Functional abdominal pain

P J Matthews, Q Aziz

Functional abdominal pain or functional abdominal pain syndrome (FAPS) is an uncommon functional gut disorder characterised by chronic or recurrent abdominal pain attributed to the gut but poorly related to gut function. It is associated with abnormal illness behaviour and patients show psychological morbidity that is often minimised or denied in an attempt to discover an organic cause for symptoms. Thus the conventional biomedical approach to the management of such patients is unhelpful and a person’s symptom experience is more usefully investigated using a biopsychosocial evaluation, which necessarily entails a multidisciplinary system of healthcare provision. Currently the pathophysiology of the disorder is poorly understood but is most likely to involve a dysfunction of central pain mechanisms either in terms of attentional bias, for example, hypervigilance or a failure of central pain modulation/inhibition. Although modern neurophysiological investigation of patients is promising and may provide important insights into the pathophysiology of FAPS, current clinical management relies on an effective physician-patient relationship in which limits on clinical investigation are set and achievable treatment goals tailored to the patient’s needs are pursued.

Pain is a symptom commonly reported by most patients with functional gastrointestinal disorders (FGD) and while abdominal pain is the most common symptom in irritable bowel syndrome (IBS) it is associated with other physiological events such as disordered defecation, which is not the case in functional abdominal pain syndrome (FAPS). Hence while the mechanisms of pain in FAPS and other FGDs including IBS are likely to be similar, these mechanisms may not explain the associated disorders of physiological function. To be concise we have focused on the mechanisms of pain in FAPS but the reader should be reassured that sufficient criteria for other FGDs that would entail a multidisciplinary system of healthcare provision. Currently the pathophysiology of the disorder is poorly understood but is most likely to involve a dysfunction of central pain mechanisms either in terms of attentional bias, for example, hypervigilance or a failure of central pain modulation/inhibition. Although modern neurophysiological investigation of patients is promising and may provide important insights into the pathophysiology of FAPS, current clinical management relies on an effective physician-patient relationship in which limits on clinical investigation are set and achievable treatment goals tailored to the patient’s needs are pursued.

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Abbreviations: FAPS, functional abdominal pain syndrome; FGD, functional gastrointestinal disorders; IBS, irritable bowel syndrome; SIT, spinothalamic tract; SI, primary somatosensory cortex; SII, secondary somatosensory cortex; ACC, anterior cingulated cortex; EEG, electroencephalograph; PET, positron emission tomography; fMRI, functional magnetic resonance imaging; MEG, magnetoencephalography; TCA, tricyclic antidepressant; GI, gastrointestinal
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Spinothalamic neurones via activation of AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazoleproprionic acid) and kainite ligand-gated ion channels. Sensory information is then conducted via the spinothalamic tract (STT), spinoreticular tract, and spinoencephalic tracts to the brain. The STT terminates in the medial and posterior thalamus and thalamicocortical fibres then projects to the primary somatosensory cortex (SI) and for visceral pain predominately to the secondary somatosensory cortex (SII). This pathway forms the basis of the sensory discriminative aspect of the pain experience whereas the spinoreticular and spinoencephalic pathways eventually terminate on the medial thalamus and from thence, third order thalamicocortical fibres ascend primarily to the anterior cingulate cortex (ACC) and insula; both areas encode the reflexive, motivational, and affective properties of the stimulus with the insula additionally involved in the integration of sensory and motor activity (fig 2). Recent evidence suggests the dorsal column that is most commonly known as the conduit of fine touch and vibration stimuli also mediates visceral pain. In a functional MRI experiment Al-Chaer et al have shown that after lesion of the dorsal column at T10 in a primate model of acute visceral pain, brain activation to colorectal distension is greatly attenuated. Interestingly the converse, a lesion in the STT at T10, failed to achieve the same effect. 9

As well as the ascending pathways a number of descending inhibitory pathways play a part in the perception of normal visceral sensation. The origin of the pathways is the opioid rich ACC and from here inhibitory signals are conveyed to the periaqueductal grey either directly or via second order neurones from the amygdala. Other midbrain regions where synaptic connections are made include the locus coeruleus and rostral ventral medulla. Third order opioidergic, serotonergic and second order noradrenergic neurones connect to the dorsal horn neurones where they “gate” or modulate ascending visceral afferent signals.

This is a simplistic description but is adequate in terms of understanding where abnormalities of pain processing can occur and may contribute to functional abdominal pain.

