Masked depression and depressive equivalents

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by J. J. López Ibor*

The terms "affective or depressive equivalents" and "masked depression" are being used more and more often nowadays, especially in the Anglo-Saxon literature. Careful analysis of these two terms immediately reveals that, as KIELHOLZ[7] has aptly observed, it is possible to draw a distinction between them.

Masked depression would seem to be a form of the disease resembling the classic *depressio sine depressione;* it is being encountered in a steadily growing number of cases, not because its actual incidence is on the increase but simply because physicians have now learnt to recognise it**. To give an example, we once had to deal with a woman who came to our clinic accompanied by her husband. She accused him of no longer paying any attention to her, and this accusation of hers had given rise to difficulties in their marriage. She did not feel at all melancholy and she refused to admit that she had any symptoms referable to depression. Nevertheless, her face belied her words, and when, with the husband's help, we had pieced together her clinical history, we were able to confirm that she was in fact suffering from depression. She was given appropriate treatment, and when the depressive phase lifted, her marital problems disappeared as well. Three years later she had a second phase of typical depression, which likewise cleared in response to treatment. After a further three years had passed, a third depressive phase ensued, but was not recognised as such by her doctor even though her husband repeatedly told him about the previous phases—and the patient committed suicide by throwing herself from a balcony. In this case, weight loss was the only detectable sign of a mental disorder.

The term "affective or depressive equivalent" implies the presence of a predominant symptom which claims the patient's—and even the unwitting physician's—entire attention. But this attention-riveting symptom, which drives the patient to seek medical advice in the first place, is accompanied by a depressive state of mind that is difficult to detect. For example, the patient may complain initially of headaches, which mask, as it were, his depression.

In 1950 I published a book on "Vital anxiety"[9], the sub-title of which was "General psychosomatic pathology". In this book, I put forward the thesis that certain forms of anxiety neurosis and of so-called psychosomatic disease were

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** The following synonyms are employed in English to denote this condition: "masked depression", "hidden depression", "missed depression", "depressive equivalent", "affective equivalent". It should also be mentioned that the term "*larvierte Depression*" has long been used in German, as has "*depresión larvada*" in Spanish, and "latent depression" in English.

superimposed on a phasic alteration in the *endothymic background* of the patient's personality. I singled out a group of patients displaying relatively well-defined features which I described as indicative of *anxious thymopathy;* at that time, I was not aware that FRANK[4] had brought out a book in which he used the term "thymopathy" instead of "neurosis". My basic contention was as follows: just as there is a "vital sadness" typical of so-called endogenous depression—as pointed out by SCHNEIDER[16], who based his approach on SCHELER's classification of the emotions[15]—so is there also a vital anxiety.

Subsequently, the term "vital anxiety" came to be widely used in Spanish to denote the normal anxiety engendered by conflict situations arising as a result of our modern way of life; for this reason, I abandoned the expression and replaced it by "endothymic anxiety", using the word "endothymic" in the sense in which LERSCH employs it.

As far as psychiatry is concerned, clinical observation has shown me that the psychodynamic mechanisms underlying neuroses are very often based on an anxiety which is not reactive in the strict meaning of the word, but endothymic. When SCHULTE[17] said that patients suffering from melancholia cannot feel sadness, he was undoubtedly referring to two different types of sadness—the sadness of the healthy person who is afflicted by some misfortune which makes him sad, and the sadness of the patient with melancholia. The problem becomes even more complicated if fluctuations in mood and in mood structure, as well as, for example, the role played by sadness in the clinical picture of anxiety, are also taken into account.

It should be remembered that SCHELER never included sadness among the normal vital emotions. In his stratification of affective life, sadness figures among the psychic—i.e. reactive—emotions. Pain is classified under the heading of sensory emotions. Although other phenomenologists may disagree with SCHELER's classification, the exclusion of vital sadness from the category of the vital emotions demonstrates that the distinctions drawn between the various strata of emotional experience do alter *under pathological conditions.*

Acceptance of the thesis that there is an endothymic background in neuroses and in the group of masked depressions and affective equivalents does not imply that psychodynamic factors are not involved in the clinical morphology of these disorders, especially in the case of neurotic patients. In all such instances, one problem which has to be faced is that of the patient's personality and of the way in which his personality reacts to any upset he may encounter*. When SCHNEIDER[16] described his "underground depressions", he was referring to depressions superimposed on a background that could not be experienced by the patient. The important thing is to find out to what extent a painstaking analysis of the symptomatology can reveal the role played by the endothymic background in these cases—a background which apparently cannot be experienced by the patient himself and which is expressed by him in the guise of symptoms. By way of example, let me mention the case of a woman who one day, for no

