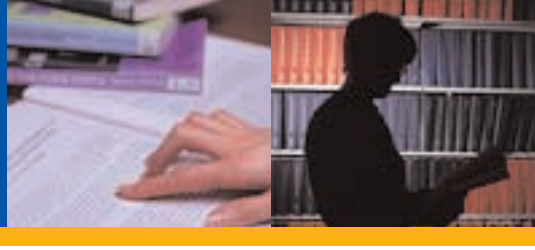
Pancreatitis



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The biological and nursing implications of pancreatitis

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in brief

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Summary
Joseph McArdle examines the physiology of pancreatitis, discussing the nurse's contribution to the patient's recovery. He addresses the need for blood glucose monitoring during an acute episode, contrasts the risks associated with venepuncture and capillary sampling and identifies good practice in capillary blood glucose sampling.

Keywords
- Accident and emergency nursing
- Intensive care nursing

These key words are based on the subject headings from the British Nursing Index. This article has been subject to double-blind review.

Aims and intended learning outcomes

This article looks at the aetiology of acute and chronic pancreatitis, the variety of treatment options available and the physiological and psychological nursing needs of patients undergoing these treatment regimes. Its purpose is to produce a balanced overview, looking at the nursing care required in the acute period and, in particular, the monitoring of blood glucose levels.

After reading this article you should be able to:

- Identify lifestyle risk factors associated with patients who have pancreatitis.
- Describe the structure and function of the pancreas.
- State what treatment options are available and their associated complications.
- Recognise the perils of capillary blood sampling during an acute episode and evaluate the associated risk to the patient's wellbeing.
- Prepare a standard operating procedure for safe capillary blood sampling.

Introduction

Acute pancreatitis is a common condition, with approximately 50 cases per 100,000 population per year in the UK (Slavin 1999). It accounts for an estimated 3 per cent of all cases of abdominal pain admitted to hospital (de Dombal 1991).

Estimates of mortality vary, depending on the severity of the episode, but it is thought to be between 10 and 15 per cent (BSOG 1998), increasing to 25 per cent if the cause is considered post-surgical intervention (Evans *et al* 1997).

To reduce this risk of death, it is necessary to detect any deterioration early. With cases that can be considered severe following either an APACHE (Larvin and McMahon 1989) or Ranson score (Ranson *et al* 1974), the British Society of Gastroenterologists advocates admission to a high-dependency/critical care setting so close observation can occur. The vital signs observed for are:

- Temperature.
- Pulse.
- Blood pressure.
- Oxygen saturation.
- Blood glucose level.
- The patient's biochemical profile.

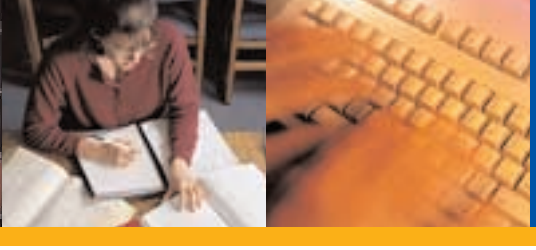
The latter two are invasive procedures associated with a degree of risk and discomfort, including pain, infection, staff injury and inaccuracy. In the US alone, there are 800,000 injuries per annum related to sharps, resulting in 6,500 cases of hepatitis B transmission (Marchitto *et al* 1998).

This article examines the optimum process and frequency of one of these tests, blood glucose monitoring, and produces a working guideline for nursing staff on the wards: the more frequent the test, the greater the risk. It examines the physiology of the pancreas, the pathology of acute pancreatitis and its impact on blood glucose control. It discusses chronic pancreatitis, treatment options and the nursing therapy required by a patient suffering an acute episode.

The article also highlights the dangers of reliance on capillary blood sugar measures, identifies current failings or complacency in practice and explores the potential of having a prescribed glucose-monitoring regime.

TIME OUT 1

Take ten minutes to note your current knowledge of the structure and function of the pancreas. Keep these notes and refer to them as you continue with this article.