Abnormal visceral pain perception: the role of peripheral and central sensitisation

Peripheral sensitisation Studies suggest a third of people after an episode of gastroenteritis will develop IBS and that these people are more likely to show increased levels of mucosal lymphocytes on colonic biopsy compared with controls. In addition animal studies, which use models of inflammation, for example GI infection with Trichinella spiralis, show that visceral afferent neurones may remain sensitised after the inflammatory insult has elapsed leading to persistent visceral hypersensitivities and intestinal dysmotility.

Modern biological techniques have characterised possible putative receptors on peripheral nerve terminals that encode noxious stimuli and include: sodium channels, for example, SNS1/SNS2; the P2X3 (purinergic) receptor that is sensitive to adenosine triphosphate (ATP); neurokinin receptors, such as NK1; acid sensitive ion channel (ASIC) receptors in particular ASIC 3 and vanilloid receptors, principally TRPV1. In inflammatory conditions these receptors’ sensitivity is increased by the release of local inflammatory mediators like the prostaglandin PGE2, bradykinin, neurotrophins, and cytokines, which can induce increased receptor expression or receptor activation by the removal of local inhibitory mechanisms. For example, after inflammation bradykinin induces activation of protein kinase C (PKC), which in turn leads to sodium channel (SNS) and TRPV1 sensitisation.

The neurotrophic factor, nerve growth factor (NGF) is absorbed by the peripheral nerve terminal and retrogradely transported to the cell body where it induces increased TRPV1 expression, these new receptors are then anterogradely transported to the peripheral terminal. NGF causes increased cyclic adenosine monophosphate (cAMP) expression via G protein coupled stimulation of adenylyl cyclase (AC). cAMP may induce sensitisation by modulation of ion channels, increased receptor expression, or further interactions with secondary messengers, for example, Ca2+.

In addition genetic differences in secondary messengers of immune cell activation, for example, G proteins, which are involved in the transduction of signals from about 80% of all

![Image](http://pmj.bmj.com/)

Figure 1 Simplified cartoon showing the major receptor groups and molecular events involved in activation and sensitisation of visceral afferents. (Top) Normal activation. TRPV1, transient receptor potential vanilloid receptor 1 sensitive to H+, noxious heat and capsaicin; mDEG1, m Degenerin (a candidate receptor for mechanical transduction); P2X3, purinergic receptor; AMPA, α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor; NMDA, N-methyl-D-aspartate receptor. (Bottom) Sensitisation. At the dorsal horn synapse increased levels of different neurone activation lead to the activation of the NMDA receptor by the removal of the magnesium ion block. BK, bradykinin; PGE2, prostaglandin. E2; G, G protein; PKC, protein kinase C; Src, tyrosine kinase Src. (See text for more details).
cellular membrane receptors, may play a part in determining susceptibility to a gut inflammation. G proteins are essential for stimulus-response coupling of receptors linked to intracellular effector systems such as the adenylcyclases, the phosphoinositide system, ion channels, as well as various protein kinases and transcription factors. A recent study has shown that a polymorphism for the gene GNB3 that encodes the 3 subunit of heterotrimeric G proteins may be increased in patients with functional dyspepsia; the authors speculate that the resultant defective G protein signal transduction in these patients may compromise normal immune cell-pathogen interactions in the gut leading to poor pathogen clearance and susceptibility to FGD. Alternatively compromised G coupled signal transduction might also interfere directly with local inhibitory adrenoceptor nerves and induce visceral hypersensitivity.

Central sensitisation
The increased afferent neurone barrage to the dorsal horn (DH) neurones of the spinal cord because of PS causes central sensitisation (CS) by increased presynaptic glutamate secretion, which leads to the removal of the magnesium ion block of the N-methyl-D-aspartate (NMDA) receptor. Additional key enzymes in the dorsal horn neurone include protein kinase C (PKC) and the tyrosine kinase, Src, which are produced as the end product of the activation of multiple G protein coupled receptors and further contribute to CS by phosphorylation of the NMDA channel. The resulting increased responsiveness of the dorsal horn neurones outlasts the initiating insult and may only require a low level peripheral input to maintain it. In addition the recruitment of surrounding DH neurones, termed heterosynaptic potentiation, makes the area from which the stimulus originated in the gut appear enlarged (see fig 1).

