Summary
This review highlights the importance of right hemisphere language functions for successful social communication and advances the hypothesis that the core deficit in psychosis is a failure of segregation of right from left hemisphere functions. Lesion studies of stroke patients and dichotic listening and functional imaging studies of healthy people have shown that some language functions are mediated by the right hemisphere rather than the left. These functions include discourse planning/comprehension, understanding humour, sarcasm, metaphors and indirect requests, and the generation/comprehension of emotional prosody. Behavioural evidence indicates that patients with typical schizophrenic illnesses perform poorly on tests of these functions, and aspects of these functions are disturbed in schizoaffective and affective psychoses. The higher order language functions mediated by the right hemisphere are essential to an accurate understanding of someone’s communicative intent, and the deficits displayed by patients with schizophrenia may make a significant contribution to their social interaction deficits. We outline a bi-hemispheric theory of the neural basis of language that emphasizes the role of the sapiens-specific cerebral torque in determining the four-chambered nature of the human brain in relation to the origins of language and the symptoms of schizophrenia. Future studies of abnormal lateralization of left hemisphere language functions need to take account of the consequences of a failure of lateralization of language functions to the right as well as the left hemisphere.

Keywords: schizophrenia; language; laterality; pragmatics

Abbreviations: LHD = left hemisphere damage; RHD = right hemisphere damage


Introduction
Many of the key symptoms of schizophrenia are either expressed in terms of language or are direct abnormalities of language functions themselves (Andreasen 1979a, b; WHO, 1993; APA, 1994). Language dysfunction has correspondingly been of great importance in schizophrenia research. To date, much of the research on language abnormalities in schizophrenia has focused on functions normally mediated by the left cerebral hemisphere. For instance, it is known that patients with schizophrenia find it difficult to process semantic aspects of language appropriately (Nestor et al., 2001), their speech is ‘disorganized’ (Kerns and Berenbaum, 2002), they demonstrate reduced comprehension accuracy according to syntax (Condray et al., 2002) and they do not perform as well as control subjects in word-stem completion tasks (Titone and Levy, 2004). This trend in the literature is understandable given the dominance of the left hemisphere for the majority of language functions (Capozzoli, 1999; Gernsbacher and Kaschak, 2003). Indeed, the recent creation of the term ‘schizophrenia’ reflects parallels between the language dysfunction in schizophrenia and that exhibited by the group of left hemisphere language disorders known as aphasia (Critchley, 1964; Oh et al., 2002), and
schizophrenia has in the past been referred to as ‘a left hemisphere disorder’ (perhaps most notably by Flor-Henry, 1976, 1983). Intimately associated with this body of research is the hypothesis that people who suffer from schizophrenia display abnormal lateralization of language and that the nuclear symptoms of schizophrenia reflect this failure to establish left hemisphere dominance (Crow, 1997a, b, 1998, 2000). Both structural and functional evidence has accumulated which suggests that schizophrenia is associated with reduced lateralization of language to the left hemisphere, with some studies reporting a reversal of lateralization, to the right hemisphere (Gruzelier, 1999; Gur and Chin, 1999; Kwon et al., 1999; Petty, 1999; Aydin et al., 2001; Sommer et al., 2001; Kircher et al., 2002; Sumich et al., 2002). Thus, in patients with schizophrenia, the right hemisphere’s apparent involvement in the mediation of left hemisphere language functions is greater than normal. At the same time, schizo-affective and affective psychoses have been associated with deficits in right hemisphere functions (Jamison, 1990) and have sometimes been attributed to a focus in the right hemisphere (Flor-Henry, 1976, 1983).

**Right hemisphere language functions**

In contrast to the left hemisphere, the right hemisphere does not normally have much responsibility for linguistic processes such as phonology, morphology and syntax. However, it is the primary mediator of a set of paralinguistic or pragmatic phenomena that accompany the words of an utterance and that can modify or influence its meaning. These higher order language functions are crucial to understanding someone’s true communicative intent and thereby integrating effectively into society. How something is said may be just as important as what is said. In the review of literature that follows we focus on studies that compared patients with unilateral left hemisphere brain damage, patients with unilateral right hemisphere brain damage and healthy controls without brain damage. Where possible, more recent evidence from neuroimaging studies is included; this latter group of studies primarily assessed right hemisphere language functions in people free from neurological and psychiatric disorders.

**Discourse comprehension and production**

Speakers of a given language have certain expectations with respect to the organization of written texts and conversations (Obler and Gjerlow, 1999). Thus, according to the philosopher Paul Grice, in a communicative exchange: (i) participants should not provide too much or too little information (Maxim of Quantity); (ii) participants should maintain a topic from one utterance to the next, with topic shifts allowed only at certain junctures (Maxim of Relevance); and (iii) information should be presented in an organized and clear manner (Maxim of Manner) (Grice, 1975). Other unconscious expectations include the choice and maintenance of an appropriate level of formality depending on the context (lexical selection) (Obler and Gjerlow, 1999). Since discourse typically spans more than a single utterance, a listener must integrate linguistic and paralinguistic information across utterances, to build a complete understanding of the speaker’s communicative intent (Brownell et al., 1992). Inferences about communicative intents are not simply made at the beginning of a conversation and then ‘forgotten’. An understanding of someone’s communicative intent is updated and modified over the period of discourse as new information is presented and the shared knowledge upon which inferences are made becomes more elaborate (Sabbagh, 1999). The forms which discourse may take are: (i) procedural: describes the procedures involved in performing an activity; (ii) expository: conveys information on a single topic by a single speaker; (iii) conversational: conveys information between a speaker and a listener or among several speakers and listeners; and (iv) narrative: a description of events (Ulatowska et al., 1990; Hough and Pierce, 1993). However, distinctions between different types of discourse may also be framed in terms of function rather than form. Thus, according to van Dijk (1997), there are three main dimensions of discourse: (i) discourse as verbal structure (‘language use’); (ii) discourse as communication of beliefs (‘cognition’); and (iii) discourse as action and intention in social situations.

Most of what we know today about the neuroanatomical basis of discourse comes from studies of patients with unilateral lesions to one hemisphere or the other following a stroke or other neurological insult. These studies clearly show an association between damage to the right hemisphere (right hemisphere damage, RHD) and discourse deficits such as difficulty generating inferences, difficulty comprehending and producing main concepts or central themes, a reduced level of informative content, and a reduced sensitivity to communicative context (Myers, 1999).

