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Review

Affective agnosia: Expansion of the alexithymia construct and a new opportunity to integrate and extend Freud's legacy

Richard D. Lane[a,b,c,*], Karen L. Weihs[a,d], Anne Herring[a,b], Alex Hishaw[a,e], Ryan Smith[a,b]

[a] Department of Psychiatry, University of Arizona, 1501 North Campbell Avenue, Tucson, AZ 85724-5002, United States
[b] Department of Psychology, University of Arizona, 1503 E. University Avenue, Tucson, AZ 85721, United States
[c] Department of Neuroscience, University of Arizona, 1040 E 4th St., Tucson, AZ 8521, United States
[d] Department of Family and Community Medicine, University of Arizona, 1501 North Campbell Avenue, Tucson, AZ 85724-5052
[e] Department of Neurology, University of Arizona, 1501 North Campbell Avenue, Tucson, AZ 85724-5023, United States

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ABSTRACT

We describe a new type of agnosia consisting of an impairment in the ability to mentally represent or know what one is feeling. Freud the neurologist coined the term "agnosia" in 1891 before creating psychoanalysis in 1895 but the term has not been previously applied to the domain of affective processing. We propose that the concept of "affective agnosia" advances the theory, measurement and treatment of what is now called "alexithymia," meaning "lack of words for emotion." We trace the origin of the alexithymia construct and discuss the strengths and limitations of extant research. We review evidence that the ability to represent and put emotions into words is a developmental achievement that strongly influences one's ability to experience, recognize, understand and use one's own emotional responses. We describe the neural substrates of emotional awareness and affective agnosia and compare and contrast these with related conditions. We then describe how this expansion of the conceptualization and measurement of affective processing deficits has important implications for basic emotion research and clinical practice.

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* Corresponding author at: Department of Psychiatry, University of Arizona, 1501 North Campbell Avenue, Tucson, AZ 85724-5002, United States. Tel.: +1 520 626 3272; fax: +1 520 626 6050.
E-mail addresses: lane@email.arizona.edu (R.D. Lane), weihs@email.arizona.edu (K.L. Weihs), anneherring@gmail.com (A. Herring), ahishaw@neurology.arizona.edu (A. Hishaw), rssmith@email.arizona.edu (R. Smith).

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1. Introduction

In 1891 Sigmund Freud coined the term "agnosia" (from the Greek meaning "absence of knowledge") referring to patients with brain lesions who had lost their ability to recognize objects despite intact perception (Freud, 1953). He proposed that this recognition failure resulted from an inability to connect the stimulus information to the mental representation of the object. In 1895 Freud turned away from the study of neurological patients to the psychological investigation of patients with conversion disorder (Breuer and Freud, 1955), in so doing creating the field of psychoanalysis. Although the concept of agnosia was not invoked in subsequent psychoanalytic writings by Freud or his followers, within neurological circles the concept has thrived, referring to failures of recognition due to brain lesions that are specific to one sensory modality (e.g. the inability to recognize a person by viewing their face [prosopagnosia] but a retained ability to recognize a person by their voice) (Frederiks, 1969). The thesis of this paper is that the mental representation of emotion, including its conscious and unconscious vicissitudes, became a central concern of psychoanalysis, although not specifically described as such, and that severe impairments in this function can and should be linked to the agnosia concept. Parallel to the inability to map visual perceptual experience on to mental representations of conceptual meaning in visual agnosias (Crick and Koch, 2000; Jackendoff, 1987; Koch and Braun, 1996; Koch, 2004; Marr, 1982; Prinz, 2012), we here describe a new type of agnosia, called "affective agnosia," that involves a deficit in the ability to mentally represent the meaning of emotional responses, which typically requires the integration of information from multiple interoceptive and somatic sensory modalities, as well as information regarding conscious/unconscious appraisals of one's situation.

The current concept most closely related to affective agnosia is alexithymia, a term coined in 1972 from the Greek meaning "lack of words for emotion" (Sifneos, 1972). Taken literally this term refers to a type of anomia. Although alexithymia has often been assumed to be something more than a simple lack of words, it has been challenging to find a more suitable term and conceptual framework to describe and measure it. In fact, although marked by notable successes, the empirical study of alexithymia has focused on milder versions of the originally observed lack of awareness of, or inability to experience, emotion (Nemiah and Sifneos, 1970), and has failed to support the original observation by the creators of the construct that alexithymia was associated with adverse physical health outcomes (Nemiah et al., 1976).

Based on a review of recent developments in the neuroscientific and psychological study of emotion, and the burgeoning literature on "theory of mind" or mentalization (Frith and Frith, 2012, 2006, 1999; Frith, 2001), we argue that such impairments in the mental representation of emotion, at the most severe end of the continuum, are more accurately conceptualized as an expression of affective agnosia. Lesser degrees of impairment are of course much more common, may be transitory and are likely grounded in related brain-based processes. By anchoring a continuum in this new way and describing a continuum of complexity of mental representation of emotion, however, the method for measuring and studying the phenomenon of "knowing one's own emotions" may be advanced. Clinically, this new perspective creates new opportunities for the psychotherapeutic treatment of impairments in emotional experience and emotional processing that are in fact quite common clinically but have previously been difficult to recognize and understand, and thus to identify and treat.

2. Origins of the alexithymia construct

As psychoanalysis became the premier school of thought within psychiatry in the early to mid 20th century, its application to medical disorders became an important new focus. The relevance of psychoanalysis to sister medical disciplines arose naturally from Freud's guiding belief that the mental phenomena addressed in psychoanalysis were an expression of brain function and that someday the biological basis of psychoanalysis would be understood (Freud, 1895).

In this context, while seeking to emulate the astonishing successes associated with the prevention and treatment of infectious diseases, Franz Alexander and colleagues formulated what came to be known as "Specificity Theory" in the 1940s (Alexander, 1950). This theory held that seven psychosomatic disorders (e.g. hypertension, asthma, ulcerative colitis) were each associated with a specific unconscious conflict. It was recognized that additional factors such as genetic predisposition played a role, but it was also strongly believed that affect, activated in the context of unconscious conflicts but not consciously processed, could induce peripheral physiological changes that had an adverse effect on physical health over time (Alexander et al., 1968; Grinker and Spiegel, 1945; Weiner, 1977).

To empirically test Specificity Theory, two young psychiatrists at Harvard, Peter Sifneos and John Nemiah, interviewed patients with the disorders specified by Alexander and colleagues in the 1960s and made a critical observation. Based on their interviews they concluded these patients were not defending against unconscious affects as described by Specificity Theory and classic psychoanalytic theory. Rather, these patients appeared to have a diminution or absence of the basic human ability to experience feelings (Nemiah and Sifneos, 1970; Nemiah et al., 1976). In 1972 Sifneos named the condition "alexithymia" from his native Greek meaning "lack of words for emotion" (Sifneos, 1972).

Their formulation of alexithymia as a deficit-based rather than conflict-and-defense based phenomenon occurred within the broader context of a mid-century shift in psychoanalytic thinking. Relative to the focus on oedipal-level conflicts characteristic of neuroses in classic psychoanalytic theory, the shift to an earlier focus on developmental deficits in the pre-oedipal period was better able to explain more severe psychopathology such as borderline (Kernberg, 1967) and narcissistic (Kohut, 1977) personality disorders. The formulation of the alexithymia construct reflected a recognition that this same distinction applied to the relationship between affect and physical disease.

As a counterpoint to Specificity Theory, the formulation of the alexithymia construct also reflected a growing interest in common personality characteristics that contributed to psychosomatic disorders. In 1948, around the time that Specificity Theory had reached its apogee, and 24 years before the alexithymia term was coined, Ruesch (1948) published a paper titled "The Infantile Personality" in which it was hypothesized that a deficit or developmental arrest in the capacity for symbolic mental representation of emotion was the core problem in patients with psychosomatic disorders. Ruesch (1948) posited a host of potential causes for the personality structure associated with psychosomatic conditions, such as a lack of consistent parenting or trauma that overwhelms the child's sense of mastery. He proposed that these experiences caused deficits in social learning and an "infantile" form of "self-expression" that manifested as somatic symptoms. As Ruesch said, in these patients "tension must be expressed through action or through organs." Similarly and independently, in the 1950s and early 1960s, Marty and de M'uzan (1963) observed a specific cognitive style ("pensee operatoire") in individuals prone to psychosomatic disorders, characterized by a lack of fantasy and a preoccupation with the concrete

details of external events. Thus, the concept of alexithymia was part of a broader movement within psychodynamic thinking to a focus on developmental deficits.

It is our contention that fundamental scientific developments since the concept of alexithymia was first put forward contribute to the need to expand the construct. First, the burgeoning of systems neuroscience, derived in part from functional and structural brain imaging in humans, has led to an increasingly complete account of the biological basis of the mind (Gazzaniga et al., 2014). We now have a much better understanding of the neural basis of emotion and emotional experience in humans. Second, decades of empirical research on emotion that began in the 1960s has advanced our understanding that emotion is more than feeling, can be conscious as well as unconscious, and that intermediary gradations exist between the two (Kihlstrom et al., 2000; Lambie and Marcel, 2002). Third, the neuroscientific study of consciousness is burgeoning. An important domain in which significant advances have been made involve the neural basis of conscious vs. unconscious visual perception (Crick and Koch, 2003; Jackendoff, 1987; Koch, 2004; Marr, 1982; Prinz, 2012). Principles developed in the context of visual perception are potentially applicable to other domains of mental activity. Fourth, six years after the term "alexithymia" was coined, the concept of "theory of mind" or mentalization was put forward by Premack and Woodruff (1978) to address the ability to understand mental states, both one's own and those of others. We believe that an understanding of "theory of mind" both psychologically and neurobiologically, in light of new knowledge about the brain and emotion, makes a scientifically tractable extension of alexithymia not only possible but even necessary. Importantly this new formulation makes it possible to put "a diminution or absence of the basic human ability to experience feelings" (Nemiah and Sifneos, 1970) on a more solid theoretical and empirical footing and permits a shift to a perspective that verbal behavior is an indicator of the problem rather than the primary problem itself. It is our belief that once this distinction is appreciated the clinical and scientific relevance of the phenomenon that Sifneos and Nemiah called "alexithymia" will be more widely appreciated by the mental health community, including psychiatry, colleagues in sister medical disciplines, and researchers interested in the neural basis of emotion and emotional experience.

3. Alexithymia research

The clinical description of alexithymia was an important and valuable start, but empirical research on it required a method to reliably identify individuals with this condition. Using the clinical description by Sifneos and Nemiah, a self-report measure of alexithymia was created by Taylor et al. (1985) to capture this clinical phenomenon. The current version of the scale, the 20-item Toronto Alexithymia Scale (TAS-20), is the most commonly used measure of alexithymia (Bagby et al., 1994a, 1994b).

Until the TAS, and ultimately the TAS-20, was created, no psychometrically sound measure existed. Other measures of alexithymia with sound psychometric properties are now available in addition to the TAS-20, including the self-report Bermand-Vorst measure (Vorst and Bermond, 2001), the Observer Alexithymia Scale (Haviland et al., 2000), an interview-based rating scale (the Beth Israel measure) (Taylor et al., 1997) and others (Gori et al., 2012; Haviland and Reise, 1996; Porcelli and Mihura, 2010). Moreover, the developers of the TAS-20 have created a clinical interview based on the same conceptual framework as the TAS-20 that involves clinician ratings rather than self-report (Bagby et al., 2005). Although these are important innovations, the self-report TAS-20 is by far the most popular measure of alexithymia. It is, therefore, timely to review the TAS-20 as the leading measure of alexithymia.