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The pancreas

The pancreas is a large gland, shaped like a silverfish, which sits in the epigastric and left hypochondria region of the abdomen (Ross and Wilson 1987) (Fig. 1). Weighing approximately 60g and with a length of 12-15cm, it is surfaced by a thin connective capsule that extends inwards as septa, partitioning the gland into lobules. These are called the head, body and tail and feed into two pancreatic ducts: the principle duct (Canal of Wirsung) and the accessory pancreatic duct, which eventually join the common bile duct and ultimately the duodenum. The pancreas has two separate roles, one as an exocrine gland and the other as an endocrine gland.

The exocrine cells form 98 per cent of the pancreatic tissue and consist of grape-like clusters called acini. The function of the acinar cells is to manufacture the following digestive enzymes in an inactive state:

- Trypsinogen.
- Chymotrypsin.
- Carboxypolypeptidase.
- Pancreatic lipase.
- Pancreatic amylase.

As well as several other enzymes, the acini also produce trypsin inhibitor, the deactivation agent that prevents the enzymes activating and attacking the pancreatic tissue. This is because trypsin, the activated version of trypsinogen, is the catalyst for activating the other enzymes.

The endocrine portion of the pancreas comprises approximately one million small clusters of cells, called islets of Langerhans. These islets are richly vascularised, allowing their secreted hormones ready access to the circulation and, although they comprise only 1-2 per cent of the mass of the pancreas, they receive about 10-15 per cent of the pancreatic blood flow. Additionally, they are innervated by parasympathetic and sympathetic neurons, which modulate secretion of insulin and glucagon.

The pancreatic islets house three major cell types, each of which produces a different endocrine product:

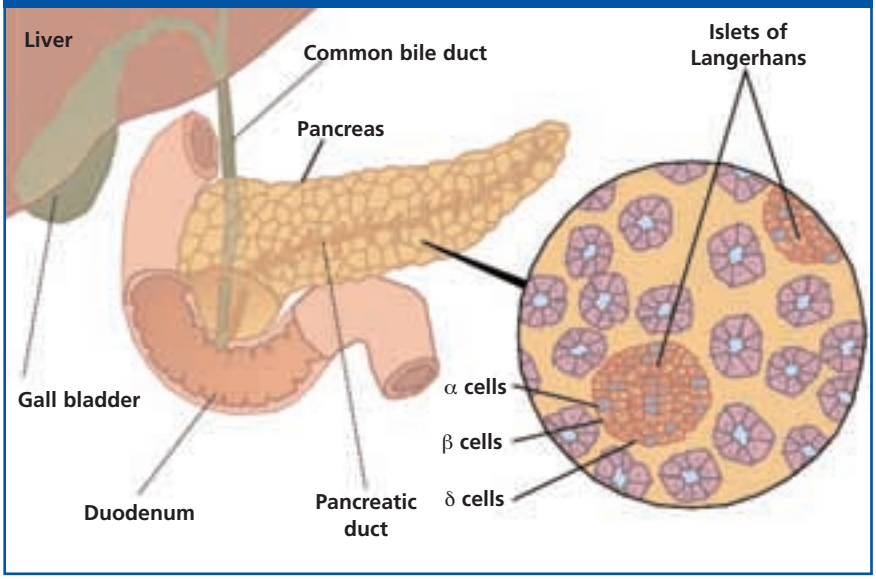
- Alpha cells (α cells) secrete the hormone glucagon.
- Beta cells (β cells) produce insulin and are the most abundant islet cells.
- Delta cells (δ cells) secrete the hormone somatostatin, which is also produced by a number of other endocrine cells in the body.

