood draining from the stomach and intestines passes (for entirely anatomical reasons) to the liver, where it is destroyed by a specific enzyme at a constant rate. Then it flows to the venous side of the heart, whence it is directed through the lungs and back to the arterial side of the heart, from which it is delivered through the arteries to all the organs and tissues of the body, *inter alia*, the brain.

From the forensic point of view, it is primarily the action of ethyl alcohol on the brain that is of practical importance. The alterations in the functions of this bodily system may interfere with the ability of a person to carry out tasks which require normal judgment and normal co-ordination of muscular movements.

Table 6.
Approximate Percentages of Pure Alcohol in Certain Beverages Commonly Consumed

<table>
<thead>
<tr>
<th>Nature of Beverage</th>
<th>Alcohol Content</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>By Volume</td>
</tr>
<tr>
<td>Light beer (lager or pilsener)</td>
<td>4 to 6</td>
</tr>
<tr>
<td>Heavy beer (ale or stout)</td>
<td>6 to 8</td>
</tr>
<tr>
<td>Natural wines (claret or hock types)</td>
<td>9 to 14</td>
</tr>
<tr>
<td>Fortified wines (port, sherry or muscadel types)</td>
<td>16 to 20</td>
</tr>
<tr>
<td>Liqueurs</td>
<td>About 30</td>
</tr>
<tr>
<td>Spirits (whisky, brandy, gin, etc.)</td>
<td>About 30</td>
</tr>
</tbody>
</table>

* In many countries, 42.7 per cent is the minimum legal limit of absolute alcohol in potable spirits, and is, therefore, also the maximum strength which is usually supplied commercially.

The action on the brain depends, among other things, on the amount of alcohol in the blood reaching the brain. This blood concentration is influenced by a variety of factors.

**ABSORPTION FROM THE INTESTINES**

It is important to appreciate that only alcohol which has been absorbed into the blood stream has any effect on behaviour. Any alcohol which is in the cavity of the stomach or the intestines is still virtually outside the body and can, therefore, be neglected in assessing the influence of this drug on behaviour. Its only significance is the evidence it provides that alcohol has been ingested.

Alcohol in the stomach may, however, diffuse into the blood and the tissues after death, and so lead to the estimation of higher blood levels than actually existed during life, if blood samples are taken from parts into which post-mortem diffusion has taken place.

Alcohol is one of the few substances which can be absorbed directly from the stomach. Its absorption, however, is slower from the stomach than from the small gut. On the other hand, its absorption from the stomach is faster when the stomach is empty than when it contains food.

Food affects the rate of absorption in varying degree, probably by delaying the emptying time of the stomach. Starch, protein and fatty foods have all been shown to delay absorption of alcohol from the intestines into the blood. A mixed meal can depress the maximum concentration of the blood alcohol by about half. Drinking milk has a similar effect. Nickolls states that with the presence of food in the stomach as much as 17 to 20 per cent of the alcohol ingested appears to escape absorption and never appears in the blood stream. Alha refers to convincing evidence that food ingested with alcohol may prevent 10 to 20 per cent of the ingested alcohol from being absorbed.

When alcohol is ingested in man in a diluted form, it is absorbed more slowly than when taken in a concentrated form. A lower maximum level is attained in these circumstances, indicating a slower rate of absorption. Not all workers agree with this observation. There is also evidence that alcohol is absorbed most rapidly at concentrations of 10 to 20 per cent, lower and higher concentrations being absorbed more slowly. Harichaux et al. state that alcohol in weak concentrations (12 per cent) accelerate gastric emptying in man, whereas high concentrations (45 per cent) retard it.

The type of beverage also affects the rate of absorption, quite apart from the difference in alcohol concentration. When whisky is diluted to the same strength as stout, the alcohol is absorbed more rapidly and rises to a higher level in the case of the whisky. This indicates that substances are present in stout which retard absorption from the gut, probably by an effect on the emptying time of the stomach. The nature of these substances is not precisely known.

This principle may be the basis for the claim that ‘mixing of drinks’ induces greater intoxication than would be expected from the amount consumed. In such circumstances, substances may be present or may be formed which affect the rate of emptying of the stomach, with more rapid absorption of the alcohol contained in the drinks.
However, absorption is generally complete in a matter of one to three hours, irrespective of the concentration of the alcohol and the type of beverage. After absorption is complete, there is an equilibrium between the alcohol concentration in the blood and in the tissues. It is from blood samples taken at this period that certain calculations are often made. Shortly after taking a drink, e.g. after 15 minutes, the level in arterial blood may be 40 to 60 per cent higher than the level in peripheral venous blood, and this may persist for the first hour of drinking. But after absorption has been completed, the alcohol level in venous blood is slightly higher than in capillary (i.e. arterial) blood. Blood samples should therefore only be taken after equilibrium has been established.

Habituation or tolerance also influences absorption. Habituated heavy drinkers absorb alcohol more rapidly than do abstainers. This may partly be due to an increased rate of absorption of the alcohol as a result of a more rapid emptying of the stomach. This difference does not apply between moderate drinkers and abstainers.

The emotional state of the subject may influence the contractions of the stomach and so affect its emptying time and thus the rate of absorption.

Drugs, e.g. benzedrine or atropine, may slow the rate of absorption of alcohol, probably, in part, by retarding the emptying time of the stomach.

Alha claims that alcohol is absorbed less rapidly (delayed maximum) by younger (under 35) than by older (over 35 years) subjects; but Lofthus found no difference in the tolerance of two such groups.

Gastrectomy may lead to a greatly accelerated rate of absorption of alcohol. Fleming et al. noted accelerated peak blood alcohol concentrations after truncal vagotomy and a drainage operation. They suggested that the acceleration in alcohol absorption was probably due to an increased rate of gastric emptying after surgery.

Absorption by Inhalation. Blood levels may also be affected by the inhalation of alcohol. Lester and Greenberg in determinations of the percentage of alcohol absorbed from the inspired air, showed that on the average about 62 per cent of the alcohol was absorbed. The concentration of alcohol attained in the blood is proportional to the concentration of the alcohol in the inspired air and to the rate of ventilation, and inversely proportional to the weight of the subject.

The inhalation of alcohol, under certain conditions, can result in intoxication.

The Weight of the Subject. When the same amount of alcohol is drunk by a group of persons, the concentration of the alcohol reached in the blood is not the same in the different persons in the experiment. This is due to an important extent to the amount of tissue in the body capable of absorbing alcohol. Bone and fat do not, apparently, absorb alcohol, as the water-containing tissues do. The fatty, bony or muscular nature of the subject is therefore of great importance. It is also recognized that in the same subject the alcohol concentration after the same dose of alcohol rises to different levels on different occasions.

Widmark attempted to calculate what fraction of the body weight can absorb alcohol. He found this fraction to be lower in women than in men and that it had
a range from 0.51 to 0.85. Other investigators have found even larger ranges. Alha\textsuperscript{19} claims that in more than 80 per cent of his subjects the fraction had a value between 0.61 and 0.80, but the range extended from 0.52 to 0.89. The fraction may also vary in one and the same person at different times and seems to be independent of the amount of alcohol drunk.