The TAS-20 consists of 20 questions each rated on a 5-point scale that capture three factors: (1) difficulty describing feelings; (2) difficulty differentiating between feelings and physical sensations; and (3) externally-oriented thinking. The TAS-20 is now widely used in normative populations and occasionally in clinical populations. The factor structure of the TAS-20 has been supported (Bagby et al., 1994a, 1994b). Authors often report that they have studied alexithymia if they used the TAS-20, even though the subjects do not resemble those described by Sifneos and Nemiah.

Clearly the TAS-20 has put alexithymia on the empirical map (for an overview of findings with the TAS-20, see Taylor et al., 1997; Taylor and Bagby, 2013). To put these and other issues in context it is important to consider several issues regarding measurement of alexithymia with the TAS-20.

The first issue is that the TAS-20 is a self-report instrument in which subjects indicate the degree to which they agree that they have the manifestations of alexithymia. Given that the concept of alexithymia originated with the goal of capturing an impairment in experiencing and describing emotions (Nemiah and Sifneos, 1970; Nemiah et al., 1976), difficulties potentially arise when attempting to use a self-report measure to accomplish that (Dunning et al., 2004). For example, items from the TAS-20 include:

> It is difficult for me to find the right words for my feelings.
>
> I am able to describe my feelings easily (reverse scored).
>
> When I am upset, I don't know if I am sad, frightened, or angry.
>
> I have feelings that I can't quite identify.
>
> I often don't know why I am angry.
>
> It is difficult for me to reveal my innermost feelings, even to close friends

Clearly a respondent may have a complex array of emotional experiences and yet endorse each of these items in the alexithymic direction. In order for the TAS-20 to be a valid measure of severe alexithymia, affected individuals need to know that they have this impairment in experience and accurately report on it (i.e. to accurately report their difficulty in self-report).

A second issue is that the TAS-20 consistently correlates positively and substantially with self-reported negative affect (Lumley, 2000). On its face, this would appear to contradict the original concept of alexithymia. While such a positive correlation is consistent with the concept that individuals who are more distressed have difficulty regulating their emotions, it clearly conflicts with the original description of alexithymia as a severe affective deficit including a deficit in the description of affective distress. Moreover, the TAS-20 cannot disentangle the actual problem of not experiencing emotions from perceiving oneself as having difficulty describing emotions despite intact experience. This is illustrated by a recent carefully-performed study in which raters who were blind to patient TAS-20 scores examined videotaped interviews of psychiatric inpatients and coded for the number of emotions expressed by the patients. The counter-intuitive finding was that subjects in this study who rated themselves as having more difficulty describing their emotions were actually better able to express their emotions than others, not worse (Leising et al., 2009). A more recent report corroborates the finding in another sample of patients with psychiatric disorders, demonstrating that the TAS-20 appears to be a measure of psychological distress (Marchesi et al., 2014). These finding are inconsistent with the hypothesis that higher scores on the TAS-20 correspond to a deficit in emotional experience and expression, and demonstrate the challenges of relying on the accuracy of self-assessments (Dunning et al., 2004).

A third issue is that most studies using the TAS-20 involve healthy individuals, as it is assumed that findings in people who

have no impairment in affective processing will usefully inform us about those who do. A key assumption here is that the emotional experience of alexithymic individuals is not fundamentally different from that of non-alexithymic individuals. This is consistent with the theory guiding the creation of the TAS-20, which is that alexithymia is a problem in the verbal description (readout or report) of experience (Taylor et al., 1997), analogous to an anomia, that reflects a continuum in the population. It is also noted that the clinical condition of alexithymia involves a failure of mental representation of emotion (Taylor and Bagby, 2013; Taylor, 2010; Taylor et al., 1997) which involves an impairment in the experience of emotional feelings, or in accessing and making use of their conceptual meaning, as in an affective agnosia. However, the use of a self-report measure in healthy individuals that correlates with reported distress means that the empirical study of alexithymia has become somewhat disengaged from the original clinical phenomenon that prompted its description.

A fourth issue is that research to date has provided only very limited evidence for an association between alexithymia as measured by the TAS-20 and medical outcomes. A recent review (Kojima, 2012) revealed that no prospective studies of TAS-20 as a predictor of medical outcome have been published. An earlier version of the TAS (Taylor et al., 1985) was used in a Finnish study (Kauhanen et al., 1996) that demonstrated an association with all-cause mortality, particularly violent death. As such, the study does not disentangle whether TAS captured a problem in "actions" (behavior) or "organs" (pathophysiology), to paraphrase Ruesch (1948). The absence of any published findings to date in which the TAS-20 is a predictor of medical outcomes could either indicate that the original observations or conclusions leading to the formulation of the alexithymia construct were wrong, or that some limitation in the TAS-20 is leading to a failure to detect an association with adverse medical outcome that actually exists.

In summary, although alexithymia was originally described as a condition or category involving a severe affective experiential deficit, the use of a self-report method to measure alexithymia has resulted in a body of research that has focused on a so-called continuum of alexithymia (Taylor et al., 1997), with an emphasis on its less severe end. This bias may also have occurred in part because individuals who are more severely alexithymic tend to avoid emotions and most likely would tend not to volunteer for research on emotion. As the evidence grows that depression (Cuijpers and Smit, 2002) and anxiety (Eaker et al., 2005) contribute to adverse outcomes including mortality in medical disorders, there is an increasing need for a measure of impaired emotional processing that is not confounded by self-reported distress. Such a measure would permit a re-examination of the core hypothesis that alexithymia, or a related concept like affective agnosia, is associated with poor medical outcome. The Levels of Emotional Awareness Scale (LEAS) (Lane et al., 1990) is a possible candidate.

4. The levels of emotional awareness construct

Nemiah and Sifneos (1970), Nemiah et al. (1976) and Ruesch (1948) before them, hypothesized that alexithymia arose from an arrest in development. The theory of "levels of emotional awareness" published in 1987 sought to describe the developmental line in question. Lane and Schwartz (1987) proposed that an individual's ability to recognize and describe emotion in oneself and others, called emotional awareness, is a cognitive skill that undergoes a developmental process similar to that which Piaget (1937) described for cognition in general. A fundamental tenet of this model is that individual differences in emotional awareness reflect variations in the degree of differentiation and integration of the schemata (implicit programs or sets of rules) used to process emotional information, whether that information comes from the external world or the internal world through introspection. Emotional awareness is considered to be a separate line of cognitive development that may proceed somewhat independently from other domains of cognition (Lane and Pollerman, 2002; Lane and Schwartz, 1987).

The model posits five "levels of emotional awareness" that share the structural characteristics of Piaget's stages of cognitive development (Piaget, 1937). The levels of emotional awareness in ascending order are awareness of physical sensations, action tendencies, single emotions, blends of emotions, and blends of blends of emotional experience (the capacity to appreciate complexity in the experiences of self and other). Level 1, somatic sensations, and Level 2, action tendencies, constitute sensorimotor expressions of emotion associated with visceromotor and somatomotor activity, respectively, whereas Levels 3 through 5 constitute progressively increasing degrees of differentiated feeling states. The foundational distinction in cognitive neuroscience between implicit (levels 1 and 2) and explicit (levels 3–5) functions maps onto this framework (Lane, 2000).

The five levels, therefore, describe the cognitive organization or complexity of emotional experience as manifested in the description of one's experiences (not one's thoughts about or appraisal of one's emotions). The levels are hierarchically related such that functioning at each level adds to and modifies the function of previous levels but does not eliminate them. For example, blends of emotion (Level 4 experiences), compared to action tendencies (Level 2 experiences), are expected to be associated with more differentiated representations of somatic sensations (Level 1). The feelings associated with a given emotional response can be thought of as a construction consisting of each of the levels of awareness up to and including the highest level attained. The trait level of function is the level at which a given individual typically functions.

Modern conceptions of cognitive development have refined Piaget's views but are still consistent with the model proposed here, such that development of knowledge proceeds through a process called "representational redescription" (Karmiloff-Smith, 1992). Cognitive development from this perspective consists of the transformation of knowledge from implicit (procedural, sensorimotor) patterns to explicit (conscious thought) representations, in part through use of language or other semiotic modes. Language facilitates one's ability to form, and make use of, more abstract and fine-grained concepts, and this allows the thoughts one has available to make sense of experience to be more flexible, adaptable and creative. The concepts one possesses can also feedback to alter one's perception of one's own emotional responses. This viewpoint is consistent with the theory that the way language is used to describe emotion is not simply a readout of one's mental state, but that it can also indirectly (via facilitation of concept acquisition) modify what one knows about emotion and how emotion is experienced consciously (Werner and Kaplan, 1963).

5. Research using the Levels of Emotional Awareness Scale

The LEAS is a written performance measure that asks a person to describe his or her anticipated feelings and those of another person in each of twenty vignettes described in two to four sentences (Lane et al., 1990). Scoring is based on specific structural criteria aimed at determining the degree of differentiation in the use of emotion words (the degree of specificity in the terms used and the range of emotions described) and the differentiation of self from other. The scoring involves little or no inference by raters. Because the scoring system evaluates the structure of experience and not its content, individuals cannot easily enhance their scores or create a socially desirable impression in their responses, as is the case with some self-report instruments. A glossary of words at each level was created to guide scoring and an on-line version for administration

and scoring of the LEAS is now available (Barchard et al., 2010) (www.eleastest.net).

The LEAS has consistently been shown to have high inter-rater reliability and internal consistency (Lane et al., 1998). The test-retest reliability at two and four weeks has been shown to be quite good. Norms for age, sex and socioeconomic status have been established.

A variety of studies (but not all, see Lumley et al. (2005); Waller and Scheidt, 2004) support the construct validity of the LEAS. The LEAS correlates moderately positively with two cognitive-developmental measures, the Sentence Completion Test of Ego Development (Loevinger and Wessler, 1970; Loevinger et al., 1970) and the cognitive complexity of the description of parents (Blatt et al., 1979). These results support the claim that the LEAS is measuring a cognitive-developmental continuum and that the LEAS is not identical to these other measures. Greater emotional awareness is associated with greater self-reported impulse control (Bréjard et al., 2012), consistent with the theory that functioning at higher levels of emotional awareness (Levels 3–5) modulates function at lower levels (actions and action tendencies at Level 2) (Lane et al., 2000a). Greater emotional awareness is also associated with greater openness to feelings (Lane et al., 1990) and greater emotion recognition ability (Lane et al., 2000b, 1996). The LEAS correlates positively with empathy ability, certain subtests of a standard measure of emotional intelligence (Barchard and Hakstian, 2004), the tendency to seek help for emotional problems and the actual amount of social support that a person has. Additionally, individuals with lower LEAS scores rated their general sense of well-being lower during experimental induction of sad mood compared to baseline ratings whereas those with higher LEAS scores had a stable sense of general well-being independent of their momentary mood (Ciarrochi et al., 2003). The latter finding highlights that the LEAS captures the ability to establish and recall mental representations of one's own typical emotional state independent of the immediate circumstances or state. Importantly, the LEAS and TAS-20 are negatively correlated but tend to show only a small degree of overlap (e.g. correlations around −0.20 [4 percent of shared variance]) (Lane et al., 1996).