These cells are not randomly distributed in the islet, rather beta cells occupy the central portion of the islet and are surrounded by an outer layer of alpha and delta cells (Bowen 1999). One further product of the islets is pancreatic polypeptide (PP), produced by the PP cell, although its function is not clear (Guyton and Hall 1996). Both glucagon and insulin regulate the serum blood glucose levels. Hence, if the pancreas becomes damaged, this regulatory system might become dysfunctional. Somatostatin, with a short half-life of three minutes, has three key roles. It:

- Acts as an inhibitor for both cells, alpha and beta, depressing the production of glucagon and insulin.
- Decreases secretion and absorption in the intestine.
- Slows the motility of the duodenum, stomach and gall bladder.

To examine the need for blood glucose monitoring in acute pancreatitis, it is necessary to understand the role of glucagon and insulin in normal regulation.

Fig. 1. The pancreas



Blood glucose control and hyperglycaemia

The pancreas produces insulin and glucagon, with each acting in the opposite way to the other. Insulin decreases blood glucose by allowing glucose to enter the muscle and adipose tissue cells and by enabling the storage of glucose in the form of glycogen in the liver (Guyton and Hall 1996). Brain tissue, however, does not rely on insulin to absorb glucose, as it is already fully permeable to it and cannot use other sources of energy. This explains why low serum blood glucose is so dangerous. Having a half-life of approximately six minutes, insulin can be totally cleared from the body within 10-15 minutes and its production is controlled via a negative feedback system. If serum glucose levels drop, so does the insulin secretion, while glucagon manufacture intensifies. In an absence of insulin, large quantities of fatty acids are released into the circulation following the breakdown of stored triglycerides. This raises the volume of fatty acids in the liver cells, which are then converted into aceto-acetic acid. This cannot be metabolised by peripheral tissues and culminates in a state of acidosis (Bowen 1999), frequently seen in severe hyperglycaemia. This can result in hyperventilation as carbon dioxide is blown off. If bicarbonate production is reduced, as in pancreatitis, then blood pH can

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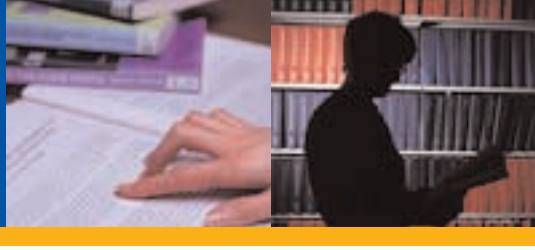

drop to below 7.0, causing death (Steinberg and Tenner 1994).

Glucagon has the opposite effect to insulin on the liver cells and through a complex chain of enzyme activation degrades the stored glycogen to release glucose. It only takes a few minutes for serum blood glucose levels to double. In only four hours, all the stores in the liver can be degraded. This is why an increased glucagon production is so dangerous and hyperglycaemia results. Glucose exerts a high osmotic pressure in extracellular fluid, leading to cellular dehydration and system dehydration secondary to osmotic diuresis by the kidneys.

This bionegative feedback mechanism is a complex balance between insulin and glucagon production, resulting in a serum blood glucose stability, although other stimuli exist in the form of amino acids which are released after a meal or exercise.

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Pancreatitis is an acute or chronic inflammatory condition of the pancreas that can range from a mild form with local gland oedema to a severe form, where glandular necrosis and haemorrhage might occur (BSOG 1998, Miller 1999, SSAT 1997). Currently, pancreatitis is diagnosed when there is severe upper abdominal pain and a raised serum amylase, although work by Slavin (1999) suggests that measuring trypsin activation peptide might be a more precise diagnostic tool. The degree of disease, however, is not clearly identifiable from these blood results, so two scoring criteria have been devised: the Ranson scale shown in Box 1 (Ranson *et al* 1974) and the APACHE (Acute Physiology and Chronic Health Evaluation) grading system (Larvin and McMahon 1989). Both look at the blood glucose level and score it for diagnostic reasons if it is greater than 10mmol/litre. Further symptoms include:

- Nausea.
- Vomiting.
- Fever.
- Tachycardia.
- Respiratory distress secondary to diaphragmatic elevation, pulmonary infiltrates or pleural effusions.

