The aprosodias: further functional-anatomical evidence for the organisation of affective language in the right hemisphere

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SUMMARY   Fourteen right-handed patients with right hemispheric strokes were examined for disorders of affective language in order to further define the clinical-anatomical correlates of the aprosodias. A bedside evaluation strategy and CT scan mappings were utilised to make these comparisons. There were six patients with motor aprosodia, one with global aprosodia, two with conduction aprosodia, one with sensory aprosodia, one with transcortical sensory aprosodia, one with pure affective deafness and two with normal examinations of affect. Functional-anatomical correlations were consistent with those predicted previously. Recovery of affective language function in selected cases was characterised by improvements in affective-prosodic repetition and/or evolution into other aprosodic subtypes. Our results lend further support to the hypothesis that the organisation of affective language in the right hemisphere mirrors that of propositional language in the left hemisphere.

Because the left hemisphere plays a major role in the neural processes governing the production and comprehension of propositional language, it has been designated as the "dominant" hemisphere. The propositional attributes of language are principally concerned with lexicon, grammar and articulation. However, there are other features of speech beyond lexicon, grammar and articulation, subsumed under the term prosody, that involve, for example, melody, pauses, timing, stress, accent and intonation. In addition to clarifying potentially ambiguous syntax and adding pragmatic factors to speech, prosody is also the vehicle by which speakers insert attitudes and emotions into their dialogue. Although Hughlings-Jackson[1] recognised the clinical importance of affective communication almost 100 years ago, it was not until the 1940s that a systematic inquiry into pathological disturbances of prosody was first undertaken by Monrad-Krohn.[2-5] More recently, the neurology of the affective components of language, encompassing attitudes and emotions, has been fur-

ther delineated.[6-24] Studies by Heilman *et al*,[11] Tucker *et al*[12] and Ross *et al*[16 17 19] suggest that these particular aspects of language appear to be dominantly governed by the right hemisphere and allow spoken language to acquire affective tone through the use of prosody and gesturing. Without them, the expression and comprehension of affectively charged speech may be seriously impaired. The different syndromes of affective deficits following focal right brain damage have been called aprosodias[16] and appear to be analogues of the aphasias which follow focal brain damage. Since the concept of aprosodias is relatively new, we decided to study patients with right hemispheric strokes in order to see if we could confirm the initial observations concerning the clinical-anatomical correlates of the various aprosodias.[16]

Patients and methods

Our study population included 14 right-handed patients with right hemisphere strokes who were hospitalised between October, 1981, and April, 1982, at the Michael Reese Hospital and Medical Center. The patients were evaluated consecutively by PBG who was a stroke fellow at Michael Reese at that time. Patients were examined within 2 weeks of stroke onset except for case 9 in which the initial evaluation was performed 2 months after the acute event.

event. Subjects that remained hospitalised for physical therapy were assessed serially over the succeeding month.

After undergoing a detailed neurological examination, each subject's affective language and behaviour were tested and tape recorded by PBG utilising the bedside technique developed by Ross.[16] Four major categories of affective language and behaviour were examined: (1) spontaneous affective-prosody and gesturing, (2) affective-prosodic repetition, (3) affective-prosodic comprehension and (4) comprehension of emotional gesturing.

Spontaneous affective-prosody and gesturing were assessed during casual conversation and upon discussion of emotionally charged events in the patient's life. Observations were made about the affective-prosodic quality of the patient's voice (for example intonation, melody and variability) and any mismatching of the affective components of language to the semantic content. Spontaneous speech was judged to have either normal affective prosody or moderate or severe loss of affective prosody, a judgement similar to the assessment of speech fluency in aphasics. In addition, observations regarding the patient's spontaneous facial and limb gesturing were also noted.