Clinically, it has been shown that patients with borderline personality disorder score lower on the LEAS than age-matched control subjects (Levine et al., 1997) and that individuals with the "disorganized attachment style" have lower LEAS scores than those with the "organized attachment style" (Subic-Wrana et al., 2007). Lower scores on the LEAS are associated with greater pain in patients with irritable bowel syndrome (IBS) (Lackner, 2005). Patients on a psychosomatic inpatient ward with somatoform disorders had lower LEAS scores than patients with depressive disorders. Somatoform patients in this study showed significant increases in LEAS scores after three months of multi-modal inpatient treatment that integrated body-based techniques with intensive group and individual psychotherapy (Subic-Wrana et al., 2005). This multimodal treatment is elaborated upon below. These findings are consistent with a developmental model of emotional awareness in which lower emotional awareness is associated with a tendency to experience emotional distress only as bodily symptoms and in which emotional awareness can improve with therapeutic interventions that facilitate the transition from implicit to explicit processing.

Variations in emotional awareness help illuminate clinically relevant developmental differences in medical and psychiatric conditions. Patients with essential hypertension had lower LEAS scores than those with hypertension secondary to other medical conditions such as renal disease (Consoli et al., 2010). Patients with eating disorders (anorexia and bulimia) were observed to have lower LEAS scores than matched controls (Bydlowski et al., 2005), consistent with Hilde Bruch's classic observation that eating disorders are associated with an impairment in interoceptive awareness of one's own emotions (Bruch, 1973). Patients with PTSD have lower LEAS scores than matched controls, and LEAS scores were inversely correlated with the severity of PTSD symptoms, particularly symptoms involving dissociation (Frewen et al., 2008). Patients with schizophrenia had lower scores on the LEAS than matched controls on those LEAS items depicting more complex social scenarios, suggesting that emotional awareness declines in these patients when resources are taxed (Baslet et al., 2009).

Individuals with generalized anxiety disorder have higher LEAS scores than matched controls (Novick-Kline et al., 2005), indicating emotional awareness can be a double-edged sword. In contrast, patients with depression were found to have decreased awareness of the emotions of others (Berthoz et al., 2000; Donges et al., 2005), consistent with the pathological introspective focus that can occur with depression. Thus, measurement of emotional awareness with the LEAS can be used to understand developmental or state differences for which targeted interventions for medical and psychiatric disorders can be developed.

The LEAS typically does not correlate with self-reported negative affect (such as anxiety or depression) either in the presence or absence of anxiety or depressive disorders (Stonnington et al., 2013), unlike the TAS-20, which typically does (Lumley et al., 2005). In three separate studies, including studies of essential hypertension (Consoli et al., 2010), eating disorders (Bydlowski et al., 2005) and somatoform disorders (Subic-Wrana et al., 2005), it has been shown that associations between lower emotional awareness and the clinical condition were not altered by removing variance due to self-reported negative affect, whereas control for self-reported negative affect rendered associations with the TAS-20 non-significant. Lack of correlation of the LEAS with negative affect means that distress does not bias the measurement of the personality attribute in question.

New findings indicate that higher LEAS scores at baseline in patients with panic disorder predict greater symptom reduction after either cognitive-behavioral or manualized psychodynamic psychotherapeutic treatment (Beutel et al., 2013), which requires that patients become aware of what they are feeling. No other studies of this kind have been completed with the LEAS, so it is possible that emotional awareness as measured by the LEAS is a predictor of better psychotherapeutic outcome in a variety of contexts.

Another recent finding in patients with somatoform disorders and medical controls is that, after controlling for self-reported positive and negative affect, the LEAS correlated positively with several measures of "theory of mind" whereas the TAS-20 did not (Lane et al., 2015). This observation strengthens the argument that the LEAS, in contrast to the TAS-20, captures the ability to mentally represent emotional states and could help to explain its predictive value in psychotherapy.

6. Freud's concept of agnosia and its application to alexithymia

Agnosia is a relatively rare neuropsychological symptom defined in the classical literature as a failure of recognition that cannot be attributed to elementary sensory defects, mental deterioration, attentional disturbances, aphasic misnaming, or unfamiliarity with external stimuli (Frederiks, 1969). The essence of agnosia is intact perception of a stimulus while not knowing or recognizing its meaning. Freud hypothesized that recognition of objects required a sequence of processing steps including sensory perception, linking the perception to a mental representation of an object, and linking that mental representation to a verbal label or name (see Fig. 1) (Freud, 1953). The condition of asymbolia or

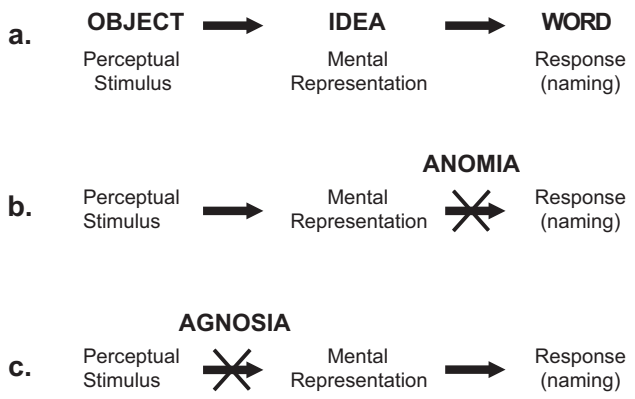

Fig. 1. Freud's concept of agnosia. (a) Freud proposed that the ability to name an object after perceiving it was mediated by a mental representation of that object. (b) If the mental representation of the object was intact but an impairment in naming was observed, that constituted an anomia or asymbolia. Under such circumstances, individuals could recognize the object and use information about it due to intact mental representation but could not name it. (c) If a mental representation deficit was present, naming would also be impaired. In addition, the affected individual would not be able to recognize or use information about the perceived object, thus constituting a more severe deficit called an agnosia. X – indicates where the transmission of information is blocked.

(equivalently) anomia consisted of interference with the final step of naming, but in that case the perception and understanding of the object were intact. Thus, in anomia, recognition was intact but the verbal label was missing. In agnosia, however, the sensory perception is not linked to the mental representation so that one can see or hear the object (perception is intact) but not be able to recognize it. As part of this recognition impairment, a failure of naming necessarily occurs. If one simply observed a failure in naming one could not distinguish between an anomia and agnosia.

Applied to emotion, the equivalent of the object is the visceromotor and somatomotor expression of the emotional response. These visceral/somatic emotional reactions, which are generated by the brain, then need to be perceived by the brain, and the emotional meaning of these perceived reactions further needs to be recognized/represented.

The concept of affective agnosia may be further refined by an important distinction in the agnosia literature. Lissauer (Lissauer and Jackson, 1988) described two clinical forms of impaired object recognition: "apperceptive" and "associative" agnosia. A central idea in Lissauer's work is that object recognition depends both on the integrity of early perceptual processes and a "gnostic" stage in which the visual impressions are combined in such a way that an internal conceptual representation is also activated. Only after the latter stage can conscious recognition occur. Apperception referred to the conscious perceptual experience of a sensory impression. Patients with apperceptive agnosia are thought to have intact basic visual perception but they are not able to consciously perceive the object, and thus fail to copy the object or match it to other related objects. The inability to copy or match raises the possibility that basic perception is not intact and its inclusion among agnosias has been questioned (Mesulam, 1985). Importantly, one variant of apperceptive agnosia involves an integrative deficit (Gazzaniga et al., 2014, Ch. 6), such that patients may visually experience the individual parts of an object but not experience the object itself as a discrete whole. In contrast, by associative agnosia Lissauer meant the inability to link an adequate percept to stored knowledge of it. Associative agnosia is characterized by the ability to copy the object or match it to similar objects (perceptual experience is intact) but affected individuals cannot identify the object and are unable to demonstrate knowledge about the meaning, purpose or use of the object. As we discuss below, these distinctions may be applied to emotional content and different subtypes of affective agnosia (*Footnote*: Lissauer used the term "Seelenblindheit" which means "mind blindness." Freud coined the term "agnosia" which was applied to subsequent translations of Lissauer's work).

A more contemporary and detailed neurocomputational model of these perceptual and recognitional capacities has been put forth by Marr (1982). Focusing on vision, Marr proposed that perceptual processing involves three hierarchically organized stages which can be classified as low-, intermediate-, and high-level stages of representation. Low-level representation is comprised of unconscious processes which detect discrete features (such as the representation of corners and edges in the case of vision). Intermediate-level representation includes processes which integrate low-level features in order to detect coherent patterns from a given viewpoint (such as the contours making up the shape of a cube from a specific vantage point). Jackendoff (1987), Crick and Koch (2003, 2000), Koch (2004) and Prinz (2012) have since extended this model in light of more recent work in cognitive science, and argued that this intermediate level of representation is the level which corresponds most to phenomenal experience, and therefore perception. Finally, the high-level of processing involves representations which are viewpoint invariant (such as a state which is active when viewing a cube from any angle). It is this high-level which is thought to allow for categorization and conceptual recognition/representation. If the high-level malfunctions (as in visual agnosia), one would be able to perceive a cube from a specific viewpoint and draw it (because intermediate-level representations are intact), but one would not be capable of seeing it *as a cube*, labeling it as such, or using the knowledge that it is a cube to plan to respond to it appropriately.

Extending the Marr model to emotion involves considering these three hierarchical levels of representation with respect to somatic/visceral emotional reactions (Damasio, 1994; Prinz, 2006). The low-level may here involve unconscious detection of activity patterns within individual organs or skeletal muscles (such as the heart or intestines, or aspects of one's bodily posture or facial expression), whereas the intermediate-level would involve the consciously experienced patterns of one's entire bodily state across organs, muscles, and so forth. Importantly the intermediate level would involve bodily expression of emotion (e.g. such as facial expression and posture) that would enable an outside observer to conclude that the subject was manifesting an emotional response. Thus, the intermediate-level, in contrast to the low-level, constitutes an intact, potentially identifiable emotional response. The high-level would then involve processes which categorize a range of somatic/visceral reactions as having the same emotional meaning (such as the states preparatory to freezing and fleeing both being categorized under the concept of fear). Just as in associative visual agnosia, if the high-level of body state representation malfunctions then one will still experience and respond to bodily states, and other people will recognize them as expressions of emotion, but one will not experience them *as emotions*, be able to label them as such, or be able to use knowledge of their emotional meaning to plan to respond to them appropriately.

In the case of emotion, however, the knowledge that one's perceived somatic/visceral reaction has a certain emotional meaning may result in even larger changes in phenomenology compared with vision. The cognitive realization that one is sad, for example, can have significant further effects on one's emotional experience, perhaps via feedback processes between emotion concept representations and interoceptive/somatic perceptual systems themselves. These feedback processes may well influence the nature of the somatic/visceral state to be perceived. Similar top-down "pattern completion" processes, in which higher-level conceptual representations alter lower-level perceptual representations, have been proposed within language processing, for example (McClelland and Rumelhart, 1981).

Finally, it appears that intermediate- and high-level representations can be either conscious or unconscious depending on the context. Experiments which use subliminal perception and backward masking, and which illustrate semantic priming effects, are both examples of cases in which such representations appear to be activated but not consciously perceived. A large body of empirical and theoretical work suggests that these representations become conscious (and affect self-reported phenomenal experience) only under conditions in which there is a top-down modulatory effect on information flow such that these representations become available to working memory and related cognitive systems, which in turn allows them to be used to guide flexible, goal-driven decision-making and behavior (Baars, 2005, 2002; Dehaene, 2014; Prinz, 2012). This empirically tractable, use-related notion of consciousness is often termed "access-consciousness."

