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Masked Depressions

The Forty-fifth Maudsley Lecture, delivered before the Royal Medico-Psychological Association, 20 November 1970

By J. J. LÓPEZ IBOR

When I received the invitation to deliver this year's Maudsley Lecture, I felt that a great honour had been conferred upon me. The series of the Maudsley Lectures provide a succession of pictures and a measure of the level attained by psychiatry, not only in Great Britain but throughout the world in recent times. Maudsley himself was a great master in the realm of clinical psychiatry. In these days psychiatry is menaced by many diverse and varied interpretations that tend towards bringing about its disintegration. Reading Maudsley's *Pathology of Mind*, and reading these lectures that have been given under his name since their foundation, constitutes an invaluable lesson that enables present-day psychiatrists to understand what their real situation is, and what is the field of action that is open to them as scientists in contemporary society.

The subject that I have chosen for my lecture today is that of *masked depressions*. *Manifest* depression is a phenomenon that today is very easy to recognize, but clinical experience shows that, side by side with this fundamental nucleus of depressions, there exist others in which the typical symptoms do not appear but which manifest themselves by means of other symptomatological equivalents. I believe that the situation of the problem will become clearer if I briefly recall the following typical clinical case.

Antonia L. is a woman aged 30, who was admitted one day in March 1968 to the Madrid University Hospital, in a state of coma as the result of having tried to hang herself. From the Intensive Care Unit she was transferred to the Psychiatric Service. With the aid of her family and the social workers it proved possible to reconstruct her history. In brief, this was as follows: there were no previous illnesses, she was married and had no problems of a matrimonial, sex, economic or other nature. She had a son 7 years of age, and her husband was a skilled worker. No other significant events or special peculiarities in her previous personality were discovered. All that the patient said was that she had been suffering from some 'strange' headaches, which had begun some months previously, and which she had treated with the usual analgesics. One day, apparently just like any other day, when she returned home from taking her son to school and buying the day's provisions, she told her mother, who happened to be in the house, that she was going to the bathroom to wash her hair. Her mother heard the water running for a short time; after a few minutes of silence she heard a strange noise—a bathroom stool that had fallen over. She ran to the bathroom door to see what was happening, and was horrified to discover that her daughter had hanged herself, using a nylon clothes-line.

When she was admitted to the psychiatric clinic, after the four days that she had spent in the Intensive Care Unit, we quickly appreciated the fact that she was depressed and that the attempt at suicide was the consequence of her depression, which until then had not been apparent to the members of her family, to her family doctor or even to the neurologist who had examined her. Frequent headaches were the only disturbance that the patient had been complaining about for some months past. There is no better name for this case than that of *masked depression*.

It is estimated that approximately only one out of every four or five cases of depression consults a psychiatrist. In a large general hospital, where I have the opportunity of work-

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ing, in a single year there were 7 suicides in the non-psychiatric services, while in the latter there were only two 'attempted' suicides. Thus, if we add to the previous figure that of the masked depressions, the number of patients who pertain to the depressive circle is much greater than that which emerges from the existing epidemiological studies.

Some authors have spoken of *depressions without depression*—'depressio sine depressione'; again, for the last twenty years in various publications I have called attention to the *thymopathic equivalents*, in which, wrapped in different symptomatological disguises, there is always found a depressive or anxiety-ridden state of the mood. The notion of the 'equivalent' goes back a long way. Billod (1850) introduced it in relation to epilepsy.

Depressions are very often masked during adolescence. In many cases of agitated adolescents with behaviour disturbances there is found a succession of depressive or cyclothymic phases (Monro, 1968). These phases hamper the maturation of the personality. If it is borne in mind that adolescence is very often an essentially tumultuous and even metaphysical phase, it is not at all surprising that such depressive phases are taken to be reactive, when essentially they are endogenous. Basing myself on my own clinical and therapeutic experience, I would go so far as to express the opinion that such depressive states are, in many cases, analogous to what Spitz (1967) has termed 'anaclitic depression', or that they have their origin in psychosomatic disturbances in early childhood and are of an endogenous nature.

In the present-day literature a great deal of discussion is devoted to the differential diagnosis between reactive or endogenous depressions, with all their intermediate degrees, such as provoked depressions, endoreactive dysthymias, etc.; but this is not the question that I wish to discuss in my lecture today.

The *masked depressions* and the depressive equivalents to which I refer pertain to the circle of the endogenous depression. Endogenous depression is not a deeper or more complicated reactive depression, but is another way of experiencing the mood-state that fundamentally is constituted by sadness; but together with this sadness there also exists a general decline of what are called vital feelings, that is to say the manner in which the Ego is aware of the state of its corporality. These vital feelings are distinguished by a *quality* that differentiates them from reactive or directed feelings, that is to say those which produce any event in the external environment that leads to sadness. Sometimes the difference is very clear for certain patients; at other times only clinical experience is capable of appreciating the qualitative differences between the two classes of feelings. If we adopt the non-Cartesian tradition, we can say that between the purely noetic and the purely somatic strata of the human person there exists an intermediate stratum which we can describe as thymopathic or endothymic. From this it follows that these feelings can be denominated *endothymic*. It is not only a question of sadness but of anxiety, of inhibition and of other states of mood which the patient himself sometimes finds it difficult to define, but which have this general characteristic (preoccupation, tedium, emptiness, despair, etc.).