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* Cf. PÖLDINGER's classification of anxiety[14] and KIELHOLZ's classification of depression[7].

apparent reason, tried to hang herself at home, but whose life was eventually saved by treatment in an intensive care unit. Nothing in this patient's past history would have led one to think that she was suffering from depression; she had never complained of any symptoms, apart from mild headaches for which she had taken some drug she had seen advertised on television. There was no conflict situation. Nevertheless, we realised that she was suffering from deep depression immediately we saw her facial expression, even though questioning failed to reveal any of the usual symptoms, whether of the nuclear or of the peripheral type. It was only our determination to discover the exact structure of the headaches experienced by the patient that put us on the right track (this case was subsequently used as the theme for an instructional film entitled "The case of Antonia L.").

Psychiatrists are steadily improving their knowledge of depression in all its forms. Moreover, doctors working in general, non-psychiatric hospitals are surprised to see that suicide is much more common among the patients in non-psychiatric wards than among those treated in psychiatric departments. In the University Psychiatric Clinic in Madrid we have had only one case of suicide in five years, as compared with six to eight cases in other departments. This fact provides considerable food for thought.

Posthumous re-examination of these patients' clinical histories with the assistance of their relatives suggested that almost all of them had been suffering from unrecognised depression and that they had presented with one predominant symptom; the department to which they were sent for examination and treatment had been selected in the light of this predominant symptom. Most of these patients had committed suicide during their first six or seven days in hospital—i.e. during the period devoted to exhaustive clinical tests. All this demonstrates that in the field of depression today there is a peripheral zone which deserves to be extensively studied; it is highly important that general practitioners and non-psychiatric specialists should always bear the possibility of masked depression in mind.

We have been investigating this problem in detail for a number of years and we have succeeded in many cases in persuading our colleagues in internal medicine or surgery to keep a look-out for this possibility and to refer any suspect patients to us before deciding on diagnosis or treatment. With this aim in view, moreover, we are planning to organise seminars for general practitioners (the University Clinic in Madrid belongs to the Social Insurance Scheme). These seminars will be run along the lines suggested by BALINT[1], but with certain modifications. BALINT adopted a psychoanalytical approach, but we intend to tackle the matter from another angle.

A number of authors have pointed out that somatic disorders often coincide with depressive phases. SCHULTE, for example, has drawn attention to the relationship between depression and certain forms of cardiac arrhythmia, cerebrovascular accidents, infectious mononucleosis, etc. Other authors have reported a similar relationship between depression and thyrotoxicosis, rheumatoid arthritis, diabetes, alopecia areata, and lupus erythematosus (MCCLARY, MEYER, and WEITZMAN[13]; COHEN and LICHTENBERG[2]). This problem, as well as that of the

"syndrome shift", has been studied in particular by SPIEGELBERG[18,19] and GROEN et al.[6] from two different aspects*.

It is very difficult to estimate, even approximately, the incidence of such disorders. Some authors—such as DOWLING and KNOX[3], and LESSE[8]—suggest that 20% of the patients treated in a general medical hospital fall into this category. These authors only consider the question of whether masked depression is a primary disorder or whether it is secondary to some organic disease for which the patients concerned have been admitted to hospital; in the majority of cases, however, a physician who has received adequate training in both general medicine and psychiatry will not find it all that difficult to distinguish between the two.

The problem of depressive equivalents has to be viewed in relation to psychosomatic disturbances. It is also important that we should discuss here the relationship between depressive equivalents and such clinical pictures as anorexia nervosa, anaclitic depression, and forms of hysteria which run a phasic course. Another point to be borne in mind is that depression does not display the same pattern in adolescents as in adults. An analysis of this whole problem would inevitably draw us into a long discussion on whether or not all these groups of disorders are of endothymic origin.

When singling out and describing many depressive equivalents—a subject which I do not intend to go into today—the basic criteria I employ are as follows: firstly, the absence of any lesions which would justify the signs and symptoms in question, and, secondly, the clinical connection between the symptomatology and depressive phases. Nuchal pain might be taken as an example of the first criterion; nuchal pain is often attributed to osteoarthritic changes in the cervical vertebrae, changes which persist unaltered in follow-up X-ray pictures even when the pain itself has completely disappeared. In cases of meralgia paraesthetica I have noticed that sometimes the symptoms fail to respond to neurosurgical operations on the external cutaneous femoral nerve; in these instances, the symptoms seem to be related to depressive phases and they disappear in response to appropriate treatment. I could quote many other examples of this type. It is therefore my belief that this sector provides a new approach to the clinical assessment of certain psychic, neurological, and psychosomatic disturbances, and should be carefully investigated; the extensive clinical material I have collected, which is based on very long follow-up periods, is extremely demonstrative in this connection.