Affective-prosodic repetition was assessed by testing the subject's ability to repeat sentences using the affective components of prosody. Each patient was instructed to repeat a declarative sentence void of emotional elements with the same affective quality conveyed by the examiner. The test sentence "I am going to the movies" was presented with happy, sad, angry, and indifferent tones of voice. The patient's ability to match the examiner's affective quality was judged to be poor, moderate or good. Each subject was also graded in a similar fashion on his ability to sing the test sentence with the same melodic quality conveyed by the examiner.

Affective-prosodic comprehension was assessed by standing behind the patient in order to avoid visual clues. A declarative sentence devoid of emotional words was presented to the patient in differing affective tones, and the subject was asked to identify the projected emotion. The test sentence "I am going to the movies" was presented in happy, angry, sad, surprise, and indifferent tones of voice. After each example the patient was given verbally, using a monotone voice in order to avoid prosodic cueing, five multiple-choice answers (outlined above) of which he was asked to select the one that most closely corresponded to the affective tone conveyed in the sentence. Four or five errors out of five on this test was considered poor comprehension; two or three errors constituted moderate comprehension difficulties while zero or one error was considered "normal".

Comprehension of emotional gesturing was assessed by "pantomiming" a series of emotional gestures involving the face and limbs, in order to produce five different affective states: happiness, anger, sadness, surprise, and indifference. The patients were asked to name or describe the emotion after each gesture. If the patient had difficulty identifying the gesture, five multiple choice answers were provided verbally by the examiner using a monotone voice, and the patient was asked to select one. The same rating scale used for affective-prosodic comprehension was also used for this test.

The tape recordings were reviewed independently by both authors without immediate knowledge of the computed tomographic (CT) scan results or each other's diagnostic conclusions. One of us (EDR) was sent the tape recordings in a completely blind fashion without any clinical information to aid in the assessment of affective prosody. Each category of affective language was graded as either being normal, abnormal or moderately impaired. Subjects were assigned to an aprosodia subtype according to the classification of Ross.[16] The two examiners' results were then compared. Any discrepancies in diagnoses were jointly reviewed, and a consensus diagnosis was reached. Joint arbitration was required in only one instance, case 12. In this case both examiners agreed that there was impairment of affective-prosodic comprehension only; however, one of us (PBG) was uncertain under which aprosodia subtype to classify this case (see below).

Each stroke subject had at least one CT scan study during hospitalisation. Scans were performed on the Pfizer FS 200. Each patient's CT scan lesion was mapped onto a right lateral or medial-hemispheric template or diencephalic template by EDR who was again blinded to all clinical and identifying information. The projection technique developed by EDR, is as follows, for producing the hemispheric templates. The lowest cut in the CT scan displaying the most prominent landmarks, that is, mid cerebellum, anterior inferior temporal lobe, gyri recti, orbits, etc, for indicating the angle of the CT scan is drawn on the template as a baseline. A perpendicular line is then erected from the baseline to the most rostral part of the brain template using a right angle triangular drafting tool. Using an eleven-arm-adjustable-equal-spacing divider (Charvoz 11–1188), the relative level of each of the remaining CT cuts above the baseline cut is notched on the vertical, and a line is drawn parallel to the baseline using a parallel ruler. The divider is then adjusted to section the antero-posterior (AP) distance of each CT cut into ten parts. The AP length and position of the stroke, as measured by the points on the divider, is noted. The divider is then expanded to fit the AP diameter of the CT cut represented on the template, and the distance subtended by the stroke is marked out. After all cuts containing the stroke are recorded on the template, the marks are connected to indicate the area and location of the stroke. (This particular projection technique differs from previously published techniques[25][26] in two ways: (1) it allows CT lesions to be mapped accurately onto a standard brain template regardless of the actual overall dimension of the imaged brain by isomorphically adjusting for any AP distance or height incongruity between the imaged brain and the template and (2) the templates are true lateral and medial views of the hemisphere so that anatomical relationships are not distorted by the mapping. Superiorly located gyri and lesions on the lateral template, therefore, appear rather small because they are viewed tangentially.) A similar technique is used for mapping the diencephalic lesions.