In studies of discourse comprehension, participants are frequently asked to listen to or read a narrative and answer questions about inferred meaning or the appropriate order of sentences. Such tasks involve the extraction of meaning from individual sentences and their integration into the context supplied by other sentences in the narrative (Hough, 1990). For example, Bryan (1988) adapted five paragraphs from an educational textbook to make them suitable for adults and devised four questions for each passage, the answers to which were not directly stated in the paragraphs but were clearly implied by its contents. Compared with healthy controls and patients with left hemisphere brain damage (LHD), patients with RHD made significantly more errors, i.e. they made inferences, but these inferences were not correct. According to Bryan, they had failed to appreciate the implications of the texts because they could not use semantic information that extended beyond individual words or sentences. Even over a short period of discourse such as the two sentence vignettes employed by Zaidel et al. (2002), RHD was still associated with the production of inappropriate inferences. Patients with RHD could not integrate and evaluate the information provided by both sentences. They more often than not based their
interpretation upon information provided in the first sentence alone. Interestingly, in a test of comprehension of a longer narrative, the magnitude of interpretation deficit corresponded directly to the size of the RHD; the bigger the lesion in the right hemisphere, the poorer the patient’s performance on this task. When asked to interpret illustrations, RHD patients focus obsessively on specific details (e.g. such as the appearance of the people depicted) and demonstrate little evidence of having understood the main concept or theme of the picture (e.g. what were the people doing in the picture) (Myers and Brookeshire, 1996). These types of deficits are even greater in stories that contain bizarre elements. Whereas healthy and LHD control groups do not usually accept these parts of narratives, patients with RHD tend to accept such explanations and even add explanations to justify them (Gardner et al., 1983). Even when told the central theme of the story, the performance of patients with RHD does not improve, suggesting that they have difficulty recognizing the correct inference about a story when it is provided, as well as a difficulty generating the correct inference when it is not provided (Schneiderman et al., 1992).

More recently, functional neuroimaging studies have supported the association between the right hemisphere and discourse comprehension. Whilst lesion-based studies have undoubtedly been informative, patient groups are not homogeneous; the location and extent of the lesion varies, as does the degree of function recovery after the insult. From such studies we can deduce which hemisphere is necessary for discourse comprehension, but from neuroimaging studies we can discover which hemisphere is sufficient for this function. From neuroimaging studies, we now know that the left hemisphere is not sufficient to mediate normal levels of discourse comprehension (Robertson et al., 2000) and temporal lobe activity is right lateralized when participants listen to stories, even when they do so passively (Tzourio et al., 1998). Other studies suggest that a more diverse network of right hemisphere regions is involved in discourse comprehension, but again the right lateralization of temporal lobe activity is clear (St George et al., 1999; Robertson, 2000). As with the correlation between lesion size and degree of deficit, it appears that the greater the difficulty of the comprehension task, the greater the right hemisphere activation (Nichelli et al., 1995; St George et al., 1999).

Narratives have been used to investigate the association between discourse production and the right hemisphere too. For example, when asked to recall the major events of a fable, RHD patients tend to focus on minute detail, recall events out of sequence, digress from the point of the fable and produce discourse that lacks clear structure (Gardner et al., 1983). Whilst they may use as many words as normal controls to generate these narratives, they fail to produce appropriate levels of content, that is to say their utterances are not particularly informative (Bloom et al., 1992). When their spontaneous discourse is analysed in an unconstrained environment, RHD patients’ performance also shows deficits. In Bryan’s study, RHD patients demonstrated difficulties such as discussion of personal or emotional issues at an inappropriate time and failing to change the subject despite cues from the listener that this was desired (Bryan, 1988). Despite the advantages of neuroimaging, there are fewer functional imaging studies of discourse production than of comprehension. One of the reasons for this paucity is the confounding influence of speech-associated movement on fMRI (Friston et al., 1996). PET does not suffer from this restriction, and Braun and colleagues have shown that the early stages of narrative production involves posterior regions of the right hemisphere, although classical left hemisphere speech regions were also activated (Braun et al., 2001).

**Metaphors, indirect requests and humour/sarcasm**

Grice (1968) was again the first to explicitly consider the importance of making correct inferences about communicative intent. Thus he noted that people do not always mean precisely what they say. As a result, he proposed a distinction between two levels of meaning in an utterance: ‘sentence meaning’ and ‘speaker meaning’. Sentence meaning is the straightforward interpretation that is derived from the linguistic content and grammatical construction, whereas speaker meaning is derived through an understanding of what the speaker intended to communicate. This dichotomy is akin to Searle’s distinction between ‘direct speech acts’, whereby the intention of the speaker is given explicitly by the linguistic form itself, and ‘indirect speech acts’, the comprehension of which requires one to understand what was said explicitly, but move beyond the literal meaning to grasp the speaker’s intent in a specific context (Searle, 1969). Once again, much of what we know about the lateralization of this group of language functions comes from studies of patients with RHD. In essence it seems that damage to the right hemisphere leads to difficulties interpreting words or phrases that have more than one meaning. RHD patients frequently choose the literal meaning over the non-literal, correct meaning. It has been suggested that right hemisphere traces are accessed when subordinate associations of a given word form become more relevant than the dominant (most frequently used) association (Coney and Evans, 1998).

Indirect requests can be thought of as examples of non-literal speech, because they require a response that does not address the literal meaning of the question (Hough and Pierce, 1993). For example, consider the situation in which someone is returning a heavy pile of books to the library. When they arrive at the library they find it difficult to open the door because they have no free hands. At that moment someone else approaches the library. The person carrying the books asks ‘could you open the door for me?’. If this request were interpreted literally, the second person would reply ‘yes’ or ‘no’. However, people who do not have difficulty interpreting indirect requests would realize that the first person’s communicative intent was actually to ask if he or she would open the door for them. In one of the prominent studies of the
neural basis of indirect requests, Foldi presented participants with line drawings in which one character delivered an indirect request to another character. Participants were then shown a second drawing that depicted the second character responding to the request either a literal or non literal (correct) manner. (Foldi, 1987). Participants’ task was to determine whether the second picture they saw depicted what the second character was supposed to do in that scenario. In comparison with healthy controls and patients with LHD, patients with RHD made far more literal scenario identifications than non-literal. This pattern of results has been replicated in studies in which the both the stimuli and response were verbal (Lakoff and Johnson, 1980). Of note are the results of a second experiment by Foldi (1987), in which participants were required to judge actor’s responses to direct commands. In this control task patients with RHD performed no worse than controls or patients with LHD.

A second example of a language function that requires sensitivity to non-literal meanings is the understanding of metaphors. Metaphors are used frequently in everyday conversation, thus an inability to discern their correct meaning could have a major impact on the success of social encounters (Weylman et al., 1989). As with the processing of other forms of non-literal language, patients with RHD experience great difficulty processing metaphors. In a clear demonstration of the link between the right hemisphere and the processing of metaphors, Winner and Gardner (1977) presented participants with a metaphorical expression and then asked them to select which of two drawings best represented the meaning of the expression. For example, one of the verbal expressions participants saw was ‘he had a heavy heart’. Corresponding to this expression was a picture of a person crying (the metaphorical or non-literal meaning) and a picture of a person stumbling along under the burden of a heavy heart tied to his back (the literal meaning). RHD patients selected the inappropriate literal meaning far more frequently than either the LHD patients or healthy controls. However, some researchers have questioned the use of pictorial stimuli, suggesting that the deficits of RHD patients could reflect difficulty interpreting the pictures themselves rather than the metaphors (Joanette, 1990). However, similar patterns of results have been observed with paradigms that used verbal stimuli alone (Bryan, 1988; Brownell et al., 1990). Further support for the supposition that the understanding metaphors is right lateralized comes from functional imaging studies. When Bottini et al. (1994) controlled for brain regions activated by lexical decisions, the prefrontal cortex and middle temporal gyrus regions activated by metaphor comprehension were both in the right hemisphere.