The existence of physical symptoms during depressions has been frequently recognized. Cleghorn and Curtis (1959) have published a very interesting paper on this subject. The patients are sent in the first place to the general physician or to a surgical clinic, and finally it is seen that their disturbances cannot be understood as purely somatic illnesses. The presence of physical symptoms is more evident in those depressive patients who are initially sent to a doctor or surgeon than in the other patients who themselves have decided to go direct to a psychiatrist. Such symptoms include the following: nauseas, cephalalgias, wandering pains in different parts of the body, oppression, and always in the background a lack of appetite; at times there is even a slight feeling of being depressed, but very often the majority of these patients do not speak about this sensation of depression unless they are questioned in detail about it. This demonstrates the need for extending the channels of communication between the general physician and the psychiatrist, and also for basic communication between patient and doctor, not, as sometimes happens, limited to various analyses and explorations without the

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doctor having previously listened to the patient's manner of describing his symptoms.

In speaking of affective equivalents or of masked depressions I refer especially to those patients in whom the presence of the depression is not at all clear even to themselves. Sometimes we are surprised at the energy with which they deny the feeling of being sad or of suffering from anxiety or any other of the symptoms that pertain to the category of the thymopathic. The fact is that basically it is a question of the greater or lesser perceptive capacity that they have with regard to the state of their corporality. Here, the difference between some patients and others is enormous.

Neurologists are very well acquainted with the problem of anosognosia, and, just as this exists for certain neurological disturbances of organic character of what they call the corporal schema, there exists in these other patients a kind of anosognosia of their deep-down mood state with respect to the perception of the state of their corporality.

When it comes to interpreting the physiology of the affects and of the emotions, a fundamental physiological principle that Albert Haller (1754) discovered many years ago is frequently forgotten. This principle is concerned with the difference between nervous excitability and nervous irritability. Excitability is triggered off by external stimuli; irritability is something specific; it is that which determines that the response to the stimulus is not automatic, but contains something proper to the organism. By transferring this schema to the physiology of the emotions we shall thus come to understand how there can exist states of depression and anxiety determined by frustrations or wants existing in the external environment, and how there can exist analogous states of endothymic character.

There is in the psychiatry of all countries a common basis. The psychic changes that occur because of somatic changes do not come under discussion, but in what are termed endogenous or cryptogenic psychoses, however you may wish to describe them, the differences between the diagnostic and genetic criteria become more widely separated. Treatments with convulsants and insulin have led to the hypothesis of the presence of an unknown and unidentified *somatosis* (K. Schneider, 1960). Advances in the field of pharmacology have proved to be decisive in favour of this hypothesis. Before the Second World War a multitude of works on heredity were published. In subsequent years the number of such publications decreased enormously as a consequence of Hitlerism. The whole situation changed little by little, and now there are many publications on the subject. Also the biochemistry of endogenous psychoses has made some progress. On the other hand, psychotherapy has abandoned its utopian aims, and social psychiatry is coming to occupy a more important place. The situation of a psychiatrist at the present time can prove to be more prudent the more eclectic it is. There are many who desire to overcome a desolating dichotomy. The efforts to find new techniques of research, diagnostic nominalism, socialization of psychiatry and, last but not least, the overload of administrative tasks are engendering a dangerous fact: that of the diminished attention that is devoted to the clinical side. I can quote a significant example: the World Psychiatric Association has created numerous Sections, from that concerned with the psychiatry of adolescence to that dealing with the higher activity of the nervous system; but as yet no Section has been specifically created to deal with clinical psychiatry. It is my hope that it will soon realize that this is a key subject for the future of psychiatry, because at the centre of it is the patient in all his complex singularity.

I should now like to present, in general terms, the most frequent types of these masked depressions or depressive equivalents. The fundamental groups that we have been able to establish are the following:

I. Pains and paraesthesias;
II. Agoraphobic or thymopathic vertigo;
III. Psychosomatic disturbances;
IV. Anorexia and hysteria.

In these four groups we can classify the depressive or thymopathic equivalents, such as I described them for the first time in my book *La Angustia Vital* (Vital Anxiety) (1950), and which were subsequently included in another book of mine *Las Neurosis como Enfermedades del Animo* (Neuroses as Diseases of Mood) (1960). In the doctoral thesis of López-Ibor Aliño (1970)

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an analysis is given of such cases catamnestically and from a statistical point of view. A. F. da Fonseca (1959) has concerned himself in great detail and very fruitfully with the *depressive equivalents*, taking his clinical material from the Bethlem Royal and Maudsley Hospitals through the Genetics Unit of the Institute of Psychiatry of the University of London. The investigation of 60 patients affected by these symptoms, twins of the same sex who had reached the adult age, incline him to support the thesis of Slater (1936, 1938) concerning the existence of a genetic unit in the affective disturbances, although naturally the dominant gene is characterized by its incomplete penetrance and in many cases does not signify more than a predisposition to affective disorders without being the unique and complete cause responsible for them. Referring to our previous works on the subject, he groups as depressive equivalents a certain number of disturbances of a more or less cyclic nature:

(A) Rheumatoid and neuralgic attacks, and periods of lumbago which appeared periodically in 5·5 per cent of the twins constituting the sample, fathers and brothers.