In patients suffering from pain the physician bases his diagnostic approach on the peripheral and radicular distribution of the nervous pathways, except in cases of referred pain or of so-called thalamic pain. In its distribution, as well as in the manner in which it is elicited and experienced, thalamic pain most closely resembles thymopathic pain, although it also differs from the latter in some respects. As a rule, the patient complains of a "dull ache" which he finds very difficult to describe; one can spend hours or even days observing and questioning the patient and still not succeed in obtaining a detailed description of

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* Cf. the thesis on "Depressive equivalents" by J. J. LÓPEZ-IBOR ALIÑO[12].

his sensations. The pain, moreover, may not follow the expected pattern; it may change its site not only from day to day, but even while the physician is carrying out his examination. Meralgia paraesthetica, for example, sometimes fails to correspond in its extent to the distribution of the external cutaneous femoral nerve and may even assume an alternating form in which it affects the left thigh for a few days and then switches to the right thigh.

More than a hundred years ago, in one of his inaugural lectures at the Berlin Clinic, GRIESINGER[5] gave a masterly description of thymopathic frontal headaches which he designated as "frontal anxiety" or "frontal dysthymia". "The patients complain", he said, "of a sensation in the frontal part of the head. Their entire disease consists solely of this sensation. They search for words to describe it. They all say it is not a pain, and some add: 'If only it were a pain'. Many of them refer to it as a torment, a weight, etc. To indicate the localisation of the disorder, many patients draw lines across the root of the nose. When they experience this sensation, they are incapable of thinking and are prostrate with anxiety. The sensation is much more impressive than any other pain and exerts a much more marked effect on the patient's psyche. One elderly patient who consulted me after his second attack told me he had been so despondent as a result of this painful sensation in the head that he had attempted to commit suicide, but had fortunately been stopped in time."

It is possible in some cases to analyse the peculiar nature of these forms of paraesthesia and to conclude that they represent a sensation equivalent to the fear of dying evinced by some patients usually diagnosed as suffering from anxiety neuroses. Such an analysis, however, which might be described as a "transphenomenological analysis", is only feasible if the patient possesses a certain capacity for self-observation and if the psychiatrist has sufficient patience and psychological insight. I have often seen cases of "paraesthetic brachialgia" which, as WARTENBERG[20] has reported, occurs in the morning and disappears again as soon as the patient begins to move his arms. Akinesia algera and a whole series of similar manifestations are likewise commonly encountered. Even akathisia, in cases where it is not due to treatment with neuroleptic drugs, may often be included among these thymopathic equivalents.

A long time ago, when the only treatment for depression was electroconvulsive therapy, I was faced with the problem of treating a severely depressed patient. He improved a little in response to the first two E.C.T. sessions, but after the third he developed such marked akathisia that he could no longer stay in bed. He insisted on getting up not only during the day but also during much of the night, despite the fact that he was given hypnotics to help him sleep. I was extremely puzzled by this behaviour, but as neither the patient's past history nor the results of current examinations provided any clue to the cause of the akathisia, I decided to continue the treatment. In response to the fourth E.C.T. session an improvement set in, and after the fifth the akathisia disappeared completely. Following two more E.C.T. applications the patient's depression also lifted. I have followed up this patient ever since, and he has never had any recurrence of akathisia. Fortunately, moreover, his depression has not returned either.

More common than this major form of akathisia is the phenomenon described as "restless legs", which has recently been much discussed in the literature. Its cause has been interpreted in a number of different ways. Although I certainly do not deny that this symptom may be due to various aetiopathogenic factors, I can affirm, in the light of my long experience, that many patients with restless legs belong to the group we are concerned with here.

We are at present studying other similar neurological syndromes, all of them relatively rare ones. I should like to point out, for example, that what is often diagnosed as vertigo of the Ménière type is simply another thymopathic equivalent which should really be called agoraphobic vertigo or thymopathic vertigo. I have seen a number of cases of this kind in which the patient has even undergone a surgical operation. The operation failed to elicit a response because the patient's condition had not been correctly diagnosed.

It is important that general practitioners should be fully aware of the existence of latent or masked depression, which manifests itself exclusively in the form of non-depressive symptoms or depressive equivalents, and that they should in general be familiar with this marginal zone of classic depression, because they probably encounter even more cases of this type than do psychiatrists. This sector constitutes, as it were, a bridge between general medicine as practised by family doctors, and psychiatry.

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