Widmark's findings indicate that corpulent subjects have a lower fraction than have thin subjects. As it is difficult to assess the proportion of fat and bone in any person, the body weight of the subject is not necessarily a reliable guide to the amount of tissue into which the alcohol is absorbed.

It is a not uncommon practice to assume that about two-thirds of the body weight is available for the absorption of alcohol in a person of average weight, e.g. 150 lb or 70 kg, under hypothetical average conditions. The results of such calculations are of interest as generalizations but do not necessarily have any application to an individual case.

McCallum and Scroggie\textsuperscript{20} have shown that, in fact, the blood alcohol concentration does not decrease uniformly as the weight of the subject increases. These observations invalidate procedures for correcting the alcohol value obtained by adjusting it according to a formula depending on the body weight of the subject. This unwarranted adjustment is, for example, recommended in reports published by the British Medical Association.\textsuperscript{21,22} It is also the theoretical but unjustified basis of the calculations in the Tables drawn up by Sapeika\textsuperscript{23} for the so-called simple determination of the amount of alcohol ingested by an individual.

It is thus impossible to make accurate calculations and probably undesirable to attempt to do so.

THE EXCRETION OF ALCOHOL

Alcohol is eliminated through all the bodily routes of excretion. About 5 per cent of ingested alcohol is excreted in the breath and about 5 per cent in the urine. Thus, in ordinary circumstances, with the ingestion of large doses of alcohol, about 10 per cent is eliminated in the breath and the urine. Negligible amounts are eliminated in the sweat and the faeces. The small amount excreted in the saliva is usually swallowed and therefore does not affect the position.

Newman\textsuperscript{24} has pointed out that under extreme conditions, e.g. in the tropics or in a boiler room, the secretion of sweat may reach such proportions as to be a significant factor in the excretion of alcohol.

FACTORS INFLUENCING THE DESTRUCTION OF ALCOHOL IN THE BODY

In general, about 90 per cent of the alcohol ingested is destroyed in the body. This is effected mainly in the liver, where the alcohol is broken down via acetaldehyde and acetic acid (vinegar) to carbon dioxide and water. From this it is clear that the accumulation of aldehydes and acetic acid in the body, from sources other than alcohol, may wrongly be estimated as representing alcohol when non-specific methods of alcohol estimation are employed.
In practice, hardly any factors influence the rate at which alcohol is destroyed in the body. Proteins and the amino acids derived from them may have a specific effect in that, when given with alcohol, a lower maximum is reached in the blood and the decline to zero is also more rapid.\textsuperscript{25} Starches, fats, the inhalation of oxygen or carbon dioxide, the presence of a fever and drugs such as barbiturates and caffeine (contained in tea and coffee) have no appreciable effect on the rate of destruction of alcohol in the body.

There is no good evidence that habituation to alcohol increases its rate of destruction in the body of the habituated subject.

Insulin, however, may accelerate the fall of the blood alcohol concentration appreciably—a phenomenon not observed in patients with cirrhosis of the liver (an organ where presumably the main destruction of alcohol takes place and which is one of the important sites of action of insulin).\textsuperscript{26}

Intravenous infusions of fructose have been claimed to accelerate alcohol oxidation.\textsuperscript{27} This has been challenged by Camps and Robinson.\textsuperscript{28}

Carpenter \textit{et al.}\textsuperscript{29} have shown that muscular exercise does not have any effect on the rate of destruction of alcohol in the body, a conclusion abundantly confirmed by Nyman and Palmlov.\textsuperscript{30}

The rate of combustion of alcohol is generally regarded as constant, but it varies in different persons and in the two sexes. Newman\textsuperscript{31} has also postulated that this constancy does not hold above a concentration of 100 mg per 100 ml. At higher levels the rate at which alcohol is metabolized is increased. Widmark found the rate of combustion in man to vary from 4.1 to 11.1 g per hour. He gives an average of 7.3 g for males and 5.3 g for females. The regulation of the rate of metabolism of ethyl alcohol has been reviewed by Wallgren and Barry.\textsuperscript{32}

Bonnichsen \textit{et al.}\textsuperscript{33} state that the average hourly oxidation rate of alcohol is affected by the body weight of the subject. The oxidation rate increases by about 0.7 g per hour with each 10 kg of body weight.

Mellanby\textsuperscript{34} reported in dogs an average rate of oxidation of 2.5 ml (i.e. 2 g) of alcohol per hour (i.e. 0.185 ml per kg body weight per hour); but in man this can obviously be, according to Widmark, as high as almost 14 ml per hour and as low as 5 ml per hour.

Isselbacher and Greenberger,\textsuperscript{35} reviewing the rate of alcohol metabolism, state that alcohol can be metabolized at a rate of 100–200 mg per kg body weight per hour. This rate may be as high as 240 mg per kg per hour. The blood alcohol level can decrease between 10 and 25 mg per 100 ml per hour.

When this considerable range is taken into account, the arbitrary nature of statements can be appreciated which claim that 10 to 12 ml of alcohol can be destroyed per hour by ‘the average man’. It is the assumption about these arithmetical averages that underlies the claim that ‘the average man can destroy the equivalent of a small whisky per hour (one fluid ounce)’.\textsuperscript{36}

**THE LEVEL OF ALCOHOL IN THE BLOOD**

As a result of the factors influencing absorption, metabolism and excretion, the alcohol concentration rises steeply in the blood, followed by a more gradual
upward slope to reach a maximum, which is more or less distinct. After this peak there is an irregularly curved fall to the period of diffusion equilibrium. This takes place over 15 to 30 minutes. Then the alcohol level in the blood falls progressively in a rectilinear fashion (elimination phase) until it has all been eliminated from the body.\textsuperscript{37}

The typical blood alcohol pattern following the consumption of a single alcoholic drink is shown in Fig. 15.1.

![Figure 15.1](image_url)

**FIG. 15.1.** Typical blood alcohol pattern following consumption of a single drink. (After Alha, op. cit., p. 12).

Extensive studies by various authors have established that the peak concentration in the blood is reached within about 1 to 2 hours after ingestion. It may be reached earlier or even later; and not all persons react in the same way as is illustrated in the case of Fig. 15.1. This\textsuperscript{38} is illustrated in Figs. 15.2A and 15.2B.

**Type 1:** There may be a steep rise, with a distinct peak.

**Type 2:** There may be a steep rise, but without a distinct peak.

**Type 3:** There may be a slow rise without a distinct peak.

**Type 4:** There may be a distinct and high peak with a subsequent depression.

Alha\textsuperscript{39} found in 12 per cent of his blood alcohol curves, considerable irregularities in the post-absorptive phase. Shumate \textit{et al.}\textsuperscript{40} have made similar observations. They noted repeatedly an unstable period during the first hours after ingestion of the alcohol was completed (Fig. 15.3). The steeple or peak effect observed by Alha and by Shumate \textit{et al.} is not peculiar to alcohol. It can also occur after the absorption of glucose, as may be seen when glucose tolerance tests are carried out.\textsuperscript{41}
FIG. 15.2A. Variations in the type of blood alcohol curve (after Alha). 