Recently Willert et al have shown in healthy volunteers that the NMDA antagonist ketamine can reverse acid induced oesophageal hypersensitivity mediated by CS suggesting a role of the NMDA receptor in human visceral hypersensitivity. Clinical evidence that CS may mediate FAPS comes from studies that show that patients with FGD have an increased likelihood of developing fibromyalgia or to have multiple functional GI syndromes at the same time, or both, for example, functional dyspepsia or non-cardiac chest pain. These clinical findings of a “globally” sensitised somatic and visceral afferent nervous system suggest CS, occurring at the level of the spinal cord is an important mechanism of viscerosomatic and viscerovisceral sensitisation.

THE NEUROPHYSIOLOGICAL INVESTIGATION OF VISCERAL PAIN
Various neurophysiological techniques have been used to explore normal and abnormal pain processing within the central nervous system. Primarily these techniques have focused on the examination of GI afferent pathways and elucidation of the brain areas involved in the cognitive interpretation after a visceral stimulus:

Cortical evoked potentials (CEP)
In this technique an electroencephalograph (EEG) is recorded during runs of stimulation (usually electrical or mechanical) of the viscera undergoing investigation. Recording of EEG is phase and time locked with the stimulus and after successive stimuli extraction of background randomly occurring EEG waves can be achieved leaving the desired stimulus evoked brain activity. This technique can be used to assess visceral afferent pathway integrity and may give an indication of whether these pathways have been sensitised to promote visceral afferent transmission to the central nervous system.

Positron emission tomography (PET)
This cerebral scanning technique permits measurement of cerebral blood flow, a proxy of neuronal activity, after the intravenous injection of a radioisotope, for example, labelled
ences in cortical activation of the brain than that induced by
suggests that noxious visceral stimuli produce some differ-
evaluation of pain pathway activation and patterns of
offer the exciting prospect of providing a second by second
advances allowing deeper regions of the brain to be studied
stimulation. This latter quality and the technological
dynamic interpretation of brain area activation after visceral
as having comparable spatial resolution to PET and fMRI,
helmet-like apparatus that covers the subject’s head. As well
sensitive sensors known as SQUIDS (super conduction
minute magnetic fields. These can be detected with highly
Visceral stimulation leads to cortical activation that generates
Magnetoencephalography (MEG)
water (H215O) by the detection of positron emitting radio-
nuclides. Block design experiments are used that compare the perfusion images taken when the subject is at rest with those when the subject is experiencing a visceral stimulus.

**Functional magnetic resonance imaging (fMRI)**

fMRI detects increases in blood flow in areas of heightened neuronal activity by the detection of oxygenated haemoglobin. Paradoxically increased neuronal activity and hence blood flow correlates positively with levels of oxyhaemoglobin, as spurts of neuronal activity are associated with anaerobic brain metabolism. Thus increased oxyhaemoglobin at the sites of increased neuronal activity produces an increased signal that can be interpreted to show the structures activated by the visceral stimulus. fMRI has a superior temporal resolution compared with PET of between four and eight seconds and because no radioisotopes are used permits repeated studies on the same subject.

**Magnetoencephalography (MEG)**

Visceral stimulation leads to cortical activation that generates minute magnetic fields. These can be detected with highly sensitive sensors known as SQUIDS (super conduction quantum interference device), which are contained within a helmet-like apparatus that covers the subject’s head. As well as having comparable spatial resolution to PET and fMRI, MEG has a superior temporal resolution that permits a more dynamic interpretation of brain area activation after visceral stimulation. This latter quality and the technological advances allowing deeper regions of the brain to be studied offer the exciting prospect of providing a second by second evaluation of pain pathway activation and patterns of aberrant pain processing.

The evidence from studies using the above techniques suggests that noxious visceral stimuli produce some differences in cortical activation of the brain than that induced by somatic stimuli. These studies show that cortical processing of visceral sensation entails initial processing in the primary and secondary somatosensory cortices (for sensory discrimination), with subsequent involvement of the anterior cingulate (ACC) and pre-frontal cortices for affect and cognition respectively. This shows that it is possible not only to localise the site of our gut sensation but it is also possible to assign emotional value to it. Furthermore, the study by Phillips et al is of particular relevance to FAPS as it shows that even in normal subjects, visceral stimuli presented within a negative emotional context (that is, oesophageal stimuli presented at a time when subjects were viewing fearful facial expressions on a screen) are rated as more unpleasant and cause significantly more activation of brain areas involved in cognitive pain processing than those stimuli paired with neutral faces. This suggests that the psychological state of the person plays an important part on visceral sensory perception and the brain processing.

