

READING SUB-TEST – QUESTION PAPER: PARTS B & C

CANDIDATE NUMBER:

LAST NAME:

FIRST NAME:

MIDDLE NAMES:

PROFESSION:

VENUE:

TEST DATE:

Candidate details and photo will be printed here.

Passport Photo

CANDIDATE DECLARATION

By signing this, you agree not to disclose or use in any way (other than to take the test) or assist any other person to disclose or use any OET test or sub-test content. If you cheat or assist in any cheating, use any unfair practice, break any of the rules or regulations, or ignore any advice or information, you may be disqualified and your results may not be issued at the sole discretion of CBLA. CBLA also reserves its right to take further disciplinary action against you and to pursue any other remedies permitted by law. If a candidate is suspected of and investigated for malpractice, their personal details and details of the investigation may be passed to a third party where required.

CANDIDATE SIGNATURE: _____

TIME: 45 MINUTES

INSTRUCTIONS TO CANDIDATES

DO NOT open this **Question Paper** until you are told to do so.

One mark will be granted for each correct answer.

Answer **ALL** questions. Marks are **NOT** deducted for incorrect answers.

At the end of the test, hand in this **Question Paper**.

DO NOT remove OET material from the test room.



HOW TO ANSWER THE QUESTIONS

Mark your answers on this **Question Paper** by filling in the circle using a 2B pencil. **Example:** (A)



Part B

In this part of the test, there are six short extracts relating to the work of health professionals. For **questions 1-6**, choose the answer (**A**, **B** or **C**) which you think fits best according to the text.

Fill the circle in completely. Example:   

1. The manual informs us that the Blood Pressure Monitor

- (A) is likely to interfere with the operation of other medical equipment.
- (B) may not work correctly in close proximity to some other devices.
- (C) should be considered safe to use in all hospital environments.

Instruction Manual: Digital Automatic Blood Pressure Monitor

Electromagnetic Compatibility (EMC)

With the increased use of portable electronic devices, medical equipment may be susceptible to electromagnetic interference. This may result in incorrect operation of the medical device and create a potentially unsafe situation. In order to regulate the requirements for EMC, with the aim of preventing unsafe product situations, the EN60601-1-2 standard defines the levels of immunity to electromagnetic interferences as well as maximum levels of electromagnetic emissions for medical devices. This medical device conforms to EN60601-1-2:2001 for both immunity and emissions. Nevertheless, care should be taken to avoid the use of the monitor within 7 metres of cellphones or other devices generating strong electrical or electromagnetic fields.



2. The notice is giving information about

- (A) ways of checking that an NG tube has been placed correctly.
- (B) how the use of NG feeding tubes is authorised.
- (C) which staff should perform NG tube placement.

NG feeding tubes

Displacement of nasogastric (NG) feeding tubes can have serious implications if undetected. Incorrectly positioned tubes leave patients vulnerable to the risks of regurgitation and respiratory aspiration. It is crucial to differentiate between gastric and respiratory placement on initial insertion to prevent potentially fatal pulmonary complications. Insertion and care of an NG tube should therefore only be carried out by a registered doctor or nurse who has undergone theoretical and practical training and is deemed competent or is supervised by someone competent. Assistant practitioners and other unregistered staff must never insert NG tubes or be involved in the initial confirmation of safe NG tube position.



3. What must all staff involved in the transfusion process do?

- A check that their existing training is still valid
- B attend a course to learn about new procedures
- C read a document that explains changes in policy

'Right Patient, Right Blood' Assessments

The administration of blood can have significant morbidity and mortality. Following the introduction of the 'Right Patient, Right Blood' safety policy, all staff involved in the transfusion process must be competency assessed. To ensure the safe administration of blood components to the intended patient, all staff must be aware of their responsibilities in line with professional standards.

Staff must ensure that if they take any part in the transfusion process, their competency assessment is updated every three years. All staff are responsible for ensuring that they attend the mandatory training identified for their roles. Relevant training courses are clearly identified in Appendix 1 of the Mandatory Training Matrix.