Type 1. A steep rise with a distinct peak; Type 2. A steep rise without a distinct peak; Type 3. A slow rise without a distinct peak; Type 4. A distinct and high peak with a subsequent depression.

The curves in Fig. 15.2A were obtained after an alcohol dose of 1 g per kg body weight.

FIG. 15.2B. Variations in the type of blood alcohol curve (after Alha). 

Type 1. A steep rise with a distinct peak; Type 2. A steep rise without a distinct peak; Type 3. A slow rise without a distinct peak; Type 4. A distinct and high peak with a subsequent depression.

The curves in Fig. 15.2B were obtained after an alcohol dose of 1.25 g per kg body weight.
These studies indicate that it is fallacious, in theoretical calculations derived from a known value of the blood alcohol at a particular time, to apply to an individual, conclusions which are based on statistical generalizations.

Herbich and Prokop emphasize the difficulty or impossibility of determining the amount of alcohol consumed or the course of the blood alcohol curve from a single subsequent estimation. Bayly and McCallum also conclude that 'an accurate assessment cannot be made of the blood alcohol concentration in an individual at some time prior to the taking of a blood sample'.

Bowden, reporting on the situation in Sweden, states:

The position has been reached where scientifically it has been decided that it is not possible to determine what the blood alcohol level was at the time of driving from an analysis of the blood at a period later. This is a highly important point, properly appreciated in Sweden. It is believed at the Government laboratory in Stockholm that unsatisfactory assumptions have to be made before a scientist can start to calculate back to the time of driving, and an investigation of many cases has shown that the information produced, upon which the assumptions are made, is not reliable. In the words of the authority here: 'We will not do it; we cannot say with scientific accuracy what his blood (level) was at the time of driving'.

A Report of a Special Committee of the British Medical Association entitled The Medico-Legal Investigation of the Drinking Driver states, on the question of back calculation:

we advise strongly against the court permitting any 'back calculation' to determine how much higher the blood alcohol concentration must have been at the material time. In fact the rate of elimination of alcohol, both between different individuals and in the same individual at different times, varies to some extent and an exercise of this kind cannot, in our opinion, be justified, although we are aware that it is the accepted practice in some other countries. Conversely, if the suspect is known to have taken alcohol just prior to being detained the possibility must be borne in mind that the blood alcohol concentration was still rising at the time the sample was taken.


THE CLINICAL CONSEQUENCES OF DRINKING INTOXICATING LIQUOR

The disturbance in behaviour is due to the influence of alcohol on the central nervous system. This depends primarily on the extent to which the brain is affected.

The Clinical Signs and Symptoms

These will, on the whole, be more marked as the alcohol concentration in the tissues increases. They will therefore vary, inter alia: (1) with the amount of alcohol consumed; (2) with the period of time after the ingestion of the alcohol; (3) with the tolerance of the subject to alcohol.

It is claimed that the degree of intoxication for the same level of alcohol is more marked during the phase of getting drunk than in the phase of sobering up.46

The departure from normal is usually tested by a clinical examination of the subject, directed particularly to the way in which the nervous system has been affected in respect of acute mental and motor deterioration.

Non-medical evidence about the conduct of the person concerned may be extremely important and may, very properly, be considered by the Court in relation to the diagnosis, especially as it may indicate the nature of the behaviour at a time well before the clinical examination was made by the medical practitioner.

Alcohol acts pharmacologically as a depressant of the central nervous system. The normal discipline and controlled conduct of the average person is due to restraining influences which the higher centres of the brain exercise. When sufficient alcohol has been drunk to affect these higher centres, the normal restraints and inhibitions are removed. This is one of the earliest effects of alcoholic intoxication and may show itself in a variety of ways, e.g. garrulity, impairment of judgment, recklessness, etc.

As the degree of alcoholic intoxication advances, other symptoms and signs manifest themselves, mainly in relation to the effect on the nervous system.

Statutory forms are usually a guide to the medical practitioner for the examination of a person alleged to be drunk or under the influence of liquor. An analysis of such forms reveals that the observations to which the practitioner directs his attention deal largely with external appearances and the evidence of disturbances of the nervous system.

It is by now fairly generally recognized that the smell of alcohol on the breath, the pulse rate, the dilatation of the pupils and the colour of the face give no measure of the degree (if any) of intoxication.47–50

The Smell of Alcohol on the Breath

Smith50 states that one can have the odour of an alcoholic beverage on the breath at a time when there is no alcohol in the body. It is not the alcohol per se which imparts an odour to the breath, but the other non-alcoholic constituents in the beverage that give it the characteristic flavour of a wine, whisky, liqueur or beer. The odour of these substances can persist in the breath for many hours after the alcohol has left the body.
Reflexes

Observations on the pupils are completely subjective and consequently not of much value except in cases of coma (when the clinical problem is smallest). Kestenbaum\textsuperscript{51} states that the normal pupil may react to light by contracting promptly or sluggishly.

Tendon reflexes may normally vary from being unobtainable to very brisk. They are therefore of no particular value in these circumstances.

The Conjunctiva

The appearance of the conjunctiva also presents difficulty as an absolute sign. It is well known that, e.g. fatigue and exposure may cause reddening of the conjunctiva. The reddened conjunctiva may also be a normal appearance.

Co-ordination

Very important observations are those made on the integrity of the nervous and the muscular systems, when tested for the co-ordination of fine movements and of grosser movements, e.g. balance, gait and speech.

There is, however, a remarkable variation in the degree of response from subject to subject. Jetter\textsuperscript{52} states:

When it is realized that at 0.40 per cent alcohol, a concentration close to the lethal point, an individual may show so little effects as to be adjudged sober by our criteria, or, on the other hand, that he may be in coma at this same level, it becomes evident that there exists a marked variation in the individual's reaction to alcohol.

A further study of an additional 800 cases confirmed Jetter in his views about individual tolerance.\textsuperscript{53}

The same person may also vary in his response to alcohol at different times. Rabinowitch\textsuperscript{54, 55} cites examples of intoxication at as low a level as 50 mg per 100 ml, and on another occasion no evidence of intoxication at 291 mg per 100 ml.

He also reported\textsuperscript{18} the effects of alcohol in the original Siamese Twins (Chang and Eng, born in 1811; died in 1874). At the time of the initial post-mortem dissection of the twins, the post-mortem injection of a dye revealed that it passed freely from one liver into the other. It is known that Chang not only drank to excess, but was frequently ‘drunk’, but that, despite this, Eng never felt the effects of the drunkenness. On the basis of this initial report, Rabinowitch states that the twins provide ‘unique evidence of different effects of the same amount of alcohol in the same person’. However, reference to the complete autopsy report as published in 1875 by Allen,\textsuperscript{18a} shows that a careful dissection of the injected blood vessel proved it to be a terminal twig of the portal system of Chang, did not pass, as such, across the band and broke up into minute branches before reaching the liver of Eng. There were some minor vascular anomalies in the band, but it is apparent from Allen’s report that Chang and Eng had independent circulations. This fact is supported by Luckhardt\textsuperscript{18b} who states that the findings would seem to indicate ‘that there was no free interchange in their circulations’. Controverting the view of Rabinowitch, Luckhardt states: ‘Chang drank pretty heavily—at times getting drunk; but Eng never felt any influence from the debauch of his brother—a seemingly conclusive proof that there was no free interchange in their circulations.’