(B) Attacks of asthma in 3 per cent of the cases.

(C) Attacks that appear to be peptic ulcers with severe gastric symptoms and with improvements and relapses in 3·5 per cent of the cases.

(D) Attacks of eczema, erythema, psoriasis, neurodermatitis, dermatosis, of recurrent nature, in 3 per cent of the cases.

Naturally, these data do not mean than these diseases do not exist on another basis, but when a relationship is established between these studies of inheritance of the depressive equivalents and the evolution and displacement of the syndromes throughout life, one increasingly acquires the conviction of the relationship that exists between them, a conviction which is also confirmed by therapeutic successes in these cases.

Group I

With regard to the definition of pain, there is no unanimity among the many authors who have concerned themselves with this subject. For example, in the 39th Maudsley Lecture, Stengel (1965) defined pain accepting the terms of Merskey (1965): pain is an unpleasant experience that we generally associate primarily with a physical lesion or describe as the sequel to or in terms of this same organic lesion. Slater (1966) refuted this definition, basing himself on what Wolff and Wolf (1968) had done, referring it to a sensorial experience transmitted by specific nervous structures distinct from those of touch, heat, temperature, etc.

In brief, there is a tendency today to consider different sensations, such as those of pain, touch, heat and cold, as a total somataesthetic experience that makes use of different information channels. Pain is nothing other than the unpleasant aspect of this somataesthetic experience.

But our difficulties increase if we refer to clinical experiments such as those of Beecher (1948) who, during the Second World War and subsequently, calmed very intense pains produced by serious traumas by means of the intravenous injection of pentothal sodium. And the fact is that in those pains anxiety constituted their overwhelming and decisive component.

Again, we scarcely need to stress that pain does not always play a prophylactic role, since there are illnesses that consist of pain alone. Patients, even when they are uneducated and find it difficult to express themselves, make a distinction between this type of internal pain not connected to any lesion and the pain emanating from an external agency. To describe what they are experiencing they make use of singular expressions such as that of 'slow pain'. In other cases the patient may say, 'I have a terrible pain in the nape of the neck.' 'What is the pain like?' we ask him. 'I don't know, it's a pain that is not like other pains; it is a strange sensation that originates here and spreads through the whole of my head as though it were going to stupefy me or to make me faint.'

Side by side with pain we can locate, there is this expressive ambiguity, the paraesthesias. There is a certain type of paraesthesia well known to classic neurology, and on which I do not propose to dwell. What is of interest in my context is another type of paraesthesias, such as those

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referred to by some of the following patients.

From the point of view of the psychopathology of pain, the error that, in my opinion, exists is that of considering as psychogenic any pain that does not have an organic explanation. In the classification of Allan Walters (1967) a distinction is drawn between the psychogenic increase of physical pain, psychogenic muscular pain, pain as the emotional expression of fear, and even the pain of a somatic hallucination in a psychosis; but the fact to be stressed, as I see it, is that pain which is not physically caused cannot be explained in very many cases by the concept of 'conversion reaction', and still less can it be considered as being hysterical.

Among the psychoanalysts, Rangell (1953) supports this limitation of the field of psychogenic pain. In reality, pain is not a sensation, but, as Stumpf (1928) has said, a 'Gefühlsempfindung', which we may translate into English by the term 'feeling-sensation'. What is certain is that some elementary sensations, including pains produced by physical causes, are always accompanied by an enhanced state of malaise. Scheler (1912) included this fact in his 'stratification of the emotional life', making a distinction between somatic, vital and psychoreactive feelings (leaving apart those which he called metaphysical feelings); that is to say, following Pascal, he affirmed the existence of a 'logic of the heart', based on reasons of which the intellect is unaware.

Pain, considered as a depressive equivalent, is very frequently accompanied by paraesthesia and profound anxiety. The distinction between pain and anxiety is very difficult to establish in some cases.* The most frequent localization of these thymopathic pains is as follows: headaches of diffuse character, but which very often have a tendency to spread towards the nape of the neck, then producing a sensation which few patients can manage to describe and which, definitely, is as if they were about to faint. With the same character there appear scapulalgias, lumbalgias and, at times, pains that suggest sciatica or something else, apart from the precordial pains that give rise to the problem of their differential diagnosis with respect to coronary affections.

For a long time past there has been much in vogue the diagnosis of headache due to muscular tension, although at the present time many of the former diagnoses of this type have been replaced by that of psychogenic cephalalgias. It is thought that anxiety and emotional tension are the causes of this type of headache, although at the same time other authors have invoked muscular tension as a cause. This form of headache is frequently described as a sensation in the posterior cervical muscles that subsequently becomes extended as though it covered the rest of the head. There appear paraesthesias, sensations of prickling or of burning in different regions, but especially in some regions such as the occipital, so that frequently this type of headache has been related to occipital neuritis. What is curious is that the syndrome begins, as do depressions, with a circadian cycle. When these patients are questioned in detail, replies are obtained which are very similar to those of patients suffering from typical thymopathic headache. It is a special tension which they feel has different varieties, all of which are only ways of expressing the manner in which, at a given moment, a profound experience of thymopathic, vital or endothymic character that has been lived through reaches the consciousness.