Results and comments

There were eight black patients (six men and two women) and six white patients (two men and four women) (see table 1). The average age of the subjects was 65·7 years, with a range of 36–82 years. All patients were right-handed. There were two cases of large artery thrombosis, seven cases of cerebral

Table 1 *Patient characteristics**

Case	Age	Race	Sex	Stroke mechanism	Site of vascular involvement
1	74	W	F	Embolism	MCA
2	62	B	F	Embolism	MCA
3	74	W	F	Embolism	MCA
4	60	W	F	Embolism	MCA
5	61	B	M	Uncertain	MCA
6	66	B	M	Large artery thrombosis	MCA
7	73	W	F	Embolism	MCA
8	57	W	M	Embolism	MCA
9	67	B	M	Embolism	MCA
10	71	B	F	Larger artery thrombosis	MCA
11	36	B	M	Hypertensive intraparenchymal haemorrhage	LS
12	82	W	M	Lobar intraparenchymal haemorrhage	PCA
13	70	B	M	Hypertensive intraparenchymal haemorrhage	LS
14	66	B	M	Uncertain	PCA

*All patients were right-handed.
B = black.
W = white.
F = female.
M = male.
MCA = middle cerebral artery territory.
ACA = anterior cerebral artery territory.
LS = lenticulostriate artery territory.
PCA = posterior cerebral artery territory.

Table 2 *Summary of affective behaviour*

Case(s)	Spontaneous affective prosody & gesturing	Affective-prosodic repetition	Affective-prosodic comprehension	Comprehension of emotional gesturing	Type of aprosodia
1–6*	A	A	N	N	Motor
7	A	A	A	A	Global
8, 9	N	A	N	N	Conduction
10†	N	A	A	A	Sensory
11	N	N	A	A	Transcortical sensory
12	N	N	A	N	Pure affective deafness
13, 14	N	N	N	N	Normal

*Initial evaluations in Case 5 demonstrated a global aprosodia which rapidly evolved over several days into a motor aprosodia.
†Initial evaluations demonstrated a sensory aprosodia which weeks later evolved into a transcortical sensory aprosodia.
N = normal, A = abnormal.

embolism and three cases of intraparenchymal hae-morrhage (see table 1). Ischaemic stroke mechanism was uncertain in two patients. The sites of vascular involvement were: the middle cerebral artery stem, major divisions or branches (nine cases); lenticulo-striate arteries (two cases); anterior cerebral artery (one case); and the posterior cerebral artery (two cases).

Bedside examination of affective behaviour
In our study population 12 patients had aprosodias (cases 1–12), and two patients had normal exam-inations of affect (cases 13 and 14). There were six subjects with motor aprosodia (cases 1–6), one with global aprosodia (case 7), two with conduction apro-sodia (cases 8 and 9), one with sensory aprosodia (case 10), one with transcortical sensory aprosodia (case 11), and one with pure affective deafness (case 12). Table 2 summarises the affective behav-ioural features for each patient. Case 10 evolved into a transcortical sensory aprosodia over several weeks after being diagnosed initially as having a sensory

aprosodia. Case 5 evolved over several days into a motor aprosodia after first being diagnosed as a global aprosodia. For classification and correlation purposes, he was considered to have a motor apro-sodia (see below) since this change occurred during the acute stages of his infarct.