In evaluating the clinical effects of alcohol consumption, it is desirable to dis-
tistinguish between evidence indicating that alcohol has been ingested and evidence indicating that the functions of the central nervous system have been affected adversely.

THE CORRELATION OF THE CLINICAL DEGREE OF INTOXICATION WITH THE BLOOD ALCOHOL LEVEL

The diagnosis of the alleged drunken state is often beset with genuine difficulty. It is generally agreed that the problem would be simplified very considerably, in certain cases, if the blood of the accused person were examined chemically to determine its alcohol content. If the result of the chemical test is negative, or nearly so, it will substantiate the claims of the innocent; if it is positive, it may (in certain limited circumstances) assist the Court in checking the accuracy of statements made about the amount of drink alleged to have been consumed. The chemical result may also, but not always, confirm the result of the clinical examination.

Bowden records the following case in a jurisdiction where breath tests were used:

In one case in particular a driver was arrested and charged with drunk driving. This man's condition was such that he was incapable of standing without assistance. There was an odour of alcohol on his breath, but he maintained that he had had but one drink prior to his arrest . . . [the Breathalyzer test] indicated that he had less than an ounce of whisky in his body at the time of test. This man was sent to hospital at about 9.00 p.m. that evening. About an hour later the doctor at the hospital called and questioned the Breathalyzer operator, for he was of the opinion that the man was drunk. It was not until about 5.00 a.m., after conducting various tests, that they found the man was suffering from a virus infection of the nervous system.

Except in extreme cases, a blood alcohol test is unlikely to be reliable as a simple and single test for drunkenness. It is a special investigation which must be interpreted in relation to the rest of the evidence, including the medical examination.

The intoxicated state shows itself in a profound disturbance of behaviour due entirely to the influence of the alcohol on the nervous system of the subject. Therefore, any other drugs, or any disease processes affecting the same parts of the nervous system in the same way, are likely to produce disturbances in behaviour similar to those produced by alcohol. The subjects of high blood pressure, for example, may suffer transient minor strokes leading to unsteadiness and thickness of the speech, and if the victim of such a seizure happens to have the faintest smell of alcohol on his breath, he may quite unjustly be accused of drunkenness, especially by the inexperienced. The differential diagnosis must include, inter alia, such conditions as virus infections, petit mal, acute carbon monoxide poisoning, head injuries, shock and the difficult problem of hypoglycaemia. The diagnosis of coma presents additional complexities (poisoning, diabetes mellitus, uraemia, head injuries, cardiovascular accidents, epilepsy, psychosis, etc.).

It seems desirable for the medical examiner to make his clinical diagnosis uninfluenced by any knowledge of the results of the chemical test. Lofthus, reporting on the examination of drivers detained by the police on suspicion of being
under the influence of alcohol in the Oslo area during the period 1930 to 1945 states:

At first a blood sample was taken . . . Then the suspect underwent a clinical examination according to a definite authorized schedule. The result of this examination was recorded immediately, so as to make it independent of the result of the blood analysis, which would not be known until a day or two later.

When an attempt was made to depart from this procedure in Johannesburg (Case No. T1193/58. 1), the magistrate remarked, *inter alia*, at a resumed hearing of the case:

Under the Mental Disorders Act, as far as I remember, the section reads that in deciding whether or not a person is mentally disordered from a magisterial point of view, the magistrate should summon to his aid, or must summon to his aid, two medical practitioners?

Yes.

And they each conduct a separate and completely independent examination, and they each express a separate and completely unrelated conclusion?

Yes.

And on those certificates and on the other evidence the magistrate has, he then issues his temporary Reception Order. Now I mention that because I want to see whether there is any comparison between the position of the district surgeon examining the man clinically, and the scientist conducting his scientific examination in his laboratory. This affidavit which is produced, is produced because of a very special provision in the Criminal Procedure Code. In the normal course of events the scientist's affidavit would be inadmissible, he would have to come here himself to court?

Yes.

And he would himself have to say: 'Look here, I got a specimen of blood, I carried out certain examinations on that specimen and I find it contains this percentage of alcohol'. This affidavit which the district surgeon sees, is merely a procedure which was, I should imagine, of course I can't be sure, but which was initiated for convenience. I don't think it was primarily initiated for the assistance of the district surgeon to enable him to come to a conclusion. It was to dispense with the necessity of a scientist coming to give evidence of the result of his scientific examination, and I imagine that the primary object was that that evidence should be made available to the court in the most convenient form. I don't know whether one of its objects was to make that affidavit available to the district surgeon. Do you follow what I mean?

Yes, yes perfectly.

Now I come to where I started, and I want you please to understand that I am not criticizing anybody, I am simply looking for information, in the interests of justice generally and in the interests of this particular accused. Isn't the district surgeon, in effect, calling another witness to his aid, a witness that the court should really and would really have called had it not been for the specific machinery which is introduced into the Criminal Code to enable the production of the affidavit, rather than the witness's appearance in court where he would normally have appeared and in which case the district surgeon would not have had the benefit of knowing what his conclusion was in order to assist him to come to his own?

Yes, well I quite understand your worship's argument, but if that were to be brought into effect, then I would have to say that the district surgeon would have to wait until the blood alcohol tests were proved in court, and that having then received that information he would then be in a position to consider his own diagnosis. Because, your Worship, this I have brought into effect, mainly for the protection of the accused. I have seen cases where doctors have diagnosed drunkenness when in fact no such thing existed, and where later it has been proved that the accused was not drunk at all but was suffering from some other condition which simulated drunkenness. I have seen cases where a doctor has diagnosed drunkenness when the blood alcohol report has come back naught; in other words, there was no alcohol at all. That particular man may have been subjected to the expense of procuring counsel and all the rest of it, whereas if we know that his blood alcohol is naught, then we would not certify even though the signs of drunkenness may have existed. They were in fact due to some other condition. That was one of the reasons why I thought it in the interest of justice that the diagnosis should not be made until it is absolutely confirmed by the presence of a certain amount of alcohol in the blood.

Doctor, I see your point of view completely, and I have no quarrel with it at all, with your point of view, but I am still concerned with the probative value of a conclusion expressed in these cir-
cumstances as compared with the probative value of a conclusion expressed by the doctor on his clinical findings, independently of the blood alcohol, and then confirmed in so far as need be or possible by the blood alcohol report.

The smell of alcohol on the breath is a subjective and extremely fallible index of the amount of liquor taken or of its presence in the body. For this reason, *inter alia*, the usual medical examination carried out by a medical practitioner cannot be dispensed with because it is designed to discover or exclude those disease conditions which may closely simulate the drunken state. There are likely to be cases, however, where the clinical investigation may not settle the matter beyond doubt. At its best, the medical practitioner's diagnosis of alcoholic intoxication is only the most reasonable inference he can draw from his clinical observations. It is, therefore, proper that the doctor's reasonable inference should be tested to see whether it is consistent with the independent and scientific testimony of the chemist. The doctor as an expert witness must not usurp the functions of the court, which must assume the final responsibility of making the decision.