An implication of this framework is that an affective agnosia could actually arise in three ways. First, an apperceptive affective agnosia would involve a deficit in moving from low-level representations of discrete elements of one's somatic/visceral reactions to intermediate-level representations of coherent patterns across elements of such reactions. Such an individual, for example, might have discrete somatic complaints such as stomach pain and muscle tension, but not perceive these complaints as related (i.e. parts of a coherent bodily reaction). Second, as discussed above, there could be a deficit in moving from intermediate-level representations of one's somatic/visceral emotional reactions to high-level conceptual representations of their emotional meaning. Third, however, one might accurately represent emotional meaning (i.e. activate high-level representations appropriately), but such representations could remain unconscious. This could happen if such representations were not modulated via top-down signals in the right way to make them accessible to working memory and related cognitive systems associated with flexible, goal-directed behavioral control. In this latter case one might still observe semantic priming effects associated with emotional meaning, but one would still not verbally report awareness of the emotional nature of one's bodily reactions. This bottom-up "failure to connect" representations of emotional meaning to working memory may be one example of what Janet referred to as dissociation (Van der Kolk and Van der Hart, 1989) as opposed to an active process of inhibition corresponding to the defensive processes described by Freud (Taylor and Bagby, 2013).

This neurocomputational framework can also offer an account of affective anomia that is entirely consistent with Freud's original conception of the distinction between agnosia and anomia. An affective anomia would involve appropriate activation of high-level representations of emotional meaning, and appropriate conscious access to that emotional meaning, but an inability to map emotion concept representations to linguistic representations of appropriate terms within language production systems.

The distinction between anomia and agnosia is one that, to our knowledge, has not been made or previously considered in relation to alexithymia or affect. In 1971 (just one year before alexithymia was described), Nauta in his discussion of frontal lobe processes, discussed "interoceptive agnosia" (Nauta, 1971). He defined this as an impairment of the individual's ability to integrate certain information from his internal milieu with external information processing. Although interoceptive agnosia appears quite related to what we are describing as "affective agnosia," Nauta was aiming to define what is impaired in frontal lobe lesions generally and did not specifically discuss affective responses or their mental representation.

It is our contention that the phenomenon that Sifneos and Nemiah described involved a severe deficit in recognition, mental representation or knowing one's own emotional responses or states – an affective agnosia. This would explain why the patients appeared to have a lack of awareness or inability to experience emotion – they were not aware of their emotions and did not know how they felt. Naturally these patients had difficulty describing their emotions. If they had been told by others that they had this problem they might indicate on a self-report inventory such as the TAS-20 that they had difficulty describing how they felt. But such a report on an inventory might be indistinguishable from someone who *did* experience a range of emotions but felt that they had difficulty describing how they felt, as in the two studies of psychiatric inpatients described above (Leising et al., 2009; Marchesi et al., 2014). The key point is that the concept of agnosia highlights the importance of mental representation of the emotional meaning of one's perceived interoceptive/somatic emotional reactions states as a prerequisite for both understanding one's experience and naming it – a phenomenon that opens up a whole new range of opportunities for both clinical intervention and research. The link to a developmental model is critical because it means that when emotion is not mentally represented appropriately, the emotional response is simply understood as a bodily response, and is not perceived as a differentiated feeling of the type that would be required for use in planning adaptive responses to emotion-eliciting situations or related considerations pertaining to specific experiences in the past.

7. Caveats regarding the concept of affective agnosia

There are at least three ways in which affective agnosia differs from other agnosias. The first, and perhaps most obvious, is that in the case of emotion there is currently no practical objective means of fully demonstrating what a subject knows or does not know about their own emotional reactions. The current limitations in objectively measuring and verifying a subject's actual somatic/visceral reactions, as one can with a concrete object in visual perception, does not make scientific investigation of such reactions intractable, however. Further, any difficulties in the measurement and verification of a subject's *consciously perceived* emotional state also applies to states of consciousness more generally, but that does not preclude them from being studied scientifically (Searle, 1998) either, nor does it prevent them from being an important component of clinical evaluation or the primary focus of clinical intervention in mental health contexts.

A second way in which affective agnosia differs from traditional agnosias is in the relationship between the perceiving or knowing apparatus and that which is perceived or known. In traditional agnosias the object to be perceived exists in the external world and the perceptual apparatus is in the person's brain. In the case of affective agnosia, that which can potentially be perceived is the predominantly subcortically-generated emotional response, and the perceiving apparatus is instantiated in paralimbic and cortical structures in interaction with subcortical structures. Thus, in affective agnosia the perceiver and the perceived interact so that what becomes known changes by virtue of the process of mental representation. As we discuss below, recruitment of the medial prefrontal cortex in the act of mentally representing the emotional meaning of one's unconsciously generated bodily reactions, and having conscious access to such representations, both may have top-down modulatory effects on the neural hierarchy mediating basic emotional responses such that the experience and physiology of emotion could (and likely does) become more differentiated in the process of coming to know what one is feeling. This variable nature or "moving target" aspect of an emotional state that becomes known by virtue of it being mentally represented would typically disqualify it from consideration for inclusion within the clinical neurosciences. However, mentalization is of fundamental importance in the successful social and self-regulatory functioning

of human beings (Allen, 2013). As alluded to immediately above, the attribution of a mental state to a person based on his or her behavior is an inference that cannot be verified or proven. Yet, there is work which suggests that our awareness of our own conscious mental states, our capacity for self-reflection and our ability to know what others are thinking and feeling may all derive from this capacity for mentalization, which in its advanced form (that transcends what can be directly observed by an external observer) may be a uniquely human attribute (Steklis and Lane, 2013). Moreover, the capacity for sharing mental states is the basis for collaboration, without which we would not have science, culture or indeed civilization itself (Tomasello et al., 2005).

A third way in which affective agnosia differs from other agnosias is that it is typically attributable to a failure in development (Lane and Garfield, 2005; Subic-Wrana et al., 2011) as opposed to an acquired brain lesion (although the latter etiology is possible; for further discussion of this point see below). Affective agnosia, therefore, constitutes the extreme end of a developmental continuum. It is notable that the development of this cognitive skill depends upon the mentalization skills or emotional awareness of caretakers during a child's development (Allen, 2013; Gergely and Watson, 1996). If a child does not have adequate recognition and mirroring of its own mental and emotional states during development, that child will not be able to know how he or she feels, a condition that we are calling "affective agnosia."

8. The "Blindsight" neural model of alexithymia

Conceptualizing alexithymia "as a lack of words for emotion" rather naturally led to a neural model that posited mediation by a lack of transfer of emotional information from the right hemisphere, presumed to mediate emotion, to the left hemisphere, known to be dominant for language in almost all right-handers and most non-right-handers. This "functional commissurotomy" model of alexithymia has received some empirical support (Tabibnia and Zaidel, 2005). However, a disadvantage of this model is that it does not explain the physiological mechanisms by which the deficits associated with alexithymia might contribute to adverse physical disease outcome. Another possible disadvantage is that it would not explain why emotional information that is itself represented within the left hemisphere cannot be accessed by language production systems.

In 1997 one of us published a paper called "Is alexithymia the emotional equivalent of blindsight?" (Lane et al., 1997a). Blindsight is a phenomenon resulting from lesions in area V1 of the primary visual cortex (Cowey and Stoerig, 1991; Cowey, 2010; Weiskrantz, 1986) in which patients claim to be blind, but respond with high accuracy on visual tracking and other select visual tasks. Thus, these patients behave as if they can see even though they are blind. Similar phenomena have been noted in other sensory modalities, such as touch (Paillard et al., 1983) and olfaction (Schwartz et al., 1994). These cases have in common a lesion in or near the primary sensory cortex specialized to process that modality along with accurate behavioral responses suggesting that some aspects of sensory perception remain intact. Based on the previous discussion of the three-level Marr model, this could be explained by saying that a subset of low-level visual representations, but not those at the intermediate- and high-level, were functioning properly in blindsight patients.

Drawing upon evidence that individuals who were more emotionally aware activated the dorsal anterior cingulate cortex (ACC) during emotional arousal to a greater degree than those who were less aware (Lane et al., 1998), it was proposed that the neural basis of alexithymia might consist of a disconnection syndrome in which subcortical emotion-generating mechanisms do not communicate adequately with cortical mechanisms including the ACC involved in explicit processing (Lane et al., 1997a, 1997b). This paper called attention to the distinction between implicit and explicit cognitive processes, suggested that this same distinction applied to emotion, and proposed that analogous to blindsight, alexithymic individuals manifest implicit expressions of emotion but exhibit little or no awareness of the emotions that have been activated. The absence of conscious processing of affect was proposed to arise due to disuse during early stages of development when such connections are still forming (Munakata and Pfaffly, 2004). Consistent with Ruesch's (1948) proposal that psychosomatic disorders result at least in part from neglectful parenting or childhood trauma, disuse of such connections could result when parental figures fail to engage the child in forming such connections or when the child avoids acknowledging, representing and processing arousal that is aversive and overwhelming.

Since then, a variety of studies have found evidence that alexithymia is associated with altered ACC activity (Heinzel et al., 2010; Kano and Fukudo, 2013; Moriguchi and Komaki, 2013) and structure (Grabe et al., 2014; Gündel et al., 2004). However, the neuroimaging findings using the TAS-20 have been inconsistent, sometimes showing positive associations between TAS-20 and ACC structure and function and sometimes showing significant negative associations (Van der Velde et al., 2013). In a neuroimaging context, the distinction between emotion that is consciously experienced and reportable (explicit) vs. not consciously experienced and therefore not reportable (implicit) is quite important, and, as noted above, the TAS-20 fails to distinguish between these two. Given that the TAS-20 correlates strongly and positively with self-reported negative affect (Lumley, 2000), a positive correlation between TAS-20 and ACC activity would be expected, potentially accounting for this observation in a recent meta-analysis (Van der Velde et al., 2013). This failure to distinguish between conscious and unconscious emotion reinforces the need for a method of detecting an emotion processing deficit not confounded by reported distress.

In a similar vein, Ricciardi et al. (2015) showed that alexithymia scores on the TAS-20 are often elevated in patients with neurological diseases. Many studies showed covariation of alexithymia with affective disturbance such as depression and anxiety. However, this association with affective disturbance was not always present, suggesting that perhaps in some patients there is some impairment in emotional functioning other than depression or anxiety that is due to the neurological abnormality in question. These findings raise the possibility that impairments in emotional processing, potentially consistent with affective agnosia, could result from neurological lesions later in life. Our neural model suggests that this could be the case if the neurological lesions affect specific subregions of the medial prefrontal cortex involved in the representation and conscious processing of affect (or perhaps also functionally connected basal ganglia structures; Alexander et al., 1986; Postuma and Dagher, 2006). For example, Hornak et al. (2003) observed a blunting of subjective emotional experience in patients who had lesions involving the ventral ACC and Brodmann area 9 of the medial prefrontal cortex.