4. The guidelines establish that the healthcare professional should
- (A) aim to make patients fully aware of their right to a chaperone.
 - (B) evaluate the need for a chaperone on a case-by-case basis.
 - (C) respect the wishes of the patient above all else.

Extract from 'Chaperones: Guidelines for Good Practice'

A patient may specifically request a chaperone or in certain circumstances may nominate one, but it will not always be the case that a chaperone is required. It is often a question of using professional judgement to assess an individual situation. If a chaperone is offered and declined, this must be clearly documented in the patient's record, along with any relevant discussion. The chaperone should only be present for the physical examination and should be in a position to see what the healthcare professional undertaking the examination/investigation is doing. The healthcare professional should wait until the chaperone has left the room/cubicle before discussion takes place on any aspect of the patient's care, unless the patient specifically requests the chaperone to remain.



5. The guidelines require those undertaking a clinical medication review to

- (A) involve the patient in their decisions.
- (B) consider the cost of any change in treatments.
- (C) recommend other services as an alternative to medication.

Annual medication review

To give all patients an annual medication review is an ideal to strive for. In the meantime there is an argument for targeting all clinical medication reviews to those patients likely to benefit most.

Our guidelines state that 'at least a level 2 medication review will occur', i.e. the minimum standard is a treatment review of medicines with the full notes but not necessarily with the patient present. However, the guidelines go on to say that 'all patients should have the chance to raise questions and highlight problems about their medicines' and that 'any changes resulting from the review are agreed with the patient'.

It also states that GP practices are expected to

- minimise waste in prescribing and avoid ineffective treatments.
- engage effectively in the prevention of ill health.
- avoid the need for costly treatments by proactively managing patients to recovery through the whole care pathway.



6. The purpose of this email is to

- (A) report on a rise in post-surgical complications.
- (B) explain the background to a change in patient care.
- (C) remind staff about procedures for administering drugs.

To:

All Staff

Subject:

Advisory Email: Safe use of opioids

In August, an alert was issued on the safe use of opioids in hospitals. This reported the incidence of respiratory depression among post-surgical patients to an average 0.5% – thus for every 5,000 surgical patients, 25 will experience respiratory depression. Failure to recognise respiratory depression and institute timely intervention can lead to cardiopulmonary arrest, resulting in brain injury or death. A retrospective multi-centre study of 14,720 cardiopulmonary arrest cases showed that 44% were respiratory related and more than 35% occurred on the general care floor. It is therefore recommended that post-operative patients now have continuous monitoring, instead of spot checks, of both oxygenation and ventilation.



Part C

In this part of the test, there are two texts about different aspects of healthcare. For **questions 7-22**, choose the answer (**A**, **B**, **C** or **D**) which you think fits best according to the text.

(A)
(B)
(C)
(D)

Fill the circle in completely. Example:

Text 1: Sleep deprivation

Millions of people who suffer sleep problems also suffer myriad health burdens. In addition to emotional distress and cognitive impairments, these can include high blood pressure, obesity, and metabolic syndrome. 'In the studies we've done, almost every variable we measured was affected. There's not a system in the body that's not affected by sleep,' says University of Chicago sleep researcher Eve Van Cauter. 'Every time we sleep-deprive ourselves, things go wrong.'

A common refrain among sleep scientists about two decades ago was that sleep was performed by the brain in the interest of the brain. That wasn't a fully elaborated theory, but it wasn't wrong. Numerous recent studies have hinted at the purpose of sleep by confirming that neurological function and cognition are messed up during sleep loss, with the patient's reaction time, mood, and judgement all suffering if they are kept awake too long.

In 1997, Bob McCarley and colleagues at Harvard Medical School found that when they kept cats awake by playing with them, a compound known as adenosine increased in the basal forebrain as the sleepy felines stayed up longer, and slowly returned to normal levels when they were later allowed to sleep. McCarley's team also found that administering adenosine to the basal forebrain acted as a sedative, putting animals to sleep. It should come as no surprise then that caffeine, which blocks adenosine's receptor, keeps us awake. Teaming up with Basheer and others, McCarley later discovered that, as adenosine levels rise during sleep deprivation, so do concentrations of adenosine receptors, magnifying the molecule's sleep-inducing effect. 'The brain has cleverly designed a two-stage defence against the consequences of sleep loss,' McCarley says. Adenosine may underlie some of the cognitive deficits that result from sleep loss. McCarley and colleagues found that infusing adenosine into rats' basal forebrain impaired their performance on an attention test, similar to that seen in sleep-deprived humans. But adenosine levels are **by no means the be-all and end-all** of sleep deprivation's effects on the brain or the body.