**Emotional prosody**

As is the case for the other higher order language functions discussed so far, accurate interpretation of someone’s emotional state is essential to successful social integration. In addition to what a person says, one of the most potent cues to someone’s emotional state is their tone of voice. Deficits in emotional communication skills may impair the ability to interpret the emotional states of others and to behave in a socially appropriate manner (Trauner et al., 1996), and the ability to interpret emotional cues plays an important role in maintaining successful relationships and healthy psychological functioning (Carton et al., 1999). The use of tone of voice to convey emotional information is more formally known as emotional prosody, prosody being ‘that faculty of speech which conveys different shades of meaning by means of variations in stress and pitch—irrespective of the words and grammatical construction’ (Monrad-Kohn, 1947). Whilst most of the research on the neural basis of emotional processing has focused on detecting facial emotion, there is little clear evidence that such processing is an asymmetrical function. In contrast, there is strong evidence that the processing of emotional prosody is lateralized and, in particular, that it is lateralized to the right hemisphere.

The abilities of patients with right hemisphere lesions once again provide the main source of evidence as to the lateralization of this function. Patients with RHD find it difficult to identify the emotion conveyed by a speaker’s tone of voice (Ross and Mesulam, 1979; Ross, 1981; Ross et al., 1981; Heilman et al., 1984; Code, 1987; Gorelick and Ross, 1987; Blonder et al., 1991; Starkstein et al., 1994; Schmitt et al., 1997). Depending on the location of the lesion, patients with RHD may also have difficulty expressing emotional prosody (Gorelick and Ross, 1987; Brådvik et al., 1991; Pell, 1999a, b). Thus, whereas the left hemisphere encompasses the neural substrates of sensory (Wernicke’s area) and motor (Broca’s area) phonology, the right hemisphere apparently includes corresponding substrates for interpreting and generating prosodic intonation. On the basis of evidence such as this and studies demonstrating an association between the right hemisphere and gesturing, Bowers and colleagues have suggested that the right hemisphere houses a ‘non-verbal affect lexicon’, analogous to the verbal lexicon in the left hemisphere (Bowers et al., 1993). When RHD patients are given additional visual cues in video clips of actors presenting emotional prosody stimuli, their performance is still below that of LHD patients and healthy controls (Schmitt et al., 1997). It is worth noting at this stage that the right hemisphere does not seem to have control over the ability to use and understand emotional words themselves, since patients with RHD are still able to identify the emotion conveyed by verbal content when the prosody is neutral (Blonder et al., 1991; Lalande et al., 1992).

It has been suggested that the deficits of RHD patients may be due in part to the distractions of concurrent semantic information (Bowers et al., 1987). Although RHD patients’ performance is indeed worsened when the emotions conveyed by verbal content and prosody are incongruent (compared with when they are congruent) (Bowers et al., 1987; Lalande et al., 1992), so is that of LHD patients, albeit to a lesser degree. Furthermore, when the speech frequencies that carry semantic information are electronically filtered
out, patients with RHD are unable to deduce the emotion conveyed by these ‘pure’ emotional prosody stimuli (Bowers et al., 1987; Lalande et al., 1992). Thus, there is some evidence of a distraction deficit as a consequence of RHD, but this occurs in addition to an emotional prosodic processing deficit; it is not a substitute explanation. However, since RHD patients’ performance is worse in the incongruent condition, one can infer that the more difficult the emotional prosodic processing task, the greater the demands on right hemisphere resources. This inference is supported by Tompkins and colleagues’ demonstration of an increase in RHD patients’ deficits as the cognitive load of their task was increased by progressing from the identification of emotion conveyed by ‘pure’ emotional prosody, through same/different judgements of pairs of emotional prosody stimuli, to labelling judgements from four alternative emotions (Tompkins and Flowers, 1985; Tompkins, 1991). Conversely, the bigger the right hemisphere lesion and the bigger the consequent reduction in right hemisphere processing resources, the worse the emotional prosodic comprehension deficit (Brådvik et al., 1991). Similar gradations in severity of deficit have been observed in patients with RHD performing emotional prosody expression tasks (Ross et al., 1997).

Although pitch is probably the main element of prosody that enables us to determine emotion (Van Lancker and Sidis, 1992; Pell and Baum, 1997a), and the perception of pitch is known to be right lateralized (e.g. Riecker et al., 2002), it is not the case that the processing of all prosody is right lateralized. Stress prosody is a non-emotional type of prosody in which changing the placement of stress can alter the meaning of an utterance, e.g. from a declarative statement to a question. Patients with LHD are able to detect the emotion conveyed by emotional prosody but they cannot detect the effects of stress prosody on meaning (Shapiro et al., 1985; Emmorey, 1987; Pell and Baum, 1997b). Comprehension of stress prosody is therefore left lateralized. It may be the case that certain basic components of prosody are processed in the right hemisphere, but that complete processing of stress prosody relies on lexical information stored in the left hemisphere.

In normal participants, complementary studies reinforce the association between the right hemisphere and emotional prosodic processing. For instance, dichotic listening studies have shown a distinct left ear advantage for the comprehension of emotional prosody (Behrens, 1985; Shipley-Brown et al., 1988; Herrero and Hillix, 1990; Grimshaw et al., 2003). Owing to the crossed nature of the auditory pathways, a left ear preference can be interpreted as indicating right hemisphere superiority. In these studies, auditory stimuli are presented separately to the left and right hemispheres. When the task is to make a judgement about the semantic meaning of the stimulus, participants respond faster and more accurately if stimuli are presented to the left ear/right hemisphere. Administration of the Wada test is commonly used to determine language lateralization in patients with epilepsy prior to surgery to alleviate their condition. First developed by Wada, it involves the injection of sodium amobarbital into the left or right carotid artery, thereby anaesthetizing one hemisphere whilst the functions of the other hemisphere are studied in isolation (Wada and Rasmussen, 1960). Application of the Wada test to healthy right-handed people has shown that without intact functionality of the right hemisphere, participants are unable to modulate their tone of voice to convey emotion (Ross et al., 1988). Whilst the dichotic listening test procedure may be criticized on the grounds that the auditory pathways are not fully crossed and the Wada test ignores the possibility of inter-hemisphere cooperation, functional imaging does not suffer from either of these weaknesses. Both PET and fMRI confirm the right lateralization of emotional prosodic processing. When controlling for lexical processing, the comprehension of emotional prosody activated brain regions in the right hemisphere, and when the activity of the left and right language centres is compared directly, the region of interest in the right hemisphere is more active than that in the left (George et al., 1996; Buchanan et al., 2000).