At the Second International Neurological Congress held in London, Wartemberg (1932) gave a very detailed description of 'brachialgia statica paraesthetica' as a form of acroparaesthesia. This description was mainly concerned with adult patients whose average age was 43. This disease is more frequent in women and in certain professions. With regard to the symptoms, brachialgia coincides with the acroparaesthesias in the existence of this form of dysaesthesias, without any positive finding coming to light in the course of exploration.

What is curious about brachialgia is that it presents itself during the night, and consequently is observed on awakening ('waking numbness'); the patient is unable to refer the symptoms to anything definite, but the pain and the paraesthesias present themselves in oscillating form for some weeks on awakening, and subse-

* The French speak of 'psychic pain' and 'moral pain' (sadness, anxiety).

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quently there are many nights that are not followed by these sensations. As a general rule the paraesthesias begin in the hand, especially on the left side and in the fourth and fifth fingers, and are subsequently accompanied by discomfort, and particularly by a kind of dull pain in the rest of the arm as far as the deltoid muscles. Sometimes it is only the pain that appears, without the paraesthesias. Although the latter appear to become extended into the territory of root C8, sometimes as far as C5, nevertheless no fixed limit for them can be established. On some nights the sensations are so strong that they cause the patient to wake up. The most noteworthy feature of these paraesthesias is the variability of the picture, which is at times confused with the syndrome of the 'painful shoulder'. They do not generally appear during siesta, and they often completely disappear when the patient begins to move his limb, as though it were the immobility during sleep which caused them. It has sometimes been considered that they are due to compression in the position of sleep, while at other times they have been attributed to a brachial neuritis due to some local infection, or to a rheumatic neuritis. Some authors have spoken of angiospastic neuritis, but, as we have previously said, the pains are rather slow, there are no acute pains, there is no hypersensitivity when the nerves are subject to pressure or when they are stretched, there is no cutaneous hyperaesthesia not hypoaesthesia, etc. Explorations of the cervical column often show quite normal results, or if anomalies present themselves they cannot be considered as aetiological, because the paraesthesias disappear indefinitely if they are properly treated, while the anomalies in question continue to exist. Consideration has also been given as an aetiological factor to the presence of anomalies in the cervical rib. The similarity of the picture with that of 'paraesthetic meralgia' and with the 'acroparaesthesias' is evident.

Although Déjerine (1916) said that the formication of which the patients complain seems to be analogous to that which is produced when a nerve is compressed, what is certain is that the difference between both dysaesthesias is quite clear to a good observer. What is curious is that in his first work on the subject Wartemberg (1932) favoured the aetiological hypothesis of the posture during sleep as the cause, and that in the book that he has written on neuritis and neuralgias (1958), in order to explain their erratic and irregular character and the lack of correspondence with the data known concerning peripheral innervation, he establishes an entity that has no anatomico-pathological or clinical basis, constituted by a kind of *errant or migratory neuritis*.

Wartemberg (1932) has also described as a mononeuropathy the *cheiralgia paraesthetica* which also pertains to this group of pains, and which, like the others, is accompanied by paraesthesias, and dysaesthesias, and on the other hand, objective alterations in the sensorial field are scarcely found. It affects territories that correspond to the innervation of the radial. He also described digitalia paraesthetica and gonyalgia paraesthetica (1954). Fernandes (1957) has described a glossodynia thymopathica.

To these paraesthesias there must be related the 'acroparaesthesias' described by Schultze (1892), and the 'meralgia paraesthetica' generally attributed to a lesion of the external femoral nerves, to which Roth (1895) devoted a study. The disturbance consists of unpleasant dysaesthesias which present themselves above all in the hands, and more rarely in other parts of the body. They do not coincide in their distribution with the radicular or peripheral dermatomes; as a general rule they manifest themselves in the form of gloves on the hands, sometimes in the form of acroparaesthetic fingerstalls, while at other times they appear to be limited, without exactly coinciding with this topography, to the cubital zone of the hand; in the feet they present themselves in the form of a 'sock' and they also appear around the mouth, on the ears or on the cheek.

Very curious is the existence of paraesthesias of this type in the teeth, where they are generally extraordinarily pertinacious.

When we read the different authors, we see that it is necessary to distinguish two types of acroparaesthesias, one being those of Schultze (1892) and those of Bernhardt (1895) in which there are no neurological changes and the exploration of sensitivity always gives normal results, even when the most detailed methods

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possible are employed; the same is the case with regard to the exploration of the vegetative functions, such as that of sweating. On the other hand, in the old descriptions of Déjerine (1904) we find the datum that acroparaesthesia is accompanied by objective disturbances of sensitivity, although Cassirer (1906) considered that in these cases it was not a question of pure forms of acroparaesthesias. In neuralgias, as is natural, the possible presence of an organic cause of these disturbances must always be eliminated by means of the appropriate explorations. Some authors tend to attribute brachial acroparaesthesias to disturbances of the sympathetic cervical system or to changes in the cervical column itself; but in the same publications of these authors it may be seen that the therapy employed in relation to this assumed pathogenesis fails to produce an effect. Van Bogaert, Tombeur and de Wandels (1946) published a study giving details of treatment by means of anaesthesia of the stellate ganglion, which, however, did not produce any result.