Studies have also shown that patients with functional gastrointestinal disorders either display an increased activation in brain areas involved in processing visceral sensation or show different areas of cortical activation than control subjects. However, these studies are preliminary and do not shed light on specific pathophysiological mechanisms of heightened pain perception in patients with functional gastrointestinal disorders. (For a detailed review of the literature on functional brain imaging in functional gastrointestinal disorders please see Hobson and Aziz). Table 1 describes the results of such studies and the method used in each.

Therefore in summary the mechanisms involved in symptom production in FAPS and other functional digestive disorders are potentially multiple and interconnected. The scientific challenge will be to correctly interpret the results of studies, in the knowledge that no method is without its

<table>
<thead>
<tr>
<th>Reference</th>
<th>Patients (n)</th>
<th>Normal subjects</th>
<th>GI tract studied</th>
<th>Technique</th>
<th>Principal findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furlong et al</td>
<td>NA</td>
<td>Oesophagus</td>
<td>MEG</td>
<td></td>
<td>Non-painful stimulus with balloon distension caused activation of SI, SI, insular cingulate cortex and the ACC. Painful and non-painful stimulation. Inferior border of SI activated with both stimuli. ACC activation with painful stimuli.</td>
</tr>
<tr>
<td>Aziz et al</td>
<td>NA</td>
<td>Distal oesophagus</td>
<td>PET</td>
<td></td>
<td>Painful and non-painful stimulation. Inferior border of SI activated with both stimuli. ACC activation with painful stimuli.</td>
</tr>
<tr>
<td>Silverman et al</td>
<td>18</td>
<td>Rectum</td>
<td>PET</td>
<td></td>
<td>Normal subjects: actual or simulated painful stimuli evoked activation of ACC. IBS subjects left prefrontal cortex activated but no ACC activation.</td>
</tr>
<tr>
<td>Mertz et al</td>
<td>42 (IBS) 23</td>
<td>NA</td>
<td>Rectum</td>
<td>fMRI</td>
<td>Female IBS subjects showed greater activation in the ventromedial prefrontal cerebral cortex, right ACC, left amygdala. Male IBS subjects showed increased activation in right dorsolateral prefrontal cerebral cortex, insula and PAG. Conclusion: Different sex IBS subjects differ in activation of brain areas after visceral stimulation.</td>
</tr>
<tr>
<td>Naliboff et al</td>
<td>9 (IBS) 57</td>
<td>Women 19 men</td>
<td>Rectum</td>
<td>fMRI</td>
<td>Rectal distension and somatic heat stimulation compared between IBS and normal controls. Increased neural processing evoked to both stimuli in IBS patients and at different cortical levels, suggesting increased differential activation, such as, selective aberrant central nervous processing.</td>
</tr>
<tr>
<td>Verne et al</td>
<td>9</td>
<td>Rectum</td>
<td>fMRI</td>
<td></td>
<td>Study in which oesophageal stimuli were presented with visual stimuli (neutral or fearful faces). Significantly greater oesophageal discomfort and activation of left dorsal anterior cingulated gyrri and bilateral anterior insulai occurred with fearful faces.</td>
</tr>
<tr>
<td>Phillips et al</td>
<td>8</td>
<td>Oesophagus</td>
<td>fMRI</td>
<td></td>
<td>Painful and non-painful stimulation. Inferior border of SI activated with both stimuli. ACC activation with painful stimuli.</td>
</tr>
</tbody>
</table>

Table 1 Methods and results of the studies on patients with functional gastrointestinal disorders.
limitations, to produce an organised objective understanding of different phenotypes within this heterogeneous population and from thence to evaluate the potential genetic susceptibilities responsible for them.

**CLINICAL EVALUATION OF THE FAPS PATIENT**

**History**

The initial evaluation of FAPS patients in primary and secondary care should be essentially the same and begins when the patient enters the consulting room. A careful history should be taken that takes into account not only the chronology of pain events, for example, relation to surgery or adverse or traumatic life events but attention should be paid to the emphasis and style with that these are described by the patient. Pain is often described by patients in more emotional terms than those with structural disease with terms like “agonising” and “sickening” rather than crampy, sharp, or stabbing. In addition, abdominal pain often covers a large area and may be one of several pains suffered by the patient suggesting concurrent somatisation disorder. The intensity of abdominal pain seldom varies with maximal pain being experienced most of the time, which contrasts with structural disease in which the pain intensity varies and is also more likely to affect other GI activity, for example, motility.