Furthermore, Mitchell et al. (2003) have demonstrated that emotional prosodic processing is right lateralized in a wide range of paradigms. Passive listening to emotional prosody versus rest, emotional prosody versus neutral prosody and ‘pure’ emotional prosody versus rest, all activated the right lateral temporal lobe to a greater extent than the left. Even when participants were presented with stimuli in which the emotions conveyed by verbal content and prosody were incongruent, the resultant brain activity was still right lateralized. Active attention to emotional prosody also induced a right lateralized pattern of brain activity, whereas attention to the verbal content of the same stimuli induced left lateralized activity.

**Right hemisphere language functions in schizophrenia**

Previous authors (e.g. Cutting, 1990, 1994; David, 1994) have argued for a role for the right hemisphere in the pathophysiology of schizophrenia. In our search for evidence as to the right hemisphere language capabilities of patients with schizophrenia, we concentrated on studies that specifically focused on this hemisphere’s language functions and in which patients were recruited that met standard operational diagnostic criteria. Studies were preferred in which patients and controls were matched for age, gender and number of years of formal education.

**Difficulties comprehending and producing discourse**

Whilst most other research on language dysfunction in schizophrenia has concentrated on one or two specific abnormalities, it may be equally important to consider the...
whole range of a speaker’s output/listener’s understanding; it may be just as important to examine patients’ ability to engage in discourse (Hotchick and Harvey, 1986). One of the principal early works on discourse in schizophrenia is the book *Crazy talk* (Rochester and Martin, 1979). The authors focused on what Bleuler (1911) termed ‘looseness of associations’, in particular cohesive ties (connections between clauses) and references (cue word or forms that indicate that the listener needs to retrieve and use previously presented information). Their analyses showed that patients with schizophrenia make less use of cohesive ties than normal controls, and that they failed to provide adequate references (patients presumed they had given information crucial for the listener’s understanding but they had not). Rochester (1978) summarized this work as follows. ‘Normally we would expect that speakers maintain some constant lag between an item and its referent in the verbal context. The lag may be of the order of two to three independent clauses, and it may vary with the situation and the interpersonal relationship between the speaker and listener. However, in a brief conversation the lag should be small and approximately constant. For the schizophrenic and especially the thought disordered schizophrenic, we would expect the lag to be broad and variable. If the speaker does not recall clearly how and when an item was referenced, he/she should confuse the listener by the use of widely diverging referencing distances.’

A second early seminal work was that of Chaika (1974). According to Chaika, the abnormal nature of schizophrenic discourse was best described as ‘a disruption in the ability to order linguistic elements into meaningful structures’. Among the more noticeable abnormalities in the speech samples she collected were ‘production of new sentences according to phonological and semantic features of previously uttered discourse rather than according to topic’ and ‘failure to monitor own speech’. The findings of modern analyses of schizophrenic discourse have confirmed these early assertions. Noël-Jorand and colleagues’ application of computer-assisted discourse analysis again demonstrated a lack of cohesion in patient speech samples, particularly the presence of ‘language satellites’ consisting of short secondary discourse that had no relevance to the main discourse (Noël-Jorand et al., 1997). Thus, it seems that the speech of patients with schizophrenia violates Grice’s maxims of successful discourse discussed above. Hoffman et al. (1982) have attributed these abnormalities to a failure to generate a plan that reflects the gist or intention of what will be said. Indeed, Docherty et al. (2000) have demonstrated that discourse abnormalities in the speech of patients with schizophrenia (particularly confused, missing, incorrect or vague references) are strongly correlated with poor performance on a conceptual sequencing task. A systematic series of tests by Barch and Berenbaum (1997) demonstrated that not only do patients with schizophrenia find it difficult to generate a discourse plan (as evidenced by a drop in performance when given no cues to plan a discourse versus when given background information on what they would be asked), but once engaged in conversation they also find it difficult to maintain discourse (as evidenced by a drop in performance when not given structured questions to initiate a dialogue versus when prompted with structured questions).

Parallel experimental studies have sought to test specific aspects of patients’ understanding of discourse by systematically manipulating certain discourse parameters. Kuperberg et al. (1998) employed an on-line word monitoring task in which participants were first presented with a written target word and then asked to listen to prerecorded sentences and press a button when they heard the target word being spoken. Some of the test sentences contained linguistic violations in which the verb preceding the target word was replaced by another verb that made the sentence pragmatically implausible e.g. ‘the crowd was waiting eagerly; the young man buried the guitar’. The comparison sentences did not contain pragmatic violations, e.g. ‘the crowd was waiting eagerly; the young man grabbed the guitar’. Whereas healthy controls took longer to recognize words preceded by such linguistic anomalies, patients with schizophrenia appeared to be relatively insensitive to linguistic violations since they exhibited much smaller differences in reaction time. In another study, Tényi et al. (2002) used test stimuli that violated the expected relevance of written discourse such that the first sentence in a vignette described a relationship between two people and implied the suitability of person A to comment on some facet of person B, but what person A then said unexpectedly implicated a negative opinion behind their utterance. For example, in one of the vignettes a professor was asked to give his opinion about his junior lecturer and answered ‘she is a female’. In contrast to healthy controls, patients with schizophrenia made significantly more mistakes when trying to decode person A’s true communicative intent.

It has been suggested by some researchers that discourse abnormalities are diagnostic of schizophrenia; that is to say that the profile of discourse production in schizophrenia is distinct from that in other psychiatric disorders (Morice and Ingram, 1982; McPherson and Harvey, 1996). Ceccherini-Nelli and Crow (2003) have gone so far as to suggest that these language disturbances are superior to traditional nuclear symptoms in discriminating schizophrenia from other psychoses. Evidence of discourse abnormalities in direct relatives of patients suggests that these dysfunctions may be associated with a familial predisposition towards schizophrenia (Docherty et al., 1998, 1999; see also Loftus et al., 1998). Whilst studies such as these have led to the claim that discourse abnormalities are a trait effect of schizophrenia rather than a state effect within the illness (Condray et al., 1995), current evidence suggests that their severity may be linked to symptomatology. The exact nature of this relationship remains unclear. Discourse abnormalities have at times been linked to the prevalence of delusions (Robbins, 2002), thought disorder (Barch and Berenbaum, 1996), positive symptoms as a whole (Allen and Allen, 1985), negative symptoms (Corcoran and Frith, 1996) and global measures of function (Byrne et al., 1998). Alternatively, it has also been
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proposed that discourse abnormalities are not primary consequences of schizophrenia per se, but that they are secondary consequences of some other aspect of the disease. For instance, Melinder and Barch (2003) have demonstrated that in patients with schizophrenia, discourse cohesion deficits correlate with working memory deficits, and that when discourse production competed for working memory resources in a category monitoring task, patients’ deficits worsened. Thus, discourse abnormalities in schizophrenia could be related to concurrent working memory dysfunction. They could also be a consequence of more general context processing deficits, since patients with schizophrenia demonstrate an inability to act appropriately on contextual information across a range of cognitive tasks, including lexical disambiguation in the context of written discourse (Cohen et al., 1999). Harvey et al. (1986) propose a third possibility, that discourse abnormalities are related to attentional deficits via an inability to deal with distracting information. In Harvey et al.’s study, ambiguous references in patients’ discourse were associated with poor performance on a task requiring them to ignore the presentation of distracting inter-stimulus numbers and only identify target numbers. The difficulty in assessing these alternative proposals (category monitoring, working memory and attentional deficits) is to relate these functions to linguistic capacity, and indeed to determine what the neural basis of the latter (human-specific) faculty is.