One of my recent women patients had a *paraesthetic meralgia* that on some days manifested itself in the right thigh and on other days in the left thigh; at night the picture was combined with the phenomenon of 'leg jitters'. This patient was submitted to all kinds of examinations, having in mind the possibility, in view of her age, of a cancerous affection of the spinal column. She did not feel depressed, but suffered from a strange malaise which she was unable to attribute to anything specific, and which lasted for the whole day and had a circadian rhythm. Again, the reconstruction of her history made it possible to discover that she had suffered from a depression some years previously and that, on being given antidepressive treatment, at the end of a week she had got up, after having remained in bed for the previous three months. Naturally we cannot discard the possibility that specific affections exist in the nerves cited, which require the appropriate treatment, but it must also be stated that the great majority of cases are presented in patients in a state of sub-depression or of marked depression, since all their attention is concentrated on these disturbances.

In all these cases our attention is called to the difficulty, in these algias, of finding the famous points of Valleix which were of such importance in the classical clinical examination; this fact does not stand alone, but in paraesthetic meralgia the exploration of sensitivity does not show objective findings; again, if the doctor sets out to do so, he can produce in some patients zones of anaesthesia of suggestive character, and if explorations are carried out on different days or at different moments of one and the same day, the limits of the zone are also different. A clinical fact worthy of note is that the patient is capable of marking with the tip of his finger the zone of paraesthesia, and that, on the contrary, if it is the doctor who tries to locate this zone with any of the normal clinical procedures employed in the exploration of sensitivity, he finds himself faced with a series of phantasmal variations. The patient ends by explaining that it is not an external sensation, but that it is something internal, difficult to locate, difficult to describe, but extraordinarily tormenting.

Akathisia today constitutes a clinical picture much better known that when it was described for the first time by Haskoveč (1901, 1904), due to the presence of states of akathisia in the treatment of psychoses by means of psychotropic drugs. Haskoveč described it as belonging to the clinical picture of hysteria, and it was Bing (1923) and Wilson (1925) who subsequently found it in some cases of post-encephalitic parkinsonism. But akathisia appears in some patients without being directly connected with any organic lesion, but only with a sub-stratum of depression and anxiety as occurs in the other depressive equivalents. The patient manifests only this motor restlessness without having a clear perception of his internal state of anxiety and of depression. At other times, on the contrary, when the patients are questioned about the state of their mood they reply by describing it as one of anxiety and sub-depression.

Closely connected with akathisia is another phenomenon which Brissaud (1902) described as '*muscular impatience*'. Subsequently this phenomenon has been given various names, such as that of 'unruhige Beine' in German, and that of 'restless legs' in English (Ekbom, 1945, 1950).

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This is a special dysaesthetic sensation that presents itself at night when the patient is preparing for sleep. The dysaesthesia or paraesthesia is so intense that it often compels the patient to get up and walk until after a certain time it spontaneously begins to diminish, and then the patient can go to sleep.

This clinical picture was described as far back as 1695 by Thomas Willis under the name of '*anxietas tibiarum*'. This restlessness sometimes also presents itself in the upper limbs, as in some cases recorded in our statistics. Allison (1943) gave a detailed description of these disturbances under the name of 'leg-jitters'. Ekbom (1945, 1950), who has devoted an interesting monograph to this subject, distinguishes two forms: one called '*asthenia crurum paraesthetica*', characterized by paraesthesias in the form of formication and especially with a sensation as though worms were moving within the body, and another clinical form that he calls '*asthenia crurum dolorosa*', in which, together with the paraesthesias, pain exists. These paraesthesias are generally limited to the leg and rarely invade the posterior part of the foot or the thigh. They are deeply localized. Very often the patient does not know how to define this vague localization and refers it to the bones or the deep muscles; it always produces an irresistible need to move the lower limbs (or the upper limbs, when it is located in this region).

The pathogenesis of this disturbance has always been considered to be very obscure. Ekbom stresses the frequency of the disease in pregnant women and the important role played by heredity. In all these cases, the most detailed neurological exploration, including radiograms of the spinal column, myelogram and analysis of the cerebrospinal fluid are completely negative; for this reason Ekbom suggests the possibility of the intervention of a vasomotor factor, and other authors attribute it to an iron deficiency or refer to the possibility of an increase of serum cholinesterase.

The very meticulous classical descriptions of all these cases often call attention to the presence of certain alterations of the personality which, as I have said, are included in neurasthenia or in hysteria. In the cases that have come within my experience, my attention has been always attracted by the following data:

(1) The absence of any organic lesion to which the presence of these phenomena may be attributed (it should be borne in mind that many of these patients have been under observation for years).

(2) The phasic course that they often show, although in some cases the phases are not clearly separated; there are oscillations which disappear and reappear after a temporary silence, and they generally last longer than the average duration of the depressive phases in 27 per cent of my cases, while in the remainder the duration is shorter.

(3) The existence of a basic sub-depressive state, with all the characteristics of a vital or endothymic sub-depression, and not of reactive character; not even situations of stress or fatigue produce it; but on the contrary it is these same paraesthesias which produce situations that are so unbearable that they lead some patients to give up their normal working activity, as in the case of a patient who recently consulted me who has resigned from an excellent position in the Civil Service because of not being able to bear his dental paraesthesia.