In some jurisdictions the chemical test is done on the blood and not on the breath or the urine. It is the most direct evidence obtainable in the living. It has been amply proved in animal experiments that there is a fairly constant relationship between the amount of alcohol in the blood and in the brain tissue supplied by the blood, once equilibrium has been established between the alcohol in the blood and the tissues. A blood alcohol determination, therefore, will give us a measure of the amount of alcohol in the brain itself. This is most important information to have, because the brain tissue is the site of action of the alcohol, and it is the disturbance of its functions by this drug that results in the behaviour we call 'drunken'.

There is no such simple and constant relationship between breath or urinary alcohol and blood alcohol. Haggard *et al.* instance the example of a person given 250 ml of whisky at 10 p.m. He retained his urine until 8 a.m., when his blood alcohol on direct test was zero but as calculated indirectly from the amount in the urine it was 110 mg per 100 ml. Alha and Tamminen have reported high levels of alcohol (up to and even over 200 mg per 100 ml) in the urine of recently deceased persons in whom no alcohol was present in the blood. Alcohol may pass through the lining of the bladder in either direction after death, as well as in life. The direction of diffusion is determined by the relative concentrations of alcohol in the blood and the urine at the time of death.

Attempts to estimate the amount of alcohol affecting the brain, when based on analyses of breath or urine, may therefore be quite fallacious and misleading. This is unfortunate as these biological materials are easy to obtain. More recently it has been claimed that more reliable techniques have been developed for analysis of the breath, but they require skill and special training. Surveys of breath tests for alcohol have been made, *inter alios*, by Denny, Landauer and Milner and Milner and Landauer.

A blood alcohol determination may acquaint us with the amount and concentration of alcohol circulating in the body of the subject, i.e. within the flexible range which has been established for this type of investigation. By a simple calculation it may be possible to determine (again within fairly wide limits) how
much alcohol must have been imbibed within a certain period of time to produce the level found in the blood at the time of examination. This, of course, can only be attempted after absorption has stopped and equilibrium between the blood and the tissues has been attained. With the aid of simple Tables (e.g. Table 7), this information can then be translated into terms of tots of whisky, brandy, wine or beer. This information has the advantage of being objective, although not very precise. Used with caution, it would give the Court a limited opportunity of testing the reliability of the story told by the accused. It would assist the doctor in confirming his already reasonable suspicions of what was the matter. It would also enable the Court to accept a purely clinical diagnosis of drunkenness without any reluctance. It might serve to resolve conflicting reports given by non-medical and even medical witnesses. Any such evaluation must distinguish between the general statistical probabilities and the position in a particular individual case. Data treated statistically to give average results may smooth out the many variables which may make the individual case markedly different from the average.

Table 7. Blood Alcohol Concentrations in Relation to the Amount of Liquor Consumed

<table>
<thead>
<tr>
<th>Amount of ethyl alcohol per 100 millilitres of blood (tissues and blood concentrations being in equilibrium)</th>
<th>Amount of ethyl alcohol in a man of 70 kg in weight (11 stones)</th>
<th>Minimum amount of liquor consumed, in one of the following forms, by a man weighing 70 kilograms (11 stones)</th>
<th>Approximate time required for removal of alcohol from the body</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Whisky</td>
<td>Wine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alcohol 40% by volume = 30° U.P.</td>
<td>Alcohol 16% by volume</td>
</tr>
<tr>
<td>Milli-grammes</td>
<td>Milli-</td>
<td>Milli-</td>
<td>Fluid</td>
</tr>
<tr>
<td></td>
<td>litre</td>
<td>litres</td>
<td>ounces</td>
</tr>
<tr>
<td>50</td>
<td>0.063</td>
<td>26.4</td>
<td>0.9</td>
</tr>
<tr>
<td>100</td>
<td>0.126</td>
<td>52.9</td>
<td>1.8</td>
</tr>
<tr>
<td>200</td>
<td>0.252</td>
<td>105.8</td>
<td>3.7</td>
</tr>
<tr>
<td>300</td>
<td>0.378</td>
<td>158.7</td>
<td>5.5</td>
</tr>
<tr>
<td>400</td>
<td>0.504</td>
<td>211.6</td>
<td>7.4</td>
</tr>
<tr>
<td>500</td>
<td>0.630</td>
<td>264.6</td>
<td>9.3</td>
</tr>
<tr>
<td>600</td>
<td>0.756</td>
<td>317.5</td>
<td>11.1</td>
</tr>
</tbody>
</table>


Precautions in Taking Specimens

As emphasis has been placed on the blood as the most reliable biological material which should be investigated in the living (in the dead the brain itself is available), it is important to appreciate that the usual sterile precautions taken by medical practitioners (when removing blood from a vein for various routine test purposes) are sufficient to eliminate all risks from the procedure. Thousands of blood samples are taken daily in many clinics as a matter-of-fact routine. The accused person is not, therefore, subjected to any particular risk when he submits to this special examination. The only precaution which the medical practitioner must take is to ensure that the needle and syringe employed have not been sterilized with alcohol. These instruments should be sterilized either by dry heat
or else be boiled up in the usual way. For the same obvious reasons the skin over
the site of the vein to be tapped should not be cleansed with alcohol in any form.
(See also p. 374). If these obvious precautions are observed, a blood sample may
be taken without medical risk to the patient.

It is desirable and preferable to use packaged sterilized disposable equipment
for taking blood samples. This practice is followed in many parts of the world.

Indeed, it would be as well for the medical practitioner, as a routine, to incor­
porate, either in his affidavit or in his evidence, the fact that he actually observed
these precautions before taking the blood sample. Hollopeter\textsuperscript{64} records the
following:

It sometimes happens that following an accident, a blood sample is taken in hospitals at the
direction of a police officer, but occasionally it is done in a crude, wholly unscientific manner.

A man \textit{was} charged with felonious drunken driving and felonious manslaughter following the
death of his passenger. The defendant driver himself received serious injuries and was taken to
hospital. On his admission, on the direction of the police, a sample of his blood was taken . . . the
conclusion of the chemist who subsequently examined the blood was excluded because of the
extreme carelessness exercised in making the withdrawal. Under cross-examination it developed
that the nurse who actually withdrew the blood, under the direction of an attending doctor, first
swabbed the area from which the sample was to be taken with rubbing alcohol and then inserted the
syringe to withdraw the blood. Obviously any analysis for alcohol of blood so taken would be
invalid.

\textbf{Correlation Studies}

A very comprehensive study of the correlation between the blood alcohol level
and the degree of intoxication has been carried out by Jetter.\textsuperscript{65} His investigation
has been quoted extensively in the literature and forms the basis of an evaluation
of blood alcohol findings (Fig. 15.4).

It is important to appreciate once again that the correlations observed are
based on statistical generalizations and do not apply to an individual case. For
example, according to Jetter's criteria, 47 per cent of persons may be regarded as
under the influence of alcohol at a blood alcohol concentration of 150 mg per
100 ml. In other words, 53 per cent of persons would, by the criteria employed,
be sober.