Over a century of sleep research has revealed numerous undesirable outcomes from staying awake too long. In 1999, Van Cauter and colleagues had eleven men sleep in the university lab. For three nights, they spent eight hours in bed, then for six nights they were allowed only four hours (accruing what Van Cauter calls a sleep debt), and then for six nights they could sleep for up to twelve hours (sleep recovery). During sleep debt and recovery, researchers gave the participants a glucose tolerance test and found striking differences. While sleep deprived, the men's glucose metabolism resembled a pre-diabetic state. 'We knew it would be affected,' says Van Cauter. 'The big surprise was the effect being much greater than we thought.'



Subsequent studies also found insulin resistance increased during bouts of sleep restriction, and in 2012, Van Cauter's team observed impairments in insulin signalling in subjects' fat cells. Another recent study showed that sleep-restricted people will add 300 calories to their daily diet. Echoing Van Cauter's results, Basheer has found evidence that enforced lack of sleep sends the brain into a catabolic, or energy-consuming, state. This is because it degrades the energy molecule adenosine triphosphate (ATP) to produce adenosine monophosphate and this results in the activation of AMP kinase, an enzyme that boosts fatty acid synthesis and glucose utilization. 'The system sends a message that there's a need for more energy,' Basheer says. Whether this is indeed the mechanism underlying late-night binge-eating is still speculative.

Within the brain, scientists have glimpsed signs of physical damage from sleep loss, and the time-line for recovery, if any occurs, is unknown. Chiara Cirelli's team at the Madison School of Medicine in the USA found structural changes in the cortical neurons of mice when the animals are kept awake for long periods. Specifically, Cirelli and colleagues saw signs of mitochondrial activation – which makes sense, as 'neurons need more energy to stay awake,' she says – as well as unexpected changes, such as undigested cellular debris, signs of cellular aging that are unusual in the neurons of young, healthy mice. 'The number [of debris granules] was small, but it's worrisome because it's only four to five days' of sleep deprivation,' says Cirelli. After thirty-six hours of sleep recovery, a period during which she expected normalcy to resume, those changes remained.

Further insights could be drawn from the study of shift workers and insomniacs, who serve as natural experiments on how the human body reacts to losing out on such a basic life need for chronic periods. But with so much of our physiology affected, an effective therapy – other than sleep itself – is hard to imagine. 'People like to define a clear pathway of action for health conditions,' says Van Cauter. 'With sleep deprivation, everything you measure is affected and interacts synergistically to produce the effect.'



Text 1: Questions 7-14

7. In the first paragraph, the writer uses Eve Van Cauter's words to
- (A) explain the main causes of sleep deprivation.
 - (B) reinforce a view about the impact of sleep deprivation.
 - (C) question some research findings about sleep deprivation.
 - (D) describe the challenges involved in sleep deprivation research.
8. What do we learn about sleep in the second paragraph?
- (A) Scientific opinion about its function has changed in recent years.
 - (B) There is now more controversy about it than there was in the past.
 - (C) Researchers have tended to confirm earlier ideas about its purpose.
 - (D) Studies undertaken in the past have formed the basis of current research.
9. What particularly impressed Bob McCarley of Harvard Medical School?
- (A) the effectiveness of adenosine as a sedative
 - (B) the influence of caffeine on adenosine receptors
 - (C) the simultaneous production of adenosine and adenosine receptors
 - (D) the extent to which adenosine levels fall when subjects are allowed to sleep
10. In the third paragraph, what idea is emphasised by the phrase 'by no means the be-all and end-all'?
- (A) Sleep deprivation has consequences beyond its impact on adenosine levels.
 - (B) Adenosine levels are a significant factor in situations other than sleep deprivation.
 - (C) The role of adenosine as a response to sleep deprivation is not yet fully understood.
 - (D) The importance of the link between sleep deprivation and adenosine should not be underestimated.