CT Scan correlations
Motor Aprosodia Figure 1 is a composite template of the six patients diagnosed as having motor apro-sodia. CT abnormalities were characterised predom-inantly by ischaemic infarctions involving the frontal opercula. Some patients had lesions that extended into the anterior parietal opercula (cases 1, 3, 4 and 5) and two patients also had infarctions involving the very anterior portions of the temporal lobe (cases 3 and 4). However, the posterior and superior mid-temporal regions were spared in all instances. In one patient (case 2) with a past history of rheumatic heart disease, atrial fibrillation and myocardial infarction, a left occipital infarction, undiagnosed previously, was also noted. In addition, although this subject's overall
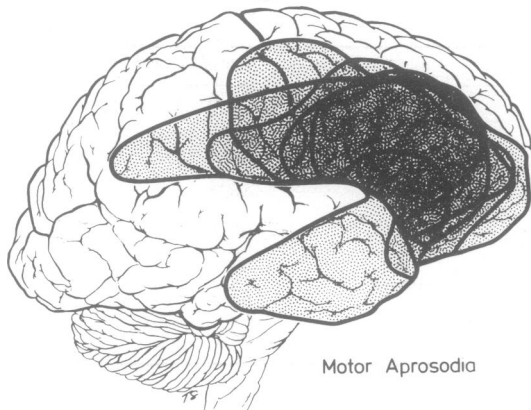
Fig 1   *Composite template showing the CT lesions in the 6 patients with motor aprosodia (cases 1–6). Note that no lesion affected the posterior and mid-superior temporal lobe.*
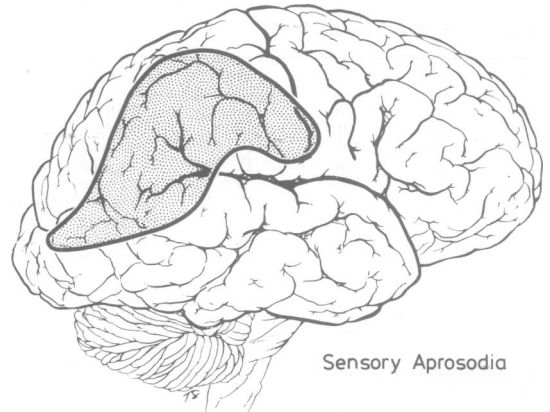

Fig 3   *Template showing the CT lesion in the patient with sensory aprosodia (case 10).*

speech was flat due to motor aprosodia, she was still able to display extreme emotional behaviour, in particular crying, when she was discussing emotionally charged life events.

In a prior study, Ross[16] suggested that motor aprosodia was the right hemisphere functional-anatomical counterpart of Broca's aphasia. In our patients with motor aprosodia, the CT lesions were localised predominantly to the right frontal opercula which would be consistent with the left hemisphere lesions associated with Broca's aphasia.[27–30] These results lend further support to Ross's hypothesis.

Global aprosodia   One patient was diagnosed as having a mild global aprosodia. There was no lesion noted on the acute CT scan, which occurs occasionally if the scan is done too early in the course of cerebral infarction.[31] Unfortunately, a follow-up CT scan was not performed. Since no CT lesion was detected, we cannot make any statements regarding clinico-anatomical correlations.

In our other case of global aprosodia, which rapidly evolved into motor aprosodia after several days (see case 5), a peri-Sylvian fronto-parietal infarction was observed. On initial evaluation, he showed only moderate difficulties with affective comprehension. The CT abnormality correlated with the predicted site of involvement for motor aprosodia. Reversible temporal lobe ischaemia, undetectable by CT, may explain the initial clinical presentation.

Conduction Aprosodia   CT lesions in two patients with conduction aprosodia are shown in fig 2. This type of aprosodia was predicted by Ross[16 24] but not encountered previously. Case 8 (stippled lesion) had an extensive infarction involving the temporal and parietal lobes. Although one would have predicted a sensory aprosodia based upon prior observations,[16] this was not found. This preliminary finding suggests that the anatomical locus of injury for conduction aprosodia may be as diverse or as wide spread as that of its left hemisphere functional counterpart conduction aphasia.[32 33] In the left hemisphere, pathological lesions causing conduction aphasia have been demonstrated to involve the supramarginal and insular cortex and underlying white matter, the dominant auditory association cortex, or a combination of the two sites. Similarly, the anatomical locus of injury for conduction aprosodia may include homologous right temporal and parietal brain regions which mirror those encountered in the left hemisphere for conduction aphasia.
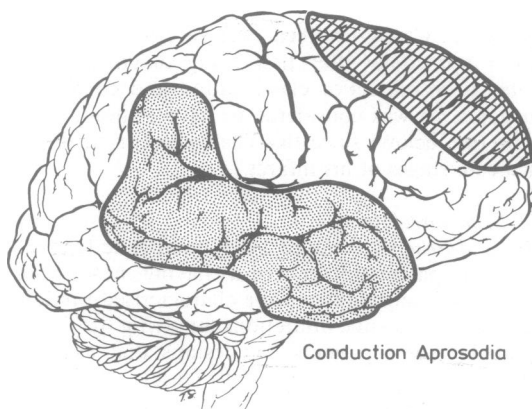