Since that "blindsight" paper (Lane et al., 1997a) was published there has also been a burgeoning of information about the ACC and the frontal lobe in two important domains. First, it has become increasingly clear that the ACC and medial prefrontal cortex are involved in representing and regulating emotion. Reviews by Amodio and Frith (2006), and Lee and Siegle (2009), and more recent empirical work (Waugh et al., 2014), demonstrate that different parts of the ACC and medial prefrontal cortex are involved in different aspects of conscious processing, attentional mechanisms, working memory maintenance, and motor regulation related to emotion. More generally, somatic/visceral emotional responses

(that are predominantly subcortically-generated) appear to be processed perceptually within insular and parietal regions (Craig, 2009, 2002; Gazzola et al., 2012), followed by the activation of representations of the emotional meaning of those responses within the rostral ACC and surrounding regions of the medial frontal lobe (Kalisch et al., 2006; Peelen et al., 2010; Smith et al., 2014). Second, studies show that these same medial prefrontal and paralimbic (ACC) structures have monosynaptic connections to the hypothalamus, periacqueductal gray and other brainstem structures involved in visceral regulation (Price, 1999), and that the medial prefrontal cortex and ACC have a top-down modulatory influence on visceral functions, including autonomic, neuroendocrine and immune variables in a variety of disease contexts (Lane and Wager, 2009). These two aspects of medial frontal lobe function have been integrated in what is known as the "neurovisceral integration model" (Thayer and Lane, 2009, 2000). A corollary of this model is that failure to establish high-level representations of one's emotional state (associated with lack of engagement of the medial prefrontal cortex, for example), could well result in the simultaneous inability to know what one is feeling and visceral dysregulation resulting in adverse physical disease outcomes.

The "blindsight" model of alexithymia, therefore, proposed a shift away from the original definition of alexithymia involving a lack of words for emotions and its parallel neural substrate involving an impairment in the communication between the right and left hemispheres (the functional commisurotomy model). An advantage of the "blindsight" model, which focused on the vertical rather than horizontal axis in the brain, is that it inherently addressed the physiological dysregulation (resulting from the lack of top-down modulation discussed above) that may link alexithymia and physical disease (Lane et al., 1997a). However, although this new model suggested that "alexithymia" was not the most appropriate name for the condition, a more appropriate name was not specified in that paper. We turn next to consider why "affective agnosia" might be the most proper label for this condition.

9. Neural basis of emotional awareness

Although much has been written about the neural basis of emotion within the neuroimaging and animal neuroscience literature, those aspects of emotion involving conscious experience and cognitive accessibility have received considerably less attention. The current model does not attempt to address basic aspects of consciousness independent of emotion, or the process of emotion generation itself. The rudimentary elements of our model and its schematic mapping onto the brain is presented in Fig. 2.

A natural starting point for understanding the instantiation of conscious emotional experience in the brain is the ventromedial prefrontal cortex (VMPFC), an area Damasio highlighted in his landmark book *Descartes' Error* (Damasio, 1994). In that book Damasio made the bold but now well-validated claim that feelings are not mere epiphenomena of emotion with no real meaning or evolutionary importance, but rather have a specific neuroanatomical basis that differentiate this conscious experiential state from the generation of basic emotional responses. Williams and colleagues (Williams et al., 2006) showed that fearful faces that were presented under experimental conditions such that they could not be consciously perceived nevertheless activated the VMPFC. One can think of this area as participating in the ongoing evaluation of emotional significance of stimuli in the environment in communication with cortical structures such as the insula and subcortical structures such as the amygdala, and generating representations of the emotional meaning of one's situation (Roy et al., 2012) that are potentially accessible to conscious processing (but may only be reportable if attended to or reflected upon) (Lane et al., 2013).

Damasio suggested that this brain area is responsible for registering a "somatic marker" or the gut feelings that occur when emotions have been activated but have not yet reached full conscious awareness (Damasio, 1994). This area of the frontal lobe is part of the medial visceromotor network that has direct monosynaptic connections to brainstem areas such as the periaqueductal gray and the lateral hypothalamus that participate in orchestrating autonomic and neuroendocrine responses generally, including in emotional contexts (Price, 1999). The top-down and bottom-up traffic in this network fits well with its role in registering somatic markers. This area is a critical hub in the medial visceromotor-brainstem axis that increasingly is being implicated as the principal neural basis of mind-brain-body interactions (Lane and Wager, 2009).

The complex pathways through the brainstem and the mechanisms of linkage with autonomic and neuroendocrine outflow that interact with bodily organs have been articulated elsewhere (Lane and Wager, 2009; Lane et al., 2009a, 2009b). The principal source of visceral afferent information to the brain from the body is the vagus nerve, which itself is predominantly afferent (Cameron, 2002). This afferent information is processed through a complex subcortical network that culminates in the anterior insula (Craig, 2009, 2002), as depicted in Fig. 2. The insula is a predominantly sensory structure that registers and remaps bodily information and sensations into conscious somatic sensations, including the bodily component of emotions. This corresponds to the intermediate level of processing within the Marr framework discussed above. The insula transfers this information to a variety of other structures including the various sectors of the anterior cingulate cortex (ACC) (Deen et al., 2011). The ACC is a predominantly motor structure that coordinates interoceptive and exteroceptive information and regulates autonomic and somatomotor activity in anticipation of the behavioral response needed to respond to the eliciting stimulus (Medford and Critchley, 2010).

Fig. 2 highlights structures in the medial frontal lobe that participate in different aspects of conscious emotional processing. The dorsal anterior cingulate cortex (dACC, or anterior middle cingulate cortex, according to Vogt, 2009) plays an important role in allowing perceived bodily/emotional states to motivate the selection of specific motor responses (Critchley, 2005; Medford and Critchley, 2010), and is also involved in autonomic regulation (Critchley et al., 2003) and attention to one's bodily states (Farb et al., 2013). Two different functional neuroimaging studies have demonstrated that higher LEAS scores are associated with greater activity in this region during the activation of emotion (Lane et al., 1998; McRae et al., 2008). This finding suggests that when emotional responses are triggered (and subsequently processed in interoceptive/somatosensory cortical regions), dACC may have greater attentionally-mediated access to such responses in people who are more emotionally aware. The rostral or pregenual anterior cingulate cortex (rACC) appears to specialize in the representation of emotional meaning, particularly meaning that is concept-driven, by integrating highly processed interoceptive and exteroceptive information (Kalisch et al., 2006; Peelen et al., 2010; Roy et al., 2012; Smith et al., 2014). This suggests that the rACC may instantiate the high-level within the Marr framework discussed above. An fMRI study showed that during recall of life-threatening experiences healthy subjects who did not have PTSD showed greater activity in the rACC as a function of greater scores on the LEAS (Frewen et al., 2008). The rACC, rather than the dACC, may have been more engaged in the latter study as a function of emotional awareness because of the complexity of the narrative and the integration of stored information from which emotional meaning was constructed. The dorsomedial prefrontal cortex (DMPFC) also appears to play an important role in holding mental representations of one's own thoughts and feelings (and those of others) actively in

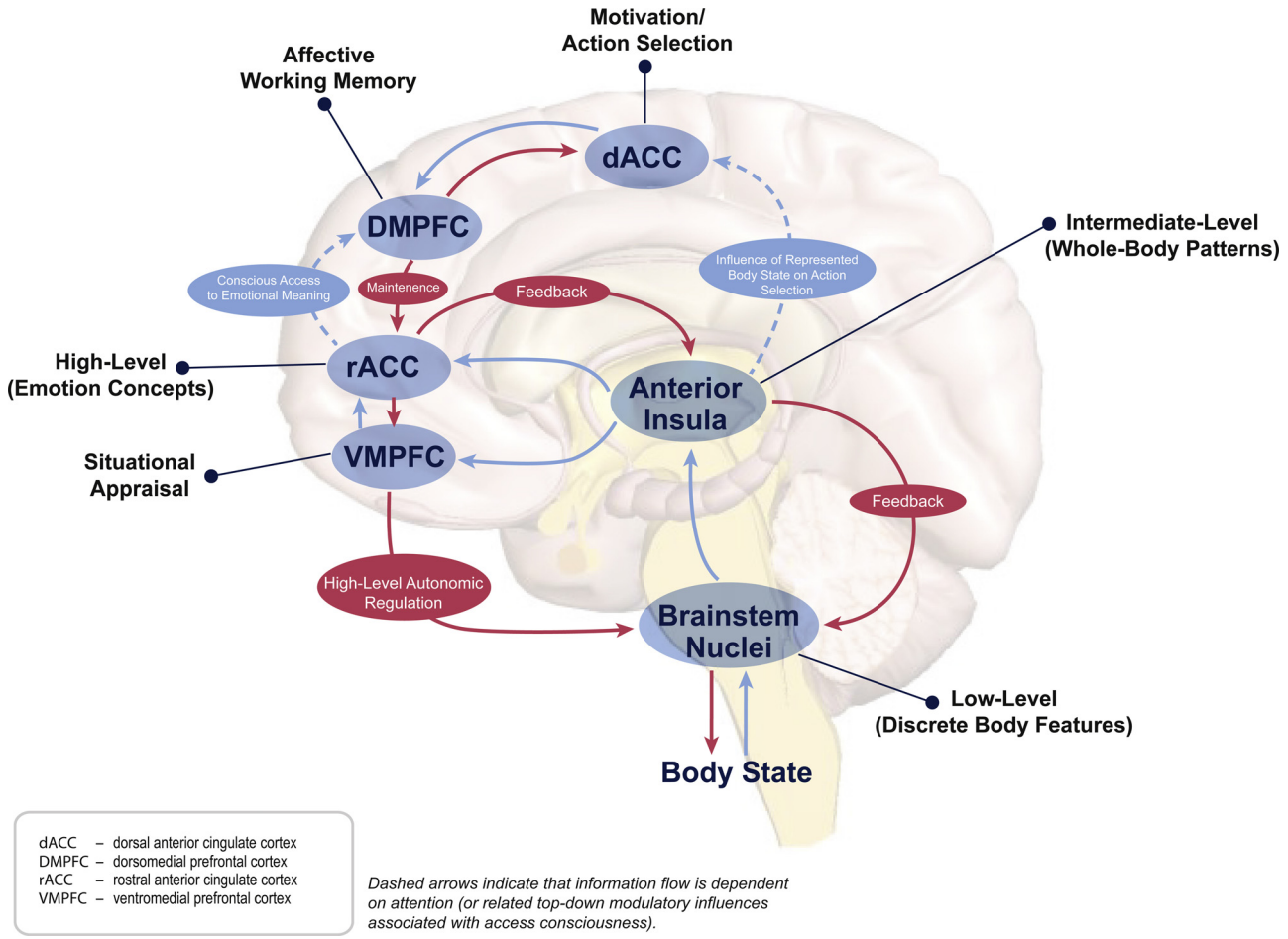

Fig. 2. Schematic diagram of the neural basis of emotional awareness. The principal loci participating in the hierarchical processing of bodily state information in the context of emotional arousal are displayed: Low level (discrete bodily features): brainstem nuclei; Intermediate level (whole body patterns): anterior insula; and High level (emotion concepts): rACC. VMPFC participates in situational appraisal and high level autonomic regulation. DMPFC participates in affective working memory, and the maintenance of emotion concepts in working memory through interaction with the rACC. dACC participates in motivation and action selection. Linkage between insula and dACC and between rACC and DMPFC are enhanced by attention and contribute to different aspects of conscious awareness. Blue arrows indicate bottom-up (afferent) information flow; red arrows indicate top-down (efferent) information flow. Dashed arrows indicate that information flow is dependent on attention (or related top-down modulatory influences associated with access consciousness). DMPFC: dorsomedial prefrontal cortex; rACC: rostral anterior cingulate cortex; dACC: dorsal anterior cingulate cortex; VMPFC: ventromedial prefrontal cortex. (For interpretation of the references to color in figure legend, the reader is referred to the web version of the article. Red arrows are darker gray and blue arrows are lighter gray.)

working memory, such that this information can be used to guide deliberative decision-making processes (Waugh et al., 2014). These structures all interconnect with the VMPFC, which as noted above, plays a major role in both appraising the emotional significance of one's current situation and in subsequent autonomic and neuroendocrine regulation (Price, 1999; Roy et al., 2012).