The most common causes are alcohol and gallstone obstruction in the ducts, which account for between 66 and 75 per cent of cases (BSOG 1998, Steinberg and Tenner 1994). Viral infections, toxins and drugs, hereditary factors and abdominal injuries are rarer causes, with up to 10 per cent of the initial triggers being idiopathic (Miller 1999). Recent research has shown that a genetic mutation in the form of a single cystic fibrosis copy is present in many of these idiopathic cases (Cohn *et al* 1998). After an initial event, the trypsin inhibitor agent becomes overwhelmed and the enzymes become activated so autodigestion occurs, resulting in proteolysis, oedema, haemorrhage, vascular damage, fat and parenchymal cell necrosis.

Numerous complications can occur, including shock, pseudocysts, coagulopathy, hyperglycaemia, acute renal failure and respiratory failure leading to the patient requiring ventilation. This is because the release of phospholipase degrades the surfactant of the lungs (Steinberg and Tenner 1994).

The clinical management depends on the degree of severity, but is mainly supportive. Analgesia, antibiotic therapy, correction of fluid and electrolyte disturbances, endoscopy to remove the obstruction, suppression of pancreatic secretion and intensive monitoring and gastrointestinal rest form the main course of action. Banks (1997) suggests that good hydration reduces the risk of further necrosis, but many other studies have not shown conclusively that any of these treatments are fully effective. The role of the nurse, as well as delivering essential nursing care, is to monitor the patient frequently and promptly identify any improvement or deterioration in his or her condition.

TIME OUT 2

Think about the frequency of observations you would carry out on the patient. Write up a short rationale on why you would change this frequency.

Chronic pancreatitis

The most overwhelming problem for patients with chronic pancreatitis is pain, which is difficult to manage. There is a danger that they can become typecast as addicts if they frequently request painkillers, with the extent of their pain not fully appreciated by the team who are treating them (Pasero 1998).

TIME OUT 3

Discuss with another colleague the most painful experience of his or her life. Write a short statement on how you would describe this pain to others so that they can understand the impact it had on your colleague.

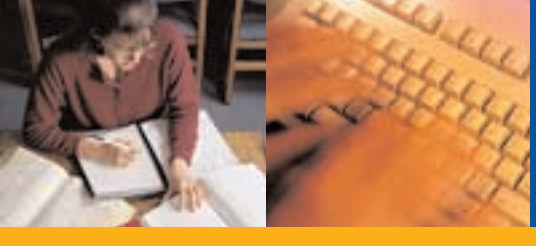
Patients might also have repeated episodes of nausea and vomiting, develop diabetes and steatorrhea (foul-smelling, greasy faeces that noticeably float in toilet water) secondary to the inability of the digestive tract to breakdown fat. This will be the reason for radical surgery.

Recent studies have shown that familial pancreatitis results from a defect in the gene that controls synthesis of the pancreatic enzyme trypsinogen. Roughly 30 per cent of patients with chronic pancreatitis have no identifiable cause for their disease (idiopathic pancreatitis). Diagnosis is often made on patient history and

Box. 1 Ranson's criteria of severity

At admission/during initial 48 hours:

Age:	>55 years
Decrease in haemoglobin of:	>10 per cent
White cell count:	>16,000
Urea:	>16mmol/l
Glucose:	>10mmol/l
Ca++:	<2mmol/l
Lactate dehydrogenase:	>700IU/l
PaO_2:	<60mmHg
Aspartane amino transferase:	>200IU/l
Fluid loss:	>6l



Pancreatitis

symptoms, supported by clinical tests, such as CT and ultrasound, particularly if a complication such as pancreatic pseudocysts is suspected. Some organisations are also able to carry out 48-hour urine-based pancreatic function tests.

The clinical management of chronic pancreatitis depends on four factors: cause, which segment of the pancreas is implicated, the presence or absence of symptoms, and the size of the pancreatic duct. Abstinence from alcohol is essential for patients with alcohol-induced chronic pancreatitis. Supportive therapy providing replacement pancreatic enzymes is also given.