Fig 2   *Composite template showing the CT lesions in the 2 patients with conduction aprosodia (cases 8–9). The lesion involving the superior frontal lobe is hatched because this patient was seen 2 months after his initial stroke making clinico-anatomical correlations tenuous. Historically, he most likely had a motor type of aprosodia that evolved into a mild conduction aprosodia.*
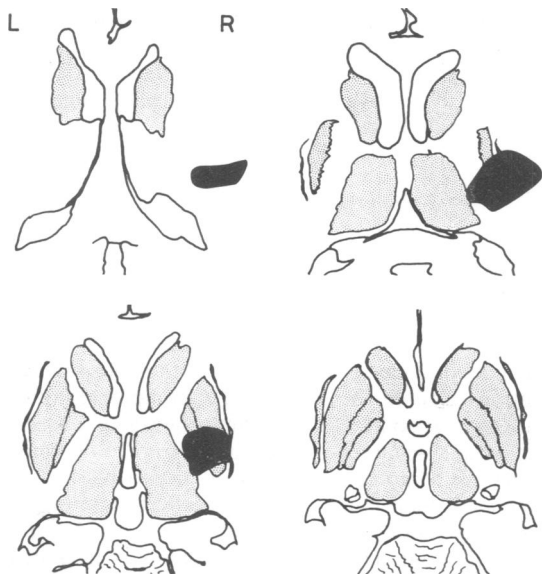
Fig 4 *Template showing the distribution of the diencephalic haemorrhage found on CT in the patient with transcortical sensory aprosodia (case 11).*

Although case 9 had elements of a conduction aprosodia, the deficits in affective repetition were minimal. This patient was examined 2 months after the stroke and history gathered from the family suggested that he initially had marked flattening of speech which resolved over time. Therefore, his lesion is mapped on the template with hatch marks since we believe his mild conduction aprosodia most likely evolved from a motor type of aprosodia. Transposition of the latter lesion onto the left hemisphere
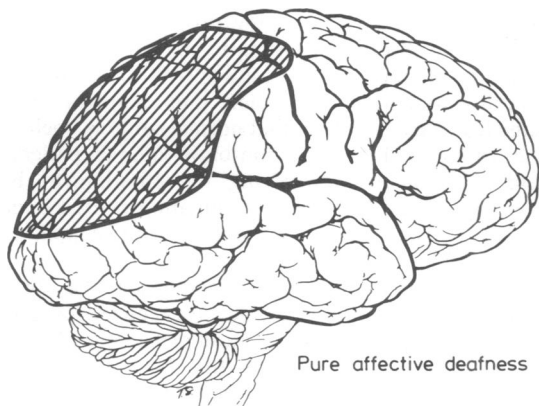

Fig 5 *Template showing CT lesion associated with pure affective deafness (case 12). The lesion is hatched since it was a haemorrhage with tracking oedema making clinico-anatomical correlations tenuous.*

would be expected to result in a transcortical motor aphasia.[34 35]