(4) When we follow up the history of patients who come to consult us for the first time, for example after 50 years of age, we see that they have previously had minor depressions or depressive equivalents of another nature, and on subsequently observing them this same fact is confirmed.

(5) Both neuralgias and thymopathic pains and this group of paraesthesias do not respond to the customary treatments for pains or for paraesthesias. Many of such cases are treated by general physicians or by other specialists with antalgics, on some occasions with general hygienic measures and on others with minor neurosurgical operations. For instance, an algia which did not have the typical characteristics of sciatica, but which was so diagnosed and attributed to a prolapsed disc, was treated by operation but continued to manifest itself for a year afterwards, yet in the following four or five months it disappeared suddenly and spontaneously without any other medical intervention.

(6) The simultaneous presence of some of these pains and paraesthesias with very typical

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depressive pictures, or the appearance of such algias and paraesthesias at the end of depressions or as an initial syndrome of the latter.

(7) The therapeutic results obtained through the use of thymoleptic drugs.

How can these paraesthesias and pains and a localized anxiety be explained, and what significance should be attributed to them? Some patients speak of them as though they were the preliminary symptoms of approaching death. Others do not dare to express this idea. But what is certain is that they signify, as does pain itself, a modification in the perception or awareness of the condition of corporality.

To say that our living or animated body is essentially different from a corpse may seem to be a trivial commonplace, but its triviality disappears when we ask ourselves how we sense our own bodies. I have previously spoken of an overall somataesthetic experience in which all the various sensory qualities are integrated. With reference to our intra-body we could apply a similar scheme which would differ from the concept of Höffding (1893), when he spoke of coenaesthesia, or that of Leibnitz when he referred to 'minute perceptions'. It is certainly difficult to accept that fact that every cell of our bodies transmits a message along a special cable in order to give an account of its state. But what is certain is that this phenomenon manifests itself at times slowly and gradually and at other times as a sudden impact in the form of a sensation of profound malaise or of notable wellbeing (euphoria). On some occasions this state will be produced by something that comes from without, such as an influenza infection or the taking of a small quantity of alcohol. To this immediate global perception of how we sense ourselves in relation to the world and to ourselves, there pertains what Scheler (1912, 1913) termed the stratum of the vital feelings, and which I prefer to call thymia, humour or state of mood.

Well known to psychiatry are the major disorders manifested by states of euphoria and of depression and of manic-depressive psychoses, and it is also well known that the great majority of these affective oscillations are not determined by any external event or stimulus; they are like great positive or negative waves that occur on the flotation level of the thymia. The question why the negative waves are more frequent than the positive is bound up with the mystery of life itself, just as is the fact that it is sadness rather than happiness that colours the day-to-day state of the mood. This special global perception of the state of awareness of the body could be compared on the noetic plane to that expression which today has fallen into disuse in scientific language, but which signified so much in other epochs: 'common sense' as the ante-room of the region in which intelligence itself exercises its activity.

Endothymic sadness has characteristics which differ so widely from normal sadness that many patients are aware of these differences, as is the case, as we have seen, with pain. To such an extent is this so that, as Schultze (1930) says, many melancholiacs complain that they are unable to become sad. Happiness, sadness, anxiety, tedium, emptiness, fullness and many other states belong to this stratum of our being, and are so well known that it is not necessary for us to discuss them in detail.

Group II

Psychosomatic medicine has invaded the field of modern medicine and has made its own extensive zones of diseases attributed to other sectors of medicine. Nevertheless, it is advisable to add new points of view to the interpretation of many problems of psychosomatic medicine, especially with regard to changes in the circulatory and respiratory apparatus, the digestive and urinary systems, hypochondria, etc. It is not possible for me to make here a detailed exposition of its relation to the affective equivalents; I should only like to call attention to the fact that many authors, such as von Uexküll (1952) and Bilz (1940), base themselves, in order to explain the 'simultaneous psychosomatic correlation' (Mitscherlich, 1960) on the existence of a pathological background such as that which emerges in psychosomatic symptomatology. Of all the states of mood that serve as a basis for the emergence of neurotic and psychosomatic disturbances, the most typical is *anxiety*, but it is not the only one since side by side with it there exists nausea, tedium, vertigo, sadness, etc.

The specificity of the symptoms is bound up

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with the relationship between anxiety and corporality: where do we perceive our corporal ego? Or rather, expressed in other words, where do we locate the ego within the corporal schema? Corporality enjoys and participates in the *intentional character* that the whole of the psychic life has. The psychosomatic symptom appears at that point at which the personality notes the possible dehiscence which produces anxiety. The symptom is located where the anxiety is, and the anxiety is located where the symptom is. Instead of thinking that the symptom is located in the most inferior organ, according to the thesis of Adler, or in that which is considered to be most charged with libido, as in the thesis of Freud, the symptom is located where the experiencing of the unity of the personality feels itself to be most threatened, and this place is functionally specific. The dynamics of the psychosomatic disturbances which appear against the background of vital anxiety are analogous to those of the phobias and obsessions, which move away from the primary anxiety nucleus and have a tendency to the objectification of the disturbance, that is to say, to a neutrality of the mood. Crises of anxiety are accompanied by a series of symptoms which are projections of these crises on the somatic and visceral planes; once the critical phase has passed, the vegetative symptoms also become automatic and independent and constitute 'vicious circles'. The work of Denis Leigh (1967), for example, shows the relationships between neuroses and asthma, and I have previously pointed out the importance of the endothymic factor in the appearance of neuroses.