**Difficulties processing alternative meanings**

It has been known since the 1940s that patients with schizophrenia find it difficult to discriminate the correct meaning of metaphorical statements (Kasanin, 1944). Chapman (1960) demonstrated that the basic deficit in patients with schizophrenia was a tendency to make literal interpretations rather than figurative ones. This pattern has been replicated many times (Cutting and Murphy, 1990; Drury et al., 1998; Goldberg and Weinberger, 2000) and it appears to be specific to schizophrenia, since these deficits are not present in related psychiatric disorders such as schizotypal personality disorder (Langdon and Coltheart, 2004). Other test paradigms suggest that patients’ dysfunction is genuine (and not an artefact of the literal/figurative choice paradigm) and that the nature of their deficit is broader than was first thought. Thus patients with schizophrenia are poor at judging whether a sentence represents a plausible or implausible metaphor (Corcoran, 1999), they find it difficult to choose the metaphor that best matches the meaning of a proverb (De Bonis et al., 1997) or to judge whether a metaphor is appropriate given a particular situation (Langdon et al., 2002), they still favour literal meanings when asked to choose a picture that best represents the meaning of a metaphor (Anand et al., 1994) and they are unable to complete a sentence to create a metaphor by selecting a word from a selection of alternatives (Drury et al., 1998). Since difficulty appreciating metaphors is seen in first-episode psychosis, it has been suggested that it is a direct consequence of the disease itself rather than a consequence of chronicity factors such as long-term medication effects (Anand et al., 1994). However, Drury and colleagues’ results suggest that difficulty appreciating metaphors may be more accurately thought of as a consequence of acute schizophrenia. Their study of patients who had suffered schizophrenia for at least 5 years showed that metaphor appreciation was normal during remission (Drury et al., 1998). This explanation is consistent with evidence relating metaphor appreciation difficulty to symptoms strongly associated with acute schizophrenia such as delusions (Rhodes and Jakes, 2004), but other researchers claim an association with negative symptoms (Langdon and Coltheart, 2004). Associated cognitive phenomena in patients with schizophrenia are purported to include memory dysfunction (Corcoran, 1999), theory of mind dysfunction (Langdon et al., 2002) and general executive dysfunction (Langdon and Coltheart, 2004). Again, the question arises of the relationships between theory of mind and executive interpretations and the components of working memory, and the relationship of the latter to the capacity for language.

There are fewer studies of the ability of patients with schizophrenia to infer communicative intent from indirect requests; however, the evidence that does exist clearly suggests that patients have difficulty interpreting this class of multiple meanings too. In 1995, Corcoran explicitly developed the ‘hinting task’ to assess this function in patients with schizophrenia (Corcoran et al., 1995). In the hinting task, participants are required to listen to a short passage presenting an interaction between two people, especially the hint at the end implying what person A wants person B to do. If the participant fails to infer the real request, an even more obvious hint is given and the participant is again asked what person A wants person B to do. The score of patients with schizophrenia performing this task was significantly lower than that of healthy controls even when between-group differences in IQ were taken into account. Of the different symptom subgroups studied, patients with negative symptoms performed the worst. In a later study, patients with schizophrenia again performed worse than controls on the hinting task (Corcoran, 2003). This second study demonstrated that patients’ performance was positively correlated with their performance on other cognitive tasks including a story recall test, a problem solving task and an inductive reasoning task. These results therefore suggest that the inability of patients with schizophrenia to infer meaning in indirect requests may be related to more general thought or verbal memory dysfunctions. Although data on the symptom status of patients was provided (primarily positive symptoms, primarily reality distortion symptoms or remitted), patients’ ability to infer from indirect requests was not analysed according to symptom group. It is therefore unclear how this deficit relates to schizophrenic symptomatology.

As discussed above, humour, sarcasm and irony are a third class of language functions that require the listener to evaluate multiple meanings and discern which meaning best
reflects a speaker’s communicative intent. Clinical anecdotes frequently report a distorted sense of humour in patients with schizophrenia, and experimental studies suggest that this dysfunction is again related to patients’ preference for literal (non-humorous) meanings over figurative (humorous) interpretations. It has been demonstrated experimentally that when presented with cartoon jokes, patients with schizophrenia often fail to ‘get’ the joke (Rosin and Cerbus, 1984; Corcoran et al., 1997). In Corcoran et al.’s study, patients were presented with cartoon jokes and asked to explain them. As predicted, there was a significant difference between controls and patients in the number of jokes they were able to explain. When presented with jokes that required a viewer to make inferences about a character’s state of mind, these deficits were worse, independent of symptomatology. Corcoran et al. (1997) therefore suggested that humour appreciation deficits in schizophrenia are related to dysfunctional theory of mind abilities. The performance of patients in remission was, however, much closer to that of healthy controls. Drury et al. (1998) have similarly found that the inability to appreciate irony is attenuated during remission, although they question whether these deficits are related to theory of mind dysfunction in all patients. Evidence that patients with schizotypal personality disorder also have difficulty appreciating irony strengthens the association between this dysfunction and the schizophrenic illness (Langdon and Coltheart, 2004). Rosin and Cerbus’ work suggests that not only do patients have difficulty appreciating humorous material, but conversely that they often find material humorous that is not meant to be so (Rosin and Cerbus, 1984). When the authors presented participants with photographs and asked them to rank 14 possible captions in order of humour, patients perceived the task as stressful and claimed that they did not find the captions amusing. When asked to produce their own captions for the photographs, patients produced captions that were later deemed by college students to be low in humour. Patients with schizophrenia may therefore have difficulty appreciating and producing humour.