The neural basis of somatization and related disorders is not known. However, based on the clinical and psychological characteristics described above, it may be hypothesized that affective agnosia involves a failure to engage the medial prefrontal areas involved in the conscious experience and representation of emotional distress (e.g., rACC), and/or the ability to hold this information in mind for use in complex cognitive processes such as mentalization and to guide goal-directed action selection (e.g. DMPFC). Instead what may occur is a "short circuit" whereby somatic sensations at the intermediate level of processing within the insula are consciously experienced, and interpreted by the VMPFC as a danger signal that in turn drives arousal mechanisms (see Fig. 3). This short circuit is associated with a relative failure to engage the rACC, dACC and DMPFC and the related functions of emotional awareness. Under such circumstances, bodily feedback from this arousal may further amplify somatic sensations in a vicious cycle because the top-down modulatory vagal mechanisms that would have been recruited by these medial frontal lobe structures (Thayer et al., 2012) are not engaged. This may help to explain the at times intense state of bodily arousal that is not recognized by the individual as an emotional response but rather as a symptomatic bodily state. Cognitive-behavioral therapy for somatization focuses on recognizing the source of such arousal and reappraising it in order to down-regulate it (Kroenke and Swindle, 2000).

According to the neurocomputational model of perceptual processing described above, as it applies to emotion, the low-level most likely involves unconscious brainstem- and possibly other subcortical-level neural states which induce and detect activity changes within individual organs, changes in circulating hormone levels, or other discrete visceral and somatic changes. The intermediate-level would involve regions which detect coherent patterns in whole body states across the discrete features detected at the low-level, and, as noted briefly above, the insula (and perhaps related parietal somatosensory processing regions; Gazzola et al., 2012) may be a prime candidate for instantiating such representations. The intermediate level is the level that most likely corresponds to conscious perception of specific bodily states when these representations are attended to and thus made available to working memory and executive control systems (Craig, 2009, 2002; Crick and Koch, 2000; Jackendoff, 1987; Koch, 2004; Prinz,

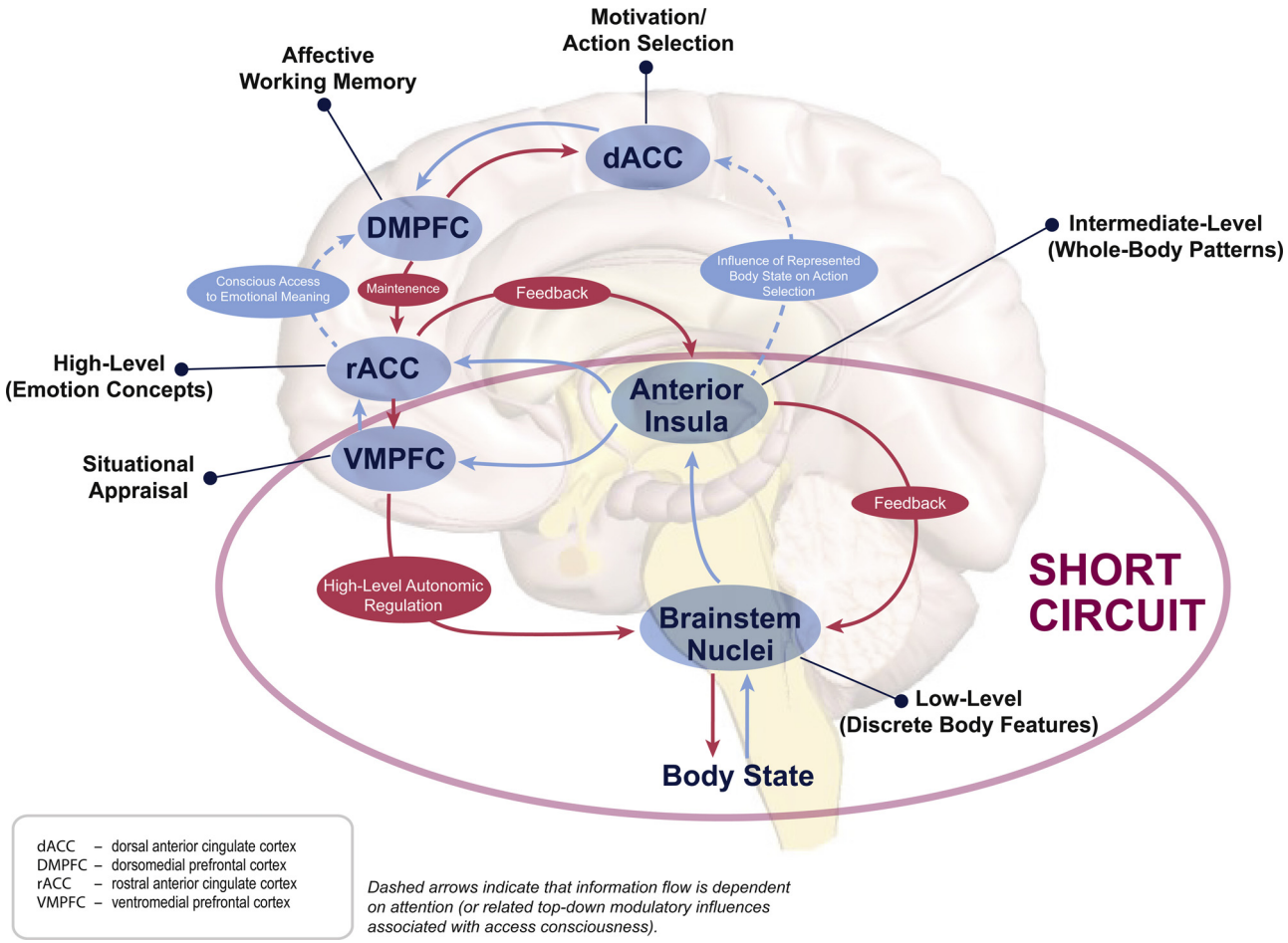

Fig. 3. Schematic diagram of the neural basis of affective agnosia. A short circuit is created by virtue of the exclusion of rACC, DMPFC and dACC in the processing of bodily state information. Processes outside of the depicted "short circuit" boundary are dysfunctional in affective agnosia, and illustrate how, because emotional insula-mediated representations of one's body state are interpreted as a sign of increased situational danger by VMPFC, body state arousal and distress about it can continue to increase within a positive feedback loop (in the absence of negative feedback arising from rACC- and DMPFC-mediated understanding of the emotional meaning of such bodily reactions). Dashed arrows indicate that information flow is dependent on attention (or related top-down modulatory influences associated with access consciousness). Blue arrows indicate bottom-up (afferent) information flow; red arrows indicate top-down (efferent) information flow. DMPFC: dorsomedial prefrontal cortex; rACC: rostral anterior cingulate cortex; dACC: dorsal anterior cingulate cortex; VMPFC: ventromedial prefrontal cortex. (For interpretation of the references to color in figure legend, the reader is referred to the web version of the article. Red arrows are darker gray and blue arrows are lighter gray.)

2012, 2006). The high-level would involve neural states which integrate intermediate-level information in order to represent one's perceived somatic/visceral reactions as having a specific emotional meaning, and this level of representation appears to correspond to the function of rACC (Kalisch et al., 2006; Peelen et al., 2010; Smith et al., 2014). The supramodal nature of this representation in rACC was demonstrated in a study in which emotion was activated by the face, voice and bodily movement and common areas of category-specific emotion activation were identified; the only other area activated in addition to rACC was left superior temporal sulcus (Peelen et al., 2010). Rostral ACC also plausibly integrates information about emotional appraisals of one's situation (through interactions with VMPFC) in arriving at these representations of the emotional meaning of one's somatic/visceral reactions. DMPFC may further function to send top-down signals which maintain these rACC representations in an active state such that they can be used to adaptively guide one's goal-directed behavior (Waugh et al., 2014). Thus DMPFC may play an important role in maintaining conscious access to the emotional meanings of one's bodily states, and thus allow this information to be used. Together the combination of rACC and DMPFC function may account for how the medial frontal lobe participates in mentalization, by representing the emotional states of self and other (rACC) and maintaining/manipulating this mentally represented information in working memory (DMPFC) such that the content of these representations can guide further deliberative thought and action.

Thus, in the present model, rACC/DMPFC dysfunction, possibly as a result of the developmental problems discussed above, would hinder one's ability to categorize a given consciously perceived body state *as a certain emotion*. As depicted in Figs. 2 and 3, without engaging high-level representations in rACC, or the DMPFC functions which allow these rACC representations to be used to guide deliberative thought, the proposed short-circuit occurs in which one only consciously perceives intermediate insula-level representations of bodily states without being aware of their emotional meaning. This could plausibly result in somatization symptoms. The effects of such rACC and/or DMPFC dysfunction would also likely alter the VMPFC's appraisals of one's current situation, leading to increases in the intensity of the autonomic and endocrine reactions it initiates. It follows from this that psychotherapy might successfully be aimed at enhancing high-level representations through labeling bodily states as emotions and understanding their meaning (Kroenke and Swindle, 2000; Subic-Wrana et al., 2011). Without recognizing emotions as such, one may be unable to use information about one's current emotional state or those of others to alter, regulate, respond to or otherwise adapt to them.

10. Differential diagnosis from a neuroanatomical perspective

To be a clinically useful concept, affective agnosia needs to be differentiated from other related phenomena. Below we compare and contrast the neural basis of affective agnosia with that of repressive coping, depersonalization disorder, psychopathy and emotional numbing. Although relatively little is known, preliminary distinctions can be made based on findings to date, and these distinctions constitute hypotheses to be further tested.

Repressive coping, a phenomenon that Sifneos and Nemiah believed could not explain what was observed in alexithymia, is a defensive process associated with a focus on positive emotions and a focus away from negative emotions (Weinberger et al., 1979). Repressive coping is thought to arise from a two-stage process consisting of vigilance and avoidance (Derakshan et al., 2007). Vigilance involves a hyper-attentiveness to threatening stimuli followed by avoidance or lack of attention to those same stimuli. The combination constitutes a defense in that recognized threats are kept out of conscious awareness. There has been relatively little neuroimaging research on repressive coping. However, one fMRI study by Rauch et al. (2007) showed that subjects with a repressive coping style, relative to those who were sensitizers (who are especially attentive and responsive to environmental threats), showed less amygdala activity and greater frontal lobe activity in response to fearful faces. Thus, repressive coping is associated with knowledge of threats but a lack of emotional experience due to top-down inhibitory processes, whereas affective agnosia is due to a developmental failure in the capacity for high-level mental representation and experience of emotion in rACC and DMPFC, potentially resulting in a relative deficit in appropriate top-down inhibition of overly intense somatic/visceral reactions by VMPFC.