Group III

The *agoraphobic vertigos* and *agoraphobia* itself, described by Westphal (1872), are often classified among the thymopathic equivalents, and at times the equivalence is demonstrated because the corresponding symptomatology is imbricated. A female patient, for example, notes that the disease begins with an alteration of the visual sensation, as if things were dancing in front of her eyes; immediately this dancing effect of things becomes more rapid, until everything comes to disappear from the perceptive field and there then irrupts, as a third act more dramatic than the previous ones, the sensation of anxiety, 'as though her heart were being compressed'. In some crises the patient loses consciousness for a few seconds, and when she comes round 'she continues to experience an oppression in the chest and a desire to weep'. Another female patient frequently had a sensation of vertigo by day and by night. This sometimes happened to her even when she was seated, and then she would get up and begin to run distractedly. In spite of her fear of fainting, she had never fallen down.

As we have seen, the web of the symptoms in these crises is so complicated that only artificially can they be differentiated from one another.

The patients speak of dizziness, but if they are obliged to define the sensation precisely they do not know whether to refer it to the plane of anxiety or to that of vertigo. The vegetative symptomatology is found embedded in the thymopathic vertigo within the same vertiginous crises. The patient speaks about a state of undefined malaise, of *intemperies nervosa*, of fear of the rupture of the state of equilibrium at any moment, a fear which at times takes on a more diffuse character, such as a fear that something will happen to him, as the sufferer from anxiety says. And, connected with this state of 'emotive drunkenness', as Féré (1890) called it, the patient feels that his legs are weak, his stomach empty, and experiences perspiration in the hands, palpitations, etc.

When the patient walks no objective disturbance is observed; at most we see that the patient seeks to extend the basis of support when he walks or that he is afraid to lengthen his steps. He can even be made to walk with his eyes closed, but suddenly a violent gesticulation shows us that the crisis has arrived. Many patients do not like to walk on a floor that is too smooth, but rather prefer one which gives them the sensation of adherence, firmness and security. It is interesting to stress the relationship of these crises with the digestive apparatus. There is no doubt that some of the vertigos described are due to 'stomaco leso' (Trousseau, 1812) belong to this class. Many patients feel better after a meal, and it is possible that this state of well-being is related to the postprandial vagotonia. In the first case of Westphal (1872),

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the patient felt worse in the morning, before taking any food, and felt better after meals or after having taken a little wine or beer. A copious meal or an excessive amount of drink can, on the contrary, provoke or aggravate the crisis.

To this group of equivalents there belong the vertigos, which are sometimes called agoraphobic, but which for some time past I have called thymopathic because they affect above all the functioning of this stratum of the personality. At times they are diagnosed as 'ménièriform' vertigos and even as actual Ménière's vertigos, and in my files there are records of some patients operated on; nevertheless, their nosological attribution is beyond doubt. Let us consider an example:

The patient is a woman aged 38; the symptoms began a year and a half ago after a slight cold; she noted a continuous whistling in the right ear, and she immediately began to suffer from vomiting and a sensation of fainting; this sensation gave her a feeling of absence as though she were in a cloud, floating in the air, as though she lacked the ground on which she stood. This sensation disappeared after a few hours, but there remained a persistent pain in the nape of the neck. She was diagnosed as a case of Ménière's vertigo. The very day after the diagnosis there appeared in her a continuous fear of going mad and of dying, and of leaving her children unprovided for, and also the fear of having another new sensation of vertigo. She has had three further attacks in the course of the last two years, but what brings her to consult us now is a clear state of depression with a feeling of tiredness, sadness and a sensation of incapacity to continue living, to continue to manage her home, etc. The pain in the nape of the neck persists, as does the buzzing in the ear like a pain, like blows on an anvil, etc., but the sensations of vertigo do not persist. She suffers from very severe cephalalgias which sometimes prevent her from resting her head on the pillow or oblige her to get up out of bed and walk around her bedroom.

Group IV

Other diseases that we should study here in their relations to the problem of the depressive equivalents and of masked depressions are those of *anorexia nervosa* and of *hysteria*. Anorexia nervosa has been the subject, on the one hand, of existential anthropological interpretations of great intellectual depth, and, on the other hand, of psychoanalytic interpretations no less interesting than the former. Long experience in the treatment of patients of this class and in their psychopathological study permits me to include them in the borderline of depressions, although they have their own characteristics from many points of view which would take up too much time to enumerate now; what I can say, however, is that the most efficacious therapy in our hands has been the existing energetic antidepressive therapy which must be practised in severe cases of depression.

Hysteria, as Slater (1960) pointed out in a previous Maudsley Lecture, is a syndromic complex; and, even though briefly, I should like to draw your attention to the hysterical phases within this syndromic complex. Symptomatology is one way the patient has of expressing not only his anxiety but also his depression, and the most noteworthy fact is, according to our most recent clinical investigations, that in many of these cases we are concerned with adults who have suffered a lesion at birth, of those denominated 'minimal brain damage', which has not prevented them from leading their normal lives, especially if they are women; and that there arrives a moment in their lives in which, because of the appearance of a depressive phase that is scarcely apparent, they react with hysterical crises.