Harger and Hulpieu\textsuperscript{66} have tabulated the results of 7 investigators, each of
whom had his own criteria for diagnosing drunkenness (Table 8). The list in­
cludes the observations of Jetter.\textsuperscript{65} The average of the combined findings of the 7
investigators closely approximates the observations recorded in Jetter's series
(Fig. 15.4).

Even when the statistical chance of intoxication increases, it has been es­
tablished that certain subjects remain sober at very high blood alcohol levels.
Rabinowitch\textsuperscript{54, 55} has shown that the same subject may be intoxicated at a low
blood alcohol level and, on another occasion, may be diagnosed as sober at very
high levels, e.g. 273, 282 and 291 mg per 100 ml. Lofthus\textsuperscript{67} reports a case where
the subject was considered sober at a level of 257 mg per 100 ml. On the other
hand, he refers to a person without any alcohol in his blood who was found to be
not sober (by Kristensen) 'possibly as a result of nervousness in the test
situation'.

The foregoing observations indicate the fallibility of allowing the diagnosis of
alcoholic intoxication to rest on the chemical determination alone. In fact, the blood alcohol level is merely one item in the evidence which must be considered in relation to other evidence about the behaviour of the subject at material times.

![Graph of Blood Alcohol Concentration vs Percentage Intoxicated](image)

**Fig. 15.4.** Relationship between blood alcohol concentration and percentage of subjects intoxicated. Jetter adopted the following criteria for the clinical diagnosis of acute alcoholic intoxication: 
(a) Gross gait abnormality; 
(b) In addition, at least 2 of the following tests must be positive: 
(i) Abnormality of speech; 
(ii) Flushed face; 
(iii) Dilated pupils; 
(iv) Alcoholic odour of the breath. Fig. 15.4 has been drawn from data published by Jetter and Harger and Hulpicu.

This evidence may be medical as well as non-medical and, in this context, the blood alcohol level is only of value when it is consistent with the other non-chemical observations made.

The vagaries of the post-absorptive behaviour of alcohol in the body must make theoretical calculations of variable significance. They must not be invested with undue and unwarranted precision.
Table 8. *Blood Alcohol Level and Frank Intoxication*

<table>
<thead>
<tr>
<th>Investigator</th>
<th>No. of Subjects</th>
<th>Blood Alcohol (mg per 100 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0-50</td>
</tr>
<tr>
<td>Widmark(^a)</td>
<td>1942</td>
<td>0</td>
</tr>
<tr>
<td>Schwarz(^b)</td>
<td>905</td>
<td>2</td>
</tr>
<tr>
<td>Harger <em>et al.</em>(^c)</td>
<td>140</td>
<td>0</td>
</tr>
<tr>
<td>Jetter(^d)</td>
<td>1000</td>
<td>10</td>
</tr>
<tr>
<td>Andresen(^e)</td>
<td>1712</td>
<td>10</td>
</tr>
<tr>
<td>Alha(^f)</td>
<td>54</td>
<td>48</td>
</tr>
<tr>
<td>Prag(^g)</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>Total:</td>
<td>5853</td>
<td>10</td>
</tr>
</tbody>
</table>

Note: Percent Subjects Diagnosed as Drunk.

---


FEATURES OF THE CLINICAL EXAMINATION

The time taken to complete the examination of the arrested person should be adequate for the performance of a reasonably full clinical investigation, e.g. a period of four to five minutes would not be adequate. It is usual for at least fifteen to twenty minutes to be required for this type of examination, although admittedly very experienced observers may take less time.

Effect of Alcohol on General Behaviour

Rentoul, Smith and Beavers\textsuperscript{68} found that the effect of alcohol on general behaviour was:

... so variable that it rapidly became apparent that no reliance can be placed on this in estimating alcohol consumption. Our subjects varied from individuals who showed no external sign of the effects of alcohol other than smell, to those who rapidly became semi-comatose and remained so for three to four hours. This was a surprise to us. We knew that a tolerance could be acquired to alcohol, but not to anything like the extent which these experiments showed. Ten ounces of whisky can be a dangerously toxic dose to someone unaccustomed to alcohol, whereas to a seasoned whisky-drinker it appears to be little more than an appetiser. An interesting observation was that no one showed any sign of exhilaration or ebullience. There appears to be no doubt that this factor in the effects of alcohol is related to companionship and surroundings. The practical importance of this is that when car accidents occur after parties, the party spirit may well be a factor in addition to the alcohol. It may even be a major factor.

The Value of Certain Tests

The tests, especially those for the co-ordination of delicate movements, must be reasonable. A corpulent subject may have difficulty in picking up a pin from the floor, even though he is sober. The Court may well comment upon the unreasonable nature of the tests applied:\textsuperscript{69}

In R. v. Bradley, Mr. Justice Streatfield commented on the tests given by a young, provisionally qualified doctor to a man who appeared at Worcestershire Assizes accused of being in charge of a car while under the influence of drink. The accused, aged 34, a car salesman, was found not guilty.

Dr. R. said he was 23 when he carried out the tests in July, and was then awaiting his full registration as a medical practitioner, and acting as house physician. He examined the accused's reaction to light by putting his hand before Bradley's eyes, then moving it away. Bradley's reaction was slow.

Counsel defending: Do you know that the usual test in this case is to shine a torch into the eyes?

Dr. R: That is a dangerous method. I was reprimanded as a student by a senior neurologist in the hospital for testing someone's reaction to light in that manner.

The doctor said he also asked Bradley to multiply seven by seven by eight. Bradley did not do it properly.

'I thought', said Dr. R., 'that as he was a salesman and dealing with figures it would be a fair test'.

As another test he asked Bradley to stand on each leg alternately with his eyes shut. 'Bradley practically fell over', said the doctor. 'I came to the conclusion that he was under the influence of alcohol'.

The judge said he thought the jury would agree that the doctor was doing his best. But of the eye test he said, 'As long as I remember in this profession almost all doctors when examining people have applied the test of shining a very bright light into a man's face. This is the first time that I have heard that that was a dangerous or misleading test or an improper one. Unfortunately, this doctor adopted a different technique. He simply covered this man's eyes with his hand and then took it away in the ordinary light of the room. Then, he told me, the eyes reacted rather slowly. Well, of course they did, if there was no particularly bright light there. You may think that through no fault of his own that test somewhat misfired'.

Of the multiplication test he said: 'How many sober people could give an answer quite quickly?'

On the third test he commented, 'I wonder how many of us, unpractised, could suddenly close our eyes and without swaying stand on one leg?'

Rentoul\textit{ et al.}\textsuperscript{68} state (op. cit., p. 4):
The 'picking up coins' test appears to have little value. A considerable number of completely sober people fumble the coins. Several variations on the standard tests were also used, e.g. arranging matches in fixed patterns and also the finding of numbers in special patterns. None of these tests, however, showed alterations in performance sufficiently consistent to be of any use in demonstrating the effects of alcohol. In fact several people showed slight improvement at some of these tests after taking alcohol.