Some behaviour traits may come to light during a consultation that might lend weight to a diagnosis of FAPS. These would include reluctance on the part of the patient to consult that might lend weight to a diagnosis of FAPS. Mental state should be assessed in a sensitive manner that takes into account their belief systems. Patients should be provided with a positive diagnosis of their suffering. Another cause of worry is the possibility of missing structural disease especially cancer. Several retrospective and prospective studies suggest that diagnostic failures occur in only 0% to 4.5% and this should be borne in mind in the follow up of such patients especially if symptoms change.

**Management**

Several aspects of management are worth clarifying. Patients should be provided with a positive diagnosis of their condition and a full explanation of the diagnosis in an acceptable manner that takes into account their belief system.

**Physical examination**

While characteristically examination in a FAPS patient should fail to discover abnormality, several features should be noted, for example, the absence of autonomic arousal, presence of abdominal scars, and closed eyes sign; in which the subject closes their eyes during examination, which is the opposite of the more fearful anticipation displayed by sufferers of an acute abdomen.

Distraction can be used with a stethoscope pressing with firm pressure on the abdomen when previously the examining hand may have elicited pain at the same spot. Finally Carrett’s test may be performed, in which a painful area is palpated before and after the patient’s abdomen by essentially performing a sit up against the resistance of the examiner’s hand on the subject’s forehead. If the subject experiences pain with palpation against tense abdominal musculature, this suggests the cause of the pain is not intra-abdominal and may be musculoskeletal in nature.

**Investigation**

A limited investigation may be warranted in selected cases and could include screening laboratory studies, for example, full blood count, erythrocyte sedimentation rate, serum biochemistry, and C-reactive protein. In those patients in which alarm features such as rectal bleeding, weight loss, or anorexia are present then another diagnosis should be considered and investigated accordingly. Naturally secondary care providers will feel under pressure to provide endoscopic investigation by both the virtue of ease of access to endoscopy within a district general hospital and the FAPS patient’s reliance on such procedures to provide reassurance and explanation to their symptoms. Another cause of worry is the possibility of missing structural disease especially cancer. Several retrospective and prospective studies suggest that diagnostic failures occur in only 0% to 4.5% and this should be borne in mind in the follow up of such patients especially if symptoms change.

**Key points**

- Functional abdominal pain syndrome (FAPS) is one of the functional gastrointestinal disorders (FGD) defined as abdominal pain in the absence of an identifiable disorder.
- FAPS patients are a heterogeneous group whose symptoms are likely to be attributable to multiple causes.
- Disorders of pain processing that include peripheral and central sensitisation, defective descending modulation of afferent pain pathways, and hypervigilance towards normal visceral sensations are likely to be involved in the initiation and maintenance of FAPS.
- Clinical management should include a thorough history including a comprehensive account of psychosocial problems and limited investigations.
- Treatment options include low dose tricyclic antidepressants and psychological approaches, for example, cognitive behavioural therapies.
More resistant cases might benefit from psychological treatments.

**Cognitive therapy**

In cognitive therapy, patients are encouraged to explore the maladaptive beliefs about their pain and are encouraged to re-conceptualise their pain and take a more active role in its management. Keeping a diary record with pain sampling re-conceptualise their pain and take a more active role in its management. Keeping a diary record with pain sampling helps to establish a relationship between daily activities and their impact on pain. Relaxation and stress management techniques may be useful as part of a behavioural therapy programme. The emphasis here is on ignoring pain and reinforcing activity related behaviour. Biofeedback has a limited role (if at all) but relaxation and stress management techniques may be useful as part of a behavioural therapy programme.

**Behavioural therapies**

Therapy is aimed at increasing activities in a graded way without necessarily involving the self analytical approach detailed above. The emphasis here is on ignoring pain and reinforcing activity related behaviour. Biofeedback has a limited role (if at all) but relaxation and stress management techniques may be useful as part of a behavioural therapy programme.

**Hypnosis**

This is perhaps a marginalised area of functional gut disorder research and most of the available evidence pertains to IBS patients. Hypnosis is used to induce general relaxation and also suggestions are made whereby patients are encouraged to use imagery or visualisation in an attempt to assert control over gut sensation/function. Hypnotherapy leads to improved clinical outcomes but also improvements in physiological markers are seen, for example, rectal sensitivity to balloon distension are seen to normalise after hypnotherapy in IBS subjects with reported rectal hypersensitivity.