11. What was significant about the findings in Van Cauter's experiment?
- (A) the rate at which the sleep-deprived men entered a pre-diabetic state
 - (B) the fact that sleep deprivation had an influence on the men's glucose levels
 - (C) the differences between individual men with regard to their glucose tolerance
 - (D) the extent of the contrast in the men's metabolic states between sleep debt and recovery
12. In the fifth paragraph, what does the word 'it' refer to?
- (A) an enzyme
 - (B) new evidence
 - (C) a catabolic state
 - (D) enforced lack of sleep
13. What aspect of her findings surprised Chiara Cirelli?
- (A) There was no reversal of a certain effect of sleep deprivation.
 - (B) The cortical neurons of the mice underwent structural changes.
 - (C) There was evidence of an increased need for energy in the brains of the mice.
 - (D) The neurological response to sleep deprivation only took a few hours to become apparent.
14. In the final paragraph, the quote from Van Cauter is used to suggest that
- (A) the goals of sleep deprivation research are sometimes unclear.
 - (B) it could be difficult to develop any treatment for sleep deprivation.
 - (C) opinions about the best way to deal with sleep deprivation are divided.
 - (D) there is still a great deal to be learnt about the effects of sleep deprivation.



Text 2: ADHD

The American Psychiatric Association (APA) recognised Attention Deficit Hyperactivity Disorder (ADHD) as a childhood disorder in the 1960s, but it wasn't until 1978 that the condition was formally recognised as afflicting adults. In recent years, the USA has seen a 40% rise in diagnoses of ADHD in children. It could be that the disorder is becoming more prevalent, or, as seems more plausible, doctors are making the diagnosis more frequently. The issue is complicated by the lack of any recognised neurological markers for ADHD. The APA relies instead on a set of behavioural patterns for diagnosis. It specifies that patients under 17 must display at least six symptoms of inattention and/or hyperactivity; adults need only display five.

ADHD can be a controversial condition. Dr Russell Barkley, Professor of Psychiatry at the University of Massachusetts insists; 'the science is overwhelming: it's a real disorder, which can be managed, in many cases, by using stimulant medication in combination with other treatments'. Dr Richard Saul, a behavioural neurologist with five decades of experience, disagrees; 'Many of us have difficulty with organization or details, a tendency to lose things, or to be forgetful or distracted. Under such subjective criteria, the entire population could potentially qualify. Although some patients might need stimulants to function well in daily life, the lumping together of many vague and subjective symptoms could be causing a national phenomenon of misdiagnosis and over-prescription of stimulants.'

A recent study found children in foster care three times more likely than others to be diagnosed with ADHD. Researchers also found that children with ADHD in foster care were more likely to have another disorder, such as depression or anxiety. This finding certainly reveals the need for medical and behavioural services for these children, but it could also prove the non-specific nature of the symptoms of ADHD: anxiety and depression, or an altered state, can easily be mistaken for manifestations of ADHD.

ADHD, the thinking goes, begins in childhood. In fact, in order to be diagnosed with it as an adult, a patient must demonstrate that they had traits of the condition in childhood. However, studies from the UK and Brazil, published in JAMA Psychiatry, are fuelling questions about the origins and trajectory of ADHD, suggesting not only that it can begin in adulthood, but that there may be two distinct syndromes: adult-onset ADHD and childhood ADHD.

They echo earlier research from New Zealand. However, an editorial by Dr Stephen Faraone in JAMA Psychiatry highlights potential flaws in the findings. Among them, underestimating the persistence of ADHD into adulthood and overestimating the prevalence of adult-onset ADHD. In Dr Faraone's words, 'the researchers found a group of people who had sub-threshold ADHD in their youth. There may have been signs that things weren't right, but not enough to go to a doctor. Perhaps these were smart kids with particularly supportive parents or teachers who helped them cope with attention problems. Such intellectual and social scaffolding would help in early life, but when the scaffolding is removed, full ADHD could develop'.