It should be borne in mind that a chronic alcoholic subject when sober may not be able to perform tests for co-ordination as well as when he has actually had several ounces of alcohol to drink.

French in discussing Ataxia due to Mental Shock or Fright states:

There are other cases of purely emotional ataxy in which there has been no traumatic factor. One speaks of 'staggering news', of having a 'staggering blow' on the exchange, and so on. Any extreme emotion, pleasant or unpleasant, may cause temporary ataxy; the worse the emotion the longer the ataxy is apt to last; and after extreme fright, panic or the like, it may be permanent though functional. These facts may become of great importance in connection with charges of drunkenness associated with motor driving accidents.

French also states that ataxia (i.e. inco-ordination of muscular movements) may be hysterical (with staggering as the only manifestation), traumatic, emotional (mental shock or fright), or due to chemicals or drugs (insulin, carbon monoxide, Veronal). Veronal may be followed by a staggering gait for weeks.

Fregly et al. have observed that maximum ataxia may occur sooner than the maximum blood alcohol level and that the ataxia may begin to improve while high blood alcohol levels are still sustained.

Alha also points out that fatigue occurs in the stage of decreasing drunkenness and produces a general retardation of functions which may therefore enhance the appearance of intoxication.

Difficulties do not usually arise in extreme cases, i.e. where the subject is clearly drunk or patently sober. It is the marginal cases and those with intermediate degrees in the disturbance of behaviour that create the clinical problem.

It is important that the observations made in the medical report should be consistent. For example, a severe degree of Rombergism with a staggering gait is, prima facie, incompatible with a faultless performance of a delicate test for fine co-ordination of movements. Similarly, a subject with a swaying, staggering gait cannot have a weakly positive Romberg test.

It should also be appreciated that the medical examination is extremely subjective, many of the observations made being incapable of objective or quantitative record, especially under the conditions governing the examination. In any event, whether the practitioner is dealing with a case of coma or an ambulatory subject, he must consciously direct his mind to a consideration of all the other reasonable causes which may produce the same disturbance as that produced by alcohol. He must pay particular attention to the exclusion of diseases or disorders which may be present and which may be an adequate explanation of the symptoms and signs observed.

A cursory and incomplete clinical examination may lead to serious error. Although the state of integrity of the nervous system is the main object of the investigation (to determine the relationship to alcohol ingestion) it is essential to conduct a complete and thorough general examination.

An arrested person is likely to be suffering from fear and acute anxiety, often
complicated by fatigue-states which may produce signs which may wrongfully be attributed to alcohol. A full general examination may, for example, provide evidence of liver disease. Brown, in a study of liver–brain relationships, has stressed the neurological changes which may be present in a patient without coma. He found positive signs in 67.5 per cent of his cases. The signs included inco-ordination, positive Rombergism and nystagmus.

**Nystagmus (Alcoholic Gaze Nystagmus: AGN)**

An undue amount of emphasis appears to have been placed on the presence of this sign (as observed clinically) by some medical examiners as evidence of alcoholic intoxication. It is by no means a constant or a common sign. Watt found it present in about 10 per cent of persons who had consumed varying quantities of alcohol. One subject who had drunk fourteen tots of whisky in about four hours had no evidence of nystagmus or nystagmoid movements. Nystagmus on lateral gaze at the normal position of the head with open eyes may occur at an average blood alcohol concentration of 0.08 per cent (range: 0.04 per cent to 0.1 per cent). It may be absent at an average concentration of 0.06 per cent (range: 0.05 per cent to 0.08 per cent). Nystagmus is thus by no means always an early sign of a disturbed nervous system and by itself it certainly cannot be regarded as enough for the clinical diagnosis of alcoholic intoxication.

Rauschke regards certain types of nystagmus as having a good correlation with the clinical symptoms of intoxication; but Prestwich has never found nystagmus in persons known to have taken the 'extra one or two'. He is critical of the way police surgeons produce (as if they were 'the Laws of the Medes and the Persians') nystagmus and other signs suggestive of intoxication and listed in the British Medical Association pamphlet *The Recognition of Intoxication* (1954).

A physiological nystagmus occurs in about one in five of normal persons. Nystagmus can be brought on by fatigue, emotion or postural hypotension, e.g. after or before a faint. It has been reported after head injuries, and Brown has found it in association with liver disease. The British Medical Association in its pamphlet *The Recognition of Intoxication* describes it as a fine lateral nystagmus. At p. 16 of the revised (1958) edition of this pamphlet, the reference to the nystagmus as fine has been abandoned without any explanation. Such apparent clinical indecision may well cast doubt on the authoritative value of this pamphlet in this connection. A distinguished Mayo clinic team says that sedative drugs, including alcohol, produce nystagmus of the cerebellar type. The eyes are steady on forward gaze, but nystagmus appears on looking to the sides, the quick component being in the direction of gaze.

* State vs. Rosen & another (4th December 1961). In this matter the senior district surgeon examined the male accused (No. 1) at 6.30 a.m. on 15th October, 1961. The accused had been arrested at 3.40 a.m. The district surgeon found no evidence of disease. The Romberg test was moderately positive, hand movements were slightly unco-ordinated and the pupils seemed to react rather sluggishly to light. There was a slight stagger in the gait on turning (the accused went off balance), his tongue was dry and furred and there was a somewhat sour smell on his breath which the district surgeon recorded as 'a faint smell of liquor'. The district surgeon stated that a state of anxiety or fatigue might show all the signs he found. Blood from accused No. 1 was examined for its alcohol content. The result was negative.
Positional Alcohol Nystagmus (PAN)

Aschan et al. have demonstrated the existence of a special type of nystagmus which can be elicited after alcohol consumption with the head in certain positions. It is known as Positional Alcohol Nystagmus (PAN) and it occurs in 2 phases (PAN I and PAN II). The phenomenon has more recently been reviewed by Goldberg.

With the subject supine, and the head in the right or left lateral position, the eye movements were recorded under laboratory conditions, mainly behind closed eyelids, with special recording apparatus.

PAN I appears about half an hour after the intake of a single dose of alcohol, lasts for 3 to 4 hours, and its duration is independent of the dose. With the head on the right side, the slow component is upwards (anti-gravity) and the rapid component is downwards (i.e. to the right), changing to the opposite direction in the left lateral position. The second phase (PAN II) begins about 1 to 2 hours after the disappearance of PAN I and usually some 5 to 6 hours after the ingestion of the alcohol. Its beat is in the reverse direction to PAN I (i.e. the fast component beats to the left in the right lateral position and to the right in the left lateral position). The duration and intensity of PAN II, depending on the dose of alcohol taken, range from 5 to 10 hours or more. PAN II, however, lasts for many hours after alcohol has disappeared from the blood. It is claimed to be a true, objective after-effect and part of the so-called hang-over syndrome.

Goldberg points out that in clinical practice it may be of value to make a tentative diagnosis about the nature of an observed nystagmus, i.e. whether it is alcohol-induced or not. To assess a possible positional alcohol nystagmus (PAN), existing mostly in lateral positions behind closed eyelids, the patient will have to be put on his back, his head turned laterally to one and the other side, and the eyes closely watched. An existing positional alcohol nystagmus, even if not recorded, may disclose itself by the observer seeing, and feeling, the cornea moving behind the closed eyelids. Opening of the eyes will block the positional alcohol nystagmus, if its intensity is low, but will disclose it if the intensity is high.