Silent pancreatitis is where the symptoms are mild or absent so no treatment is indicated. For patients with disabling symptoms, however, treatment is clearly indicated.

If the patient's symptoms are severe, surgery might be required. The surgical options are as follows:

- Chemical thoracic sympathectomy – the surgical destruction of the sympathetic nerves that feed into the pancreas. This procedure, carried out under X-ray guidance, will be tested initially by inhibiting the nerve conduction by local saturation in analgesia.
- A Peustow procedure, where the dilated duct is decompressed by suturing it to the intestine.
- Beger's procedure, where part of the pancreas is removed in those patients who have marked damage to one lobe of the pancreas or who have developed pseudocysts. They might not become diabetic because compensation for the damaged tissue might have already occurred.
- On some occasions, where damage to the pancreas is diffuse and non-dilated pancreatic ducts exist, a total pancreatectomy might be required. It is important that the patient is aware that he or she will be diabetic following this operation.

TIME OUT 4

Brainstorm for ten minutes on what role you would expect to fulfil for a patient who has pancreatitis or who has undergone pancreatic surgery. Then list ten key aspects in priority order and mark those that you would expect someone caring for you to be able to explain to your family. Think about whether you could provide this information to your patients.

The role of the nurse This can be summarised as:

- To do the patient no harm and to protect themselves and others, especially from sharp injuries.
- To monitor closely and accurately O_2 blood saturation levels, vital signs, blood glucose and compliance with treatment.
- To carry out basic care, including hygiene and hydration.
- To administer analgesia and review effectiveness.
- To provide information and education to the patient, particularly in relation to reducing alcohol intake.

Capillary blood sampling

As we have seen in acute pancreatitis and post-operatively in pancreas surgery, there is a need to monitor blood glucose due to the potential of hyperglycaemia and its associated complications. As part of the APACHE score, a blood glucose level greater than 10mmol/litre is one factor that might indicate a poor prognosis for the patient (Clark and Kumar 1998). There is nothing in the literature that describes the rate of serum glucose growth during an acute episode of pancreatitis. This is because there are so many other contributing factors that impact on these serum levels: previous diet, current nutrition status, degree of autogenesis, previous liver disease, consciousness levels relating to its uptake in the brain and whether any insulin had been prescribed for the patient. It is impossible, therefore, to prescribe a set frequency regime; it can only be determined by the clinical status of the patient (AARC 1994). This is also replicated in diabetes with the condition diabetic ketoacidosis. The British Diabetic Association (1997) (now known as Diabetes UK), in its guidelines for practice, advised a glucose monitoring regime of between one and four-hourly. They also stated that any alteration to the glucose regime must be followed by a measurement one hour later, whereas Aronson (1998) maintains that a six-hourly blood glucose testing regime would be sufficient.

To determine the best technique to monitor serum glucose, the risk of capillary sampling (with point of care tests, which have become commonplace), must be balanced with that of routine venepuncture. As technology has improved the devices used to measure blood glucose, it might be argued that the risk of staff complacency could increase.

Hazard Notice 13 (DHSS 1987) and more recently Safety Notice MDA S/N 9616 (MDA 1996) outline the risks of misleading results obtained from point of care testing and the need for adequate training. This training must include:

- The basic principles of measurement and the expected results in normal and pathological states.
- A demonstration of the use of devices used to measure blood glucose in accordance with the manufacturer's specification.
- An explanation of the consequences of improper use.
- Instruction in the process of sampling, including health and safety implications.
- Emphasis on the importance of complete documentation, including any quality assurance tests carried out.

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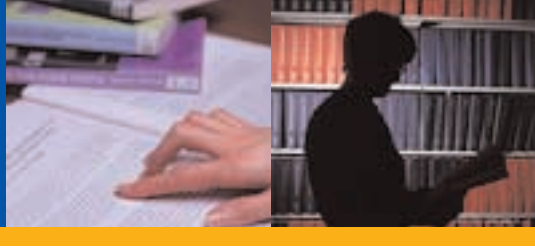

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- Any quality control and calibration methodologies required.
- Practical experience of the procedure under supervision that allows assessment of competence.