Sensory aprosodia The CT lesion in one patient with sensory aprosodia is shown in fig 3. A temporoparietal infarction, the predicted site of injury for sensory aprosodia,[16] is observed. This is also the site in the left hemisphere most commonly associated with Wernicke's aphasia.[27 28] On follow-up examination several weeks later a transcortical sensory aprosodia was noted. This example of improvement in affective-prosodic repetition is reminiscent of improvement in speech repetition that can be observed during the recovery phase of certain aphasias.[36]

Transcortical sensory aprosodia The one patient with transcortical sensory aprosodia had a deep haemorrhage involving the right posterior neostriatum and posterior limb of the internal capsule, producing only a slight mass effect (fig 4). The subcortical focus of injury on CT scan in this case is comparable to the sites of injury reported recently for transcortical aphasias.[37 38] In addition, transcortical motor aprosodias have also been encountered following right basal ganglia injury suggesting, as with aphasia, that language has both neocortical and subcortical representation.[16 17 24]

Pure affective deafness One patient was diagnosed as having pure affective deafness. The CT scan (fig 5) showed a lobar haemorrhage involving the occipitoparietal region with oedema extending more anteriorly. The lesion and oedema are shown in fig 5 using hatch marks, since the case is not really appropriate for making firm clinical pathological correlation because of the nature of the lesion. This aprosodia subtype has never been described in isolation although the combination of motor aprosodia with pure affective deafness from a lesion involving the right frontal and mid-anterior temporal opercula has been reported.[16] Its aphasia counterpart, pure word deafness, is usually the result of lesions involving the left mid-anterior temporal opercula.[39-42]

Normal affective behaviour Two patients had no detectable abnormality at bedside testing of affective behaviour. CT scans (fig 6a and b) in these two patients revealed: (1) a right diencephalic haemorrhage involving the superior-posterior putamen, superior lateral thalamus and the posterior limb of the internal capsule (case 13) and 2) an inferiormedial occipital infarction that spared the right peri-Sylvian opercular structures, the angular gyrus and splenium of the corpus callosum (case 14). The absence of affective language impairment in these cases may reflect lack of involvement of critical affective language loci or left hemisphere dominance for this neural process. In case 14, the lesion was in a region that one might have expected loss of visual
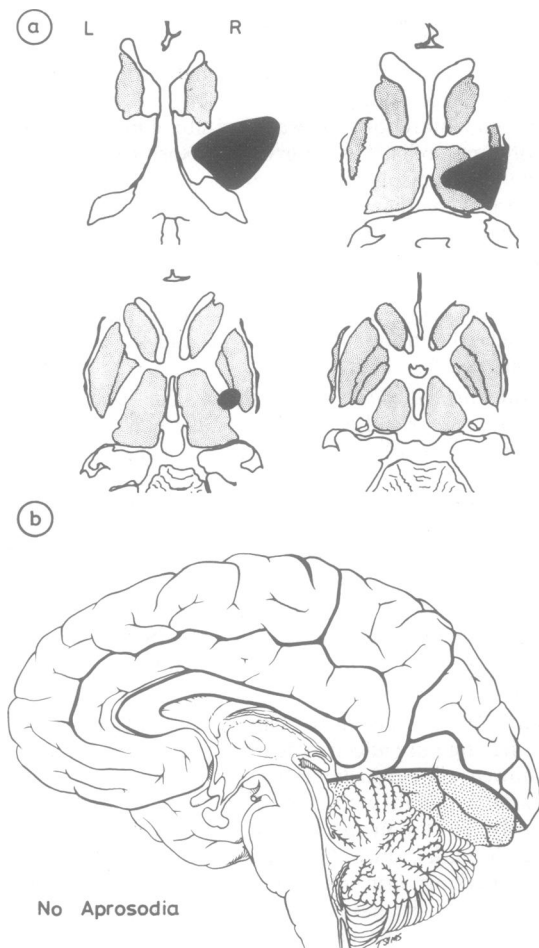
Fig 6 *(a) Template of one of the patients with no disturbances in affective modulation who suffered a diencephalic haemorrhage (case 13). (b) Template showing the lesion in the second patient with normal modulation of affective language (case 14). The lesion spared the peri-Sylvian structures, angular gyrus, and the splenium of the corpus callosum.*