What remains to be seen is the objective effect of hypnosis in the modulation of visceral pain. Recent evidence from an fMRI study of normal subjects hypnotically induced to feel a somatic thermal pain stimulus showed activation within the thalamus, ACC, and insula cortex of greater magnitude than a control group who were requested to recall a previously experienced somatic pain stimulus. This suggests that activation of certain cortical structures can induce pain regardless of peripheral input and may be of great relevance to the cause of visceral hyperalgesia.

The elucidation of the mechanisms underlying functional bowel disorders is an exciting area of gastroenterology research and no doubt the solution will be as complex as the problem. FAPS patients represent a heterogeneous group with multiple mechanisms responsible for the initiation and maintenance of symptoms. Therefore the correct identification of these subgroups remains a priority both to clinicians and scientists. Modern imaging and molecular techniques offer the tools for the elucidation of these mechanisms by identification of central nervous system pathways that have undergone sensitisation and modification. Future treatments may entail the activation or attenuation of putative receptors within the GI tract, for example, TRPV1 antagonists; spinal cord or brain, for example, NMDA antagonists, which are felt to contribute significantly to symptom production. Furthermore, pharmacogenetics may help to identify those patients with susceptibility to developing FAPS and those most likely to respond to the above treatments.

**MALFUNCTIONAL CHOICE QUESTIONS (TRUE (T)/FALSE (F); ANSWERS AT END OF REFERENCES)**

1. True or false

   (A) Functional abdominal pain syndrome occurs in about 5% of the population.

   (B) FAPS patients quite often feign symptoms to obtain secondary benefit.

   (C) Visceral hypersensitivity may be an important physiological finding in determining the cause of FAPS symptoms.

   (D) Visceral pain is transduced in exactly the same pathways as somatic pain.

   (E) Central sensitisation can occur at the level of the dorsal horn by the reversal of the magnesium block of the AMPA receptor.

2. Concerning normal visceral pain transduction

   (A) Most painful visceral sensations are conducted to the central nervous system via the vagus nerve.

   (B) TRPV1 receptors are activated by protons or capsaicin and cause a characteristic burning sensation.

   (C) Second order spinthalamic neurons are stimulated via activation of AMPA receptors.
(D) Glutamate is the major excitatory signalling peptide at the dorsal horn synapse.

(E) Serotonergic and noradrenergic neurones are involved in modulating ascending visceral afferent signals at the level of the dorsal horn.

3. Regarding peripheral sensitisation

(A) A history of gastroenteritis may be reported in up to two thirds of IBS patients

(B) During inflammation NGF release increases TRPV1 expression in the neurone cell body.

(C) Increased levels of c AMP may be responsible for afferent neurone sensitisation.

(D) Animal models of GI inflammation often use nematode infections to simulate short lived inflammation.

(E) G protein polymorphisms may be increased in functional gastrointestinal disorders leading to immune cell dysfunction.

4. Regarding functional neuroimaging in FGD studies

(A) PET is an imaging modality that is ideal for repeated scanning of study subjects

(B) MEG has a superior temporal resolution in comparison with fMRI.

(C) Visceral stimuli paired with negative emotional visceral stimuli cause greater activation of brain areas involved with processing cognitive aspects of the pain experience.

(D) Increased activation of brain areas on fMRI is analogous with increased levels of oxyhaemoglobin at these sites.

(E) The somatosensory cortices are involved with sensory discrimination of painful stimuli whereas the anterior cingulate and pre-frontal cortices are involved in affect and cognitive processing of visceral pain.

5. Concerning the clinical management of FAPS

(A) A history of previous abdominal surgery may be relevant.

(B) Extensive GI investigation is warranted in all cases.

(C) Carnett’s test may be useful in discriminating between musculoskeletal pain and visceral pain.

(D) A trial of high dose tricyclic antidepressants should be used in most cases.

(E) Studies of cognitive therapy and hypnosis suggest these therapies are of no benefit in patients with FGDs

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ANSWERS
1. (A) F, (B) F, (C) T, (D) F, (E) F; 2. (A) F, (B) T, (C) T, (D) T, (E) T; 3. (A) F, (B) T, (C) T, (D) T, (E) T; 4. (A) F, (B) T, (C) T, (D) T, (E) T; 5. (A) T, (B) F, (C) T, (D) F, (E) F.
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