Until this century, adult ADHD was a seldom-diagnosed disorder. Nowadays however, it's common in mainstream medicine in the USA, a paradigm shift apparently driven by two factors: reworked – many say less stringent – diagnostic criteria, introduced by the APA in 2013, and marketing by manufacturers of ADHD medications. Some have suggested that this new, broader definition of ADHD was fuelled, at least in part, to broaden the market for medication. In many instances, the evidence proffered to expand the definitions came from studies funded in whole or part by manufacturers. And as the criteria for the condition loosened, reports emerged about clinicians involved in diagnosing ADHD receiving money from drug-makers.

This brings us to the issue of the addictive nature of ADHD medication. As Dr Saul asserts, 'addiction to stimulant medication isn't rare; it's common. Just observe the many patients periodically seeking an increased dosage as their powers of concentration diminish. This is because the body stops producing the appropriate levels of neurotransmitters that ADHD drugs replace – **a trademark** of addictive substances.' Much has been written about the staggering increase in opioid overdoses and abuse of prescription painkillers in the USA, but the abuse of drugs used to treat ADHD is no less a threat. While opioids are more lethal than prescription stimulants, there are parallels between the opioid epidemic and the increase in problems tied to stimulants. In the former, users switch from prescription narcotics to heroin and illicit fentanyl. With ADHD drugs, patients are switching from legally prescribed stimulants to illicit ones such as methamphetamine and cocaine. The medication is particularly prone to abuse because people feel it improves their lives. These drugs are antidepressants, aid weight-loss and improve confidence, and can be abused by students seeking to improve their focus or academic performance. So, more work needs to be done before we can settle the questions surrounding the diagnosis and treatment of ADHD.



Text 2: Questions 15-22

15. In the first paragraph, the writer questions whether
- (A) adult ADHD should have been recognised as a disorder at an earlier date.
 - (B) ADHD should be diagnosed in the same way for children and adults.
 - (C) ADHD can actually be indicated by neurological markers.
 - (D) cases of ADHD have genuinely increased in the USA.
16. What does Dr Saul object to?
- (A) the suggestion that people need stimulants to cope with everyday life
 - (B) the implication that everyone has some symptoms of ADHD
 - (C) the grouping of imprecise symptoms into a mental disorder
 - (D) the treatment for ADHD suggested by Dr Barkley
17. The writer regards the study of children in foster care as significant because it
- (A) highlights the difficulty of distinguishing ADHD from other conditions.
 - (B) focuses on children known to have complex mental disorders.
 - (C) suggests a link between ADHD and a child's upbringing.
 - (D) draws attention to the poor care given to such children.
18. In the fourth paragraph, the word '**They**' refers to
- (A) syndromes.
 - (B) questions.
 - (C) studies.
 - (D) origins.



19. Dr Faraone suggests that the group of patients diagnosed with adult-onset ADHD
- (A) had teachers or parents who recognised the symptoms of ADHD.
 - (B) should have consulted a doctor at a younger age.
 - (C) had mild undiagnosed ADHD in childhood.
 - (D) were specially chosen by the researchers.
20. In the fifth paragraph, it is suggested that drug companies have
- (A) been overly aggressive in their marketing of ADHD medication.
 - (B) influenced research that led to the reworking of ADHD diagnostic criteria.
 - (C) attempted to change the rules about incentives for doctors who diagnose ADHD.
 - (D) encouraged the APA to rush through changes to the criteria for diagnosing ADHD.
21. In the final paragraph, the word 'trademark' refers to
- (A) a physiological reaction.
 - (B) a substitute medication.
 - (C) a need for research.
 - (D) a common request.
22. In the final paragraph, what does the writer imply about addiction to ADHD medication?
- (A) It is unlikely to turn into a problem on the scale of that caused by opioid abuse.
 - (B) The effects are more marked in certain sectors of the population.
 - (C) Insufficient attention seems to have been paid to it.
 - (D) The reasons for it are not yet fully understood.

**END OF READING TEST
THIS BOOKLET WILL BE COLLECTED**



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