The positional alcohol nystagmus (PAN) can be differentiated from a gaze nystagmus (AGN) by testing when the eyes look forward, and not only in lateral gaze direction; also by the direction of the fast component which changes with the position of the head and with the time after intake (PAN I or PAN II respectively).

Systematic studies have to be carried out, combining clinical observations of possible nystagmus with objective recording by EOG (electro-oculography). The results have to be related to the whole course of blood alcohol in one and the same individual, and not only to single levels in different individuals.

These studies must be carried out in individuals with varying alcohol habits, i.e. in moderate consumers as well as in alcoholics, in order to serve as a basis for possible recommendations of value for clinical use.

Drugs and PAN

Goldberg states:

Whereas meprobamate, like all other CNS-depressant agents studied from antihistamines to
buclozine and chlorpromazine, increases ROM (roving ocular movements) and decreases PAN, chlordiazepoxide is so far the only drug known that reduces both PAN and ROM.

Thus the interaction between ethanol and drugs depends not only on the type of drug administered, but also on the timing with regard to the phase of alcohol metabolism.

Congeners and Nystagmus (PAN)
Murphree et al.\textsuperscript{85} state that a significant nystagmus-inducing effect may be produced by the congeners in alcohol beverages. These congeners include higher alcohols (fusel oil), organic acids, esters and aldehydes. In their experiments the nystagmus was recorded under laboratory conditions with bipolar leads from the outer canthus of each eye to the mid-forehead, permitting separate recordings for each eye.

DIFFICULTIES IN THE CLINICAL EXAMINATION
The difficulty of a clinical diagnosis of alcoholic intoxication, in border-line cases where the matter is not obvious, must not be under-estimated.

The medical examination is usually conducted some considerable time after the traffic accident in which the accused has been involved, and it would be extremely hazardous for the clinical examiner to dogmatize about the accused's condition and capacity at the time of the accident, with the limited amount of information at his disposal. He may not know, with any certainty, whether the accused is in the sobering-up phase or \textit{vice versa}; nor can he know with any accuracy what quantity of liquor the accused has imbibed.

In discussing whether a clinical (physical) examination can assist in deciding the ability to drive a car, except when the degree of intoxication is gross, Smith and Popham\textsuperscript{86} state that available clinical tests would not appear sensitive enough. Penner and Coldwell\textsuperscript{87} considered that:

The medical examination did not prove to be a sufficiently accurate method for assessing alcoholic impairment in relation to car driving. Generally, the medical examination was not sensitive enough, but this was not consistent since several subjects were considered impaired by medical examination but not on driving performance.

Indeed, in certain circumstances, e.g. where the accused has been injured, or has suffered from concussion, or is in addition suffering from emotional shock (he may have seriously injured or killed a passenger or a pedestrian), the clinical examination as a means of diagnosing alcoholic intoxication may be quite inadequate for medico-legal purposes.

The vagaries of medical examiners have not been investigated very extensively. Lofthus\textsuperscript{88} states: 'A number of investigators have stressed the individual variation from one clinical examiner to another'. He reports Liljestrand as having found, in a group of seven observers, that the blood alcohol level at which half the subjects were diagnosed as being under the influence of alcohol, varied from 50 to 140 mg per 100 ml. Lofthus draws attention to the following individual factors in the medical examiner:

- \textit{a.} State of mind (temperament, overwork, called repeatedly the same night, etc.);
- \textit{b.} Conscious attitude towards the law and the examination (liberal–rigid);
- \textit{c.} Experience and confidence;
- \textit{d.} Incidental errors of judgment.
Lofthus also reports data showing that the diagnostic standards of medical examiners tend to change with the passage of years. He suggests that this may be due to increasing experience—not necessarily a valid hypothesis.

Andresen noted that 2.9 per cent of 170 motorists examined by police surgeons in Denmark were found clinically to be intoxicated, although chemical analysis proved that they had no alcohol in their systems.

Penner and Coldwell also found 'poor agreement between the conclusions reached by two independent medical doctors'. This disagreement consisted of differences of opinion in the various parts of the medical examination and also variations in the conclusions as to impairment drawn from similar observations.

Coldwell et al. analysed the results of 82 medical examinations made by each of two doctors (identified as MD1 and MD2) on the same subjects on the same days:

In 54 their opinions were identical. Of the remainder, MD1 concluded the subjects were impaired on 4 occasions and unimpaired in 24; MD2 came to exactly the opposite decision. Since the blood alcohol levels at the time of examination by each doctor were practically identical, these differences must be due to differences in clinical opinion.

The individual variation in the conduct of medical examinations is reflected also in an analysis performed in another jurisdiction.

A panel of four doctors took it in turn to be on call for the examination of persons in circumstances where there was a possibility that the subjects were under the influence of alcohol. There was no selection, therefore, of the clinical material examined by the individual doctors, and it is reasonable to assume that, over the (approximate) two-year period of the analysis, each practitioner saw a random sample of the population being examined. Doctors A and B roughly coincided in having a score of about 25 to 30 per cent of positive diagnoses for intoxication. Doctor C, however, had a score of over 70 per cent, and Dr. D had a score approximately between these two extremes.

The foregoing factors make it clear that the medical report is only one item in the total data available to the Court from which inferences can be drawn.

Amnesia

Amnesia may be total or incomplete, i.e. there may be patchy islands of recollection. Slater and Roth state (at p. 397):

On the mental side severer degrees of intoxication are often accompanied by increased irritability, outbursts of rage and violence after solitary brooding. For most of the events and actions in this stage the subject usually remains amnesic after return to sobriety.

Frame, discussing pathological alcoholic states, describes the alcoholic palimpsest as follows:

A condition not infrequently seen amongst alcoholics and occasionally in the non-addictive drinker is the sudden onset of behaviour resembling the 'black-outs' in anoxaemia.* I have seen many authentic cases of this type but none in my experience who had committed crimes during the phase. After only moderate ingestion, without showing any signs of intoxication, the person may carry on conversation and perform fairly well-organized and elaborate acts purely on an automatic level with indications only of patchy vague memory at intervals during this period of amnesia on the following day. This patchy amnesia which is not associated with unconsciousness has been called by Bonhofer† the 'alcoholic palimpsest'. The condition has been well described by the

Alcoholism Sub-Committee of the World Health Organization's Expert Committee on Mental Health.

One patient of my acquaintance recently presented himself for treatment because of the onset of this symptom. Just before admission he had left an hotel, apparently perfectly sober, with the intention of driving home to the northern suburbs for dinner. About 4 hours later he found himself in open country having safely traversed the traffic of two major cities (35 miles apart) at their peak hours. He had no recollection beyond a point where he had dropped a friend on his way home.

Alcoholic palimpsests (so-called blackouts) may occur in some alcoholics 'in response to rather moderate amounts of alcohol far short of the dose sufficient to cause stupor or "passing out"."