It is also clear from the UKCC *Code of Conduct* (UKCC 1992) that, as nurses, we should not harm our patient (Clause 2) or carry out activities we are not competent to fulfil (Clause 4). It is essential, therefore, that we ensure capillary blood sampling is carried out in a safe and accurate manner, and we cannot afford to be complacent. The process of good capillary blood sampling will incorporate adherence to local policies and current legislation.

TIME OUT 5

Identify any local policies and national standards pertinent to your clinical area that encompass capillary blood glucose monitoring. Reflect on what strategies could be used to ensure that all your colleagues comply with them.

When taking a capillary blood sample the following process might be followed:

1. Collect the appropriate equipment, including alcohol swab, sterile gauze, an adhesive plaster, a lancet rather than an ordinary needle (restricting puncture depth), correct glucometer and reagent strip, sharps box and a clearly identified method of transporting this equipment safely to the patient. Examination gloves may need to be worn to comply with universal precautions.
2. Explain to the patient the purpose of the test and that it might sting. Identify the site for sampling and ensure it is socially clean before cleaning with alcohol swab. Allow the alcohol to dry. The site should be clear of bruising, infection or puncture wounds.
3. Ensure the patient's palm is facing upwards and the hand is below the heart line. Hold on to the finger to be used while pressing your thumb, in a gentle rolling movement, from the knuckle of the selected finger to its tip, stimulating the flow of blood.
4. To minimise pain, gently pierce the side of the fingertip with the lancet. Wipe away the first drop of blood to promote blow flow. It must be noted that this first drop of blood contains interstitial tissue fluid, which would give inaccurate results (CDHS 1990).
5. If necessary, further gentle pressure can be applied at the distal joint of the finger to restrict blood flow from the fingertip. This must not be excessive or further interstitial fluid might be expressed and would compromise the results.
6. Ensure sufficient blood is formed in a droplet (any blood that runs down the finger cannot be used as it might lead to a low sample volume being applied to the reagent stick). Then, in a quick and efficient manner, apply the bead of blood to the reagent stick, ensuring that the sample site is equally covered. Dry off surplus blood on the stick without sucking any off the reagent strip. This is to prevent smearing of the reflective glass reader. (It must be noted that some manufacturers insist on wiping away all the blood prior to inserting the reagent stick into the glucometer.)
7. Stop the bleeding by applying pressure and apply the adhesive plaster. This will help prevent others from using the same site within 24 hours. Ensure residue or waste is disposed of safely and appropriately.

The advantages of capillary blood glucose sampling are as follows:

- It can be carried out at the patient's bedside with a relatively instantaneous result.
- It has a low financial cost to the organisation.
- It is a simpler task to delegate to junior or non-professional staff.

Capillary blood glucose sampling cannot be carried out on patients with the following complications:

- Hypotension or those patients who are peripherally shutdown – a common outcome of severe pancreatitis.
- Peripheral vasoconstriction or vascular disease, as an insufficient sample specimen might be obtained, giving an erroneous result.
- Polycythemia, as clotting time would be reduced, hence any metered reading devices would become difficult to measure (Hackett 1997).

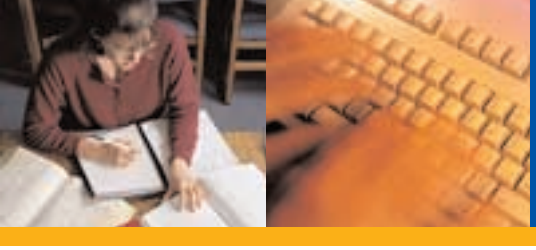
The National Committee for Clinical Laboratory Standards (1992) identified that capillary punctures should not be performed at or through the following sites:

- The posterior curvature of the heel, as the device might puncture the bone or calluses might form on it.
- The fingers of neonates because of the risk of nerve damage.
- Previous puncture sites.
- Inflamed, swollen, or oedematous tissues.
- Cyanotic or poorly perfused tissues.
- Localised areas of infection.
- Peripheral arteries (Meites 1989).