naming and comprehension of emotional gesturing similar to the left hemisphere syndrome of alexia without agraphia.[27] However, the lack of supracalcarine and callosal involvement probably preserved the critical peristriate and visual interhemispheric connections to allow visual recognition (right hemisphere) and visual naming (left hemisphere) of emotional gesturing. Crossed dominance in right handers has been described previously in aphasia.[43-45] In such cases, the neural processes governing propositional language are located in the right rather than in the left hemisphere. Similarly, the neural processes regulating affective prosody in some

individuals may reside in the left rather than the right hemisphere. Although crossed aprosodia has not been reported previously, we speculate that its frequency of occurrence parallels that of its functional counterpart of crossed aphasia.[43-45] Further observations will be required to substantiate this hypothesis.

Discussion

Recent studies of affective language have focused attention on the right hemisphere. Heilman[11] and colleagues provided evidence that right temporo-parietal lesions caused defects in the comprehension of affective speech. Tucker[12] and colleagues observed that right temporo-parietal lesions not only caused affective comprehension deficits in patients but also rendered them unable to evoke affective intonation on a repetition task. Ross and Mesulam[14] described loss of spontaneous affective behaviours in patients with right frontal opercula lesions even though these patients could feel emotions inwardly and comprehend affective behaviours in others. Based on their patients and the work of Heilman[11] and associates, they proposed that the right hemisphere dominantly modulates affective language and behaviour and that this modulation is organised in an analogous fashion to the left hemisphere's role in propositional language. In a follow-up study, Ross[16] provided additional confirmatory evidence of the functional-anatomical organisation of the affective components of language in the right hemisphere. The resulting disturbances of affective modulation for which he coined the term *aprosodias*, were classifiable and characterised by distinct anatomic loci of injury.

The bedside examination and diagnosis of the aprosodias are easy once the proper technique for making the observations is mastered. With just a minimum of practice the first author (PBG) was able to wilfully modulate his voice and gestures with sufficient clarity of affect that normal subjects, including young children and non-brain injured patients had no difficulty identifying the emotion to be communicated. Thus, this examination can be done at the bedside just as one does a routine aphasia examination.

In addition to being easily diagnosed, the aprosodias are common, as would be predicted by previously published clinical[14 16] and physiological studies.[13] In our series 12 of the 14 consecutively examined patients with right hemisphere stroke had an aprosodia. Over the same 6 month period, 15 consecutive patients with left hemisphere stroke were admitted to the Michael Reese Hospital and evaluated by PBG. Of these, 12 had aphasic syndromes.

These data suggest that the aprosodias are as common as the aphasias.

We have shown, as have others,[11 12 14 16-24] that various disturbances of affective language may result following focal right hemisphere injury either from predominantly cortical or diencephalic lesions[16 17] and that recovery patterns, similar to the aphasias, may also occur.[16] Our data, therefore, lend further support to the hypothesis that the organisation of affective language in the right hemisphere mirrors that of propositional language in the left hemisphere. Additionally, we have encountered two aprosodia subtypes not reported previously (conduction aprosodia and pure affective deafness), and have observed that extreme *emotional* displays congruent with the patient's feeling state, for example crying in case 2 in the presence of motor aprosodia, may occur in the context of loss of graded spontaneous affective behaviour. The latter observation coupled with prior evidence[16 19] suggests that emotional experience and the display of extreme emotions are dissociable from the expression of graded affective behaviour.[24] This implies, therefore, that the neuroanatomical substrates for these behaviours have different localisations. Current data suggest that extreme emotional displays are organised outside the right neocortex probably by the temporal limbic system, basal forebrain and diencephalon[24] while emotional experience seems to be associated with temporal limbic structures, in particular the amygdala and hippocampus.[46]

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