**Difficulties comprehending and producing emotional prosody**

It has been estimated that up to 84% of patients may display deficits of either comprehension or expression of emotional prosody (Ross et al., 2001). Several studies have demonstrated that when patients with schizophrenia are asked to identify the emotion conveyed by a speaker’s prosody, their performance falls well below that of psychiatrically normal people (Jonsson and Sjöstedt, 1973; Leff and Abberton, 1981; Fricchione et al., 1986; Murphy and Cutting, 1990; Leentjens et al., 1998; Edwards et al., 2001; Ross et al., 2001). This deficit is present at the sentence level and persists right down to the lowest levels of task demand, i.e. it is still present at the monosyllable level (Ross et al., 2001). A non-specific emotion-labelling deficit can be excluded, since schizophrenics also perform poorly when asked to discriminate which of two emotions is conveyed by prosody (Ross et al., 2001). Indeed, many other alternative explanations have been systematically ruled out. When the potential confound of verbal content is removed by filtering out the speech frequencies that carry semantic information, patients with schizophrenia are still unable to reliably identify the emotion conveyed by prosody (Ross et al., 2001). Where a difference in number of year’s formal education exists between patients and controls and this information is entered into the analysis as a covariate, the deficit remains significant (Leentjens et al., 1998). A general prosodic comprehension deficit can be ruled out for two reasons: patients with schizophrenia have no difficulty comprehending non-emotional prosody (Murphy and Cutting, 1990) and when tested on their perception of individual components of prosody such as tone, duration and amplitude, their discriminatory performance is also normal (Jonsson and Sjöstedt, 1973). There are some suggestions that patients’ performance might be related to symptomatology, particularly to the severity of their negative symptoms (Fricchione et al., 1986), but other studies have not upheld such a link (Jonsson and Sjöstedt, 1973; Ross et al., 2001). Edwards et al. (2001) have addressed the idea that emotional prosodic comprehension deficits may be an artefact of illness chronicity or institutionalization, but their study of patients in the first episode of schizophrenia found no evidence to support this hypothesis. In summary, patients with schizophrenia seem to have a genuine difficulty identifying the emotion conveyed by prosody; this deficit is a trait rather than state effect.

Deficits in the expression of emotional prosody may be as pervasive as the comprehension deficits displayed in schizophrenia. When asked to repeat words or phrases in a specific emotional tone of voice and this performance is rated by a panel of judges, patients with schizophrenia perform badly compared with controls (Fricchione et al., 1986; Murphy and Cutting, 1990; Leentjens et al., 1998; Ross et al., 2001). Again, this deficit is present at both the sentence and word levels (Ross et al., 2001) and again it appears to be unrelated to medication (Edwards et al., 2001) or number of year’s formal education (Leentjens et al., 1998). Murphy and Cutting (1990) suggest that this expression deficit cannot be explained as a general prosodic expression deficit, since non-emotional prosody appears to be expressed correctly. Studies that have monitored patients’ general use of prosody in conversations suggest that patients may sometimes speak in a monotone voice (Leff and Abberton, 1981; Fricchione et al., 1986; Leentjens et al., 1998). In these studies of everyday language, patients’ spontaneous use of prosody is monitored during an initial encounter or following a prompt to discuss a specific topic. Patients’ performance is then scored according to certain criteria, including speech volume, pitch, tone, intonation, vocal quality and rate or rhythm of speech. Comparisons of patients with and without flat affect suggest that spontaneous use of prosody may be particularly attenuated in patients who suffer from flat affect (Leff and Abberton, 1981; Alpert et al., 2000). This pattern of results highlights that schizophrenia is not a homogenous disorder. It has been
suggested for example that there are two (positive and negative symptoms; Crow, 1980; Crow, 1985) or three (positive and negative symptoms and thought disorder; Liddle, 1987) principal dimensions of variation in the psychopathology of schizophrenia, and that these have differing neural correlates. Future studies must therefore consider symptomatology and/or subtype of schizophrenia more closely than has been the case so far (Edwards et al., 2002). One possibility is that negative symptoms (poverty of speech, flattening of affect) reflect motor (frontal) deficits and positive symptoms sensory (occipito-temporo-parietal) anomalies, while thought disorder represents an overall failure of sensorimotor integration with respect to language. For example, reduced spontaneous use of prosody may be limited to patients with flat affect or prosodic comprehension abilities may be selective to patients with positive symptoms, e.g. delusions. Current evidence is that the specific deficit of emotional prosody expression is not as strong as it is for comprehension. However, these uncertainties do not negate the fact that most patients have difficulty expressing emotion through their tone of voice.

The implications of right hemisphere dysfunction for abnormal lateralization in schizophrenia

It has sometimes been argued that the psychological deficits associated with schizophrenia are generalized, i.e. that patients perform worse than control subjects on most cognitive tasks (Blanchard and Neale, 1994; Heinrichs and Zakzanis, 1998). According to this view, the deficits are compatible with a non-specific cortical dysfunction, and some evidence from imaging (Wright et al., 2000) and post mortem studies (Harrison et al., 2003) is consistent with a non-selective process. We argue on the contrary that the more one considers the function that is most characteristic of Homo sapiens, i.e. language and the feature that we take to be the defining characteristic of the human brain—the cerebral torque, the more one can make sense of the individual symptoms and the more readily a possible anatomical basis can be discerned. Thus, the core symptoms can be considered as anomalies of the individual components of language (see below), the anatomical changes can be construed as losses or reversals of shape or volume asymmetry of particular structures such as the planum temporale (Pett, 1999; Sommer et al., 2001), the superior temporal (Highley et al., 1999), fusiform and parahippocampal (McDonald et al., 2000) gyri, and the functional changes as losses or reversals of lateralized activation of areas of association cortex relating to particular components of perception or production of language (Mitchell et al., 2004).

Applicability of pre-existing neuropsychological theories

In the neuropsychology of language literature there are frequent examples of cases whereby following a neurological insult to language processing regions in the left hemisphere, the brain is able to reorganize itself somewhat, and homologous structures in the right hemisphere consequently play a greater role in the mediation of that function. However, the right hemisphere structures previously subserved by the new mediator are inevitably affected by this reorganization and are therefore compromised. Could this phenomenon explain the effects of abnormal lateralization for language and right hemisphere dysfunction in schizophrenia?

The transmigration of certain language functions from the left to the right hemisphere in schizophrenia could thus be the cause of the right hemisphere dysfunctions reviewed above. For instance, using fMRI, Woodruff et al. (1997) demonstrated that patients with schizophrenia (who experienced auditory hallucinations) revealed a reduced left superior temporal gyrus response to external speech, along with an increased right middle temporal gyrus response. Mitchell et al. (2003) have shown that the right middle temporal gyrus is associated with the comprehension of emotional prosody in external speech, and as reviewed above, patients with schizophrenia are well known to have difficulty with this right hemisphere language function.

However, a causative link between these three phenomena has not yet been demonstrated. Furthermore, this model assumes that in patients with schizophrenia, language functions normally mediated by the right hemisphere retain the same neural basis and remain right lateralized (hence the detrimental effect of left hemisphere function transmigration). This may not be true. It is not yet possible from functional neuroimaging to determine empirically whether right temporal lobe functions such as prosodic processing remain in the temporal lobe in schizophrenia and even whether they remain in the right hemisphere (although see our suggestions on this point below).