Depersonalization is a phenomenon closely associated with dissociation, which is defined as a disruption in the integration between consciousness, memory, identity and perception (Lemche et al., 2007). Dissociation leads to a fragmentation of the unity and continuity of the sense of self. Depersonalization is a particular type of dissociation involving a disrupted integration of self-perceptions with the sense of self, so that individuals are in a subjective state of feeling estranged, detached or disconnected from their own being. The core symptoms of depersonalization disorder include emotional numbing, visual derealization and altered bodily experience (Simeon, 2004). Several imaging studies in depersonalization disorder have shown decreased activity in the anterior insula and increased activity in the right ventrolateral prefrontal cortex (Sierra and David, 2011; Simeon, 2004). Lemche et al. (2007) observed increased activity in the dorsal prefrontal cortex associated with decreased activity in the amygdala when individuals with depersonalization disorder were exposed to facial expressions. Thus, it is believed that increased activity of the right prefrontal cortex inhibits activity in the insula, thus separating the individual from the (intermediate level) bodily basis of emotional experience. These findings have led to the hypothesis that frontal inhibition of limbic structures may mediate its characteristic hypoemotionality (Simeon, 2004) or "de-affectualization (Medford, 2012). Depersonalization appears to be similar to repressive coping by virtue of frontal inhibitory mechanisms but differs from it, and affective agnosia, by virtue of the inhibition of the insula and its essential role in creating a bodily felt sense of emotion. To the extent that the agnosia version of alexithymia is associated with heightened bodily experience (De Gucht and Heiser, 2003), depersonalization is in some ways the polar opposite.

Psychopathy is a complex disorder involving behavioral impulsivity, actions that violate social norms, and a lack of empathy and concern for others (Diagnostic and Statistical Manual of Mental Disorders, 2013). It is well established that psychopathic individuals have reduced emotional responses when other people are harmed or injured whereas emotional responses are intact when threats are directed at the psychopathic individual (Anderson and Kiehl, 2012). It appears that there are at least two fundamental problems in the brain of psychopaths: an under-functioning amygdala (specifically, reduced empathy-related amygdala responses to cues of distress in others), and a deficit in VMPFC (and connected striatal circuits) associated with reinforcement learning and decision-making (Anderson and Kiehl, 2012; Blair, 2013, 2003; Vogt and Lane, 2009). These deficits will limit both implicit and explicit processes. Relative to affective agnosia, both conditions are also associated with reductions in context-appropriate recruitment of the ACC associated with reduced emotional awareness (Vogt and Lane, 2009). A critical difference is a relative lack of emotional responding, particularly of the amygdala, in psychopathic individuals compared to those with affective agnosia. Psychopathic individuals also have intact emotion concept representations and mentalization functions regarding the mental states of others (Blair, 2008), which facilitates their manipulation and deception of others. Thus, while affective agnosia involves intact emotional responses and a deficit in emotion concept representations, psychopathy appears to instead involve deficient emotional responses combined with intact emotion concept representations.

Emotional numbing is a term that is often associated with post-traumatic stress disorder. It has been described as a markedly diminished interest in significant activities, feelings of detachment or estrangement from others, and restricted range of affect (Diagnostic and Statistical Manual of Mental Disorders, 2013). Some investigators describe it as a loss of the ability to become interested in previously enjoyed activities, or loss of the ability to feel emotions of any type, especially those associated with intimacy, tenderness, and sexuality (Litz and Gray, 2002). In a study of women with PTSD, increasing emotional numbing symptoms predicted decreased response within the dorsomedial prefrontal cortex during imagery of positive and negative events that were specifically social in nature (Frewen et al., 2012), which contrasts with anhedonia, which specifically refers to a deficit in positive affect. Emotional numbing and affective agnosia appear to share a similarity in diminished participation of the medial prefrontal cortex in representing emotional experiences. It could be that there is overlap in the neuroanatomical substrates of the two conditions. A key difference, however, may be that emotional numbing may be more of a temporary state, whereas affective agnosia may be an enduring trait. Emotional numbing may occur when emotional arousal is particularly intense (Litz and Gray, 2002), which differentiates it from apathy, in which there is a lack of emotional responding (Starkstein and Pahissa, 2014). This hypothesis is entirely consistent with the known positive correlation between vagal tone and activity in the medial prefrontal cortex: when vagal tone is low (i.e. physiological arousal is high), medial prefrontal cortex activity is low (Thayer et al., 2012), which could explain why alexithymia has at time been considered to be a temporary state in some people rather than an enduring trait (Lumley et al., 2007, page 239). Vulnerability to emotional numbing among people with PTSD, reflecting a lower threshold for the medial prefrontal cortex to "go offline" during high arousal states, may be the degree to which affective agnosia was present prior to the traumatic stress. This is consistent with the narrowed "window of tolerance" concept associated with traumatic stress, especially that in early life (Corrigan et al., 2011).

Given our emphasis on bodily perception in the low and intermediate levels of emotion perception it might appear that we are strongly advocating for a James-Lange model of emotion (James, 1894, 1884; Lange, 1885), which holds that the experience of emotion consists of the perception of bodily sensations. It is not clear, however, whether the visceral and somatomotor patterns associated with emotion have sufficient specificity to allow for the

full range of reported affective experiences (Levenson, 2014). Thus we also emphasize that high-level representations of one's own emotional responses in rACC may be further influenced by VMPFC appraisals of the emotional meaning of one's situation. Given the critical role of the VMPFC in the proposed emotion perception hierarchy and its major role in situational construal (Roy et al., 2012), it should be evident that our proposed model draws heavily from the Schachter Singer model (Schachter and Singer, 1962), which states that the nature of emotional experience is determined by the combination of physiological arousal and situational construal. When it is considered that construals of both the situation and one's bodily state are strongly influenced by preexisting concepts or schemas, models which emphasize the constructive nature of emotional experience (Averill, 1980; Lindquist and Barrett, 2008) make important contributions as well.

11. Implications for treatment

As noted above, Freud coined the term "agnosia" in 1891 prior to his discovery/initial creation of psychoanalysis in 1895. His work with Breuer titled *Studies on Hysteria* (Breuer and Freud, 1955) assigned a pathogenic role to repressed emotions in the context of traumatic (psychologically overwhelming) experiences in the past. For the remainder of his career Freud focused almost exclusively on mental contents that had been previously mentally represented which were once known but were disguised or barred from consciousness due to defenses such as repression (Levine, 2012). Freud assumed that emotions, for example, were either conscious or repressed (or otherwise defended against) (Solms and Panksepp, 2012). The concept of alexithymia was therefore a theoretical advance in addressing how emotions could be excluded from awareness by virtue of a deficit (in cognitive processing) rather than a defense. However, as described above, the distinction between an anomia and an agnosia has not previously been put forward in relation to alexithymia. As noted, anomia involves intact mental representation of emotion, consistent with the traditional Freudian psychoanalytic model, whereas we are emphasizing the phenomenon of emotional responses that have not been previously mentally represented, consistent with the Freudian neurological model of agnosia. It is difficult to know why Freud did not connect agnosia to affective experience, but this could be related to the traditionally rigid conceptual boundary between emotion and cognition that has only recently begun to crumble in the wake of functional neuroimaging findings in healthy volunteers (Lane et al., 2000a).

The distinction between agnosia and anomia in relation to affect has enormous clinical implications because a deficit in mentalization is now understood to be present in a wide variety of clinical contexts for which the traditional Freudian drive and defense model has proven inadequate. These contexts include borderline personality disorder, somatic symptom disorders, addictions, eating disorders, self-injury and the emotional consequences of early life adversity, to name a few. In many of these contexts alexithymia has been observed (Taylor and Bagby, 2013). Whether a given person has the anomia or agnosia version of alexithymia, however, will have a major bearing on how it should be treated therapeutically.

It is commonly accepted that alexithymia is notoriously difficult if not impossible to treat (Ogrodniczuk et al., 2011; Taylor and Bagby, 2013). The difficulty stems from a variety of factors. Alexithymic individuals tend to focus on somatic states rather than feelings when emotions are activated, have a long-standing pattern of avoidance of experiencing and attending to emotions, and at-times have ingrained maladaptive behavioral patterns (addictions, self-injury) that are used for emotion self-regulation. Research also indicates that many alexithymic individuals grew up in invalidating environments and they expect the same in their adult lives (Paivio and McCulloch, 2004). Thus, there is a reluctance or difficulty in trusting the therapist, helping to explain why establishing a therapeutic alliance can be so challenging, and why the usual approach to emotion processing and meaning creation does not work so well with these patients.

Adopting the perspective of affective agnosia, one might say that the issue is not one of the helping patients to "put emotions into words" as much as helping them to establish mental representations of their emotional states, i.e. helping them to know what they feel. Here the distinction between emotion and feeling, or between implicit and explicit processes, is critical (Lane, 2008). Emotional responses typically occur automatically and are often associated with subjective experiences. However, many emotional responses do not reach the level of conscious emotional awareness (Lama and Ekman, 2008), and if they do, they may be either quite contrary to one's belief system (e.g. associated with the thought that feeling a certain way is unacceptable) or to the preferences of important others (e.g. anger at someone who is abusive would not be tolerated by the abuser). Not uncommonly, emotions in this context are ignored, disavowed, suppressed or otherwise avoided so that the patient does not know what s/he is feeling or why s/he is feeling it. Importantly, mentally representing emotional experience requires an active process of representation that during development does not occur automatically but typically requires the assistance of another person who is caring and empathic (Gergely and Watson, 1996). For reasons too complex to discuss in detail here, highly arousing, "pathogenic" emotional responses are often undifferentiated and unsymbolized at the time of their first occurrence (Lane et al., 2015), contributing further to the absence of mental representation of differentiated emotional states present in higher functioning individuals. Related to the concomitant failure of top-down regulation discussed above, another important reason why emotions are not attended to is that they are at times too intense and the person does not have confidence that s/he can feel the feeling, make use of it and act appropriately in a problematic social context (Greenberg, 2002). Practicing such avoidance over many years results in under-development of the skill of being emotionally aware. Failure to attend to one's emotions prevents one from making use of the adaptive information that is inherent in emotion. This information can be derived from knowing what one feels and what that emotion indicates about what one needs in a given situation (Greenberg, 2002).

An important reason for promoting the concept of affective agnosia is that a variety of treatment methods have been created in recent year that promote mental representation of emotion. These techniques have not been applied to the alexithymia context to date, in part because alexithymia tends to be conceptualized as an anomia, and because it has not previously been directly linked to the concept of agnosia. Dialectical Behavior Therapy, originally designed for the treatment of borderline personality disorder, has a number of important components, including identifying what one is feeling and learning to regulate those feelings (Linehan et al., 1999). Mentalization-based therapy by Fonagy and colleagues (Bateman and Fonagy, 2004) aims to repair the deficit in reflective capacity that is thought to underlie borderline personality disorder. A recent innovation is to apply mentalization-based techniques to the treatment of patients with functional somatic disorders (Luyten et al., 2012). Emotion Focused Therapy developed by Greenberg (2002) within the Gestalt tradition helps people to experience, understand and transform their emotions in the treatment of depression and other symptomatic conditions. We propose that all of these techniques promote the ability to know what one is feeling and are therefore potentially applicable to the treatment of alexithymia/affective agnosia.