It would take up a great deal of time to speak about the treatments of hysteria, so I shall only call your attention to this fact, which in many cases provides us with the clinical and therapeutic key.

It is possible to think that the inclusion of these groups of patients in the borderline area of depressions masks the *distinction between neuroses and depressions*. Despite the fact that my first steps in psychiatry were influenced by the phenomenological psychopathology that makes a more rigid distinction than do other schools between neuroses and psychoses, including the endogenous depressions among the latter, clinical experience has compelled me to

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formulate another perspective that consists not only in the acceptation of intermediate types such as the 'endo-reactive depressions' and others of analogous nature but in the affirmation that in every neurosis there also exists an *endothymic base of neurodynamic origin which does not imply the denial of the existence of more or less conscious reactive superstructures*. At the outset of my investigations I limited myself to indicating the existence of some cases of neuroses determined by a vital or endothymic anxiety, just as in depressions there existed a vital sadness, by qualifying that group as 'anxiety thymopathy'; but subsequent clinical experience has convinced me of the existence of an *endothymic sub-structure in the neuroses*.

There also exists a zone of confluence between depressions and psychosomatic disturbances; but what is most important is to point out the presence of a '*syndrome shift*', that is, the fact that throughout the life of a person there are found phases of depression or of varied depressive equivalents after more or less long intervening periods. Spiegelberg (1968) has in a recent paper also called attention to this, and the fact has a great clinical and pathogenic significance; it has been referred to from the psychoanalytic point of view by Bastiaans (1957) and other authors. It is of interest to note the age differences between the patients with depressive equivalents and endogenous depressions without treatment, as has been shown, among other facts, by J. López-Ibor Aliño (1970). The depressive equivalents that present themselves with the aspect of psychosomatic disturbances occur in younger persons than do the pure depressions. Spiegelberg (1968) has made a detailed study of the relationship between thymopathic equivalents with stomach ulcers, mucomembranous colitis and other diseases.

In speaking of depressions and neuroses, I should like to recall a famous statement by Mapother (1926): 'The distinction between what are called neuroses and psychoses has really grown out of practical difficulties, particularly as regards certification and asylum treatment. It has become customary to call those types and degrees of mental disorder which rarely call for such measures by the name of neurosis. I know no other basis for the distinction; neither insight nor co-operation in treatment, nor susceptibility to psychotherapy will serve.'

And Lewis, in his meticulous and documented study of the problem (1934), speaks of the difficulties in establishing a distinction when the anamnesis of the patient is known in detail: 'The patient whose illness appeared on first sight to have been precipitated by bereavement proved to have had an incipient mood change beforehand; the patient overwhelmed with guilt and self-reproach at one stage was full of indignation and resentment at another; the patient depressed and retarded one day was anxious and agitated the next.'

Recently, Gurney, Martin Roth, Kerr and Schapira (1970) have studied in 154 patients the nosological relationships between states of anxiety and states of depression from the therapeutic point of view. The patients were assigned to one or other of the two diagnostic groups in an independent manner, and the therapeutic response was evaluated in terms of the clinical social state of the patients at the time when they were discharged from the clinic and six months afterwards. The treatments employed were electroconvulsive therapy, tricyclic antidepressives, monoamine oxidase inhibitors and sedatives or tranquillizers. The study shows that electroconvulsive therapy and the tricyclic antidepressives were prescribed more frequently for the patients with depression, while the sedatives and tranquillizers were prescribed more for the patients with anxiety states. These latter patients were not only prescribed the drugs mentioned above, but also monoamine oxidase inhibitors. The response to electroconvulsive therapy was better in the patients with depression than in those with anxiety, and the same occurred with the tricyclic antidepressives. This would naturally signify it is a question of two different groups, but the clinical picture is very varied and it must be borne in mind that all possible transitions exist, all the more so since, for example, within the tricyclic antidepressives there are variations in the responses according to the clinical picture of the patient and in relation to the composition of the drugs themselves.

The experience of our clinic coincides in

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general with that of Sargant and Slater (1944), Kalinowsky and Hippius (1969), Rees (1953) and many others. In the cases in which anxiety prevails, the monoamine oxidase inhibitors sometimes prove to be very efficacious. This is not so in those cases in which vital sadness predominates; but this should only be considered as a general rule, and in each case the therapeutic plan should be individualized. Anxiety and sadness thus appear to be two therapeutic key symptoms, but this should only be taken as nothing other than an undefined schema.

That which today we call depression was formerly called melancholia, but the word melancholia and its diagnosis have not always covered the same area. From Hippocrates until the end of the nineteenth century the majority of the delusional states were included in melancholia. But the depressions, although they include cases of secondary delusional states, have extended their field towards the symptoms of corporal or physical character. I believe that the study of masked depressions and of depressive equivalents opens up new paths for us, although, like everything new, at the same time it creates new doubts—for the same reason as Solomon said and Burton repeated: 'Even in the midst of laughing there is sorrow.'

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Prof. J. J. López Ibor, *Institut of Neuropsychiatry, Av. Nueva Zelanda, 78, Madrid-35 (Spain)*

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