The committee also decided capillary punctures should not be performed on babies who were less than 24 hours old because of poor peripheral perfusion.

To make a relative comparison with regular venepuncture, the associated complications or possible harm from capillary blood sampling must be understood, as well as the factors that might invalidate the readings, especially with blood glucose reagents (Box 2).

The most important factor to be aware of is the potential for inappropriate patient management as a result of relying on a blood test that might be suspect. For example, the blood test might not be correct if the machine has not been serviced or cleaned, if staff have not been trained in its use or if they have used it



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sub-optimally (Courtney *et al* 1990, Garvey *et al* 1999). The storage environment, particularly heat and altitude, could affect the results.

The sampling process itself is fraught with difficulty. A sufficiently deep incision needs to be made by overcoming tissue resistance to allow formation of 0.5-1ml of blood; this is approximately 2.5mm for an adult (Louis 1999), but different lancets have different settings. Nurses also frequently use a freehand approach that can cause pain and interstitial fluid contamination. A second, deeper cut might need to be made if unsuccessful the first time. An excessive number of puncture sites could lead to infection.

When contemplating routine venepuncture, however, consideration must be given to its associated complications. These are:

- The increased length of time to complete the procedure safely.
- Greater risk of infection, especially if the patient is immunosuppressed.
- Risk of nerve damage.
- Haematoma.
- Increased difficulty for operator of technique.
- Greater volumes of blood required.
- Higher associated costs.

These disadvantages might be outweighed by the increased accuracy of sampling when a poor capillary technique is used.

TIME OUT 6

Think about the glucometer or another medical device you use in your clinical area. When was it last serviced or cleaned? Write a short statement that would demonstrate to an observer that you are competent in using it.

Conclusion

Pancreatitis is a life-threatening disease with many causes. Nursing staff have a responsibility to maintain the safety of patients and to do them no harm. When nurses cause pain to a patient it must be minimal and in their best interest. Understanding the process of capillary blood sampling and how it might harm the patient will help to minimise this risk – this comes from good ward education programmes.

Blood glucose levels can change rapidly during the acute period of pancreatitis, so capillary sampling is required hourly, particularly if insulin infusion has been commenced as therapy. However, with good technique and changing of fingerprick sites to those areas with minimal nerve endings, associated complications, pain and infection can be reduced.

Fortunately, insulin infusions for pancreatitis are no longer common (BSOG 1998, SSAT 1997, Steinberg and Tenner 1994) as any damage to the islets affects insulin and glucagon production equally. There is also no evidence that the replacement of natural somatostatin would be of benefit in acute pancreatitis (McKay *et al* 1993) and as this, too, equally suppresses glucagon and insulin production, blood glucose balance remains evenly hindered. If the condition of the patient was cardio-respiratory stable, then four-hourly measurements would be sufficient as this is the time it would take for all liver glucose supplies to be used up and action would be required to restore the balance.

With chronic pancreatitis, however, the key issue is managing the patient's symptoms. The most important symptom to deal with is pain that is often resistant to opioid therapy. Patients who express dissatisfaction with analgesia cover risk being labelled as painkiller addicts (Pasero 1998). It is important to recognise that continued pain is in fact a sign of treatment failure and nurses should obtain support from specialised units and pain control teams

TIME OUT 7

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you write and submit a profile are outlined on page 53.

Box 2. Factors that could invalidate readings

Equipment
Not serviced
Not clean
Expired sticks
Lancet type
Storage
Environment

Technique
Inappropriate place
Too deep/shallow
Insufficient blood volume
Infection
Timing
Contamination with interstitial fluid

Patient
Hypovolaemia
Peripheral vascular disease
Coagulation disorders
Non-compliance
Blood-borne disorders
Intensive oxygen therapy
Jaundice
Hyperglycaemia

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