Many clinicians who are familiar with the concept of alexithymia, particularly those in training, assume that the hallmark of alexithymia is a limited or absent vocabulary of emotion words. When someone who appears to be emotionally unaware uses a few emotion words in a clinical encounter it raises the question of whether that person should be considered alexithymic. The tendency to jump to the explanation that alexithymia is a continuum obscures a critical issue and prevents this important distinction between anomia and agnosia from being made. It is therefore useful to draw from the clinical phenomenon of agnosia in a neurological sense and emphasize the importance of being unable to *use* information (in this case emotional information) as the hallmark of agnosia. If a mental representation of a concept, object or emotional state is present (and consciously accessible) one can behave and think as if the name and concept associated with the representation is present, even if it is not explicitly demonstrable at the moment (conversely, one may use an emotion word in socially appropriate ways without feeling it or understanding it). For example, having mental representations of emotions enables one to think through what happened in the past and how the emotional states of the participants contributed to what transpired, or to anticipate how possible future actions might influence one's own or others' emotional responses. This involves going beyond automatic gut feelings and being able to explicitly talk about emotional dynamics, i.e. how to *use* emotional information to promote social and occupational adaptation. It is therefore not so much the ability to utter an emotion word (which can be perfunctory and rehearsed but unfelt) that indicates the absence of agnosia; it is an ability to access representations in one's imagination and an ability to experience the associated feelings and know what they mean. The absence of imaginative representations corresponds to an impaired capacity for fantasy, which was inherent in the original alexithymia construct but was excluded from the TAS-20 for psychometric reasons (Bagby et al., 1994a, 1994b).

One of the advantages of this focus on *use* of emotional information is that it links directly to the construct of emotional intelligence, of which the ability to *use* emotional information is one of the four major components (along with perceiving, understanding and managing emotion) (Salovey and Mayer, 1989). It also links directly to the construct of access consciousness (Baars, 2002; Block, 2005, 1995), the hallmark of which is the ability to *use* the information contained in the representation. An important feature of access consciousness is that one can demonstrate knowledge of the object or concept even if one cannot name it or explicitly recall it at the moment. That is, one can have conscious access to the meaning of a given mental representation without having conscious access to the phonological representation of the linguistic term one would use to efficiently signify that meaning to others.

The cognitive-developmental model described above provides a specific guide to treatment by specifying what clinicians should focus on initially and what the next step in treatment should be (Beutel et al., 2013; Lane and Garfield, 2005; Lane and Schwartz, 1992). Following Piaget, the first step is to determine the developmental level that the patient is at. Intervention then focuses on helping the patient advance to the next level or higher levels. The distinction between implicit and explicit expression of emotion is especially relevant to this process, which involves engaging the patient at the implicit, somatic level, facilitating the transition to explicit emotional awareness and fostering the construction of more complex mental representations of emotions.

Individuals who somatize their emotions are operating at level 1, which is characterized by a lack of understanding of how internal bodily states are connected to emotions. The logic of this developmental framework dictates that a maladaptive focus on visceromotor states can be overcome by transitioning to the next level (level 2) of enactive states, such as facial expressions, gestures, patterns of behavior and behavioral scripts that are labeled by the therapist and organized around the concept of a specific emotion, such as anger, fear or sadness. Understanding how an emotion is overtly expressed in behavior, and how that behavior links to internal bodily sensations, provides a sensory-motor foundation for subsequent level 3 experiencing of specific emotions in a context-appropriate way.

Individuals who act out impulsively including in self-injurious ways are operating at level 2 (action tendencies and impulses) (Lane and Schwartz, 1987). Such behavior can be addressed by focusing on the maladaptive behavior and transitioning to the next level. This would involve helping the patient to appreciate that the acting out behavior is serving an emotion-regulatory role. Stopping the acting-out behavior makes it possible to observe what feelings are associated with the urge to act out in a particular way. The feelings that emerge may be intense and difficult to bear but are made more tolerable by virtue of a comforting therapeutic alliance and the process of coming to know what one is feeling. Such intervention helps the person become aware of the feelings at level 3 that had been associated with acting out at level 2. The feelings at level 3 do not "underlie" or precede the level 2 manifestations but rather are constructed with and emerge from them (Lane and Garfield, 2005).

As an illustration, therapists in psychosomatic inpatient units in Germany engage implicit emotional processes through art therapy, dance therapy, massage and other body-based treatments to determine what patients are aware of and to facilitate explicit experiencing, description and expression of emotion in verbal modalities including individual and group therapy (Subic-Wrana et al., 2010). Importantly, the therapists work together to first understand what is being expressed implicitly and then formulate what these expressions may foreshadow at the explicit level. This involves knowing the patient well enough to judge what they might be feeling in a given situation based on non-verbal expressions (e.g. artistic, facial or movement) and helping them to articulate and know what they are feeling and why they are feeling it in specific situations, particularly the most problematic ones (often those contexts that led to intensive treatment). This is a bit different from the classic psychodynamic model of drive and defense, which holds that once defenses are removed the emotion that is being held at bay emerges naturally. According to the current model, once defenses (e.g. ignoring, avoiding, disavowing, suppressing, and others) are overcome the critical work of coming to know (i.e. mentally represent and access) what one is feeling must be undertaken. Thus, overcoming defenses is the start of the process, not the final goal for people with affective agnosia. By contrast, people who are more highly aware and function at a higher adaptive level often are better able to reflect upon and know what they're feeling as soon as defenses are overcome, which makes therapy much easier.

Throughout this paper the foundational observation that alexithymia contributes to adverse medical outcome has been emphasized. We discussed the mechanisms by which affective agnosia may contribute to physiological dysregulation and disease. From a therapeutic perspective, we believe that this model of affective agnosia provides a principled way to integrate body-based and mind-based treatments for general medical conditions and functional somatic syndromes (Lane, 2008). Psychosomatic medicine is currently struggling with the question of whether and how to include body-based treatment approaches that fall within the domain of complementary and alternative medicine, such as massage, yoga, acupuncture, tai chi, dance therapy, art therapy, movement therapy (e.g. Feldenkreis) and other non-verbal techniques. There is also the question of how mind-based psychotherapies, including but not limited to manualized treatments such as cognitive-behavioral therapy (Kroenke and Swindle, 2000),

as well as mindfulness meditation, should be used in coordination with body-based treatments. In addition, there is also a need to incorporate psychoeducational techniques that instruct people about emotion recognition and emotion regulation skills. According to the model presented here, body-based therapies may be particularly useful for mobilizing implicit affect and making it available for explicit processing, while mind-based treatments, in conjunction with psychoeducational methods, may be used to facilitate explicit processing, such as experiencing, labeling, understanding and regulating. In some circumstances a stepwise approach may be needed. For example, in some patients with functional motor disorders, particularly those not inclined to consider psychological or emotional factors (Demartini et al., 2014), physical therapy alone may lead to some improvement in physical symptoms (Czarnecki et al., 2012), which itself could have some benefits in psychosocial functioning.

To our knowledge there are two other proposed alternatives to the alexithymia construct that address abnormalities in implicit processing. Helling proposed the concept of "apraxithymia" (Helling, 2009) involving an impairment in the execution of implicit mental operations due to psychological trauma. This concept highlights the need in therapy to directly target implicit procedural aspects of emotion processing, such as the direction of attention and inhibitory self-regulation, to facilitate explicit declarative emotion processing. A second newer concept is alexisomia (Moriguchi and Komaki, 2013), which involves an inability to describe bodily sensations perceived through interoception. The latter concept highlights the clinical need to facilitate description of bodily states. Interestingly, authors of these concepts cite previous earlier work on the blindsight model of alexithymia and neurovisceral integration by one of the current authors (RDL) in support of each of these new concepts. These newer concepts illustrate the need to do more in therapy than simply put emotions into words. The therapeutic techniques derived from the cognitive-developmental model described immediately above, which directly address therapeutic intervention at the implicit level, appear to address many of the same issues regarding how to facilitate the transition from implicit to explicit processing.

12. Implications for basic emotion research

The concept of affective agnosia as formulated above has important implications for basic emotion research. Emotion has traditionally been equated with the subjective experience of emotional feelings, but with the advent of a cognitive neuroscience approach it has come to be appreciated that emotional responses without emotional experiences are not only possible but actually common (Brosschot et al., 2010). A core concept is that subjective emotional experience has a definable neural basis and that activity within the neural network mediating emotion, and its physiological impact on the body, changes as a function of what one experiences and what knows about one's own emotions. As we have argued above, there are individual differences in the ability to experience and express emotion. Perhaps a unique autonomic or brain circuit for each "basic" emotion has not been identified precisely because of these individual differences (Lindquist et al., 2012). If so, the way to identify a unique autonomic or brain pattern of each emotion would be to study people who are more emotionally aware and more highly differentiated in their emotional experience.

Specific predictions would be that higher levels of emotional awareness would be associated with more differentiated autonomic signatures of basic emotions, more differentiated neural circuits during activation of basic emotions, a more extensive repertoire of facial expressions, and a more complex and flexible repertoire of emotion regulation strategies in real life social settings (Lane and Pollerman, 2002). Conversely, such research could reveal that the physiological patterns of basic emotions are more obscured and undifferentiated in people who are less differentiated (emotionally aware) psychologically, which may be what has been observed to date because such individual differences have not been taken into account. If so, such research could potentially lead to a better ability to identify the physiological basis of happiness, sadness, anger and fear and the psychiatric disorders associated with abnormalities of each (e.g. happiness: mania, anhedonia, and addictions; sadness: depression and complicated grief; anger: violence and suicide; fear: generalized anxiety, phobias and PTSD, to name a few). Such research could also help to demonstrate physiological differences between healthy volunteers and those with affective agnosia, as well as differentiate between the agnosia and anomia versions of alexithymia. Critically for the purposes of this discussion, such research could also help to explain the physiological basis of the link between alexithymia and physical disease progression, an association that inspired the original work of Sifneos and Nemiah on alexithymia.

13. Conclusion

After the popularity of psychoanalysis peaked within psychiatry in the mid-20th century, there has been a steady decline in the acceptance and inclusion of Freudian ideas in clinical practice. Alexithymia conceptualized as a deficit state itself represented a departure from the traditional Freudian view that impairments in awareness were due exclusively to psychological defenses. The current view that alexithymia may include a deficit in mental representation of emotion, not just in naming it, was ushered in by neuroimaging findings that suggested an impairment in emotional awareness rather than in emotional naming. The neural models of this impairment and the distinction between implicit and explicit emotion processing have been inspired by work on the conscious processing of visual information and the belief that the principles that apply to vision extend more generally to other functional systems in the brain such as emotion. The current model of affective agnosia extends the original blindsight model of alexithymia by drawing upon more current computational models of hierarchical visual processing and the concept of access consciousness. We propose that affective agnosia constitutes a supramodal agnosia consisting of dysfunction in the activation and/or use of high-level mental representations of emotional states and their meaning. This clarifies the distinction between the anomia and agnosia subtypes of alexithymia and highlights some important implications for clinical intervention, with special emphasis on using emotional information as opposed to only expressing emotions in words. Thus, by virtue of the rapid advances in the neurosciences in recent decades we find much relevance in the neurological writings of Freud a century ago and consider it possible that he might have applied the concept of agnosia to the emotional basis of certain mental disorders had this new information been available to him at the time. As such we believe that we are contributing in some small way to the realization of Freud's dream of understanding how dynamic changes in conscious and unconscious emotion are mediated in the brain and how their vicissitudes help us to better understand the origin and treatment of mental disorders as well as the role of emotion in the onset, presentation and progression of physical diseases.

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