A possible solution to the anatomical basis of schizophrenic symptoms

We maintain that the language deficits of patients with schizophrenia can still best be understood as abnormalities of lateralization, but current models need re-assessment. In patients with schizophrenia there is evidence (as reviewed by Crow 1997a; Sommer et al., 2001) that the components of language that are normally segregated to the left hemisphere are less strongly lateralized. That is to say, in schizophrenia, left hemisphere language functions are somewhat right lateralized, or more equally distributed between the hemispheres. However, any model that implicates attenuation of left hemisphere functions needs to consider the impact of any change in left hemisphere functions on language functions normally mediated by the right hemisphere. The fundamental question we suggest is which language functions are mediated by the left and which by the right hemisphere? If lateralization is the characteristic that defines the human brain (the Broca–Annett axiom; Crow, 2004b), differentiation of engrams in one hemisphere from those in the other has
enabled the evolution of a communicative system that is apparently qualitatively distinct from that of other primates. As emphasized by previous authors (Code, 1987; Beeman and Chiarello, 1998; Cook, 2002), there is no simple segregation of language to the left hemisphere, but a separation of functional components between the hemispheres. Core components in the dominant hemisphere are the motor and sensory phonological engrams in Broca’s and Wernicke’s areas respectively. While some part of the associated ‘meanings’ of these verbal engrams may be encoded in the surrounding cortex, language is not a simple reflex activity. Alternative meanings of words have to be considered (Coney and Evans, 1998) and their use appropriate to a given situation evaluated. Language is bi-hemispheric: if phonological engrams are segregated to the left this allows some part of their associations to be segregated to the right. Only in this way is the multiplicity and arbitrariness that De Saussure (1916) emphasized of the associations between the signifier (the sound pattern) and its signifieds (meanings) established.

However, any attempt to formulate a theory of the neural basis of language must take into account the anatomical implications of the torque: that it is not that one hemisphere differs in volume or overall structure from the other but that there is a bias from right frontal to left occipital across the antero-posterior axis, such that the respective quadrants appear more prominent on one side relative to the other (Witelson and Kigar, 1988). The precise nature of the bias is of great interest. It has been suggested that it is merely that the cortex on one side is thinner and broader than that on the other (Harasty et al., 2003). If this is so it presumably entails that the terminal connections of inter-hemispheric fibres of the corpus callosum and anterior commissure in the two hemispheres are different, and that this imposes a directionality on these pathways. But the important point (often overlooked) is that as a consequence of the torque, the directionality differs in relation to frontal (in principle motor) and occipito-temporo-parietal (sensory) heteromodal association cortex, the convergence being from right to left anteriorly and from left to right posteriorly. The implication is that the relationship between the two sides of the brain should be considered separately with respect to motor and sensory functions, and that the neural principle that separates the phonological engram from its associated meanings in the non-dominant hemisphere with respect to speech production differs in direction from the neural principle that relates to speech perception. Specifically, it is suggested that the process of speech production involves a transition from engrams that represent the precursors of speech (‘thoughts’ and ‘intentions’), which are at least in part located in right dorso-lateral pre-frontal cortex, to the linear phonological output that is assembled in and around Broca’s area in the dominant hemisphere, and that the perception of speech occurs in part by activation of remote associations in right occipito-temporo-parietal cortex (the extraction of ‘meanings’) of the activated primary sensory engrams in and around Wernicke’s area (Crow, 1998, 2004a).

This formulation identifies two problems that require a solution: the nature of the interaction between the motor and sensory components of the speech process in the dominant and non-dominant hemispheres. Concerning the first, it is clear that the motor engrams in Broca’s area are qualitatively different from, although closely related to, the sensory engrams in Wernicke’s area. Since the words that one can articulate are formed on the basis of the words that one hears, the two must develop together, presumably being associated through the arcuate bundle in the dominant hemisphere. However, if activation of motor engrams entails activation of sensory engrams, this clearly is not experienced as perceived speech. One does not normally hear the words that one speaks except through the ear. Conversely, activation of motor speech engrams by perceived speech is a pathological phenomenon (echolalia).

The second question concerns the differentiation of ‘meanings’ from ‘thoughts’ and ‘intentions’ in the non-dominant hemisphere. The distinction between sensory and motor aspects of these linguistic associations (de Saussure’s ‘signifiers’) must be based in part on their differing connexions (dependent upon the torque) with the primary phonological engrams in the left hemisphere. The interaction between ‘meanings’ and ‘thoughts/intentions’ presumably occurs through the arcuate and uncinate bundles in the non-dominant hemisphere, and the basis of this interaction (e.g. the directionality and filtering properties of the neural connexions) is of particular interest. On this interaction is built the structure of discourse and the use of metaphor, aspects of right hemisphere function that are disturbed in schizophrenia.

David (1994) has drawn attention to recent concepts of working memory in relation to both lateralization and the phenomena of psychosis. Whereas working memory is clearly a function that crosses species boundaries, the concept as applied to man separates two components, a phonological loop and a visuospatial sketchpad or ‘scratchpad’, conceived as ‘slave’ systems, that together support the central executive. The phonological loop has a rehearsal function that presumably includes motor and sensory engrams in Broca’s and Wernicke’s areas, respectively. The visuospatial sketchpad constitutes a transient activation of a memory store (Gathercole and Baddeley, 1993) more readily related to the right hemisphere. Both are ‘controlled’ by the decision-making central executive operating, appropriately for a motor component, from somewhere in the frontal lobes. We suggest that it is the separation of the two components of working memory and their relationship to the differential function of the two hemispheres in language that is relevant to psychosis. Thus, the phenomena of psychosis (specifically the pragmatic and prosodic anomalies) may cast light on the interaction of the visuospatial sketchpad with the phonological loop.

We propose that the components of language (and working memory) should be considered within a concept of the brain that is four-chambered with respect to areas of heteromodal association cortex, these being divided along the
antero-posterior axis into antero-motor and posterio-sensory, as well as across the midline into left and right. According to this concept, the phonological loop has motor and sensory components focusing on Broca’s and Wernicke’s areas, respectively, and the visuospatial sketchpad likewise has a sensory component in right dorso-lateral prefrontal cortex and a motor component in right dorso-lateral prefrontal cortex. Thus, in the human brain (as a result of the torque) each of these components has a distinct function. The phenomena of psychosis, the result of a deviation in the connexions between them, cast light on the nature of the segregated functions.

The principal nuclear symptoms can be interpreted (Crow, 1998, 2004a) as follows:

(i) Thought insertion and withdrawal (together with other passivity experiences) result from activation or deactivation of normally autonomous ‘thoughts’ and ‘intentions’ in right dorso-lateral prefrontal cortex by incoming sensory stimuli.

(ii) Thought echo and running commentary result from activation of neural traces in left occipito-temporo-parietal cortex that are normally engaged in speech perception by self-generated plans for speech and action that arise in right dorso-lateral prefrontal cortex.

Thus these symptoms are ‘spill-over’ phenomena that reveal aspects of the distinct roles in language of the four areas of heteromodal association cortex.

The four-quadrant concept is represented in Fig. 1. This diagram allows one to ask questions such as the following:

(i) What is the relation between working memory and the components of language?

(ii) What are the implications of the convergences and divergences in the inter-connections between the four quadrants of heteromodal association cortex?

(iii) Is there a one-way route through the four quadrants? If so what prevents back-flow?

We assume that the effect of the torque is to bias transmission within the system such that traffic of information is typically in the direction left occipito-temporo-parietal → right occipito-temporo-parietal → right dorso-lateral prefrontal → left dorso-lateral prefrontal, with the convergences and divergences representing qualitatively distinct operations. Thus the inter-hemispheric convergences from left to right posteriorly and from right to left anteriorly can be seen as associated with distillations of meaning from perceived speech and of the spoken sentence from its precursor thoughts and intentions, respectively. This leaves a function to be attributed to the intra-hemispheric expansions from posterior to anterior in the right hemisphere and from anterior to posterior in the left hemisphere. We suggest these connections provide access to a reservoir of engrams in each case: in the right hemisphere to the store of recently activated meanings of perceived events and in the left hemisphere to the store of perceived phonological engrams and their immediate associations. It should be noted that according to this scheme the form of access in the two hemispheres is different: in the right hemisphere it is to an already distilled and perhaps recently activated subset and in the left hemisphere it is to a wide set of word patterns.

The four-quadrant concept, together with evidence (Crow, 1997, 2004c) that there is a failure of development of aspects of the anatomical differentiation of the hemispheres, provides a framework for understanding the phenomena of psychosis. The production of coherent discourse, for example, is clearly a function of speech planning on the basis of an understanding of the problem in hand. The primary interaction is between the plan that is formulated in dorsolateral prefrontal cortex and the understanding that is established in occipito-temporo-parietal cortex in the right hemisphere. If these functions are not clearly differentiated, speech becomes incoherent and arguably this is the core feature of schizophrenia (Bleuler, 1911).

It was suggested by Jaynes (1979) and Nasrallah (1985) that auditory hallucinations arise in the right hemisphere, and perhaps for that reason lack the characteristic of being self-generated. On the basis of the four-chamber concept one can see that some leakage (‘back-flow’) of the neural activity associated with thoughts and intentions in right dorsolateral prefrontal cortex into right occipito-temporo-parietal cortex could present the primary perceptive cortical fields in the left hemisphere with an abnormal input. On the assumption that activations of the left occipito-temporo-parietal cortex are ipso facto treated as coming from the external world, such activation would have the characteristics of an hallucination. Conversely, leakage in the opposite direction, from right posterior to anterior, of activity with the characteristics of external input that activated motor engrams in the right hemisphere with an abnormal input. On the assumption that activations of the left occipito-temporo-parietal cortex are ipso facto treated as coming from the external world, such activation would have the characteristics of an hallucination.

Fig. 1 The normal volume bias from right frontal to left occipital across the antero-posterior axis the ‘four quadrant’ concept.
dorso-lateral prefrontal cortex could account for the passivity phenomena of thought insertion and made volitions.

David (1994) built a hypothesis of the origin of psychotic symptoms around the model of speech perception and production of Ellis and Young (1988). David’s hypothesis has similarities with the four-chamber concept proposed here. One similarity is that ‘thoughts originate in and are understood by the semantic system’, while a ‘voice is experienced at the phonological level’. A difference is that according to David’s schema there are five chambers—an auditory analysis system and a phoneme level output to accommodate speech perception and production, separate auditory input and speech output lexicons, and a common semantic system that is fed by the input lexicon and feeds out to the output lexicon—rather than the four chambers proposed here. There appear to be ambiguities in that thought is attributed to the lexical as well as to the semantic level, and that neither the semantic nor the lexical level is clearly associated with the right hemisphere.

We suggest that this scheme can be made more precise by considering the roles of specific areas of heteromodal cortex on either side of the brain as a quadripartite system under the influence of the cerebral torque. The sensory and motor phonological representations (the auditory analysis system and the phoneme level output in Ellis and Young’s terminology) are clearly located in the left hemisphere (we consider the possibility that this is the primary consequence of lateralization and that lateralization of the affective component to the right is secondary). Associated with the phonological engrams in the left hemisphere, however, are more complex neural patterns that constitute some part of the signifieds or word meanings. These make up a substantial part of what Ellis and Young refer to as the auditory input and speech output lexicons. They can perhaps be referred to as the primary lexicons. However, a second part of the lexicon, comprising more remote, variable and often affectively charged associations, is located in the right hemisphere. As is our main point, this lexicon also is divisible into separate sensory and motor components and these components are functionally distinct. Thus, in the sensory field the associations are diffuse and yield the potential to identify meaning at the level of the sentence or discourse. The motor engrams in right dorso-lateral prefrontal cortex are two stages removed from the linguistic input and thereby enjoy a degree of autonomy. They have access to the meanings that are identified in the sensory field, and on this foundation are generated the thoughts and intentions that form the substance of the individual’s sequences of speech and action.

Thus, our concept of the function of the right hemisphere is that it constitutes a secondary lexicon or a lexico-semantic store within which the separation of a motor from a sensory component gives rise to distinction between meanings on the one hand, and thoughts and intentions on the other. The separation of the hemispheres allows these components a degree of independence from the primary modules concerned with speech perception and production. In this way are generated the flexibility of representation and the diversity of association that are characteristic of human language.

We take an evolutionary view of the origins of psychosis—that schizophrenia and language have a common origin in the genetic event 100 000 to 150 000 years ago that gave rise to Homo sapiens as a species (Crow, 1995, 1997b, 2000). At that point, either asymmetry (the genetic basis of torque) was introduced as a new feature or an asymmetry that had entered at some earlier point in the hominid lineage (see Steele, 2002) was critically modified to give rise to the capacity for language. Thus the two hemispheres assumed differentiated roles in supporting the components of the capacity for language.

According to this concept, the genetic predisposition to psychosis represents a component of the variation (probably epigenetic in character) in the population associated with cerebral asymmetry and that capacity for language. The nuclear (Schneiderian) symptoms themselves represent a disintegration of the components of language, specifically the passivity phenomena reflect a failure of the transition from thought to speech production and action, and some positive symptoms (voices spoken aloud and running commentary) reflect inappropriate activation of the phonological engrams for speech perception in the dominant hemisphere (Crow, 2004a, c).

We have focused on the symptoms of typically schizophrenic illnesses because they represent the greatest departure from the mean of the normal population, but there are cogent reasons for considering schizoaffective and affective psychoses as being within the same spectrum (Crow, 1991; Kendell, 1991; Maier et al., 1993; McGorry et al., 1998): the symptoms of these states, e.g. pressure and retardation of speech, grandiosity and nihilistic content of delusions, may throw further light on the relative contribution of the two hemispheres.

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